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Recent Literature on the
Non-Auditory Effects of Noise:
The Primary Emphasis on the
Cardiovascular System

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by

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Introduction

This compilation is the result of a literature search for recent articles (primarily 1975-1977) on the non-auditory physiological effects of noise. The main area of interest in this project was the effects of noise on the cardiovascular system. A list of the investigators in the field of noise effects who provided additional information for this project has been included. The articles in the bibliography that were acquired and analyzed will be used in future work by the Environmental Protection Agency on the non-auditory effects of noise.

Informative abstracts have been completed for the 21 most relevant studies concerning the cardiovascular effects of noise. In addition to the abstract, a form summarizing the experimental design and results, including evaluations and comments, has been provided for each abstract. The form was designed to provide an easy means for comparing and analyzing the studies. The abstracts, forms, and accompanying articles are arranged alphabetically by author in sections 1-21 of this volume.

A summary form only was completed for each of 13 additional articles that cover areas other than the cardiovascular effects of noise. These studies deal with other significant non-auditory effects, especially on the endocrine system, including catecholamine excretion and plasma cortisol levels. The forms and copies of the original articles are included in sections 22-34. Both time and money limitations prevented such processing of the other 78 articles in the bibliography. Copies of the majority of these articles have been acquired by the Environmental Protection Agency.

Some of the copies of the original articles accompanying the abstracts and summary forms are of poor quality, although they are legible. They were the best copies available at the time of printing.

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(see page 9 for footnotes)

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Footnotes

- * copy of article not available at Informatics Inc.
- ** abstract, summary form, and original article in sections 1-21.
- † summary form and original article in sections 22-34.

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SECTION 1

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> J. E. Brown, II, M.S. U.S. Department of Labor OSHA Washington, D.C.		<u>Institution and address where research was performed</u> Federal Aviation Administration (FAA) U.S. Dept. of Transportation, Aeronautical Center P.O. Box 25082, Oklahoma City, OH 73125	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> FAA		
<u>Citation</u> Brown, J. E. III, R. N. Thompson, and E. D. Folk. Certain non-auditory physiological responses to noises. Amer. Ind. Hyg. Assoc. J. 36(4):285-291, April 1975.			
<u># of Ref.'s</u> 14	<u># of Fig.'s</u> 10	<u>Language</u> English	
<u>Type & duration of experiment</u> Type: retrospective case study Duration: results of annual physicals for past 7-8 years were examined.		<u>Purpose for study</u> to test for measurable non-auditory physiological changes in workers exposed to high occupational noise levels	
<u>Description of test groups (subjects, age, etc.)</u> Two groups: 1) Tests - 22 professional pilots in the FAA (male, 40-60+ years old) 2) Controls - 29 non-flying FAA executives (male) of same age and socioeconomic status as the tests.			
<u>Control of other stressors</u> None - field exposure to noise while flying		<u>Statistical Methods</u> A Randomized Complete Block Design used; t-test compared results for the two groups; F-test compared results within each group	
<u>Noise Stimulus</u> Source: aircraft noise (twin-engine transport or business aircraft; 4-engine transport; jet transport) spectral characteristics: spectra included for cockpit noise in four types of planes noise level: noise levels (shown graphically) often exceeded Damage Risk Criteria length of exposure: 6000 hours or more flying time # of trials: not applicable		<u>CVS Response Measured</u> Heart rate using EKG records blood pressure - mean systolic and mean diastolic <u>Nonauditory effects</u> CVS: no changes due to noise; fluctuations in diastolic blood pressure and heart rate in the pilots; increased systolic blood pressure in all subjects (probably due to age); decrease in heart rate in the controls. Other: both serum cholesterol levels and mean serum glucose levels declined with each measurement - no changes due to noise.	
<u>Author's conclusions</u> Results of audiograms showed noise-induced hearing loss in the professional pilots, but not in the control group. A comparison of the health records of the test and control groups did not show that occupational noise exposure produces long-term non-auditory physiological effects. The negative results could be due to insufficient noise levels or to lack of highly sensitive measurements of physiological responses.			
<u>Evaluation & comments</u> The lack of significant non-auditory effects may be due to the intermittent exposure of the pilots to high noise levels, in contrast to that of industrial exposure, such as of auto workers. Not only is the daily exposure of factory workers more continuous, but also the number of hours of exposure would be much greater than for pilots.			

Brown, J. E. III, R. N. Thompson, and E. D. Folk. Certain non-auditory physiological responses to noises. Amer. Ind. Hyg. Assoc. J. 36(4):285-291, April 1975.

A retrospective study of the nonauditory physiological effects of noise was done in a test group of 22 professional male pilots, aged 40 to over 60 years. Heart rate, blood pressure, blood glucose, and serum cholesterol measurements from annual physicals (in an executive physical program) during the past 7 to 8 years in the pilots were compared to those of a control group of 29 non-flying male executives of similar socioeconomic level and age range. Both the test and control subjects were employed by the FAA (Federal Aviation Administration). The yearly means for each of the four physiological parameters measured were analyzed for changes over time. Changes within each group and between the two groups were analyzed using the F-test and the t-test respectively. The pilots, each with over 6000 hours flying time, were often exposed to noise levels exceeding the Damage Risk Criteria (DRC) of CHABA (the National Academy of Sciences Committee on Hearing Bioacoustics and Biomechanics). Spectra of the cockpit noise for four types of aircraft (twin-engine business and transport, four-engine transport, and jet transport) are included. Audiometric histories showed noise-induced hearing damage in the pilots, but not in the controls. Although some changes occurred in the physiological parameters measured, none of the changes were the result of occupational noise exposure. There was an increase in systolic blood pressure in both tests and controls, which was probably due to age. Changes in the other parameters included insignificant diastolic blood pressure fluctuations, decreased heart rate in the controls, fluctuating heart rates in the tests, and declining glucose and cholesterol levels in both tests and controls. The authors note that the decline in cholesterol levels may have been due to changes in dietary and exercise habits of the FAA personnel due to their involvement in an executive physical program.

Chromatography

Chromatography
Ski: Col-
Chemical
by 4:367

Ski: Col-
Chemical
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Certain Non-auditory Physiological Responses to Noises

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The non-auditory physiological effects of extended exposure of pilots to high intensity noise were investigated. The health records of 22 professional pilots (FAA) were examined for recorded measurements of heart rate, systolic and diastolic blood pressure, serum cholesterol, and glucose. These data were compared to records of the same measurements from 29 non-flying FAA personnel of the same age, social and economic status as the pilots. The yearly means for each parameter were analyzed for changes with time, within the same population, and for differences between the two study groups. Audiometric histories were tabulated and compared, and noise levels inside aircraft were determined. Although it was demonstrated that FAA pilots were exposed to high occupational noise levels there was no indication that their exposure produced any significant non-auditory physiological response.

Introduction

THE SHORT-TERM NON-AUDITORY physiological effects of noise on man are relatively easy to measure and are reasonably well documented in the literature. It is generally agreed that sudden non-repetitive noise can cause alterations in voluntary muscle activity,¹ vasoconstriction of peripheral blood vessels,¹⁻³ changes in heart rate¹⁻⁴ and blood pressure,³⁻⁷ changes in respiration,¹ alterations in gastrointestinal tract activity,^{1,2} and certain endocrine⁸ and biochemical changes.^{9,10} Not always agreed upon are the magnitude, direction and persistence of these startle responses. Nor is there any real accord in regard to the significance of these responses as predictors of non-auditory effects upon individuals exposed to noise for long periods of time.¹⁰ A review of the literature reveals that much more clinical and epidemiological evidence must be gathered before any valid conclusions can be made.

The purposes of this research were: (a) to select a population of workers known or suspected to be exposed to high occupational

noise levels; (b) to confirm that they did experience such exposure; and (c) to investigate the possibility that their exposure may have produced some measurable non-auditory alterations in physiological function. It was anticipated that this retrospective study, though subject to the usual problems of "after the fact" data gathering and interpretation, would provide some valuable baseline information for the design and conduct of subsequent prospective studies.

Procedure

Selection of a test population was not a difficult decision for some rather obvious reasons. First, the Federal Aviation Administration (FAA) has a fleet of approximately 100 aircraft of many types used for a variety of missions such as monitoring of airways navigation aids, inspection of instrument landing systems, flight training, and logistics. Second, many of the pilots who operate these aircraft are based at the FAA Aeronautical Center in Oklahoma City. Next, most of these pilots have logged many thousands of hours in all types of aircraft. And finally, all of them are given annual physical examinations for FAA medical certification in the

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An "executive" annual physical examination program conducted at the Center provided control subjects. Most of the pilots are in this program and receive their medical certifications with their executive physicals. The two groups were essentially of the same socio-economic status.

Each physical examination included a complete history, routine laboratory and radiographic procedures, electrocardiography (ECG), audiometry, and biochemical tests for alkaline phosphatase, cholesterol, glucose, protein bound iodine, thymol turbidity, triglycerides and uric acid.

The data assembled and analyzed in this study were selected from the individual health records of the participants in the program. All physical examinations were conducted by the same flight surgeon and all clinical laboratory procedures were accomplished by the same team of chemists and technicians throughout the study period.

In order to accumulate as many observations as possible, only those individuals with at least eight years' participation in the program were selected. Twenty-nine pilots meeting this criterion were located and it was subsequently determined that each had accumulated 6,000 hours or more of flying time. The 29 control subjects were non-flying executives with at least eight years' participation in the program.

The health records of each person were examined for recorded measurements of heart rate, systolic and diastolic blood pressure, cholesterol and glucose. These data were analyzed for changes with time and comparisons were made between the test and control populations.

A search of the records revealed that not all subjects received a physical each year. Therefore, means of the eight most recent recordings of heart rate and blood pressure and the four most recent measurements of cholesterol and glucose were used to test for

changes with time. There was one exception; only seven mean values of heart rate were available in the control group, and consequently, comparisons between the test and control populations were made on the basis of seven consecutive heart rate determinations. A Randomized Complete Block Design with subjects as blocks was used for the statistical analyses.

Discussion

Noise Exposure Data

The nature of the noise exposure of the test population has been studied extensively by the FAA, and noise levels in crew compartments of all agency aircraft were obtained from various unpublished agency projects.¹² In Figures 1 through 4, noise spectra were plotted for comparison with the National Academy of Sciences Committee on Hearing, Bioacoustics and Biomechanics (CHABA) Damage Risk Criteria (DRC) contours.¹³ These spectra were obtained from octave band frequency analyses made on the flight decks of the various aircraft at cruise conditions. During takeoffs, pilots are exposed to higher noise levels, much higher in piston-driven aircraft.

The 8-hour DRC contour was equalled or exceeded by noise levels in the cockpits of all of the aircraft except the jet transports. In five aircraft the 4-hour DRC contour was equalled or exceeded, and in one air-

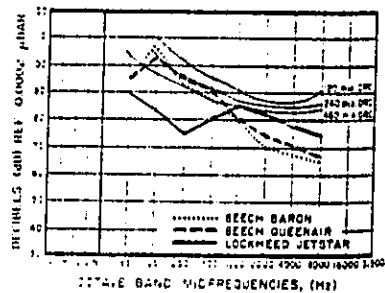


Figure 1. Cockpit noise in twin-engine business aircraft.

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craft the 2-hour DRC contour was violated. These charts offer evidence that the test population was exposed to high occupational noise levels and audiometric evaluations confirmed this fact.

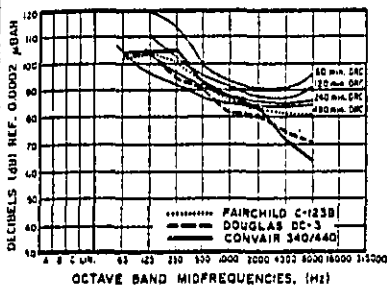


Figure 2. Cockpit noise in twin-engine transport aircraft.

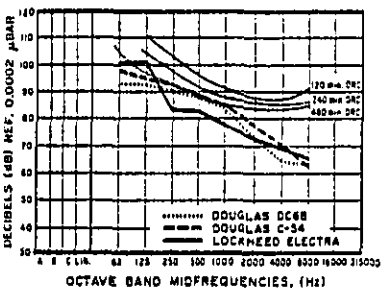


Figure 3. Cockpit noise in four-engine transport aircraft.

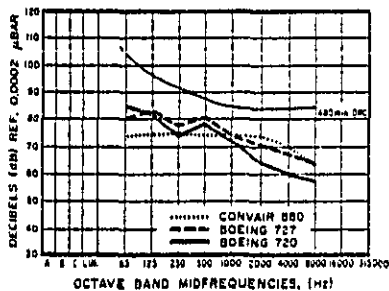


Figure 4. Cockpit noise in jet transport aircraft.

Auditory Effects

Since both temporary and permanent alterations of hearing in response to excessive noise occur most dramatically at 4,000 Hertz (Hz) and, to a lesser extent, 6,000 Hz, hearing levels in these frequencies were analyzed for evidence of such alterations in the pilot population. Also, while advanced cases of noise induced hearing loss cannot be distinguished from presbycusis, in less severe cases a 4,000 Hz "notch" (marked increase in hearing level at 4,000 Hz) followed by good residual hearing in the higher frequencies is diagnostic of noise induced hearing loss in those persons with a history of noise exposure.¹³ Presbycusis generally first affects the higher frequencies and gradually proceeds to the lower.

The study population were placed in age groups of 40 to 49 years, 50 to 59 years, and 60 years and above. The hearing levels in decibels, referenced to 1951 American Standards Association (ASA) audiometric zero, were recorded for the frequencies of 4,000 and 6,000 Hz as found on the first and last audiograms in the individual health records. The mean hearing levels were determined for each age group at each of the two frequencies, and these data were used to compare hearing loss in the pilot and control populations. The results of these comparisons are contained in Table I.

In the 40 to 49 age group, the pilots were found to have somewhat more acute hearing in the initial audiogram at both frequencies, though in the last the hearing level was nearly equal to that of the control group. The pilot group demonstrated a greater relative increase in hearing level in both test frequencies than did the controls during the study period.

The age group 50 to 59 also demonstrated an apparent noise induced hearing loss in the airmen. The pilots were found to have a substantially greater hearing level in the first audiogram at 4,000 Hz while the loss at 6,000 Hz was nearly the same in both

TABLE I
Audiometric Histories for 4,000 Hz and 6,000 Hz as Mean Hearing
Levels in dB ref. to 1951 ASA Audiometric Zero

Age Group	Audiogram				Difference	
	First		Last		4 kHz	6 kHz
	4 kHz	6 kHz	4 kHz	6 kHz		
40 to 49						
Pilots	6.25	12.50	13.75	21.35	7.50	8.75
Controls	14.60	17.30	15.00	21.33	0.40	4.03
50 to 59						
Pilots	21.14	19.32	31.82	32.18	10.68	12.86
Controls	17.22	19.81	20.14	25.00	3.52	5.19
60 +						
Pilots	30.00	28.50	36.60	38.33	6.60	10.03
Controls	19.37	24.38	24.88	34.88	2.51	10.00

population groups. As in the 40 to 49 year age group, the pilots demonstrated a greater relative increase in hearing level than did the control group.

The pilots also experienced greater hearing loss than the controls in the ages 60 and above. However, the relative increase was found to be much the same.

Audiometric data for the pilots in the age groups 50 to 59 and 60 and above demonstrated the characteristic noise induced 4,000 Hz notch in the initial tests.

These results did not differ greatly from those of Kronoveter and Somerville.¹⁴ While their study did indicate a greater degree of hearing loss in all age groups for both test and control subjects, the amount of difference between test and control subjects was almost identical.

Non-Auditory Effects

For examination of non-auditory physiological effects of noise, test and control subjects were age-grouped as previously mentioned. However, the noise exposed group contained only three pilots aged 40 to 49 and only four pilots were 60 or older. Since these age groups were believed too small to yield reliable results, only those aged 50 to 59 were given detailed consideration. Consequently, the noise exposed population contained 22 pilots and the control included 29 individuals.

A. Heart rate

The heart rates, taken from ECG records, are presented in Figure 5. The mean values of heart rate were found to fluctuate considerably without establishing a trend either

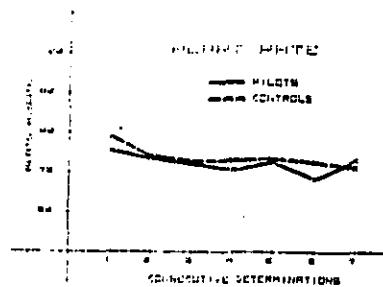


Figure 5. Mean heart rate.

toward increase or decrease. A statistical test comparing the means (F-test) indicated that the magnitude of these fluctuations was statistically significant within each population ($p < 0.05$), and a *t*-test demonstrated a significant difference in the heart rates in the two populations ($p < 0.01$).

Also, the more pronounced total decrease in the heart rate in the controls and the tendency to fluctuate in the pilots were in contrast with the general decrease in the pulse in response to audiogenic stress reported in the literature.^{3,4}

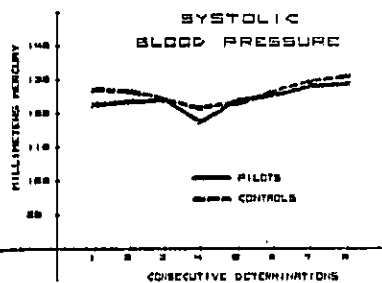


Figure 6. Mean systolic blood pressure.

B. Systolic blood pressure

The mean systolic blood pressure (Figure 6) in the test subjects rose slightly in the early determinations, but dropped in the fourth determination. This decrease was followed by an increase which continued through the final determination. The mean systolic pressure of the controls declined through the first four determinations but increased through the last four. The magnitude of change within both groups was found to be statistically significant ($p < 0.01$), while the *t*-test indicated no significant difference between the two. The observed increase may have been due to aging.

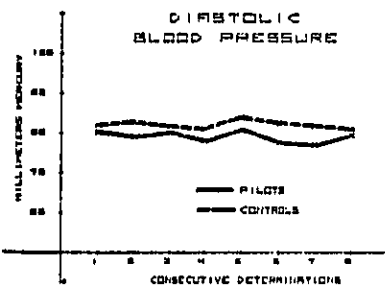


Figure 7. Mean diastolic blood pressure.

C. Diastolic blood pressure

The mean diastolic blood pressure (Figure 7) showed no appreciable change with time, but fluctuated in the same manner as the heart rates. Tests for changes within

populations and between the two populations were not significant.

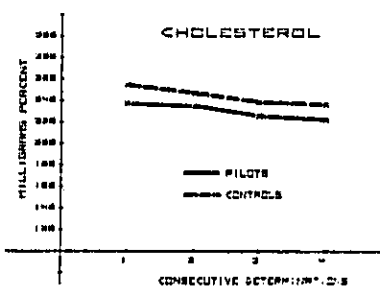


Figure 8. Mean cholesterol level.

D. Cholesterol

The mean serum cholesterol level (Figure 8) declined slightly with each successive determination in both pilots and controls. The degree of this decline, when evaluated by the F-test, did not prove significant and no difference between the two populations was indicated by the *t*-test. This decrease in the level of cholesterol was the opposite of the effect noted in earlier studies and was due, perhaps, to an increased awareness, on the part of those involved in the executive physical program, of the effect of diet and exercise on the cardiovascular system.

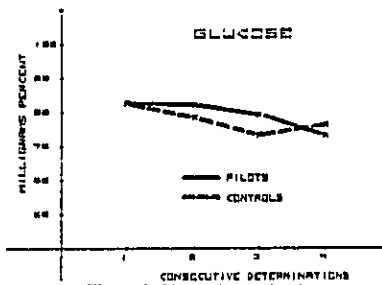


Figure 9. Mean glucose level.

E. Glucose

The mean glucose level in the blood (Figure 9) also tended to decline with each determination, though the final value in the

controls reflect a slight increase. A comparison of the means in each population yielded statistically significant results ($p < 0.05$ pilots and $p < 0.01$ controls), although no such difference was indicated when the two populations were compared. While glucose levels did change with time, there was no evidence to indicate that these changes were due to noise exposure.

Summary and Conclusions

- (1) Changes observed in the heart rate over a seven-year period, though statistically significant within population and between populations, did not show a decline in rate resulting from noise exposure nor were these changes of sufficient magnitude to be considered biologically important.
- (2) Changes observed in the systolic blood pressure, though statistically significant in the magnitude of change within populations, did not reflect a significant difference between the two populations. These changes appeared to be a normal increase with age rather than an effect of noise.
- (3) Changes observed in the diastolic blood pressure were not significant, either statistically or biologically.
- (4) Cholesterol levels decreased with successive determinations in both populations. This decrease might have been due to changing dietary and exercise habits.
- (5) Glucose levels decreased with successive determinations in all cases except the final determination in the controls. Although statistically significant alterations in glucose levels were found within each population, no significant difference between the populations was discovered. Therefore, noise did not produce a statistically detectable change in glucose levels in the pilots.
- (6) The noise exposed population demonstrated characteristics of noise induced hearing loss that were not found in the

control population.

- (7) Noise levels inside certain types of aircraft often exceeded levels of damage risk.
- (8) Three general conclusions concerning the results of this study include: (a) either noise does not produce long-term non-auditory physiological responses, or (b) the noise levels to which these pilots are exposed are not of sufficient intensity to produce such responses, or (c) the methods of measuring the physiological parameters involved are not of sufficient sensitivity to detect such responses as might be precipitated by noise.

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West Allis, WI 53227
Harold J. Conlon, M.D., Director

SECTION 2

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

Principal Investigator(s) and address Lyla B. Cartwright, M.D., Ph.D. Robert N. Thompson, Ph.D. Federal Aviation Admin., Civil Aeromedical Inst.		Institution and address where research was performed Federal Aviation Administration, Civil Aeromedical Institute P.O. Box 25082 Oklahoma City, OK 73125	
Investigator's Telephone #	Sponsoring Organization FAA		
Citation: Cartwright, L. B. and R. N. Thompson. The effects of broadband noise on the cardiovascular system in normal resting adults. American Industrial Hygiene Association Journal 36(9):653-658, Sept. 1975.			
# of Ref.'s	8	# of Fig.'s	12
Type & duration of experiment controlled laboratory (audiometric test chamber) short-term - 1 hour noise exposure 1 hour quiet-control (on different days)		Purpose for study to record statistically significant changes in blood pressure, pulse rate, and pulse pressure in humans after exposure to industrial type noise.	
Description of test groups (subjects, #, age, etc.)		1 group - each subject served as his own control 20 healthy adult human volunteers, age 17-49 years, 11 females, 9 males activity of subjects - resting, seated	
Control of other stressors laboratory conditions used		Statistical Methods: test and control values compared using the standard paired t-test. CVS Response Measured: blood pressure (BP), pulse rate, pulse pressure, EKG recorded at 2 min. intervals.	
Noise Stimulus source: random noise generator spectral characteristics: broad band (spectrum included) noise level: 91 dBA ± 1 dB length of exposure: 1 hour noise # of trials: 1 2-hour test run and 30 min quiet) 1 2-hour control (quiet) run per subject on different days.		Physiological effects CVS - transient changes occurred (attributed to chair rest, not noise exposure) Other - A temporary threshold shift (TTS) was produced by 91 dBA noise.	
Author's conclusions: No statistically significant changes in the CVS responses measured occurred in subjects exposed to 1 hour of 91 dBA noise. Two hours of chair rest did produce significant changes - decreased systolic BP and pulse pressure, decreased heart rate and increased diastolic BP under both quiet and 91 dBA noise levels.			
Evaluation & comments: The exposure time (1 hr.) was too brief to indicate the effects of industrial noise conditions on the CVS. The amount of the TTS due to noise was not reported.			

Cartwright, L. B. and R. N. Thompson. The effect of broadband noise on the cardiovascular system in normal resting adults. American Industrial Hygiene Association Journal 36(9):653-658, Sept. 1975.

The cardiovascular effects of one hour exposure to 91 dBA broadband noise were studied in 20 healthy adult volunteers, aged 17 to 49 years. The 11 women and 9 men each served as their own controls. A 2 hour test period and a 2 hour control period were run on each subject on different days in an audiometric test chamber. The test runs consisted of 30 minutes quiet conditions about 38 dBA, 1 hour of 91 dBA noise from a random noise generator, and 30 minutes of quiet (38 dBA). The control runs consisted of 2 hours of quiet (about 38 dBA). The subjects were seated in a chair and resting while in the test chamber. The blood pressure, pulse rate, pulse pressure, and electrocardiograms of the subjects were recorded automatically at 2 minute intervals in both the test and control runs. Audiograms were performed on the subjects during the pre- and poststimulus 30 minute periods in the test runs and during the same time periods in the control runs. The 91 dBA noise level was strong enough to produce a temporary threshold shift (TTS) of unspecified intensity in the subjects. The data was analyzed, using the standard paired t-test of the raw values for the subjects under both test and control conditions for corresponding units of time. No statistically significant changes due to noise were found in the cardiovascular parameters measured. The following significant changes were recorded due to the 2 hours of chair rest: slower heart rate, decreased pulse pressure and systolic blood pressure, and increased diastolic blood pressure. Graphs of the spectra under both the noise and quiet conditions are included. The raw data is presented in tabular form.

Blood pressure, pulse rate and pulse pressure were determined at 2-minute intervals on 20 healthy subjects while they were exposed to 91-dBA continuous broadband noise for 1 hour. Control data were obtained by replication without the noise. The noise produced no statistically significant changes in any of the cardiovascular parameters under study.

The effects of broadband noise on the cardiovascular system in normal resting adults

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Introduction

In 1970, Anticaglia and Cohen¹ published a review of the then available literature on the extra-auditory effects of noise on man. Their paper was especially interesting because it revealed that this aspect of noise exposure had received little attention and that much of what had been done was controversial, contradictory, and in some instances, only conjectural. They noted that, in general, American noise experts in contrast to their European counterparts, believed that environmental noise presented

but little threat to nonauditory systems. The prevailing view in this country at the time of their review was that adaptation to excessive noise minimized the likelihood of persistent alterations in physiologic function. The intent of their paper was "... to create active interest in and concern about this subject in those specialists having medical, hygiene, and safety responsibilities in industry..." and they concluded by calling for more study into the possible extra-auditory pathological effects of noise.

A review of the literature for this paper has shown that uncertainty surrounding the subject still persists, particularly in regard to the effects of noise on the cardiovascular system. It seems that investigators have generally chosen the convenient epidemiologic approach of retrospectively studying the incidence of cardiovascular disease among industrial workers exposed to noise or the less convenient but "quick result" method of measuring the startle noise effects on the cardiovascular system and extrapolating them to possible permanent effects. In both instances the inferences are somewhat tenuous.

There have been few controlled laboratory investigations into the effects of prolonged noise on the blood pressure and pulse rate of humans. Ponomarenko² studied the influence of several noises of various intensities and spectral characteristics on the cardiovascular



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functions in normal adolescent boys. He reported a lowering of the pulse pressure, a decrease in systolic pressure, an increase in diastolic pressure, and a decrease in pulse rate after 1 hour of exposure to 75-dB "wideband" noise. Identical tests, using the same subjects not exposed to noise, were used for control.

Terent'yev et al.³ exposed subjects to 1 hour of approximately 101-dB noise and reported decreases in heart rate and systolic and diastolic blood pressures. However, neither the spectral characteristics of the noise nor the activity of the subjects was reported, and no mention was made of control or the method of data analysis.

Lehmann and Tamm⁴ in 1956 published what is perhaps the most widely quoted article about man's cardiovascular response to prolonged noise. Eighteen healthy test subjects were exposed to various broadband 90-phon noises for 1 hour while blood pressures, pulse rates, and ballistocardiograms were recorded. The authors reported all their results in tables which indicated only that a particular parameter increased or decreased following exposure to noise. They concluded that there was little change in pulse rate and that pulse pressure amplitude showed a tendency to narrow, an occurrence attributable primarily to an elevation of the diastolic values. These observations were not substantiated by any numerical data.

This study was designed to try to measure any statistically significant changes (at the $\alpha = .05$ level) in blood pressure, pulse rate, and pulse pressure (the difference between systolic and diastolic blood pressure) in subjects after prolonged exposure to industrial-type noise.

Procedure

Twenty healthy adult subjects, 11 females and 9 males, ranging in age from 17 to 49, were exposed to 91-dBA (ref. 0.0002 dyne/cm²) broadband noise in an audiometric test chamber for 1 hour, during which time systolic and diastolic blood pressures, pulse rates, and electrocardiograms were automatically recorded at 2-minute intervals. Each subject served as his own control when, on another day, the same cardiovascular parameters were measured in the chamber under "quiet" conditions (approximately 38 dBA).

The 91-dBA sound level was selected because it was, at the time this study was conducted, a value just greater than that permitted

in American industry for an 8-hour workday without ear protection. This level was sufficiently intense to produce temporary damage to the ears in most subjects as detected by a temporary threshold shift (TTS) in their poststimulus audiograms. According to some physiologic theory, a stimulus great enough to produce a TTS should contain enough energy to "spill over" into another system, such as the cardiovascular system, and thereby produce a measurable alteration.

During the first 30 minutes of each test run, a 38-dBA sound level was maintained to adapt the subject to his environment and to perform the prestimulus audiogram, whereupon the noise was introduced through a 6" x 9" loudspeaker and adjusted to 91 dBA \pm 1 dB as measured 3 inches from the subject's left ear. The spectra for both quiet and noise conditions are shown in Figure 1 for comparison with the noise used by Ponomarenko.² The noise was produced by a General Radio Company random noise generator, set to the United States of America Standards Institute (USASI) mode, driving a 100-watt Bogen power amplifier. The noise was monitored and recorded continuously during all runs with a Bruel and Kjaer (B&K) microphone amplifier, type 2603, and a B&K graphic level recorder, type 2305.

At the end of 1 hour of noise, the source was silenced and a poststimulus audiogram performed. The subject remained in the quiet chamber until the 2-hour run was completed.

Systolic and diastolic blood pressures were determined automatically by using a Narco bio-systems electrospigmomanometer, model ESG-300, and were recorded on one channel of a Model 5 Grass polygraph.

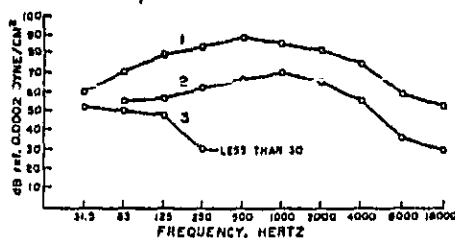


Figure 1 - Spectral analysis of: 1) noise used in this study, 2) noise used by Ponomarenko, and 3) "quiet" conditions of this study.

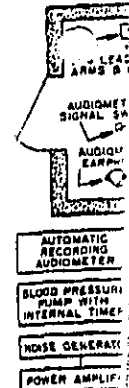


Figure 2 - system.



Figure 4 - using eq.

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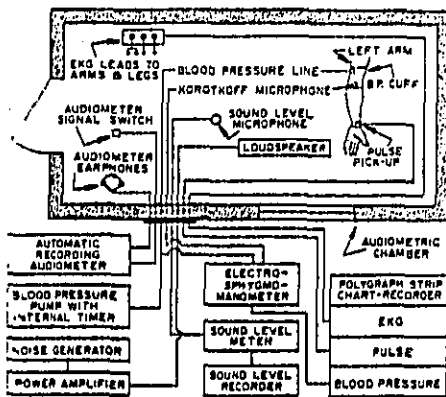


Figure 2 - Schematic diagram of experimental system.

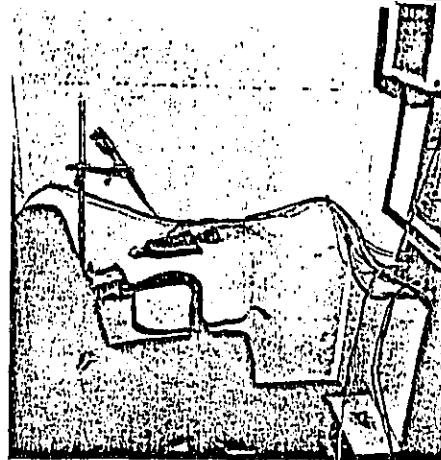


Figure 3 - View of inside of test chamber.



Figure 4 - Test subject with physiological measuring equipment attached.

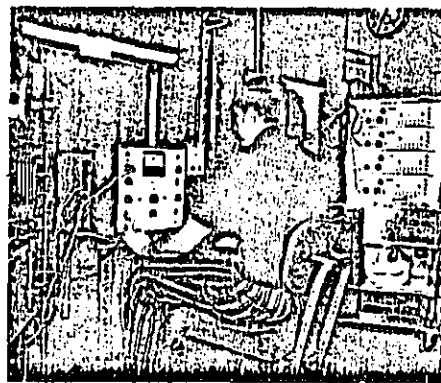


Figure 5 - External view of test chamber and operator monitoring test and recording equipment.

A standard limb lead I electrocardiogram was obtained from the arms by using large silver electrodes, and it was continuously recorded on a second channel of the polygraph.

A third channel recorded the pulse, which was detected by a piezoelectric crystal located over the left radial artery at the wrist where the artery is superficial.

All audiometry was performed with a Grayson-Stadler recording audiometer, model 1703.

The complete experimental system is shown schematically in Figure 2 and illustrated in Figures 3, 4, and 5.

Results

For each of the 20 subjects, two 2-hour runs were performed, and these provided a total of 80 hours of test and control monitoring. Recording the designated cardiovascular parameters at 2-minute intervals yielded an unwieldy 9,600 observations, which were reduced to 3,200 mean values that were time dependent as follows: For each test and control run, there were originally 60 numerical values for each parameter measured. These were converted to 20 "means of size three" so that in plotting, the first data point represented the mean of measurement made at 2, 4, and 6 minutes into

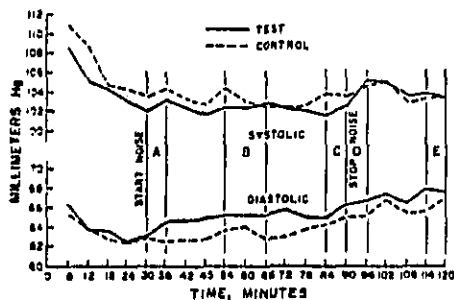


Figure 6 - Mean systolic and corresponding mean diastolic blood pressures based on raw data for all subjects during all experimental runs (20 control, 20 test).

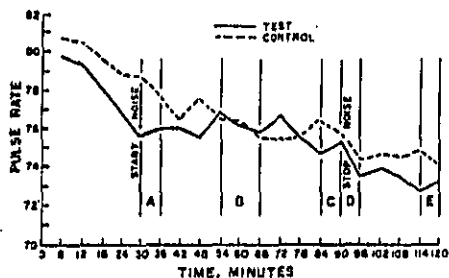


Figure 7 - Mean pulse rates based on raw data for all subjects during all experimental runs (20 control, 20 test).

a run, and data point two was the mean of measurements made at 8, 10, and 12 minutes. This conversion was continued to data point 20, which was the mean of measurements made at 116, 118, and 120 minutes, the last 6 minutes of a run.

The raw data parameters of blood pressure and pulse rate, averaged for each point in time for all subjects for both control and test conditions, are presented graphically in Figures 6 and 7. It is readily apparent that blood pressure, pulse rate, and pulse pressure did undergo changes during the course of the 2-hour run.

To determine if the observed differences between the test and control runs were not merely chance occurrences, the observed changes were evaluated by using data representing five arbitrarily chosen time periods in

the test runs and their corresponding time periods in the control runs. The five periods are designated in Figures 6 and 7 and described as follows: (A) the first 6 minutes of noise; (B) the central 12 minutes of noise; (C) the last 6 minutes of noise; (D) the first 6 minutes after cessation of noise (i.e., the start of the quiet period after the 1-hour exposure to noise); and (E) the final 6 minutes of a 2-hour run.

The change of the mean value of a parameter during each of these time periods from the mean value of that parameter during the first 30 minutes was determined for the test runs. Similar data were generated for the control runs. These test and control "change values," which represented both magnitude and direction of change, were compared by using the standard paired t test.⁵

As shown in Table I, only two of the 20 t tests were significant at the $\alpha = .05$ level, and both occurred in the systolic blood pressure test.

The most important tests were the comparisons of the changes in blood pressure, pulse rate, and pulse pressure during period C, for by then the subjects had experienced the longest exposure to the test noise. However, none of the parameters revealed a statistically significant change.

The original null hypothesis (i.e., H_0 : noise causes no statistically significant change in any of the cardiovascular parameters under study) was not rejected because the t values for all parameters for period C were too small for statistical significance at the $\alpha = .05$ level. When the null hypothesis is not rejected, the

TABLE I
Results of t testing on raw data.

TIME PERIOD	PARAMETER	t VALUE	SIGNIFICANCE
A	Systolic BP	1.08	NS
B		2.23	.05
C		0.87	NS
D		2.59	.05
E		1.81	NS
A	Diastolic BP	1.65	NS
B		1.10	NS
C		0.55	NS
D		0.61	NS
E		0.21	NS
A	Pulse Rate	0.01	NS
B		1.59	NS
C		0.85	NS
D		0.76	NS
E		0.60	NS
A	Pulse Pressure	-0.47	NS
B		0.23	NS
C		0.03	NS
D		1.89	NS
E		1.37	NS

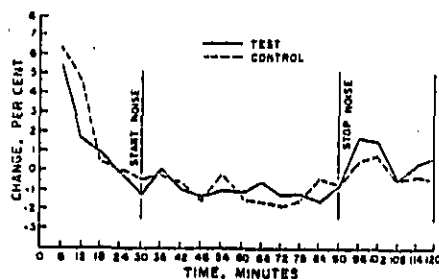


Figure 8 - Percentage change in systolic blood pressure from mean.

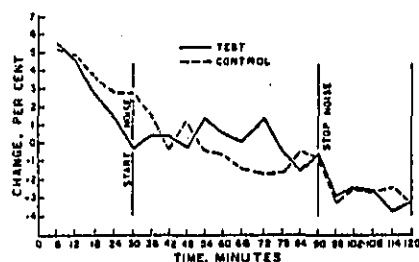


Figure 10 - Percentage change in pulse rate from mean.

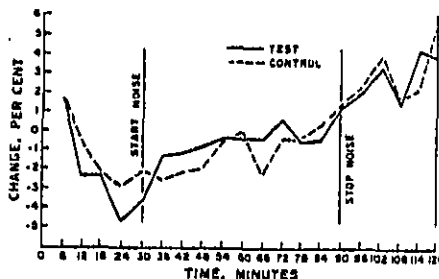


Figure 9 - Percentage change in diastolic blood pressure from mean.

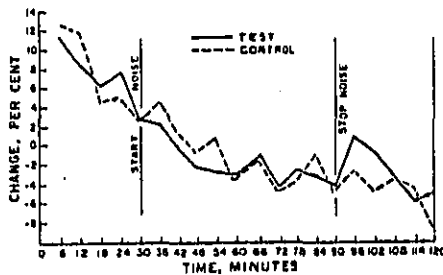


Figure 11 - Percentage change in pulse pressure from mean.

magnitude of the β (Beta) power of the test becomes extremely important, since the β power measures the probability of finding a difference between the test and control conditions if a difference is present. This difference must be specified before the β power can be determined.

The alternate hypothesis (H_A) was that noise will produce a ± 6 mm or greater change in blood pressure or a ± 6 beat-per-minute change in pulse rate. In every case the β power was greater than 97.5%, which indicated a high probability of detecting the stated difference between test and control conditions.

Among the several factors that can influence the levels of blood pressure and pulse rate are the emotional "set" of a subject on a given day⁷ and the season of the year.⁸ Consequently, it was decided to convert the raw data for each subject into percentage changes from the mean values, which would help to eliminate these additional sources of error. The results of this

conversion are shown in Figures 8 through 11. A complete examination of the methodology used and the tabulated data are contained in the work by Cartwright.⁹

It is not statistically permissible to use the same type of paired t tests on the data after such manipulation. However, the graphs do accurately illustrate the behavior trends of the various parameters. It should be noted that under the conditions of both quiet and noise, the following occurred:

- a. The systolic blood pressure decreased for about 90 minutes and then became stable. It increased, however, when the second audiogram was performed.
- b. The diastolic blood pressure decreased for approximately 30 minutes, then started to increase and continued to do so throughout the experiment. The diastolic blood pressure did not seem to be affected by the second audiogram.
- c. The pulse rate decreased rapidly during

the first 30 minutes and then decreased more slowly for the next hour. The pulse rate dipped at the time of the second audiogram, perhaps in a reflex manner in response to the systolic blood pressure increase at this point.

- d. The pulse pressure decreased throughout the entire 2 hours of the experiment, but, as would be expected, it did show a transient increase at the time of the second audiogram. (The systolic blood pressure increased at this point and the diastolic blood pressure was unchanged; hence, the pulse pressure increased.)

Eight paired t tests were performed on the percentage change data comparing each parameter's initial value to its value at the end of the experiment. All eight were significant at the $p = .001$ level, which indicated that all the factors under study were time dependent in a highly significant manner.

Conclusions

The cardiovascular parameters of blood pressure, pulse rate, and electrocardiogram were continuously monitored during both noise exposure and quiet control conditions. Because these cardiovascular parameters are constantly changing, an attempt was made to detect any difference in these changes under noise stress as compared to changes under quiet control conditions. From the results of this study, the following conclusions were made:

- a. The exposure of resting normal adult volunteer subjects to broad-band noise of 91 dBA for a period of 1 hour produces no statistically significant change in any of the parameters at the $\alpha = .05$ level. The β power of these tests was always greater than 97.5% when the alternate hypothesis (that noise would produce changes of ± 6 mm of mercury in the blood pressures or ± 6 beats per minute in the pulse rate) was applied.

- b. Two hours of chair rest in normal subjects produce the following cardiovascular changes, all of which are statistically significant at the $p = .001$ level:

- (1) a relative bradycardia;
- (2) a decrease in the systolic blood pressure;
- (3) an increase in the diastolic blood pressure; and
- (4) a marked decrease in the pulse pressure.

- c. In future investigations of this type, the fact that the blood pressure, pulse rate, and pulse pressure in normal resting human subjects do not reach a stable baseline condition in less than 1½ hours must be kept in mind.

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SECTION 3

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Deryagina, G.P. Sinitsina, T.S. Veselova, T.V.		<u>Institution and address where research was performed</u> Pavlov's Institute of Physiology Acad. Sci. USSR and Institute for Experimental Medicine/ Acad. Med. Sci. USSR, Leningrad	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Russian gov't.		
<u>Citation</u> Deryagina, G.P. et al. Effect of acoustic stimulation on lipid metabolism indices of the blood coagulation system and development of experimental atherosclerosis in rabbits. Fiziol. Zh. SSSR 62 (8):1171-1181, Aug. 1976			
<u># of Ref.'s</u> 23	<u># of Fig.'s</u> 8	<u>Language</u> Russ. (Eng. trans.)	
<u>Type & duration of experiment</u> 5 months; laboratory study		<u>Purpose for study</u> to study the effect of noise on lipid metabolism and on the development of atherosclerosis	
<u>Description of test groups (subjects, #, age, etc.)</u> 53 female rabbits, 2.7-3.2 kg.: control group--15 rabbits--ordinary diet test group--38 given 500 mg cholesterol in 5.0 ml. sunflower oil orally daily for 4.5--5 months			
<u>Control of other stressors</u> laboratory conditions--the animals were very frightened by the noise		<u>Statistical Methods</u> student t-test	
<u>Noise Stimulus</u> source: not given spectral characteristics: 3kHz noise level: 94-96 dB length of exposure: 4.5 hr./day with 2-30 min. quiet periods 14 or 28 days # of trials: 4 groups given noise stimulation		<u>CVS Response Measured</u> (1) pathological exam. of aorta, heart (2) platelet adhesiveness <u>Nonauditory effects</u> increased platelet adhesiveness & increased atherosclerotic changes due to noise. increased hypercoagulation due to noise. Increased nonesterified fatty acids due to noise. wt. loss due to noise. blood coagulation factors increased. behavior--aggressive; fear	
<u>Author's conclusions</u> Noise stimulation caused increased levels of nonesterified fatty acids and blood coagulation factors (hypercoagulation). Hypercoagulation was enhanced by cholesterol feeding. Noise alone induced microscopic arterial necrosis and other atherosclerotic changes to a lesser degree in rabbits exposed to noise for 28 days than in those exposed for 14 days.			
<u>Evaluation & comments</u> Noise may enhance the development of atherosclerosis in rabbits fed excess cholesterol levels. Individual differences between rabbits were observed. Relatively small numbers of rabbits in each group were studied.			

Deryagina, G. P. et al. Effect of acoustic stimulation on lipid metabolism, indices of the blood coagulation system and development of experimental atherosclerosis in rabbits. Fiziol. Zh. SSSR 62(8):1171-1181, Aug. 1976. (English translation)

The effects of noise and cholesterol on the development of atherosclerosis were studied in female rabbits weighing 2.7 to 3.2 kg. A total of 53 rabbits were divided into groups and treated as follows:

<u>Test Series</u>	<u>Group</u>	<u># of Animals</u>	<u>Treatment and Duration</u>
I	Test	7	No cholesterol; noise 14 days
	Control	8	No cholesterol; no noise
II	Test	12	4.5 months cholesterol; 1 month no cholesterol; 14 days noise
	Control	8	4.5 months cholesterol; 1 month no cholesterol; no noise
III	Test 1	6	4.5-5 months cholesterol (noise for 1st 14 days)
	Test 2	6	4.5-5 months cholesterol; (noise for 1st 28 days)
	Control	6	4.5-5 months cholesterol; no noise

The noise dose was 94-96 dB at 3 KHz. Daily exposure was for 4.5 hours with two 30 minute quiet periods every 1.5 hours. The daily dose of cholesterol used was 500 mg in 5 ml. sunflower oil, given orally. Blood samples were examined for changes in platelet adhesiveness and various coagulation factors, beta lipoprotein levels, unesterified fatty acid levels, and cholesterol levels. The hearts and aortae of the rabbits were examined for atherosclerotic changes. Major changes due to noise stimulation included increased platelet adhesiveness, higher levels of nonesterified fatty acids, and increased hypercoagulation. Noise both induced some microscopic atherosclerotic changes by itself and enhanced gross atherosclerotic changes in the coronary arteries caused by the atherosclerotic diets.

EFFECT OF ACOUSTIC STIMULATION ON LIPID METABOLISM,
INDICES OF THE BLOOD COAGULATION SYSTEM AND DEVELOPMENT
OF EXPERIMENTAL ATHEROSCLEROSIS IN RABBITS

By G.P. Deryagina, T.S. Sinitsina, T.V. Veselova

Fiziol.Zh. SSSR 62(8): 1171-81; Aug. 76

Laboratory of clinical and experimental cardiology
(I.E. Gapelina, director) Institute of Physiology
named after I.P. Pavlova, AN SSSR;
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Institute of experimental medicine AMN SSSR, Leningrad

The effect of sound caused an increase in the level of non-esterified fatty acids in healthy rabbits over the course of 14 days, as well as an increase of blood coagulation. Morphologically we detected hypertrophy of the heart venal* arteries, as well as focal points of necrosis in the myocardium. In the case of rabbits with experimental atherosclerosis, subjected to the effect of sound for periods of 14 and 28 days, despite the high level of lipids in the blood and hypercoagulating shifts, conditions were created facilitating resorption of the lipids on the platelets of the aorta and coronary arteries. Moreover, morphologically we also found hypertrophy and edema of the vessel walls, focal points of necrosis, focal point and diffuse adipose infiltration of the heart muscle.

Many experimental studies have shown that excessive stress to the nervous system of different types increases vascular permeability, facilitates an increase in the lipid fractions of the blood and a precipitation of the lipids in the artery walls, and causes hypercoagulation shifts in the blood coagulation system [3,8,10,16].

By means of frequent changes in the stereotypes of rabbits and dogs, P.S. Xomulo succeeded in causing atherosclerotic changes in the aorta and in the large heart arteries [15].

However, the influence mechanism of overstress of the nervous system on the development of the atherosclerotic process has not been finally explained. It is also not clear if the character of the stress effect is important in the development of the atherosclerotic process.

The purpose of the present work is to study the question concerning the influence of extended overstraining of the nervous system, caused by sound stimulation, on the development of experimental atherosclerosis in rabbits.

* This should be coronary.

Method

The work was carried out on 53 female rabbits with a weight of 2.7-3.2 kg. Of the 53 rabbits, 38 received 500 mg cholesterol in 5.0^{ml} sunflower oil per os daily for a period of 4.5-5 months, and 15 of the rabbits were kept on their ordinary feed. Some of the rabbits (34 of the 53) were subjected to the acoustical effect (94-96 dB, 3 kilohertz) for a period of 4.5 hours per day (with two 30 minute interruptions for rest every 1.5 hours of the effect). The period of the sound effect was 14 and 28 days.

3 series of studies were carried out (Table 1). 15 rabbits were used in the first series, which were kept on their ordinary feed. 7 of them were subjected to the acoustical effect for a period of 14 days (group B). 8 of the rabbits served as a control group (group A). 20 rabbits were used in the second series, which, in addition to the usual ration, received cholesterol in the indicated dose for a period of 4.5 months. Then they were transferred over to ordinary feed ("for rest"). One month after removal of the cholesterol, 12 of the 20 rabbits were subjected to the acoustical effect for a period of 14 days (group B). 8 of the rabbits (group A) were not subjected to the acoustical effect. 18 rabbits were used in the 3rd series, receiving cholesterol for a period of 4.5-5 months. For 4 months from the start of feeding with cholesterol, 12 of them were subjected to acoustical effect against a background of continued giving of cholesterol (6 in a period of 14 days--group B, 6 for a period of 28 days--group B), and 6 (group A) served as a control group (these were not stimulated).

The blood was studied twice: immediately before the start of the acoustical effect and after it was stopped in conformity with the period of stimulation. Then the rabbits were sacrificed. The rabbits not subjected to the acoustical effect (group A) were examined in the same period of time.

We determined the indicators of lipid metabolism and the system of blood coagulation: cholesterol [22], total fraction of β -lipoproteides [5], the non-esterified fatty acids [17], time of blood coagulation [20], recalcification time [21], tolerance of the plasma to heparin [23], prothrombin complex [14], fibrinogen [19], XIII factor [1], thrombin and heparin time [12], adhesiveness of the thrombocytes [18], fibrinolytic activity of the blood [2]. The blood stabilizer is 1.34% sodium oxalate. Its ratio in the blood is 1:4. The obtained numerical data were statistically processed by a different method with the use of the Student criterion t.

* This should be 5.0 ml.

Table 1.--Distribution of rabbits according to groups as a function of diet and sound stimulation.

Test series	Cholesterol general diet	Group or rabbits	No. of rabbits	Period of cholesterol feeding	Beginning of sound stimulation	Period of sound stimulation
I		A	8	--	--	--
		B	7	--	--	14 days
II		A	8	4.5 months	--	--
		B	12	Same	For 1 month after removal of cholesterol	14 days
III		A	6	"	--	--
		B	6	"	After 4 months	14 days
		C	6	5 months	Same	28 days
Total			53			

Table 2.--Change of the indicators (M±m) of lipid metabolism in rabbits without the effect (I) and under the influence of sound stimulation (II). (1) Animals; (2) Series; (3) Group; (4) Period of sound affect; (5) Cholesterol (in mg.%); (6) Total fraction of β-lipoproteids (in mg.%); (7) Nonesterified fatty acids (in ml equiv/l); (8) Studies; (9) Without the effect; (10) 14 days; (11) 28 days.

Series (1)	Group (2)	Duration of sound stimulation (3)	Duration of sound stimulation (4)	(5) Cholesterol (in mg.%)		(6) Summation fraction β-lipoproteins (in mg.%)		(7) Nonesterified fatty acids (in ml equiv/l)	
				I	II	I	II	I	II
I	A	14 days	Без воздействия (9)	75.3±14.5	73.5±14.4	189.2±41.4	205.0±48.3	625.0±73.1	645.3±47.6
			14 дней (10)	76.7±13.7	77.7±20.7	216.7±40.7	198.8±44.5	612.6±65.3	827.1±71.9
II	A	14 days	Без воздействия (9)	264.0±30.1	324.3±35.6	1050.0±252.0	734.3±187.0	077.5±114.0	750.0±66.3
			14 дней (10)	267.0±42.0	341.2±50.5	1030.0±191.0	897.9±182.2	607.5±70.9	797.9±69.1
III	A	14 days	Без воздействия (9)	503.8±67.3	520.0±63.1	2352.0±366.2	2297.1±436.0	1654.3±91.7	961.0±77.9
			14 дней (10)	520.0±69.5	521.8±93.8	2192.0±473.0	2390.1±257.4	1395.0±149.4	793.3±56.6
	B	28 days	(11)	518.5±70.3	538.5±54.8	2341.0±351.4	2249.1±409.9	1325.1±125.0	966.0±63.0

Table 3.--Changes in the blood coagulation indicators with rabbits under the influence of sound stimulation (M+m)
 (1) Animal; (2) Series; (3) Group; (4) Period of sound effect; (5) Indicator; (6) Blood coagulation time (in seconds); (7) Time of plasma recalcification (in sec); (8) Tolerance of the plasma to heparin (in sec); (9) Prothrombin complex; (10) fibrinogen (in mg.%); (11) Studies; (12) Without effect; (13) 14 days; (14) 28 days; (15) Table 3 (continued); (16) XIII factor (in %); (17) Adhasiveness of thrombocytes (in %); (18) Thrombin time (in sec); (19) Heparin time (in sec); (20) Fibrinolytic activity of the blood;

Исследования (1)		Длительность звукового воздействия (4)	Показатель (5)									
№ животного (2)	№ серии (3)		время свертывания крови (в сек.) (6)		время рециальфикации плазмы (в сек.) (7)		толерантность плазмы к гепарину (в сек.) (8)		протромбиновый комплекс (в %) (9)		фибриноген (в мг.%) (10)	
			I	II	I	II	I	II	I	II	I	II
I	A	14 дней (13)	152.5 ± 21.4	108.1 ± 25.1	126.0 ± 10.0	108.0 ± 12.3	240.0 ± 13.1	260.3 ± 14.9	65.5 ± 1.8	60.7 ± 3.5	207.5 ± 20.6	213.7 ± 16.2
	B		160.2 ± 14.8	126.5 ± 24.4	135.5 ± 16.1	84.7 ± 15.8	254.7 ± 32.5	135.8 ± 24.5	87.8 ± 3.1	92.2 ± 2.2	217.1 ± 10.4	258.5 ± 15.8
II	A	14 дней (13)	183.1 ± 30.4	186.2 ± 28.2	108.0 ± 20.6	95.7 ± 8.6	258.3 ± 37.5	190.5 ± 25.7	65.1 ± 3.4	65.6 ± 3.2	237.5 ± 8.3	211.2 ± 21.5
	B		220.0 ± 20.8	175.0 ± 19.8	139.5 ± 26.4	128.1 ± 8.5	264.8 ± 26.1	170.5 ± 28.0	68.2 ± 3.8	61.2 ± 2.0	215.0 ± 9.5	228.7 ± 12.0
III	A	28 дней (14)	168.3 ± 20.2	151.6 ± 18.5	105.0 ± 14.1	105.8 ± 14.2	178.3 ± 35.4	175.0 ± 23.8	61.0 ± 2.6	61.3 ± 2.4	260.8 ± 23.2	211.6 ± 8.3
	B		136.0 ± 10.4	152.5 ± 22.0	107.6 ± 12.1	61.8 ± 9.7	141.8 ± 31.0	107.6 ± 9.6	65.8 ± 2.5	69.1 ± 2.5	223.3 ± 22.0	211.0 ± 10.1
			161.3 ± 18.2	90.0 ± 0.4	128.0 ± 18.5	70.3 ± 5.4	168.2 ± 18.5	107.6 ± 0.1	64.0 ± 2.4	67.3 ± 1.1	220.6 ± 18.8	211.6 ± 10.0

(13) Таблица 3 (продолжение)

Исследования (1)		Длительность звукового воздействия (4)	Показатель (5)									
№ животного (2)	№ серии (3)		XIII фактор (в %) (16)		адгезивность тромбоцитов (в %) (17)		тромбиновое время (в сек.) (18)		гепариновое время (в сек.) (19)		фибринолитическая активность крови (20)	
			I	II	I	II	I	II	I	II	I	II
I	A	14 дней (13)	116.5 ± 6.9	80.3 ± 0.1	25.8 ± 2.0	23.1 ± 2.1	24.6 ± 1.4	22.8 ± 1.4	10.0 ± 0.65	9.3 ± 1.0	1.55 ± 0.16	1.20 ± 0.78
	B		111.7 ± 13.5	91.5 ± 16.0	26.0 ± 2.9	21.4 ± 2.4	23.2 ± 1.4	23.5 ± 1.8	10.6 ± 1.20	9.4 ± 1.0	1.53 ± 0.51	3.71 ± 1.47
II	A	14 дней (13)	110.0 ± 10.5	87.2 ± 11.9	26.5 ± 5.2	30.9 ± 3.0	19.8 ± 1.5	21.6 ± 2.1	8.5 ± 0.8	9.7 ± 1.8	3.17 ± 1.2	5.85 ± 1.3
	B		87.1 ± 8.2	100 ± 8.3	29.0 ± 2.7	27.8 ± 1.5	20.1 ± 1.5	19.7 ± 1.4	8.8 ± 0.8	7.3 ± 1.0	3.07 ± 0.5	4.24 ± 0.88
III	A	14 дней (13)	80.3 ± 7.3	103.1 ± 0.5	26.0 ± 5.8	22.7 ± 2.0	21.1 ± 1.3	24.3 ± 0.6	10.0 ± 0.7	9.3 ± 1.1	4.3 ± 0.9	3.0 ± 0.7
	B		85.9 ± 15.0	96.0 ± 12.5	20.7 ± 4.1	31.0 ± 2.4	27.6 ± 2.6	24.3 ± 1.0	12.8 ± 1.0	9.5 ± 1.2	5.9 ± 1.1	1.83 ± 0.10
			95.4 ± 15.2	148.5 ± 17.2	23.2 ± 3.0	36.4 ± 2.7	25.3 ± 1.3	21.1 ± 1.0	13.1 ± 1.1	8.5 ± 1.0	5.0 ± 1.05	1.75 ± 0.24

The material is fixed in 10% neutral formalin. After fixing of the aorta, everything is colored with sudan III. After composing the distribution schemes of the different sections of the aorta (arch, breast and abdominal segment), pieces from 5 to 10 are cut out (depending on the expression of the changes). The sections are prepared on a frozen microtome and colored oil red; the remaining part of the pieces were filled with paraffin-cellogin, the sections were colored according to Van Gizon and according to Veigert-Chart on elastic fibers. The hearts of the rabbits of the experimental and control groups were cut into transverse segments (4-5) which were also studied like the aorta.

Experimental Results

The rabbits subjected to sound stimulation to the end of the experiment changed their behavior. They became aggressive, attacked each other, fought frequently. At the same time, they appeared frightened. At the sight of the experimenter, they hid in the corner of the chamber. With a large part of the rabbits subjected to the effect of sound in the course of 28 days, the weight dropped 600-1000 g. In the case of the rabbits subjected to the effect of sound for 14 days, the weight drop was less.

In the first series of experiments on rabbits on ordinary feed (group B), the sound stimulation did not cause an increase in the level of cholesterol and of the total fraction of β -lipoproteids; we noticed a real increase in the level of non-esterified fatty acids from 612.8 ± 68.3 to 827.1 ± 71.0 m μ eqv/l ($p < 0.05$). In the case of rabbits found on the same ration and not subjected to the effect of sound (group A), the changes in the level of nonesterified fatty acids were not observed in the study carried out at the same time. Under the influence of sound effect, there was an increase in blood coagulation (the recalcification time was shortened from 135.5 ± 16.1 to 84.7 ± 15.8 sec., $p < 0.05$). There was an increase in the tolerance of plasma to heparin from 254.7 ± 32.5 to 135.8 ± 24.5 sec ($p < 0.05$). The other hemocoagulation indicators did not substantially differ from the values of the control group (Table 2.3).

In opening up the rabbits of the different series of experiments, sacrificed at different times, we did not find any visible changes with a general examination. Visible changes did not appear in macroscopic studies of rabbit aorta of the 1st series, either with those subjected or not subjected to the effect of sound. With a microscopic study of the coronary arteries of the heart and myocardium of group B rabbits, we found changes in the form of hypertrophy of the intramuscular wall branches and sharply expressed venous hyperemia. Small focal points of necrosis were observed near the artery changes. Such changes on the side of the vessel wall and the myocardium were not observed in the case of the rabbits not subjected to sound effects (group A).

In the IIInd series of experiments with rabbits kept on ordinary food with the addition of cholesterol for a period of 4.5 months and "rest" after its removal during the last month, the level of cholesterol and of the total fraction of β -lipoproteids before the start of experiments was higher in both groups than in either group of the first series of experiments. A substantial difference was not found in the level of nonesterified fatty acids. With repeated studies for 14 days, the cholesterol level and the total fraction of β -lipoproteids were lower in both groups, which is connected with the cholesterol metabolism. The level of nonesterified fatty acids in group B increased from 607.5 ± 70.9 to 797.5 ± 69.1 mlekvl (p < 0.05). The hemocoagulation indicators in the case of rabbits taken off cholesterol practically did not differ from their values in series I. Under the influence of stimulation (Table 2,3), the tolerance of the plasma to heparin increased from 264.8 ± 26.1 to 176.5 ± 28.0 sec (p < 0.05).

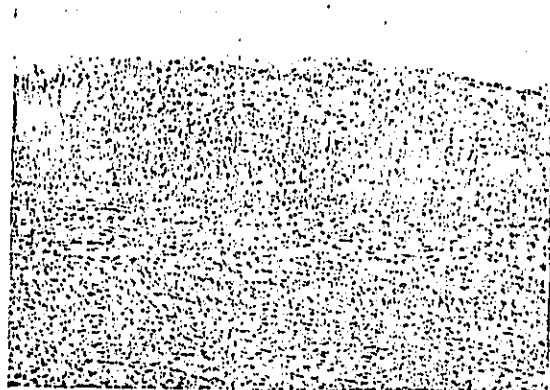


Fig. 1.--Irregular distribution of lipids in the aorta.
x 106. Rabbit No. 473

When opening up the rabbits of the IIInd series, the subcutaneous adipose layer in group B was expressed somewhat less than in group A. With microscopic examination of the aortas of the IIInd series of rabbits, we found atherosclerotic changes expressed in different degrees and they turned out to be alike in both groups. In groups B there were express changes on 3 of the 12, less clear ones on 5, slight ones on 4 of the rabbits. In group A, accordingly, on 3 of the 8, 3 and 2 of the rabbits [sic.]. With microscopic examination in the atherosclerotic platelets* on part of the rabbits of group B we observed rather clearly expressed signs of lipid resorption in the form of unequal distribution of the latter in the platelets* themselves, at places they were almost completely absent, the presence of a large number of cells with lipid inclusion in the cytoplasm (lipid macrophage) and the penetration of lipids in the middle membrane of the aorta (Fig. 1). Atherosclerotic changes, large

* This should be "plaques".

atherosclerotic platelets* were also observed in the coronary arteries of the heart and in the intermuscular and main branches of the coronary heart arteries. Also, in the aorta, in some platelets of the coronary arteries of the heart we observed an unequal distribution of the lipids, giving evidence of the start of their resorption (Fig. 2). Changes were found in the vessel wall and the myocardium, but more expressed, like in the case of the rabbits of group B of the 1st series. Considerable hypertrophy of the walls of many coronary arteries of the heart attracted attention (Fig. 3). Often we found considerable deposits of lipids in the thickened wall, not leading to the formation of platelets, however. As distinct from the intramuscular branches of the coronary arteries of the heart, in the main branches of the artery walls on the other hand we found that its lumen was widened. We turned our attention to the clearly expressed atherosclerotic damage in the coronary arteries of the heart (Fig. 4). In different sections of the myocardium, preferably close to the hypertrophied artery, we observed venous hyperemia, sometimes "old" blood effusion and very clear dystrophic changes (Fig. 5). The changes in the heart muscle appear as focal or diffuse fatty infiltration, although we must not exclude the possibility of its origin due to atherosclerotic heart damage. We also observed small focal points of necrosis and small network scars. In the case of the rabbits of group A, signs of lipid resorption in the aorta and in the coronary arteries were absent against a background of expressed atherosclerotic changes.



Fig. 2.--Large atherosclerotic platelets in the intermuscular branches of the left venal artery of a rabbit heart.

Above (a)--control, lipids with equal distribution in the platelets. x 300, rabbit No. 239; bottom (b) experimental--unequal distribution of lipids. x116, rabbit no. 473.



* This should be "plaques".

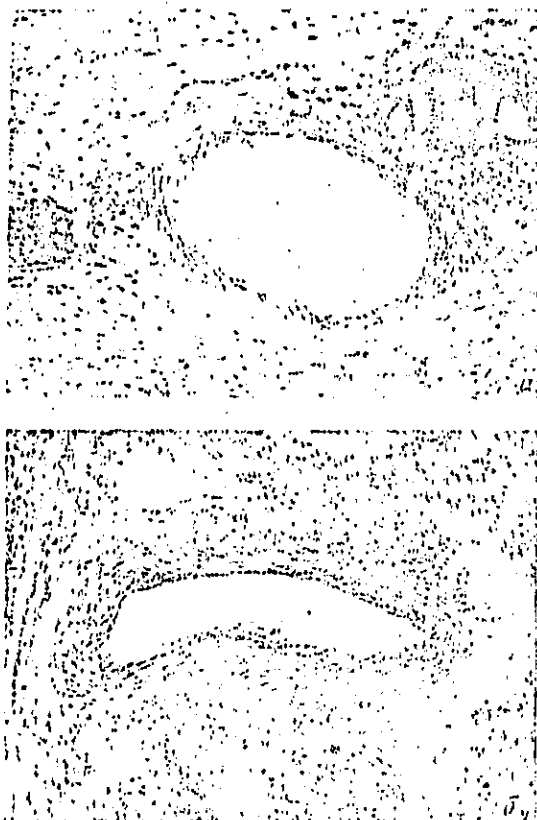


Fig. 3.--Hypertrophy of the wall of the left coronary artery, adema. Above (a)--control x140, rabbit no. 495; below (b)--experimental x106. Rabbit no. 473.

In the case of the rabbits under the influence of extended maintenance on a cholesterol diet (Table 2), the level of cholesterol, of the total fraction of β -lipoproteids, non-esterified fatty acids was considerably higher than in the case of the rabbits of the 1st and IInd series. For 2 (groups A and B) and 4 weeks (group B), the level of cholesterol, total fraction of β -lipoproteids with repeated study practically did not change. The level of nonesterified fatty acids in groups B and C dropped correspondingly from 1308.0 ± 149.4 to 793.3 ± 56.6 m μ qv/l ($p < 0.02$) and from 1325 ± 125.1 to 966.6 ± 63.6 m μ qv/l ($p < 0.01$).

Under the influence of extended feeding with cholesterol in rabbits of the IIIrd series, the heparin and fibrinolytic activity of the blood turned out to be higher than in the 1st series. In this regard, our data is closer to that observed by the other authors. [11] The applied sound stimulation facilitated an increase in the hypercoagulation properties of rabbits in groups B and C: the recalcification time was correspondingly shortened from 107.6 ± 12.1 to 61.8 ± 9.8 sec ($p < 0.05$) and from 128.0 ± 18.5 to 70.3 ± 5.4 sec ($p < 0.05$), an increase of the adhesiveness of the thrombocytes from 20.7 ± 4.1 to $31.0 \pm 2.4\%$ ($p < 0.05$) and from 23.2 ± 3.0 to $36.4 \pm 2.7\%$ ($P < 0.01$), a decrease of blood heparin activity correspondingly from 12.8 ± 1.0 to 9.5 ± 1.2 sec ($p < 0.05$) and from 13.1 ± 1.1 to 8.5 ± 1.0 sec ($p < 0.05$), a decrease of fibrinolytic activity of the blood correspondingly from 5.8 ± 1.1 to 1.83 ± 0.19 ($p < 0.01$) and from 5.9 ± 1.68 to 1.75 ± 0.24 ($p < 0.05$). In group V, in addition to these changes at the end of the sound effect, we noted a shortening of the blood coagulating time from 161.3 ± 19.2 to 90.0 ± 6.4 sec ($p < 0.01$), an increased tolerance of plasma to heparin from 168.2 ± 18.2 to 104.6 ± 6.1 sec ($p < 0.05$), an increase in the level of the XIIIth factor from 95.4 ± 15.2 to $148.5 \pm 17.2\%$ ($p < 0.05$). It should be noted that heparin and fibrinolytic activity of the blood in rabbits of the IIIrd series (groups B and C) at the end of the experiment did not exceed the values in the case of the rabbits of the 1st series.

The morphological data with the macroscopic as well as with the microscopic studies turned out to be analogous with the IInd series (in groups B and C with group B between groups A). However, the degree of expression of the atherosclerotic process turned out to be different in series III. So the atherosclerotic changes were slight in all 6 rabbits of group B which were studied morphologically. In the case of the rabbits of group B, the expressed atherosclerotic changes occurred in 2 of the 6, less expressed in 2 and weakly expressed in 2 of the rabbits. In group A, the expressed atherosclerotic changes were found in the case of 2, less expressed in the case of 1 and slightly expressed in the case of 3 rabbits. So, in the case of rabbits subjected to the effect of sound for a period of 28 days, the degree of atherosclerotic changes in the vessels was less expressed than in the case of rabbits with shorter stimulation.

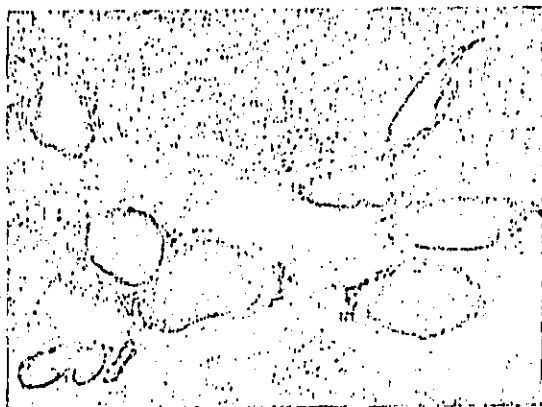


Fig. 4.--Multiple damage to the intramuscular branches of the coronary arteries. Large constricted atherosclerotic plaques (x36, rabbit no. 505).



Fig. 5.--Clear degenerative changes in the myocardium, rabbit No. 499.

Above (a)--x 130;
Below (b)--x 106.
Coloring oil red O + hematoxylin.



Discussion of the Results

As was seen, a prolonged acoustical effect exerted an influence on the changes of the animal level of nonesterified fatty acids and the indicators of the blood coagulating system. The level of nonesterified fatty acids in the experimental groups of animals changed unequally. An increase in the level of nonesterified fatty acids under the influence of the sound effect was observed with rabbits kept on ordinary food during the time of the study (1st series, group B), and in the case of the rabbits kept on ordinary feed for a period of 1.5 months (month up to the start of the experiment and during its time), but before this receiving a cholesterol diet for a period of 4.5 months (IIInd series, group B). Drop in the level of nonesterified fatty acids was observed in groups of rabbits kept on a cholesterol diet for a period of 4 months up to the start of the experiment and during the sound effect (IIIrd series, groups B and C). The changes of the hemocoagulation indicators were characterized by a general hypercoagulation tendency, more expressed in the case of rabbits on a cholesterol diet during the time of the experiment (III series, groups B and C). The level of cholesterol and of the total fraction of β -lipoproteids did not substantially change under the influence of the sound effect.

Despite the different character of the humoral shifts or their absence under the influence of sound effects in series I, II, III, the morphological changes in the wall of the heart and myocardium artery turned out to be analogous--hypertrophy of the vessel wall, interruption of blood circulation in the myocardium (plethoric blood vessels), focal points of necrosis. Evidently, the latter is connected with an increase of the sympato-adrenal activity which facilitated changes of the metabolic processes in the vessel wall and in the heart muscle. The different character of the necrosis (from fresh to scar changes) indicates that they do not emerge simultaneously, but are connected with an extended, repeated sound effect.

The data obtained on animals kept on a diet with cholesterol and subjected to sound effect (series II and III) is especially interesting. The combination of a high level of lipids in the blood and hypercoagulation shifts in the hemostasis system, on the one hand, and the presence of lipid resorption on the platelets in the aorta and coronary arteries, on the other, attests to the complexity of the organism reaction, created under conditions of extended sound effect.

The results of the obtained data in a way contradict the prevailing data concerning the increased influence of the different types of stress on the degree of atherosclerotic changes in the experiment. This is especially confirmed by the degree of expression of the atherosclerotic changes observed in a group of rabbits subjected to more extensive sound irritation (series III, group B). The reason for the fact observed by us has not yet been explained. Evidently, we may be dealing with a specific influence of the stimulation (sound) used by us. It is possible that this type of stimulation, when it is especially strong, caused special changes on the level of the hypothalamus in connection with the physical nature of the sound.

In the literature there is indication of the possibility of resorption of lipids from the platelets, created under certain conditions. One of the factors facilitating resorption of the lipids is an increase in the activity of the lipo-mobilizing factor of hypophysis whose released stimulation takes place under conditions of stress. According to the data of B.M. Lipovatakov [6,7], with the introduction of the lipo-mobilizing factor of hypophysis into the rabbits with experimental atherosclerosis, resorption of the lipids takes place in the aorta and there is an increase of lipolytic activity of enzymes in the vessel wall. The indirect support of the high lipolytic activity of the vessel walls in our observations obviously may be considered to be a large number of macrophages observed in the platelets. According to the data of Zemleni et al. [4], an increase of lipolysis in the vessel wall is connected with a great accumulation of macrophages in them, having a high enzyme activity. Possibly, a decrease in the level of non-esterified fatty acids is connected with an increase of the lipolytic activity [4, 13]. In our studies, the resorption of lipids from the platelets was accompanied by a decomposition of fat on the periphery.

So, in conditions of prolonged sound effect on animals with an expressed atherosclerotic process, despite the high level of lipids in the blood and the increased capability of the blood to coagulate, conditions are created facilitating the resorption of lipids from the platelets, against a background of the continued introduction of cholesterol. Additional studies are necessary to explain the mechanism of the obtained results.

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ВЛИЯНИЕ ЗВУКОВОГО РАЗДРАЖЕНИЯ НА ЛИПИДНЫЙ ОБМЕН,
ПОКАЗАТЕЛИ СИСТЕМЫ СВЕРТЫВАНИЯ КРОВИ И РАЗВИТИЕ
ЭКСПЕРИМЕНТАЛЬНОГО АТЕРОСКЛЕРОЗА У КРОЛИКОВ

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Звуковое воздействие на протяжении 14 дней у здоровых кроликов способствует повышению уровня холестерифицированных жирных кислот, усилению свертываемости крови. Морфологически выявлена гипертрофия венечных артерий сердца, очаги некроза в миокарде. У кроликов с экспериментальным атеросклерозом, подвергавшихся звуковому воздействию в течение 14 и 28 дней, несмотря на высокий уровень липидов в крови и гиперкоагуляционные сдвиги, создаются условия, способствующие резорбции липидов из бляшек аорты и коронарных артерий. Морфологически, кроме того, обнаружена гипертрофия и отек сосудистой стенки, очаги некроза, очаговые и диффузные жировые инфильтрации сердечной мышцы.

Многочисленными экспериментальными исследованиями установлено, что перенапряжение нервной системы различного рода усиливает сосудистую проницаемость, способствует повышению липидных фракций крови и отложению липидов в стенку артерий, вызывает гиперкоагуляционные сдвиги в системе свертывания крови [2, 8, 10, 16].

П. С. Хомуто удалось путем частой смены стереотипа у кроликов и собак вызвать атеросклеротические изменения в аорте и крупных артериях сердца [18].

Однако механизмы влияния перенапряжения нервной системы на развитие атеросклеротического процесса окончательно не выяснены. Не ясно также, имеет ли значение в развитии атеросклеротического процесса характер стрессорного воздействия.

Целью настоящей работы явилось изучение вопроса о влиянии длительного перенапряжения нервной системы, вызванного звуковым раздражением, на развитие экспериментального атеросклероза у кроликов.

МЕТОДИКА

Работа выполнена на 53 кроликах-самцах породы шиншилла весом 2,7—3,2 кг. Из 53 кроликов 28 получали ежедневно в течение 4,5—5 мес. 500 мг холестерина и 5,0 подсолнечного масла *per os*, 15 кроликов содержались на обычной корме. Четыре кролика (31 из 53) подвергались звуковому воздействию (04—06 дБ, 3 кГц) в течение 4,5 часов в сутки (с двумя 30-минутными перерывами для отдыха через каждые 1,5 часа воздействия). Продолжительность звукового воздействия была 14 и 28 дней. Выполнено 3 серии исследований (табл. 1). В 1 серии вошли 15 кроликов, содержавшихся на обычной корме, 7 из них подвергались звуковому воздействию в течение 14 дней (группа В), 8 кроликов служили контролем (группа А). Во 2 серии вошли 20 кроликов, которые дополнительно к общему рациону получали холестерин в указанной дозе в течение 4,5 месяцев. Затем они были переведены на обычную корму (подкормку). Через один месяц после отмены холестерина 12 кроликов из 20 были подвергнуты звуковому воздействию в течение 14 дней (группа В), 8 кроликов

Таблица 1
Распределение кроликов по группам в зависимости от дозы
и звукового воздействия

Серия исследования	Холестериновая область дозы	Группа кроликов	Число кроликов	Длительность инкубации холестерина	Начало звукового воздействия	Длительность звукового воздействия
I		A	7	—	—	—
		B	8	—	—	14 дней
II		A	8	4,5 месяцев	—	—
		B	12	То же	Через 1 месяц после отмены холестерина	14 дней
III		A	6	•	•	—
		B	6	•	После 4 месяцев	14 дней
		B	6	5 месяцев	То же	28 дней
Всего			53			

(группа А) не подвергались звуковому воздействию. В III серии пошло 18 кроликов, получавших холестерин в течение 4,5—5 месяцев. 12 из них через 4 месяца от начала кормления холестерином были подвергнуты звуковому воздействию на фоне продолжительной дачи холестерина (В в течение 14 дней — группа В, В в течение 28 дней — группа В), а 6 (группа А) служили контролем (не раздражались).

Кролики исследовались дважды: непосредственно перед началом звукового воздействия и после его прекращения соответственно сроку раздражения. Затем кролики забивались. Кролики, не подвергавшиеся звуковому воздействию (группы А), обелявались в те же сроки.

Определялись показатели липидного обмена и системы свертывания крови: холестерин [22], суммарная фракция β -липопротеидов [2], неэстерифицированные жирные кислоты [23], время свертывания крови [24], время рекальцификации [21], толерантность плазмы к гонарину [25], протромбиновый комплекс [24], фибриноген [18], XIII фактор [2], тромбиновое и гепариновое время [22], агрегация тромбоцитов [18], фибринолитическая активность крови [2]. Стабилизатор крови 1,34% оксалата натрия, соотношение его в крови 1:4. Получаемые цифровые данные обрабатывали статистически разностными методами с применением t критерия Стьюдента.

Материал фиксировали в 10%-м нейтральном формалине, после фиксации аорты окрашивали in toto судан III. После составления схем распределения изменений на различных отделах аорты (дуги, грудного и брюшного сегмента) вырезались кусочки от 5 до 10 (в зависимости от выраженности изменений). Срезы приготавливали на замораживающем микротоме и окрашивали oil red O; оставшуюся часть кусочков заливали в парафин—целлоидин, срезы окрашивали по ван-Гизону и по Вейгерту—Харту на эластические волокна. Сердца кроликов опытной и контрольной группы разрезались на поперечные сегменты (4—5), которые исследовались так же, как и аорта.

РЕЗУЛЬТАТЫ ИССЛЕДОВАНИЯ

У кроликов, подвергавшихся звуковому воздействию, к концу опыта изменилось поведение. Они становились агрессивными, набрасывались друг на друга, часто дрались. Одновременно появилась пугливость: при виде экспериментатора они забивались в угол камеры. У большей части кроликов, подвергавшихся звуковому воздействию в течение 28 дней, снизился вес на 600—1000 г, у кроликов, подвергавшихся звуковому воздействию в течение 14 дней, снижение веса было меньшим.

В I серии опытов у кроликов, находящихся на обычном корме (группа В), звуковое раздражение не вызвало увеличения уровня холестерина и суммарной фракции β -липопротеидов; отмечалось достоверное увеличение уровня неэстерифицированных жирных кислот (НЭЖК) с $612,8 \pm 68,3$ до $827,1 \pm 71,0$ мг/л ($p < 0,05$). У кроликов, находящихся на том же рационе и не подвергавшихся звуковому воздействию (группа А), изменений уровня НЭЖК при исследовании в те же сроки не наблюдалось. Под влиянием звукового воздействия увеличилась свертываемость крови (укоротилось время рекальцификации со $135,5 \pm 10,1$ до $84,7 \pm 15,8$ сек.,

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$p < 0.05$); повысилась толерантность плазмы к гепарину с 254.7 ± 32.5 до 135.8 ± 24.5 сек. ($p < 0.05$). Остальные показатели гемоконгуляции не отличались существенно от значений контрольной группы (табл. 2, 3).

При вскрытии кроликов различных серий опытов, убитых в разные сроки, видимых изменений при общем осмотре обнаружено не было. При макроскопическом исследовании аорт кроликов I серии, как подвергавшихся, так и не подвергавшихся звуковому воздействию, видимых изменений не выявлено. При микроскопическом исследовании венечных артерий сердца и миокарда у кроликов группы В обнаружены изменения в виде гипертрофии стенки внутримышечных ветвей и резко выраженной венозной гиперемии. Вблизи от измененных артерий наблюдались небольшие очаги некроза. У кроликов, не подвергавшихся звуковому воздействию (группа А), подобных изменений со стороны сосудистой стенки и миокарда обнаружено не было.

Во II серии опытов у кроликов, содержащихся на обычном корме с добавлением холестерина в течение 4,5 месяцев и «отдыхом» после его отмены в течение последующего месяца, уровень холестерина и суммарной фракции β -липопротеидов перед началом опытов в обеих группах был выше, чем в обеих группах I серии исследования; существенного же различия в уровне ЛЭЖК не отмечалось. При повторном исследовании через 14 дней уровень холестерина и суммарной фракции β -липопротеидов в обеих группах снизился, что связано с отменой холестерина. Уровень ЛЭЖК в группе В повысился с 607.5 ± 70.9 до 797.5 ± 69.1 мкг/л ($p < 0.05$). По-

Таблица 2

Исходные группы	Изменение показателей ($M \pm m$) липидного обмена у кроликов без воздействия (I) и под влиянием звукового раздражителя (II)				Исследования в контрольной группе		
	Холестерин (в мг%)		Суммарная фракция β -липопротеидов (в мг%)		Исследования в контрольной группе		
	I	II	I	II	I	II	
I	Без воздействия 14 дней	75.3 \pm 14.5	72.5 \pm 14.4	169.2 \pm 41.4	205.6 \pm 49.3	625.0 \pm 73.1	645.3 \pm 47.6
		76.7 \pm 13.7	77.7 \pm 20.7	216.7 \pm 40.7	188.6 \pm 44.5	612.8 \pm 68.3	827.1 \pm 71.9
II	Без воздействия 14 дней	264.0 \pm 30.1	224.3 \pm 35.6	1050.6 \pm 252.0	734.3 \pm 187.0	677.5 \pm 111.0	750.0 \pm 66.3
		287.0 \pm 42.6	241.2 \pm 50.5	1030.0 \pm 191.0	897.9 \pm 182.2	607.5 \pm 70.9	797.9 \pm 69.1
III	Без воздействия 14 дней	503.8 \pm 87.3	520.0 \pm 63.1	2352.0 \pm 386.2	2297.1 \pm 436.0	1054.3 \pm 91.7	961.6 \pm 77.9
		520.6 \pm 89.5	521.8 \pm 93.8	2192.0 \pm 473.0	2380.1 \pm 257.4	1308.0 \pm 149.4	793.3 \pm 56.6
	28 дней	518.5 \pm 79.3	538.3 \pm 54.8	2341.0 \pm 381.4	2249.1 \pm 409.9	1325.4 \pm 125.0	966.6 \pm 63.6

Таблица 3
Наименование показателей свертывания крови у кроликов под влиянием звукового раздражения (M ± m)

Группы	Возраст	Длительность звукового воздействия	Показатель									
			время свертывания крови (в сек.)		время реиндификации плазмы (в сек.)		толщина пленки в гомарину (в сек.)		протромбиновый индекс (в %)		фибриноген (в мг%)	
			I	II	I	II	I	II	I	II	I	II
I	A	Без воздействия 14 дней	152,5 ± 24,4	168,1 ± 25,1	120,0 ± 10,0	108,0 ± 12,3	240,0 ± 13,1	260,3 ± 14,0	83,5 ± 1,8	80,7 ± 3,5	207,5 ± 20,0	243,7 ± 10,2
			160,2 ± 14,8	136,5 ± 24,4	135,5 ± 10,1	84,7 ± 15,8	254,7 ± 32,5	135,8 ± 24,5	87,8 ± 3,1	82,2 ± 2,2	247,4 ± 10,4	258,5 ± 15,8
II	A	Без воздействия 15 дней	183,1 ± 30,3	186,2 ± 28,2	108,6 ± 20,6	85,7 ± 8,0	258,3 ± 37,5	109,5 ± 25,7	85,1 ± 3,3	83,0 ± 3,3	237,5 ± 8,9	241,2 ± 21,5
			220,0 ± 20,8	175,0 ± 10,8	139,5 ± 20,4	128,1 ± 8,5	264,8 ± 26,1	176,5 ± 28,0	88,2 ± 3,8	84,2 ± 2,0	245,0 ± 0,5	228,7 ± 12,0
III	A	Без воздействия 15 дней	168,3 ± 20,3	151,6 ± 18,5	105,0 ± 14,1	105,8 ± 14,2	178,3 ± 35,4	175,0 ± 23,8	91,0 ± 2,6	91,3 ± 2,4	260,8 ± 23,2	241,6 ± 8,3
			100,6 ± 10,4	152,5 ± 22,0	107,6 ± 12,1	81,8 ± 9,7	141,8 ± 31,0	107,6 ± 0,6	85,8 ± 2,5	80,1 ± 2,5	223,3 ± 22,0	241,6 ± 10,1
	B	28 дней	161,3 ± 10,2	80,0 ± 6,4	138,0 ± 18,5	70,3 ± 5,4	168,2 ± 18,5	101,6 ± 6,1	84,9 ± 2,4	87,3 ± 1,1	220,0 ± 18,8	241,6 ± 10,0

Таблица 3 (продолжение)

Группы	Возраст	Длительность звукового воздействия	Показатель									
			XIII фактор (в %)		количество тромбоцитов (в %)		тромбоциты крови (в сек.)		гепариновая проба (в сек.)		фибринолитическая активность крови	
			I	II	I	II	I	II	I	II	I	II
I	A	Без воздействия 14 дней	116,5 ± 6,9	86,3 ± 0,1	25,8 ± 2,0	23,1 ± 2,1	24,6 ± 1,4	22,8 ± 1,4	10,0 ± 0,65	0,3 ± 1,0	1,55 ± 0,16	1,26 ± 0,78
			114,7 ± 13,5	91,5 ± 10,0	26,0 ± 2,0	24,4 ± 2,4	23,2 ± 1,4	23,5 ± 1,8	10,0 ± 1,20	0,4 ± 1,0	2,83 ± 0,51	3,71 ± 1,47
II	A	Без воздействия 14 дней	116,6 ± 16,5	97,2 ± 11,0	26,5 ± 5,2	30,0 ± 3,0	19,8 ± 1,5	21,6 ± 2,1	8,5 ± 0,8	9,7 ± 1,8	3,17 ± 1,2	5,85 ± 1,3
			97,4 ± 8,2	100 ± 8,3	28,0 ± 2,7	27,8 ± 1,5	20,1 ± 1,5	19,7 ± 1,4	8,8 ± 0,8	7,3 ± 1,0	3,07 ± 0,5	4,24 ± 0,08
III	A	Без воздействия 14 дней	96,3 ± 7,3	103,1 ± 9,5	20,0 ± 5,8	22,7 ± 2,0	23,1 ± 1,3	24,3 ± 0,6	10,0 ± 0,7	9,3 ± 1,1	4,3 ± 0,9	3,0 ± 0,7
			95,8 ± 15,0	90,0 ± 12,5	20,7 ± 4,1	31,0 ± 2,4	27,6 ± 2,6	24,3 ± 1,6	12,8 ± 1,0	9,5 ± 1,2	5,8 ± 1,1	1,83 ± 0,19
	B	28 дней	95,4 ± 15,2	148,5 ± 17,2	23,2 ± 3,0	30,4 ± 2,7	25,3 ± 1,8	24,1 ± 1,0	13,1 ± 1,1	8,5 ± 1,0	5,9 ± 1,03	1,75 ± 0,24

Важнейшим фактором свертывания крови является XIII фактор (табл. 3, 3) — 26,1 до 176,5. При воздействии звука выработка фибриногена не снижается, а наоборот она увеличивается. Менее резко у кроликов 3 и 2 групп снижается фибриноген.

Рис.



отчетливо выразилось в распределении фибриногена, наглядно видно, что при воздействии звука фибриноген выделен в виде отдельных фракций. Так же как и в вальдолатовом тесте о начале их образования на I серии. Образовались фибринолитические агенты в результате образования артериальных тромбов и ее протективности сердца (рис. 4). от гипертензии, инсульта старшего (рис. 5). Намечены диффузные и возможные сердца. Н

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казатели гемокоагуляции у кроликов после отмены холестерина практически не отличались от значений их в I серии. Под влиянием раздражителя (табл. 2, 3) повысилась толерантность плазмы к гепарину с 264.8 ± 26.1 до 176.5 ± 28.0 сек. ($p < 0.05$).

При вскрытии кроликов II серии подкожный жировой слой в группе Б был выражен несколько меньше, чем в группе А. При микроскопическом исследовании аорт II серии кроликов были обнаружены атеросклеротические изменения, выраженные в различной степени, и в обеих группах она оказалась одинаковой. В группе Б резкие изменения были у 3 из 12, менее резкие у 5, слабые у 4 кроликов; в группе А соответственно у 3 из 8, 3 и 2 кроликов. При микроскопическом исследовании в атеросклеротических бляшках у части кроликов группы Б наблюдались довольно

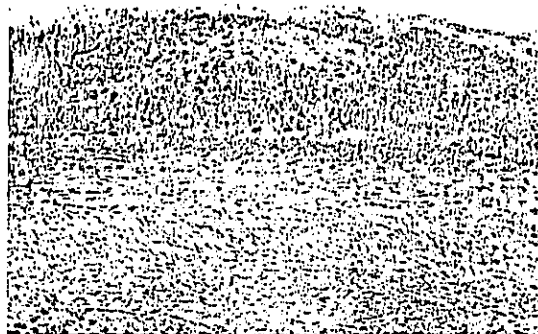


Рис. 1. Неравномерное распределение липидов в аорте.
×100. Кролик № 473.

отчетливо выраженные признаки резорбции липидов в виде неравномерного распределения последних в самой бляшке, местами почти полное их отсутствие, наличие большого количества клеток с липидными включениями в цитоплазме (липидные макрофаги) и проникания липидов в среднюю оболочку аорты (рис. 1). В венечных артериях сердца также обнаружены атеросклеротические изменения, крупные атеросклеротические бляшки во внутримышечных и главных ветвях венечных артерий сердца. Так же как и в аорте, в некоторых бляшках коронарных артерий сердца наблюдалось неравномерное распределение липидов, свидетельствующее о начале их резорбции (рис. 2). В сосудистой стенке и миокарде были обнаружены изменения, но более выраженные, как и у кроликов группы Б I серии. Обращала на себя внимание значительная гипертрофия стенок многих венечных артерий сердца (рис. 3). Порядку в утолщенной стенке определялись значительные отложения липидов, не приводящие, однако, к образованию бляшек. В отличие от внутримышечных ветвей венечных артерий сердца и главных ветвей стенки артерий, напротив, была истончена и ее просвет был расширен. Обращает на себя внимание резкая выраженность атеросклеротических поражений в коронарных артериях сердца (рис. 4). В различных отделах миокарда, преимущественно вблизи от гипертрофированных артерий, наблюдалась гипоксия гиперемия, иногда «старые» кровоизлияния и очень резкие дистрофические изменения (рис. 5). Изменения в сердечной мышце представляли собой очаговую или диффузную жирную инфильтрацию, хотя нельзя исключить также и возможности ее происхождения за счет атеросклеротических поражений сердца. Наблюдались также некротические очаги некроза и небольшие сег-

чатые рубцы. У кроликов группы А на фоне выраженных атеросклеротических изменений признаки резорбции липидов в аорте и коронарных артериях отсутствовали.

У кроликов III серии под влиянием длительного содержания на холестериновой диете (табл. 2) уровень холестерина, суммарной фракции β -липопротеидов, НЭЖК был значительно выше, чем у кроликов I и

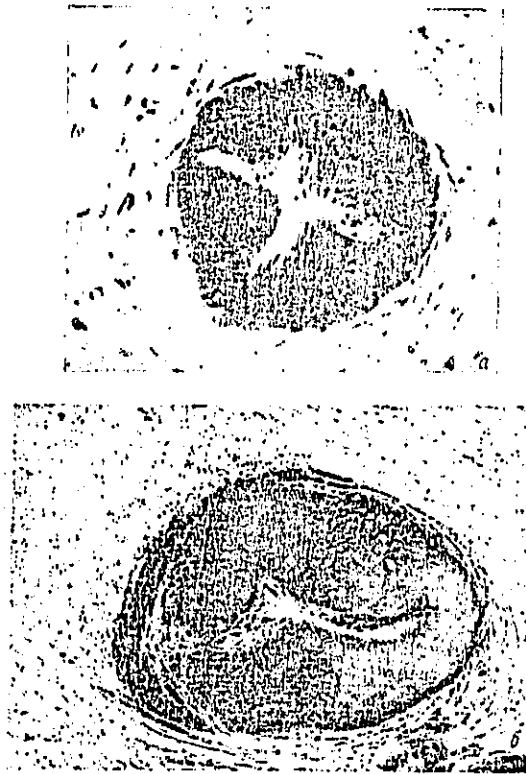


Рис. 2. Крупная атеросклеротическая бляшка во внутримышечной ветви левой венечной артерии сердца кролика. Вверху (а) — контроль — липиды равномерно расположены в бляшке. $\times 300$, кролик № 430; внизу (б) опытный — неравномерное распределение липидов. $\times 110$, кролик № 474.

II серий. Через 2 (группы А и Б) и 4 недели (группа В) уровень холестерина, суммарной фракции β -липопротеидов при повторном исследовании практически не изменился, уровень же НЭЖК в группах Б и В снизился соответственно с $1308,0 \pm 149,4$ до $799,3 \pm 56,6$ мг/л ($p < 0,02$) и с $1325,1 \pm 125,1$ до $908,6 \pm 63,6$ мг/л ($p < 0,01$).

Под влиянием длительного кормления холестерином у кроликов III серии гепариновая и фибринолитическая активность крови оказалась выше, чем в I серии. Наши данные в этом отношении близки к наблюдениям других авторов [14]. Примененное звуковое раздражение способствовало усилению гиперкоагуляционных свойств крови в группах Б и В:

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Рис.
 Вверху

($p < 0,05$) и с I
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укорочению времени рекальцификации соответственно со 107.6 ± 12.1 до 61.8 ± 9.8 сек. ($p < 0.05$) и со 128.0 ± 18.5 до 70.3 ± 5.4 сек. ($p < 0.05$), повышению адгезивности тромбоцитов соответственно с 20.7 ± 4.1 до $31.0 \pm 2.4\%$ ($p < 0.05$) и с 23.2 ± 3.0 до $36.4 \pm 2.7\%$ ($p < 0.01$), снижению гепариновой активности крови соответственно с 12.8 ± 1.0 до 9.5 ± 1.2 сек.



Рис. 3. Гипертрофия стенки левой почечной артерии, отек.
 Вверху (а) — контроль, $\times 140$, препарат № 495; внизу (б) — опыт, $\times 100$, препарат № 475.

($p < 0.05$) и с 13.1 ± 1.1 до 8.5 ± 1.0 сек. ($p < 0.05$), снижению фибринолитической активности крови соответственно с 5.8 ± 1.1 до 1.83 ± 0.19 ($p < 0.01$) и с 5.9 ± 1.88 до 1.75 ± 0.24 ($p < 0.05$). В группе В, кроме этих изменений, к концу звукового воздействия отмечалось укорочение времени свертывания крови со 161.3 ± 19.2 до 90.0 ± 6.4 сек. ($p < 0.01$), повышение толерантности плазмы к гепарину со 108.2 ± 18.2 до 104.6 ± 6.1 сек. ($p < 0.05$), повышение уровня XIII фактора с 95.4 ± 15.2 до $148.5 \pm 17.2\%$ ($p < 0.05$). Необходимо отметить, что гепариновая и фибринолитическая активность крови у кроликов III серии (группы Б и В) к концу опыта не превышала значений ее у кроликов I серии.

Морфологические данные как при макроскопическом, так и при микроскопическом исследовании оказались сходными со II серией (в группах В и В с группой Б и между группами А). Однако степень выраженности атеросклеротического процесса оказалась разной в III серии. Так,



Рис. 4. Множественное поражение внутримышечных ветвей почечных артерий. Крупные стенозирующие атеросклеротические бляшки (x30, кролик № 503).

у всех 6 кроликов группы В, обследованных морфологически, атеросклеротические изменения были слабыми. У кроликов группы Б выраженные атеросклеротические изменения были у 2 из 6, менее выраженные у 2 и слабые у 2 кроликов. В группе А выраженные атеросклеротические изменения выявлены у 2, менее выраженные у 1 и слабые у 3 кроликов. Таким образом, у кроликов, подвергавшихся звуковому воздействию в течение 28 дней, степень атеросклеротических изменений в сосудах была менее выражена, чем у кроликов с более коротким раздражением.

ОБСУЖДЕНИЕ РЕЗУЛЬТАТОВ

Длительное звуковое воздействие, как было видно, оказывало влияние на изменение у животных уровня НЭЖК и показателей системы свертывания крови. Уровень НЭЖК в опытных группах животных изменялся неодинаково. Повышение уровня НЭЖК под влиянием звукового воздействия наблюдалось у кроликов, содержавшихся во время исследования на обычном корме (I серия, группа В), и у кроликов, содержавшихся на обычном корме в течение 1.5 месяцев (месяц до начала опыта и во время него), но предварительно до этого получавших холестеринную диету в течение 4.5 месяцев (II серия, группа В). Снижение уровня НЭЖК наблюдалось в группах кроликов, содержавшихся на холестеринной диете в течение 4 месяцев до начала опыта и в течение звукового воздействия (III серия, группы В и В). Изменения же показателей гемостаза характеризовались общей гиперкоагуляционной тенденцией, более выраженной у кроликов, находившихся во время исследования на холестеринной диете (III серия, группы В и В). Уровень холестерина и суммарной фракции β -липопротеидов существенно не изменился под влиянием звукового воздействия.

Несмотря на разный характер гуморальных сдвигов или их отсутствия под влиянием звукового воздействия в I, II, III сериях, морфоло-

гические изменения сказались на состоянии слизистой оболочки желудка. Последнее, очевидно, говорит о слон...

Рис. 5. Вверху

жих до рубцово-променно, а сивством. Особенно на диете с хол III серии). Сомнионных сдвиговорбции линииговорит о слон... толького

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EFFECT OF THE SOUND STIMULATION ON LIPID METABOLISM,
PARAMETERS OF THE BLOOD COAGULATING SYSTEM
AND DEVELOPMENT OF EXPERIMENTAL ATHEROSCLEROSIS
IN RABBITS

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The 14-day sound stimulation of healthy rabbits increases the level of unesterified fatty acids and the blood coagulability. Morphologically the hypertrophy of the heart coronary arteries and necrotic foci in the myocardium are revealed. In rabbits with experimental atherosclerosis subjected to sound stimulation during 14 and 28 days, in spite of a high level of lipids in the blood and hypercoagulatory shifts, the developing conditions aid to resorption of lipids from aortal plaques and coronary arteries. Apart from that, morphological studies reveal hypertrophy and oedema of the vascular wall, necrotic foci, local and diffuse fatty infiltration of the myocardium.

гические изменения в сосудистой стенке артерий сердца и миокарде оказались сходными — гипертрофия сосудистой стенки, нарушение кровообращения в миокарде (полнокровие кровеносных сосудов), очаги некроза. Последнее, очевидно, связано с повышенным симпато-адреналовой активностью, что способствовало изменению метаболических процессов в сосудистой стенке и в сердечной мышце. Разный характер некрозов (от све-



Рис. 5. Ранние догенеративные изменения в миокарде, кролик № 499.
Вверху (а) — $\times 103$; внизу (б) — $\times 100$. Окраска oil red O + гематоксилин.

жих до рубцовых изменений) говорит о том, что они возникают по одновременно, а связаны с длительным, повторяющимся звуковым воздействием.

Особенно интересные данные получены у животных, содержавшихся на диете с холестерином и подвергавшихся звуковому воздействию (II и III серия). Сочетание высокого уровня липидов в крови и гиперкоагуляционных единиц в системе гемостаза, с одной стороны, и наличие резорбции липидов из бляшек в аорте и коронарных артерий — с другой, говорит о сложности реакции организма, создающейся в условиях длительного звукового воздействия.

Результаты полученных наблюдений как будто противоречат имеющимся данным об усиливающем влиянии различного рода стресса на степень атеросклеротических изменений в эксперименте. Особенно это подтверждается меньшей степенью выраженности атеросклеротических изменений, обнаруженных в группе кроликов, подвергавшихся более длительному звуковому раздражению (III серия, группа В). Причина наблюдаемого нами факта пока неясна. Видимо, речь может идти о специфическом влиянии примененного нами раздражителя (звук). Возможно, что этот тип раздражения оказался особенно сильным и вызывал специальные изменения на уровне гипоталамуса в связи с физическими свойствами звука.

В литературе существуют указания на возможность резорбции липидов из бляшек, создающихся в определенных условиях. Одним из факторов, способствующих резорбции липидов, является повышенная активность липомобилизующего фактора гипофиза, стимуляция выделения которого происходит в условиях стресса. По данным Б. М. Липовецкого [6, 7], при введении липомобилизующего фактора гипофиза кроликам с экспериментальным атеросклерозом происходит резорбция липидов в аорте и повышению липолитической активности ферментов в сосудистой стенке. Косвенным подтверждением высокой липолитической активности сосудистой стенки в наших наблюдениях, очевидно, можно считать большое количество макрофагов, обнаруженное в бляшках. По данным Земплени с соавт. [4], повышение липолиза в сосудистой стенке связано с большим накоплением в ней макрофагов, обладающих высокой ферментативной активностью. Возможно, с повышенным липолитической активностью связано и снижение уровня НЭЖК [8, 12]. В наших исследованиях резорбция липидов из бляшек сопровождалась и распадом жира на периферии.

Таким образом, в условиях длительного звукового воздействия у животных с выраженным атеросклеротическим процессом, несмотря на высокий уровень липидов в крови и повышенную способность крови к свертыванию, создаются условия, способствующие резорбции липидов из бляшек, на фоне продолжающегося введения холестерина. Необходимы дальнейшие исследования для выяснения механизма полученных результатов.

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SECTION 4

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> L. Verdun di Cantogno R. Dallerba P. S. Teagno L. Cocola	<u>Institution and address where research was performed</u> Dept. of Audiology and 1st Dept. of Medical Pathology, University of Turin, Turin, Italy
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> University of Turin
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Citation | di Cantogno, L.V. et al. Urban traffic noise, cardiocirculatory activity and coronary risk factors. Acta Otolaryngologica Suppl. 339: 55-63, 1976.

<u># of Ref.'s</u> none	<u># of Fig.'s</u> 12	<u>Language</u> English
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<u>Type & duration of experiment</u> Type: Controlled laboratory experiment in an Amplifon Standard G5 silent booth Duration: 105 min. per subject	<u>Purpose for study</u> to study the effects of 10 min. of traffic noise on the cardiovascular system, blood chemistry and urine levels of catecholamines
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Description of test groups (subjects, #, age, etc.)
33 male subjects aged 20 - 70 years divided into 3 test groups (which were divided into subgroups of 20 - 45 yr. olds and 46 - 70 yr. olds):
1) 11 normals (average age 36.7 yrs.) 3) 11 with heart disease (average age 52 yr.)
2) 11 diabetic or with abnormal fat levels (average age 46.4 yrs.) 1 control group - 11 normal male subjects (average age 27 yrs.)

<u>Control of other stressors</u> laboratory conditions used; blood samples taken using butterfly needle to avoid stress from procedure	<u>Statistical Methods</u> students t-test
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<u>Noise Stimulus</u> source: taped traffic noise from 10 areas of Turin spectral characteristics: not given noise level: mean Leq.=7.16dB; 88.8dB length of exposure: 10 min. # of trials: 1 per subject 15 min. quiet - 10 min. traffic noise - 80 min. quiet	<u>CVS Response Measured</u> 1) blood pressure 3) heart rate 2) electrocardiogram <u>Nonauditory effects</u> CVS: blood pressure and heart rate increased due to traffic noise; electrocardiograms - no significant changes due to noise Other: Noise stimulated increases in urinary catecholamines, blood sugar, insulin, cholesterol, uric acid.
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Author's conclusions | Traffic noise caused increased blood pressure and heart rate, and altered the blood chemistry such that this type of noise stress may be a coronary risk factor and may be involved in the development of arteriosclerosis. Blood chemistry data was too small to make definite conclusions, however.

Evaluation & comments | 1) no age-matched control group-mean age of controls is 10 years less than the youngest test group. 2) 10 min. noise stimulus - too short from which to draw any real conclusions.

di Cantogno, L.V. et. al. Urban traffic noise,
cardiocirculatory activity and coronary risk factors.
Acta Otolaryngologica Suppl. 339: 55-63, 1976.

The effects of traffic noise on blood pressure, heart rate, electrocardiogram, blood chemistry, and urinary catecholamines were studied in 33 male subjects, aged 20 to 70 years. The subjects were divided into 3 groups as follows: 11 healthy subjects (average age 36.7 years); 11 with heart disease (average age 52 years); 11 having diabetes or abnormal lipid metabolism (average age 46.4 years). A control group of 11 healthy males (average age 27 years) was subjected to all of the same procedures as the 33 test subjects, except traffic noise. Both tests and controls had normal hearing for their ages. After a 6 hour fast, the subjects were placed on a bed in an Amplifon standard G5 silent booth. Following 15 minutes of quiet, a 10-minute tape recording of traffic noise from 10 areas of Turin, Italy was introduced into the subjects' headphones. The mean noise level was 71.6dBA, and the Leq for 10 min was 73.1dBA. The subjects remained in the test chamber under quiet conditions for 80 minutes after the tape ended. Blood samples were taken 20 minutes before and immediately before the noise stimulus--the average of the 2 sets of results was used as the baseline value for each blood chemistry test. Blood samples were also taken when the tape stopped, 20 minutes later, and 50 minutes later. The blood pressures and electrocardiograms were measured before, during, and after the noise stimulus. Urinary catecholamines were measured on samples taken before and after the traffic noise. The students t-test was used to analyze the data obtained both between groups and to compare the results within groups due to age. Traffic noise stimulated catecholamine excretion in the test group with diabetes or abnormal lipid metabolism. The blood samples were analyzed for sugar, uric acid, total lipids, cholesterol, triglycerides, and insulin. Changes in blood chemistry due to noise were observed in all 3 test groups. The greatest changes occurred in the group with abnormal metabolisms. Noise induced increased blood sugar and uric acid levels, indicating that noise affects nucleic acid metabolism. Total lipid levels were the same as that of the controls in the healthy test group; total lipids increased in the other 2 test groups. Triglyceride levels increased due to noise, especially in subjects over 45 years. Cholesterol levels increased due to noise, especially in the heart patients. Insulin levels increased due to noise in the healthy subjects and in those with abnormal metabolism, whereas insulin levels decreased in the heart patients. No significant changes were found in the electrocardiograms due to noise. The heart rates and systolic blood pressures increased due to noise in all subjects. The results indicate that traffic noise may be a risk factor in the development of arterial and coronary disease.

URBAN TRAFFIC NOISE, CARDIOCIRCULATORY ACTIVITY AND CORONARY RISK FACTORS

L. Verdun di Cantogno, R. Dallerba, P.S. Teagno, L. Cocola

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SCOPE OF THE RESEARCH

The aim of this research was an assessment of the effect of road noise lasting 10' on cardiocirculatory activity and various blood chemistry indices, particularly those apparently associated with the pathogenesis of arteriosclerosis. Normal, dysmetabolic and coronaropathic subjects were examined.

While it can readily be appreciated that certain stimuli can act on the cardiovascular apparatus, leading to changes in the work of the heart and its performances, it is less easy to understand how certain psychological and emotional conditions, or certain sensorial stimuli completely mediated by the CNS, influence the heart and its vessels, thus supporting atheroma, endoarterial thrombosis or serious arrhythmias.

Acoustic stimuli offer an ideal method for the study of effects mediated by the CNS, since they can be exactly reproduced and measured in the absence of every other factor likely to influence cardiac frequency and arterial pressure, or left ventricular performance as assessed by simultaneous ECG, phonocardiographic and carotid recordings. Attention was also given to blood chemistry indices known as related coronary risk factors. Direct evaluation of cholinergic levels was not attempted, because this technique gives widely scattered results, even in normal conditions. Urinary catecholamines excreted during the test were however taken into consideration. The limitations of this method are well known.

MATERIAL AND METHODS

The absence of literature data concerning the effect of traffic noise on the parameters chosen for study suggested the advisability of preceding the experiment by a series of investigations of the effect of white noise and speech noise (100 dB and 80 dB intensity; continuous stimulation for 10' or for 1' followed by an interrupted stimulation for 9') in 16 subjects, to determine the effect of noise of different spectrum and intensity administered with different modalities. This preliminary study (details in the press) showed statistically significant differences between stimulated and control subjects. We therefore began by exposing 33 subjects aged 20-70 yr. to road noise. A further 11 normal subjects (mean age 27 yr) were studied for comparison, i.e. they were subjected to all the experimental procedures in the absence of road noise (control group).

Prior audiometric examination showed that all subjects had normal hearing for age. Only male subjects were examined, so as to ensure that responses were not affected by neurohormonal and neuropsychic factors pertinent to the two sexes.

The patients were divided into 3 groups (11 normals, average age 36.7 yr.; 11 diabetic or dyslipidemic, average age 46.4 yr.; 11 coronaropathic, average age 52 yr.). As already stated, the average age of the 11 controls was 27 yr. A division was also drawn between subjects aged more and less than 45 yr., i.e. 11 mean

57.3 yr.) and 16 (mean 32.7 yr.) respectively, excluding the controls.

Fifteen minutes after being settled on a bed in an Amplifon standard G5 silent booth erected in a room lined with soundproof panels, each subject was exposed to a tape consisting of 10 one-minute recordings of traffic noise registered at 10 different points in Turin. Its characteristics were:

abc

L_m dB	L_{eq} dB	σ dB
88.8	89.4	2.35
L_m dB (A)	L_{eq} dB (A)	σ dB (A)
71.6	73.1	3.54

L_{max}
Leq (V₀)

This noise was fed through a Uher 4400 Stereo Report IC recorder to the circuit of an Amplifon 500 Audiometer calibrated in dB SPL, and then to the subject's headphones. It was used at its true intensity, which was checked with a Brüel & Kjær phonometer fitted with a model 1613 octave filter.

Neither the normal nor the control subjects displayed basal changes in blood chemistry parameters, in blood pressure or in electrocardiographic and phonocardiographic patterns. Each patient had been fasting for at least 6 hr. Five blood samples were taken as follows: 20' and immediately before stimulation — the arithmetical mean of these values was used as the "basal value" — 10', when the tape stopped, and 30' and 90' after it started. To avoid problems of stress related to the blood samples a butterfly needle was used, kept patent with saline.

There was no significant difference between the two basal values, bearing in mind the scatter displayed by these data under physiological conditions: Sugar, insulin, uric acid, total lipids, cholesterol and triglycerides were measured in each sample, using the following methods:

— Blood Sugar: Enzymatic determination with G6-PD-hexokinase (Biochemica test combination kit).

— Blood Insulin: Richter radioimmunological kit, utilizing a "Packard" Tricarb Liquid Scintillation Spectrometer.

— Blood Uric Acid: "Urica Quant" colorimetric enzymatic method (Biochemica Test Combination).

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— Total Lipids: Colorimetric method of Zoeliner & Kirch.

— Cholesterol: Biochemica Test Combination Kit according to D. Watson, B. Zuk and H.H. Lefler.

— Triglycerides: Enzymatic determination of serum concentration with Biochemica Test Combination Kit.

— Urinary Catecholamines were determined fluorimetrically immediately before and immediately after the examination, so that their excretion during the test could be known by their concentration in the samples.

Blood pressure was measured before, during and after the stimulation with road noise 0-1'-2'-3'-4'-5'-6'-7'-8'-9'-10'-11'-12'-13'-20'-30'-60'-90' with an Erka "Diasist" apparatus, using a pneumatic cuff fitted with a microphone for the registration of Korotkoff tones. Systolic and diastolic pressure values were automatically indicated every minutes on dials with a margin of error of less than 1.5%.

ECG and polygraphic data were obtained with a 8-channel Elema Schönander "Mingograf 81", giving a simultaneous ECG, 4-frequency phonocardiogram and carotid pulse recording. Polygraphic records were taken at the same intervals as those used to record arterial pressure. Examination of the ECG, phonocardiogram and carotid pulse data gave an indication, inter alia, of cardiac frequency. The distance, in terms of time, and the relation between certain features of the heart cycle shown by these recording were than calculated, so that the influence of the traffic noise on the heart performance could be detected and studied.

The following systolic times and indices were considered:

— LVET (left ventricular ejection time): from the foot of the ascending branch of the carotid sphygmogram to its dierotic incisure).

— S_1 - S_2 (mechanical systole): from the 1st component of the first sound to the aortic component of the 2nd sound.

— PEP (pre-ejection period, or tension-time, or pre-expulsion systole) from the start of the Q wave to the foot of the ascending branch of the carotidogram, less the PTT.

— PTT (pulse transmission time): from the

aortic component

— Q_1 - S_2 (Total from the start

— Q_1 - S_1 (defor the Q wave to sound).

— ICT (isovol the first compo of the ascending mogram, less 1

— PEP/LVET formance, LVI lated to myoc

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BLOOD C

Sugar.

Road noise br in blood sug: 50% of case patients (Fig. in 50% of the they were significantly (Fig. 2).

aortic component of the 2nd sound to the dirotic incisure of the carotid pulse.

— Q_1-S_2 (Total or electromechanical systole): from the start of the Q wave to the first component of the 1st sound.

— Q_1-S_1 (deformation time): from the start of the Q wave to the first component of the first sound.

— ICT (isovolumetric contraction time): from the first component of the 1st sound to the foot of the ascending branch of the carotid sphygmogram, less the PTT.

— PEP/LVET expresses left ventricular performance, LVET/ICT seems more closely related to myocardial contractility.

The arithmetical mean of 10 beats was calculated for each systolic time corrected for frequency and compared with the normal theoretical values, to obtain significant data on myocardial performance. In addition, the product of cardiac frequency with systolic arterial pressure offers a good indication of coronary flow and myocardial oxygen needs.

RESULTS

The data were expressed as percents of the initials values referred as 100, and analyzed by applying Student's interval estimation, with $P = 0.05$. The controls were compared with each of the three groups and with the stimulated series as a whole. Comparison was also made between subjects aged less and more than 45 yr.

After illustrating the pattern displayed by each parameter separately, an account will be given of their behaviour in normal, dysmetabolic and coronaropathic subjects.

BLOOD CHEMISTRY PARAMETERS

Sugar.

Road noise brought about an immediate increase in blood sugar. This was more evident (over 50% of cases) in coronary and dysmetabolic patients (Fig. 1). At 30' values were still high in 50% of the normal subjects probably because they were younger. Values, in fact, differed significantly in function of age at 30' and 90' (Fig. 2).

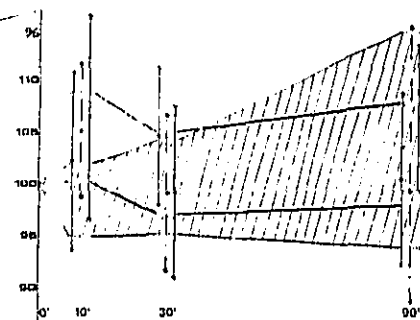


Fig. 1. Blood sugar. Normal subjects ●—●; Dysmetabolic subjects *—*—*; Coronary patients ○—○. Range of variation in unstimulated subjects *|||||*. Range of variation in stimulated subjects *-----*.

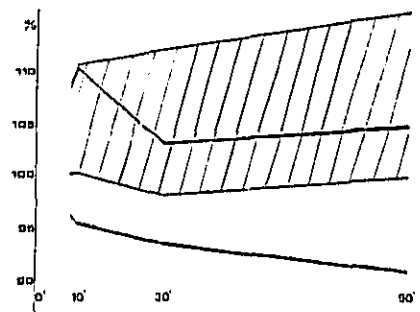


Fig. 2. Blood sugar. Subjects aged between 20 and 45 yr *|||||*; Subjects aged between 46 and 70 yr *-----*.

Insulin.

Traffic noise was accompanied by a marked increase in band width in both normal and dysmetabolic subjects. This was more evident at 90', especially in the normal cases (Fig. 3). At the end of the test, levels were well up in about 80% of normal and 50% of dysmetabolic subjects, whereas they were down in 50% of the coronary group. This, once again, may be attributed to age, since a marked, late rise in blood insulin is typical in younger subjects (Fig. 4).

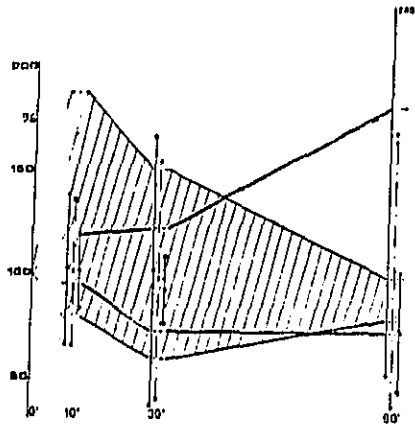


Fig. 3. Serum insulin. Normal subjects ●—●; Dysmetabolic subjects ★—★; Coronary patients ○—○. Range of variation in unstimulated subjects *|||||*. Range of variation in stimulated subjects *|||||*.

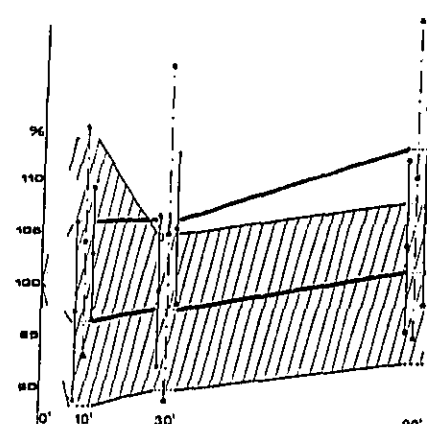


Fig. 5. Total lipids. Normal subjects ●—●; Dysmetabolic subjects ★—★; Coronary patients ○—○. Range of variation in unstimulated subjects *|||||*. Range of variation in stimulated subjects *|||||*.

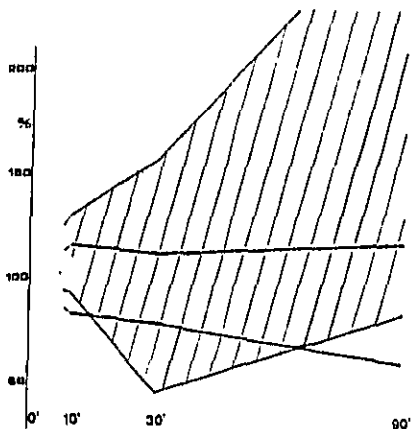


Fig. 4. Serum insulin. Subjects aged between 20 and 45 yr ●—●. Subjects aged between 46 and 70 yr ○—○. Range of variation *|||||*.

Total lipids.

At 30' and 90' both coronary and dysmetabolic subjects displayed an increase in blood lipids. Band values expanded to a much greater degree in the dysmetabolic than in the other groups (Fig. 5). In the normal subjects values were

comparable with these observed in the controls. No significant differences in function of age could be made out.

Triglycerides.

Road noise caused a fall in values that appeared earlier (10' and 30') in the dysmetabolic as opposed to the coronary subjects. At 90' there was an increase of band amplitude in the normal group (Fig. 6). The fall at 30' was more marked in patients over 45 yr. (Fig. 7).

Blood cholesterol.

Values increased after 30' and 90' in about 50% of the normal subjects. Increased band amplitude was noted from the start, especially in the coronary patients (Fig. 8). There was no significant difference in function of age.

Uric acid.

A marked increase was observed at 30' and 90', especially in the dysmetabolic group (Fig. 9). Band amplitude also increased at an early stage in the coronary subjects, and later in the normal group. Up to 30', increases were higher in the younger subjects (Fig. 10).

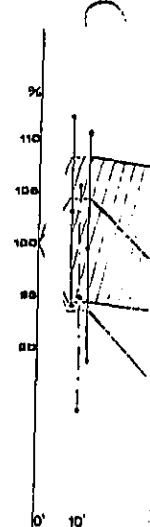


Fig. 6. Triglyceric. Dysmetabolic subjects ★—★. Normal subjects ○—○. Range of variation *|||||*. R.

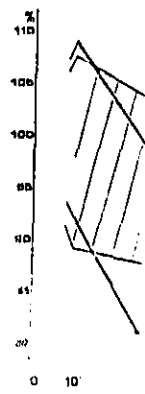


Fig. 7. Triglyceric. 45 yr. Normal subjects ○—○. Range of variation *|||||*.

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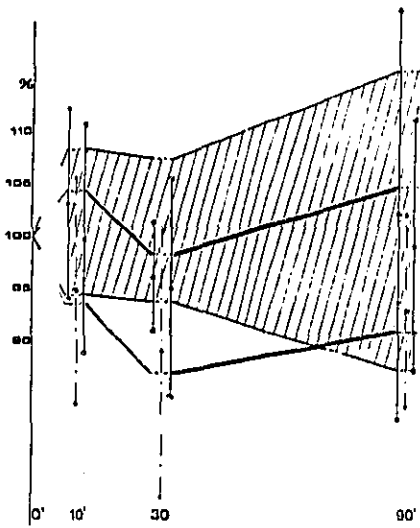


Fig. 6. Triglycerides. Normal subjects ●—●; Dysmetabolic subjects ★—★; Coronary patients ○—○. Range of variation in unstimulated subjects *hatched*. Range of variation in stimulated subjects *diagonal lines*.

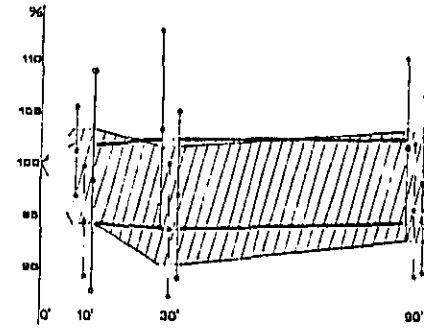


Fig. 8. Blood cholesterol. Normal subjects ●—●; Dysmetabolic subjects ★—★; Coronary patients ○—○. Range of variation in unstimulated subjects *hatched*. Range of variation in stimulated subjects *diagonal lines*.

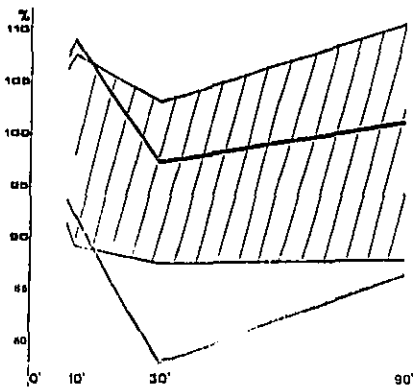


Fig. 7. Triglycerides. Subjects aged between 20 and 45 yr *hatched*. Subjects aged between 46 and 70 yr *diagonal lines*.

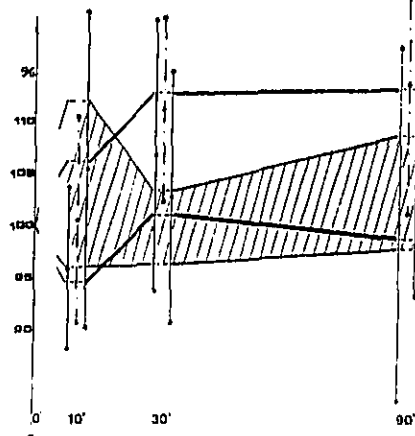


Fig. 9. Blood uric acid. Normal subjects ●—●; Dysmetabolic subjects ★—★; Coronary patients ○—○. Range of variation in unstimulated subjects *hatched*. Range of variation in stimulated subjects *diagonal lines*.

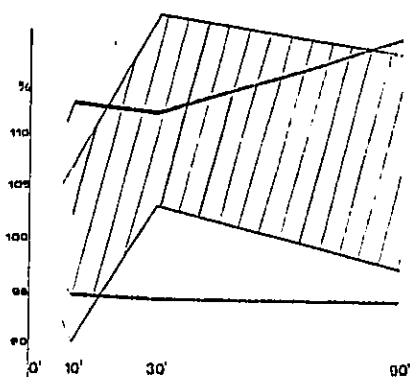


Fig. 10. Blood uric acid. Subjects aged between 20 and 45 yr (—). Subjects aged between 46 and 70 yr (hatched).

Catecholamines.

Values observed before and after the test are shown in Table 1. To obtain more accurate data it would have been necessary to keep the subjects completely at rest for 24 hr. before and after the test. This, however, was not possible. In spite of their limitation, the data showed that road noise enhanced catecholamine excretion in the dysmetabolic group. The relatively slight entity of the stimulus and its nature lead one to suppose that nothing less than direct haematochemical determination of the catecholamine content would have given an accurate picture of this parameter. The techniques required, however, are difficult to apply *in vivo*, since very low quantities are involved. Furthermore, catecholamines are very rapidly inactivated in the body.

ECG AND HEART PERFORMANCE

Road noise did not lead to any significant ECG changes, even in subjects with marked basal signs of chronic or sub-chronic myocardial ischemia. During the course of the experiment isolated atrial or ventricular extrasystole were observed in some subjects, though these appeared to be devoid of particular significance.

Left ventricular performance was indirectly assessed polygraphically by simultaneous ECG,

phonocardiographic and carotid pulse recording. Comparison was made between the length of the PEP and LVET, and of the relative importance on the ICT, with respect to the initial DT. The reciprocal relation between these parameters are indirect pointers to heart performance, especially as far as possible ischemia of the cardiac fibers is concerned. Our results did not reveal any marked variations in these and in other parameters. The noise used was apparently insufficient to influence these indices to an appreciable extent.

Changes in cardiac frequency were considered separately and together with systolic arterial pressure. As already stated, frequency times systolic pressure is regarded as one of the best pointers of coronary flow, tension time index and myocardial oxygen consumption. Traffic noise was responsible for a distinct increase of this index in the normal subjects, followed by a fall in response during the application of the stress (Fig. 11 A). A somewhat similar pattern was noted in the dysmetabolic group, though here values remained significantly high in 50% of cases until the end of the noise, after which there was a certain widening of the band (Fig. 11 B). In the coronary patients the increase was significant for a longer period in a larger number of cases (Fig. 11 C).

CONCLUSIONS

Exposure to road noise in the form employed in our experiment was followed by enhancement or depression of several blood chemistry parameters, or a wider scattering of their values, as shown by an increase of band amplitude around a more or less unchanged mean. On many occasions both responses were present, though their relative importance varied. Blood sugar displayed the earliest changes by contrast with the late response observed in the case of blood insulin.

We were particularly struck by the extent of the blood uric acid response, showing that noise has an effect on the metabolism of nucleic acids. As was to be expected, normal subjects generally presented less significant changes, especially in total lipids and triglycerides. An immediate increase in blood sugar was noted only in 50% of this group, as opposed to 80% of the other's.

Fig. 11. Cardiac subjects

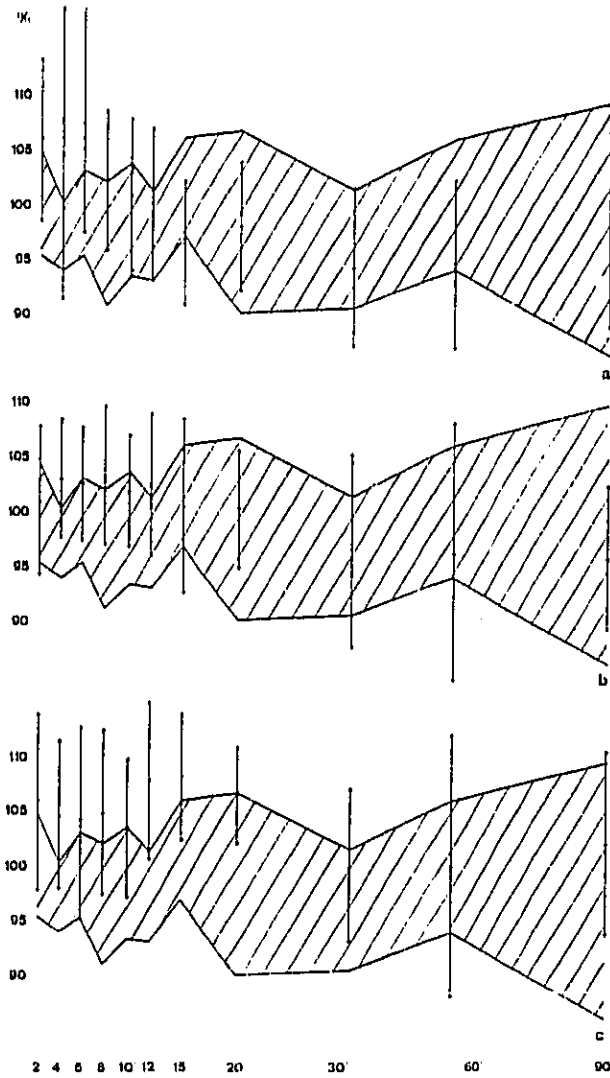


Fig. 11. Cardiac frequency times systolic arterial pressure: a) Normal subjects; b) Dysmetabolic subjects; c) Coronary patients. a1; b1; c1: Unstimulated subjects (100/60/70).

Table 1. Catecholamines excreted by patients before and after the test. Values in μg .

CATECHOLAMINES											
Controls (1)			Normal subjects			Dysmetabolic subjects			Coronary patients		
Subject	1st with- drawal	2nd with- drawal	Subject	1st with- drawal	2nd with- drawal	Subject	1st with- drawal	2nd with- drawal	Subject	1st with- drawal	2nd with- drawal
56	8.5	7.5	31	10	5.5	32	3.23	9.20	35	(*)	(*)
60	11.25	10.45	33	18	20	40	12.6	9.6	38	6.3	5.4
62	8.4	13	34	13.12	(*)	43	(*)	(*)	41	(*)	19.5
63	16.8	18	36	17.4	13.5	47	10.2	5	46	14.8	13.2
64	9.1	10.5	37	16	12.6	48	4.55	13.65	47	10.2	5
			39	10.4	14	50	11.2	27.2	54	7.2	13
			42	16.15	10.20	51	23.4	28.9	55	7	1.9
			44	(*)	(*)	53	7.8	8.04	58	20	16.19
			45	4.25	8.1	57	4.8	6.44	59	7.5	6.2
			49	10.5	12.15	61	(*)	(*)	67	15	18
			65	11.25	6	66	3.85	7.2	68	3.85	7.2
Mean value	10.81 ± 100%	11.89 ±± 109.99%	Mean value	12.7 ± 100%	11.33 ± 89.2%	Mean value	9.07 ± 100%	13.67 ± 150.7%	Mean value	10.22 ± 100%	9.89 ± 96.77%

(1) Urine samples were obtained from only 5 of the 11 controls.
(*) Missed diuresis of subjects.

whereas (1) is marked and free to display a late while its change decreases and in enlargement in Diabetic and the most significant triglycerides and the most evident in triglycerides, in total lipids at 30' and 90'.
Coronary patients though apart from were less striking were various in the case of values, shifted subjects, but can The product

whereas the late insulin response was more marked and frequent. This was the only group to display a late increase in cholesterol values, while its changes in uricemia included both decreases and increases, leading to an over 100% enlargement in band amplitude at 90'.

Diabetic and dyslipaemic subjects presented the most significant changes in total lipids, triglycerides and uric acid. This group displayed the most evident and earliest (10' and 30') fall in triglycerides, by contrast with the increase in total lipids and their wider band amplitude at 30' and 90'.

Coronary patients showed a similar pattern, though apart from blood sugar, their changes were less striking. In this group the main features were variations in band amplitude, except in the case of total lipids, whereas triglycerides values shifted less than in the dysmetabolic subjects, but earlier than in the normal group.

The product of cardiac frequency and systolic

arterial pressure — an index of change in coronary flow and the metabolic requirements of the myocardium — increased to a greater extent and for longer after the cessation of the road noise in the coronary patients, as was to be expected on theoretical grounds. This index tended to fall during the stimulation period in the normal subjects, whereas it stayed high throughout this period in the dysmetabolic group. This behaviour shows that exposure to traffic noise may be responsible for an increase in myocardial energy requirements by influencing frequency and systolic pressure.

Our blood chemistry data are too scanty to permit full interpretation of the changes observed. It can, however, be stated that road noise of the type employed can lead to distinct changes in all the parameters taken into consideration, these being of great importance as coronary risk factors in general terms, and as tending to support atheroma.

SECTION 5

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Dept. of Audiology, Otolaryngology and Internal Medicine
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Citation: Drettner, B. et al. Cardiovascular risk factors and hearing loss. A study of 1000 fifty year old men. Acta Otolaryngol. 79: 366-371, 1975

<u># of Ref.'s</u> 15	<u># of Fig.'s</u> 5	<u>Language</u> English
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<u>Type & duration of experiment</u> Type: Health examinations performed on subjects and medical histories were recorded Duration: a 2 hr. exam per subject	<u>Purpose for study</u> To determine if there is a correlation between cardiovascular risk factors and hearing loss (sensorineural)
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Description of test groups (subjects, #, age, etc.)
 1000 50 year old men in 3 socio-economic levels: 1) professionals and other highly educated people, 2) school teachers at elementary level, clerical workers, small businessmen, 3) blue-collar workers, salaried craftsmen and service workers

<u>Control of other stressors</u> no controls - smoking habits of the subjects were considered	<u>Statistical Methods</u> Chi-square test and the Kolmogorov-Smirnov test (for noise exp. and smoking habits)
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<u>Noise Stimulus</u> Noise exposure (qualitative) histories of the subjects were recorded; socio-economic level 3 had greater occupational noise exposure and hearing loss (most of the subjects in the other 2 levels had been in the military and could have some noise exposure)	<u>CVS Response Measured</u> blood pressure heart rate <hr/> <u>Nonauditory effects</u> CVS: see Author's conclusions <hr/> Other:
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Author's conclusions
 No significant correlations between cardiovascular risk factors and hearing loss were found. Further study is needed.

Evaluation & comments
 The three groups of subjects contain too many different occupations lumped together. Actual noise exposures of the subjects were too vague to make definite conclusions. The authors were not directly interested in the effects of noise on the cardiovascular system.

Drettner, B. et al. Cardiovascular risk factors and hearing loss a study of 1000 fifty-year-old men. Acta Otolaryngol. 79:366-371, 1975.

A health survey of 1000 healthy 50 year old men in Uppsala, Sweden was done to determine if a correlation exists between hearing loss (sensorineural) and various cardiovascular risk factors. The men were divided into 3 groups, according to their occupational and educational backgrounds: 1) professionals, executives, other highly trained people; 2) clerical and sales workers, self-employed craftsmen, foremen, small businessmen, elementary school teachers; 3) blue-collar workers, service workers, salaried craftsmen. Routine physical exams were performed in the morning, after the subjects had been requested not to smoke or eat after the previous midnight. Audiograms, blood chemistry tests (serum cholesterol and triglycerides, uric acid, glucose tolerance), blood pressure, and heart rates were included in the exams, as well as an assessment of the ear diseases, smoking habits, and noise exposures of the subjects. Subjects with high cardiovascular risk factors had diastolic blood pressures greater than 85 mm Hg, systolic blood pressures greater than 135 mm Hg, serum cholesterol levels higher than 240 mg per 100 ml, and smoked over 10 cigarettes per day. No correlations were found between hearing loss and high cardiovascular risk factors. Group 3, which had both greater hearing loss (sensorineural and conductive) and greater noise exposure did not have increased cardiovascular risk factors.

CARDIOVASCULAR RISK FACTORS AND HEARING LOSS

A Study of 1000 Fifty-Year-Old Men

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(Received May 16, 1974)

Abstract. The hypothesis that cardiovascular risk factors might be of importance in the development of sensorineural hearing loss was tested in a material of 1000 fifty-year-old men. No significant correlations were found. The present study confirmed the well-known observation that the left ear usually is poorer than the right. Hearing loss in the right ear was found to be related to the smoking habits in the groups with no history of noise exposure. The explanation for this is discussed. Hearing loss was more common in social class 3 than in the other social classes. This difference was principally referable to noise exposure but also to conductive hearing loss. A prospective study of this material will further analyze the question concerning a possible relationship between cardiovascular risk factors and hearing loss.

The deterioration of hearing related to aging is a well known phenomenon. It is predominantly a matter of progressive sensorineural high tone loss, which has been observed to start already during the third decade of life. Most of the available information on morphological and functional changes in presbycusis has been reviewed by Schmidt (1967). In summing up he points out: "we all carry to our senium the cumulation of harms done to our hearing acuity in a lifetime."

Among factors discussed are principally noise exposure and vascular disease.

The importance of cardiovascular disease is unclear. No relation between hearing loss and cardiovascular disease was noted by Bunch et al. (1929, 1931) or Miller & Ori (1966). On the other hand a relationship between a certain morphological type of presbycusis and vascular

disease has been suggested by Schuknecht (1964). Weston (1964) reported that the age of onset and the progress of presbycusis were related to circulatory disturbances, among other factors.

Audiological studies performed in different areas of the world have indicated a possible relationship between hearing loss and high intake of saturated fats, high cholesterol levels, atherosclerosis and coronary heart disease (Rosen et al. 1962, 1964a, 1964b, 1965, 1970a, 1970b). Great attention has focused on Rosen's reports according to which a change in the fat composition of the diet in the direction from saturated to unsaturated fat was followed not only by a reduction of the incidence of coronary heart disease but also of a diminished impairment of hearing (Rosen et al., 1970a).

An observation which is also of interest in this connection is the study by Ismail et al. (1973) showing that physical exercise which resulted in improvement in different cardiovascular parameters, did not affect the hearing thresholds, but resulted in an improved ability to recover from a temporary threshold shift induced by noise.

Against this background it was judged to be of interest to perform tone audiometry in connection with an extensive health examination survey of a large unselected material of 50-year-old men. The health examination was intended to identify risk factors for cardiovascular disease, such as hypertension, elevated blood lipids levels and smoking.

Table 1. Factors used, hearing loss

1. Systolic blood pressure
2. Diastolic blood pressure
3. Heart rate
4. Serum-cholesterol
5. Serum-triglycerides
6. Uric acid
7. Hematocrit
8. Glucose tolerance
9. Smoking habit
0. Noise exposure

The purpose of the search for possible hearing loss and cardiovascular disease as well as its relationship concerning smoking habit do not seem to have been

MATERIAL

The material consisted of 1000 fifty-year-old men who were consecutively included in a health examination survey of identifying risk factors for cardiovascular disease in healthy middle-aged men in Uppsala. The examination was performed the morning before the hearing examination. All participants were asked to fast overnight and not to smoke or drink alcohol the night before. The researchers' whole examination participation rate was 83.9%. Table 1 which have been used to describe the relationship with the amount of hearing loss.

Grouping of the material into social classes was based on the results of the health examination. The following classification (Andersen et al., 1970):

Social class 1: Professions, highly trained persons, high managers of large business

Social class 2: Clergymen, foremen, self-employed, proprietors, elementary

Social class 3: Laborers, operatives, salaried craft

Table I. Factors used for correlation studies with hearing loss

1. Systolic blood pressure
2. Diastolic blood pressure
3. Heart rate
4. Serum-cholesterol
5. Serum-triglycerides
6. Uric acid
7. Hematocrit
8. Glucose tolerance
9. Smoking habit
10. Noise exposure

The purpose of the present investigation was to search for possible correlations between hearing loss and cardiovascular risk factors, separate as well as in combinations. Studies concerning smoking habits and hearing function do not seem to have been reported earlier.

MATERIAL

The material consisted of 1000 men, aged fifty, who were consecutively selected from a health examination survey, with the special aim of identifying risk factors for cardiovascular disease in healthy middle-aged men in the City of Uppsala. The examination was performed in the morning between 7.15 and 9.00 a.m. The participants were asked to come after an overnight fast and not to have smoked after midnight. The researchers were the same during the whole examination period. The participation rate was 83.9%. Table I shows the "risk factors" which have been used to study the interrelationship with the amount of sensorineural hearing loss.

Grouping of the material according to social classes was based on interview report on occupation. The following three classes were used (Andersen et al., 1970):

Social class 1: Professionals and academically trained persons, high officials, proprietors and managers of large businesses and industry.

Social class 2: Clerical and sales workers, women, self-employed craftsmen, small business proprietors, elementary school teachers.

Social class 3: Laborers, service workers, operatives, salaried craftsmen.

METHODS

The heart rate was counted and the blood pressure measured after 10 minutes of rest in the lying position.

The serum cholesterol and the triglycerides were assayed in an Isopropanol extract of serum by using a Technicon dual-channel system (N-24 A and N-70). The hematocrit was measured with a micromethod with capillary tubes. Uric acid was determined by a wolframat method.

The intravenous glucose tolerance test (IVGTT) was performed with a glucose dose of 0.5 g per kg bodyweight administered as a 50% solution. Blood samples for determination of glucose in plasma were taken at 10 min intervals over 1 hour for estimation of glucose tolerance, which was expressed as a K -value calculated from the formula: $K = \ln 2 \times 100 / T_{1/2}$ where the $T_{1/2}$ is the time in minutes required for the concentration to be reduced by half its value. This IVGTT was performed in a random sample of 594 men in this study.

The hearing was tested with a pure tone audiometer at the frequencies 500, 1000, 2000, 3000, 4000 and 6000 Hz bilaterally. With the sound-insulated headphones used the investigation room was found to be silent enough to permit relevant threshold determinations down to 15 dB (ISO, R 389), which was regarded as non-significant hearing loss and lower values were accordingly not registered.

The individuals were interviewed by questionnaire concerning history of ear disease, noise exposure and smoking habits.

CLASSIFICATION OF THE MATERIAL AND STATISTICAL PROCEDURES

A computer IBM 370/155 was used for sorting and calculations. A check was included that all parameters fell within reasonable limits. Correlation studies were performed between hearing loss and the factors in Table I. Moreover smoking habits, history of noise exposure and of ear disease were included in the statistical calculations.

The material was grouped as follows:

Material A consisting of the total material, 1 000 males.

Material B consisting of 762 males after exclusion from the total material of 238 subjects who had possible conductive hearing loss.

The criteria for this exclusion were: (i) Hearing loss of 30 dB or more at 500 Hz in at least one ear, (ii) and/or positive answer to the question "Have you had any ear inflammation after the age of 20", (iii) and/or positive answer to the question "Have you any ear disease".

Material B was subdivided according to the following criteria:

Social classes:

1	103	(13.5%)
2	319	(41.9%)
3	340	(44.6%)

History of noise exposure:

B1	"yes"	388	(50.9%)
B11	"no"	374	(49.1%)

The following groups were selected from *Material B* with regard to smoking habits and noise exposure:

- B1. Noise exposure, never smoked, $n = 101$
- B2. No noise exposure, never smoked, $n = 105$
- B3. Noise exposure, heavy smokers, $n = 79$
- B4. No noise exposure, heavy smokers, $n = 92$

(Heavy smoking means smoking ≥ 10 cigarettes per day and smoking duration of at least 10 years. Pipe and cigar smokers were excluded unless they did not smoke ≥ 10 cigarettes a day).

Classification of hearing loss

The hearing loss of each individual was classified from the aspects given below. Each classification comprised the computing of hearing loss values of the right ear, the left ear and both ears with respect to the following classification groups 1-7:

1. Sum of hearing loss at 500-6 000 Hz.
2. Sum of hearing loss at 1 000-6 000 Hz.
- 3-7. Hearing loss at 1 000, 2 000, 3 000, 4 000 and 6 000 Hz, respectively.

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Each group was subdivided into five discrete classes of about equal size. Moreover the groups 3-7 were subdivided into three classes concerning each ear:

- Hearing loss ≤ 15 dB
- Hearing loss 20-35 dB
- Hearing loss ≥ 40 dB

Calculations in material B

The main computing program cross-classified all individuals within material B and its subgroups B1 and B11, i.e. with and without noise exposure respectively, regarding each risk factor and all groups of the hearing loss. The χ^2 -test was used to test the significance of differences. The accepted level of significance was $p < 0.05$.

The individual difference in hearing levels between both ears at 1 000, 2 000, 3 000, 4 000 and 6 000 Hz respectively were calculated for the total group B, subgroup B1 and B11 as well as for risk-groups B1-B4. The Kolmogorov-Smirnov test was applied to test for any kind of difference in the distribution between B1 and B11 and between all combinations of B1-B4.

RESULTS

Cardiovascular risk factors

The correlation studies between hearing loss in material B, classified as presented above, and the factors of Table I showed no significant correlations except for smoking habits which will be discussed below.

No significant correlations were found between the amount of hearing loss in high and low risk groups selected from material B. Subjects in the high risk group ($n = 28$) had systolic blood pressure ≥ 135 mmHg and diastolic blood pressure ≥ 83 mmHg, serum cholesterol ≥ 240 mg/100 ml and smoked more than 10 cigarettes a day. Subjects in the low risk group ($n = 35$) were non-smokers, had serum cholesterol ≥ 220 mg/100 ml and systolic blood pressure ≥ 125 mmHg and diastolic blood pressure ≥ 80 mmHg.

Social classes and hearing loss

Table II shows the distribution of hearing loss in the total material, divided into social classes.

Table II. *Distribution of hearing loss in 1000 males of hearing loss of 1 000-6 000 Hz, dB an amount means no significant*

Social class	N	1	2	3
1	130	2	1	1
2	402	2	1	1
3	468	1	1	1

With the screen hearing loss of hearing loss", it may be regarded in table II, hearing loss as most prevalent which 26.3% had, 16.0% were free of hearing loss. The corresponding percentages are 12.3% and 23.8% in distribution of hearing loss in classes 1 and 3, which are statistically significant.

It seems that there is a more prevalent class 1. Among the subjects with conductive hearing loss given previously in class 3 and only 11.5% of the responding figures were 44 and 13.5% of the subjects between the two statistically significant occurrence of hearing loss in social class 3.

Noise exposure

In the total material the amount of hearing loss was statistically significant 3 than in any of

HIN 110000



discrete groups concern-

Table II. Distribution (%) of hearing loss in social classes in 1 000 males, aged 50. The amount of hearing loss is expressed as the sum of dB hearing loss of both ears at five frequencies 1 000-6 000 Hz. With the screening level of 15 dB an amount of hearing loss of 150 dB thus means no significant hearing loss

Social class	N	Amount of hearing loss in dB					
		150-180	185-225	230-295	300-385	390-	
1	130	21.8	24.6	17.7	21.5	12.3	100
2	402	25.4	23.9	20.4	15.7	14.7	100
3	468	16.0	13.9	21.6	22.2	26.3	100

classified into sub-groups according to noise exposure factor. χ^2 -test differences, $p < 0.05$. χ^2 levels 1, 4 000 used for analysis as well as logarithmic kind BI and B1-B4.

hearing loss in noise, and significant differences which

between low risk subjects in the blood pressure 240 mg/100 mmHg, 165 mg/100 mmHg, 135 mg/100 mmHg, 110 mg/100 mmHg were 220 mg/100 mmHg.

hearing loss in social classes.

With the screening level of 15 dB, a sum of hearing loss of 150 dB means "no significant hearing loss", whereas a sum of 390 dB or more may be regarded as severe hearing loss. As seen in table II, hearing loss was most common as well as most pronounced in social class 3, in which 26.3% had severe hearing loss while only 16.0% were free from significant hearing loss. The corresponding figures in social class 1 were 12.3% and 23.8% respectively. This difference in distribution of hearing loss between social classes 1 and 3, seen in the table, was found to be statistically significant.

It seems that conductive hearing loss was also more prevalent in social class 3 than in social class 1. Among the 238 subjects, who had possible conductive loss according to the criteria given previously 53.4% belonged to social class 3 and only 11.3% to social class 1. The corresponding figures among the remaining 762 subjects were 44.6% belonging to social-class 3 and 13.5% to social class 1. This difference between the two social classes was however not statistically significant. There was also a greater occurrence of men with a history of ear disease in social class 3 than in the other two classes.

Noise exposure

In the total material a history of noise exposure was statistically more prevalent in social class 3 than in any of the other two classes.

In material B there were 388 individuals with a history of civil and/or military noise exposure, whereas the remaining 374 cases denied such exposure. The hearing loss was found to be significantly greater in the noise-exposed group, the difference being most pronounced around 4 000 Hz ($p < 0.001$). These observations were valid for the left as well as for the right ear.

In cases with asymmetrical hearing data (difference > 10 dB) the hearing loss was in most cases greater in the left ear than in the right ear. The number of cases with left-sided inferiority was found to increase towards higher frequencies to be most pronounced at 4 000 Hz. In this respect there was no difference between those who had answered "yes" or "no" to the question concerning noise exposure.

Smoking habits

In the total material there were 509 smokers and 276 who had never smoked. Neither material A nor material B showed any significant differences in hearing loss related to smoking habits per se. However, smoking and hearing loss may still have an etiological common denominator, according to the following.

Combinations of noise exposure and smoking habits

The four sub-groups in material B (B1-B4) listed above were selected for correlation studies between, on one hand, hearing loss and, on the other, history of smoking habits and noise exposure. Hearing data in the four groups are presented in Table III.

Only one indication of a possible effect of smoking was found. Among the 92 individuals (B4) who had smoked more than 10 cigarettes daily but had not been exposed to noise, the amount of right-sided hearing loss was significantly greater ($p < 0.001$) than in the 105 individuals (B2) who had never smoked and had not been exposed to noise (Figs. 1 and 2). Among these smokers, who were not exposed to noise, there was no right-sided superiority in

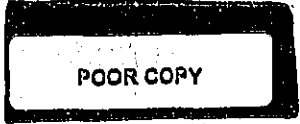


Table III. Hearing loss related to history of noise exposure and smoking habits in 377 males, aged 50

The hearing loss is given as median values in dB for the right and the left ears respectively at 2000-6000 Hz

Hz	Ear	No history of noise exposure		History of noise exposure	
		Non-smokers N 105	Smokers N 92	Non-smokers N 101	Smokers N 79
2 000	Right	18.1	19.0	18.4	18.4
2 000	Left	18.5	18.8	19.1	19.2
3 000	Right	19.2	25.7	22.8	24.7
3 000	Left	22.5	23.2	29.8	28.0
4 000	Right	23.0	31.0	34.0	34.5
4 000	Left	28.1	29.5	39.1	40.6
6 000	Right	26.3	28.3	35.8	29.6
6 000	Left	30.9	27.8	41.3	38.1

hearing but instead an insignificant tendency towards left-sided superiority. These findings refer to 3 000 and 4 000 Hz.

No similar difference was found in the noise exposed material between the 79 individuals (B3) who had smoked 10 cigarettes daily and the 101 individuals (B1) who had never smoked.

DISCUSSION

The phenomenon that the left ear usually is poorer than the right ear, has been observed

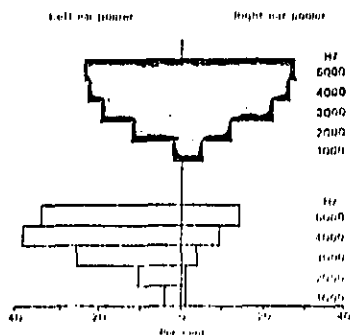


Fig. 7. The distribution of an asymmetrical hearing loss at five cardinal frequencies among 197 males, aged 50, with no history of noise exposure. The upper part of the figure shows the results from group B4 (heavy smokers) and the lower part from group B2 (never smoked). Asymmetry means a difference of >10 dB.

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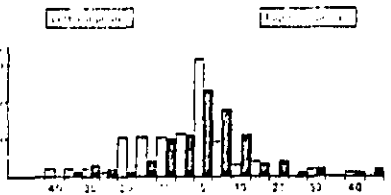


Fig. 2. The distribution of differences in hearing loss between right and left ears at 4000 Hz in 197 males, aged 50, with no history of noise exposure. White bars represent the 105 non-smokers (group B2); black bars the 92 smokers (group B4).

previously (Glorig & Roberts, 1965). Asymmetrical hearing impairment of this kind has been ascribed to noise, especially from fire arms. In our study left-sided inferiority was found not only among the individuals with noise exposure but also in those who denied noise exposure. It must be kept in mind, however, that most of the subjects who denied noise exposure had nevertheless had a period of military service in their past. The left-sided inferiority, statistically observed, might also be of a more genuine origin since the phenomenon has been observed among male children as well (Kannan & Lipscomb, 1974).

Another observation, partly connected to noise exposure was that hearing impairment was more common in social class 3 than in the other classes. This difference was found to be due not only to noise exposure but also to greater occurrence of conductive hearing loss.

A correlation between hearing impairment and cardiovascular risk factors has been discussed, not at least considering the publications of Rosen et al. (1965, 1970). They reported that a long-term change of the fat diet from saturated to poly-unsaturated fat intake was followed by a decrease not only in the incidence of coronary heart disease but also in the deterioration in hearing. In our study we did not find any correlation between hearing loss and cardiovascular risk factors or combinations of risk factors.

The only exception that the hearing in heavy smokers not only individuals who were who had never smoked statistically significant with a history of noise was found between those who had never had any obvious explanation. It may be possible that conditions in heavy deterioration of hearing discernible in the absence of a hypothetical dominantly the right ear is superior, seems obvious that it is not necessary. The question of a between cardiovascular factors motivates further of a prospective character material will offer su-

ZUSAMMENFASSUNG

Die Hypothese, dass kann die Entwicklung der Schärfe sein könnten, wurde an 197. Keine signifikanten Unterschiede stellt. Die vorliegende Studie Beobachtungen, dass das geschädigt ist als das rechte Lärmeinwirkung hervorgeht mit keiner Lärmeinwirkung doch eine Korrelation zwischen Ohres und den Rauchgefahrung dafür wird diskutiert Sozialgruppe 3 häufiger. Dieser Unterschied war hängung zurückzuführen, aber eine prognostische Bearbeitung das Problem eine zwischen kardiovaskuläre Störungen veranschaulicht.

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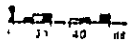
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Sweden

The only exception was the peculiar finding that the hearing in the right ears was poorer in heavy smokers not exposed to noise than in the individuals who were not exposed to noise and who had never smoked. This difference was statistically significant. Among the individuals with a history of noise exposure no similar difference was found between the heavy smokers and those who had never smoked. We cannot find any obvious explanation for this observation. It may be possible that smoking, or associated conditions in heavy smokers, can result in a deterioration of hearing, with an effect only discernible in the absence of noise trauma. Why such a hypothetical effect should involve predominantly the right ear, which otherwise usually is superior, seems obscure. It should be stressed that it is not necessarily a matter of causality. The question of a possible relationship between cardiovascular risk factors and hearing loss motivates further investigation, especially of a prospective character. The follow-up of our material will offer such a possibility.

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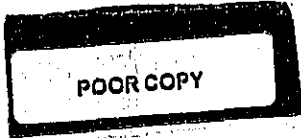
Die Hypothese, dass kardiovaskuläre Risikofaktoren für die Entwicklung der Schwerhörigkeiten von Bedeutung sein könnten, wurde an 1000 50-jährigen Männern getestet. Keine signifikanten Korrelationen wurden festgestellt. Die vorliegende Studie bestätigt die wohlbekannteren Beobachtungen, dass das linke Ohr gewöhnlich mehr geschädigt ist als das rechte, was wahrscheinlich durch die inwirkung hervorgerufen wird. In den Gruppen mit einer Lärmwirkung in der Anamnese wurde jedoch eine Korrelation zwischen Hörstörungen des rechten Ohrs und den Rauchgewohnheiten gefunden. Die Ergebnisse dafür wird diskutiert. Hörstörungen traten in der Altersgruppe 3 häufiger auf als in den anderen Gruppen. Dieser Unterschied war hauptsächlich auf Lärmwirkung zurückzuführen, aber auch auf Schalleitungsstörung. Eine prognostische Bearbeitung dieses Materials wird das Problem eines möglichen Zusammenhangs zwischen kardiovaskulären Risikofaktoren und Gehörstörungen veranschaulichen können.



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SECTION 6

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> G.R. Froehlich, Col., GAF, MC	<u>Institution and address where research was performed</u> German Air Force Institute of Aviation Medicine Fuerstenfeldbruck
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> same as above
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Citation Froehlich, G.R. The effects of ear protectors on some autonomic responses to aircraft and impulsive noise. AGARD (ADVIS Group Aerospace Res Dev) Conference Proceedings 171:CB-1--CB-5, 1975

# of Ref.'s 0	# of Fig.'s 7	Language English
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<u>Type & duration of experiment</u> laboratory experiment--short term	<u>Purpose for study</u> To test 3 different ear protectors used by the German AF, and determine their effectiveness in reducing autonomic responses produced by aircraft and impulsive noise.
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Description of test groups (subjects, age, etc.)
1 group of 25 human subjects with normal hearing; served as their own controls
control--without ear protectors
test--with each of 3 types of ear-protectors

<u>Control of other stressors</u> subjects were blindfolded, and possible random noises were masked by a continuous 50 dB(A) white noise.	<u>Statistical Methods</u> test for significance
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<u>Noise Stimulus</u> source: child's pistol (impulse noise) jet aircraft noise spectral characteristics: see graph; centered at 1000 Hz noise level: impulse--130 dB; 95 dBA (aircraft) 95 dBA (aircraft) length of exposure: 20 sec (aircraft) # of trials: not specified	<u>CVS Response Measured</u> (1) peripheral blood flow with digital plethysmography finger pulse amplitude (2) heart rate <u>Nonauditory effects</u> (no ear protectors): (1) pulse amplitudes--20 sec. jet noise: amplitude decreased to 63.3% initially; back to 54.4% at end of 20. sec. pistol shot: amplitude decreased to 62% (2) heart rate--no significant changes due to noise. <u>Other</u> : electrodermal responses--appearance of voltage due to noise
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Author's conclusions The best ear protectors (with which the amplitude response was lessened) were those that blocked out the low and medium frequencies. All 3 blocked out high frequencies well. It was concluded that autonomic nervous system responses are more affected by low and medium noise frequencies.

Evaluation & comments
This is one of the many studies that shows peripheral vasoconstriction as a response to noise.

Froehlich, G. R. The effects of ear protectors on some autonomic responses to aircraft and impulsive noise. AGARD (Advis. Group Aerosp. Res. Dev.) Conf. Proc. 171: C8-1-C8-5, 1975.

The effectiveness of 3 different ear protectors in blocking autonomic responses to noise was studied in a group of 25 human subjects with normal hearing. The responses measured were heart rate, fingerpulse amplitude, and electrodermal response. Each subject was blindfolded and a background of continuous 50 dBA white noise was present throughout the study to eliminate other sensory stimuli. The subjects served as their own controls-- responses with and without ear protectors were recorded. The noise doses were of 2 types: impulse noise from a child's pistol at a level of 130 dB and 20 sec. of jet aircraft noise at a level of 95 dBA with spectra centered at 1000 Hz. No significant changes were noted in heart rate due to noise. Definite electrodermal and fingerpulse amplitude responses due to noise were observed. With no ear protectors, there were fingerpulse amplitude reductions of 63.3% plus or minus 12.6% from the jet aircraft noise and 62% plus or minus 14% from the impulse noise. The ear protectors that were most effective in reducing the fingerpulse amplitudes and the electrodermal responses to noise were also more efficient in blocking the low and medium noise frequencies. Since all 3 ear protectors could block high frequencies equally well, it was concluded that the autonomic nervous system responds more to low and medium noise frequencies.

THE EFFECTS OF EAR PROTECTORS ON SOME AUTONOMIC RESPONSES
TO AIRCRAFT- AND IMPULSIVE NOISE

N76-12794

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Flirstenfeldbruck

INTRODUCTION

After extensive studies of the aural effects of noise, in recent years there has been an increasing interest in the non-auditory physiological effects of noise on man. The first investigators were primarily concerned with the effects of intense noise on the circulatory system. Here we encounter as the most reliable finding peripapral vasoconstriction together with more variable other cardiovascular changes. In conjunction with respiratory and endocrinological changes, all these effects are physiological responses within the frame of ergotropic mechanisms. Since all these responses depend primarily on the intensity, duration and spectral character of noise, the wearing of ear protectors must decrease the physiological responses. We therefore have chosen as acoustical stimuli an impulsive noise and jet aircraft noise together with three different types of ear protectors currently in use in the German Armed Forces (Fig. 1).

Selectone K

1b

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Com-Fit

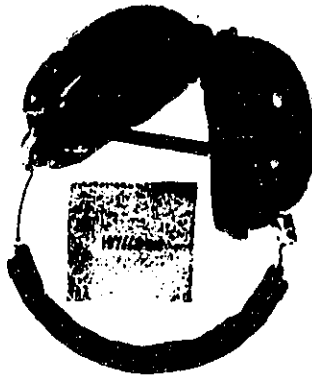


Fig. 1

The ear protectors described in the text.

METHODS

The SELECTONE K earplug contains two tiny holes, one connecting the inner cavity of the earplug with the outside air, the other with the air volume enclosed in the ear canal. Thus it acts as a two-section low-pass filter with low attenuation for the higher frequencies.

The COM-FIT earplug is a very efficient ear protector, providing excellent attenuation for all frequencies.

The HILSON Sound Barrier Earmuff SB 250 is widely known and provides high attenuation already at 1000 Hz. The attenuation characteristics are shown in Fig. 2.

The impulsive noise was produced by discharging a children's pistol at a distance of 30 cm from the left ears of the normal hearing subjects, thereby producing 130 ± 2 dB(1m). In order to avoid permanent hearing damages, we dispensed with firing real pistols in the closed reverberating room. All four situations have been equally distributed to avoid habituation effects.

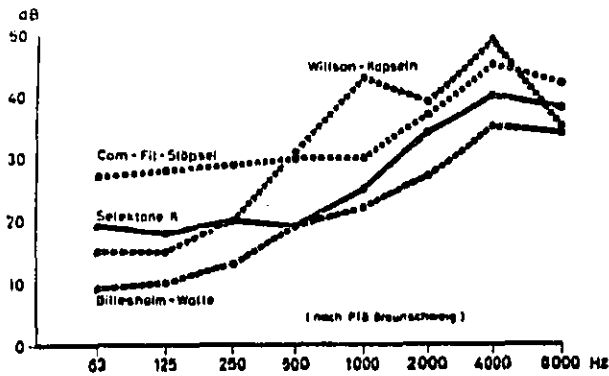


Fig. 2
Attenuation provided
by the ear protectors

As typical aircraft noise we have used a 20 sec F-104(0) jet aircraft noise of 95 dB(lin) = 95 dB(A), as demonstrated in Fig. 3. Since there is a discrete frequency in the octave band centered at 1000 Hz, there is a very annoying, shrieking sound within a broadband noise. The noise levels and spectra in the subjects' ear canals were calculated by subtracting from the ambient noise level the attenuation values of the three ear protectors.

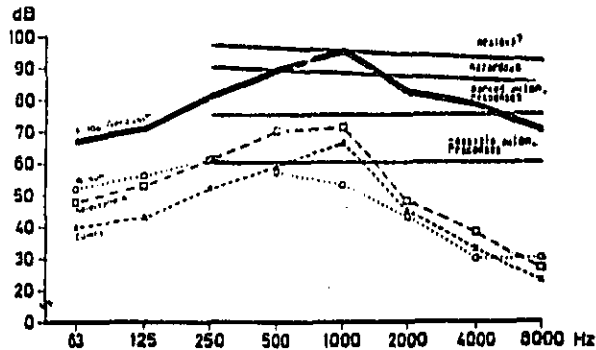
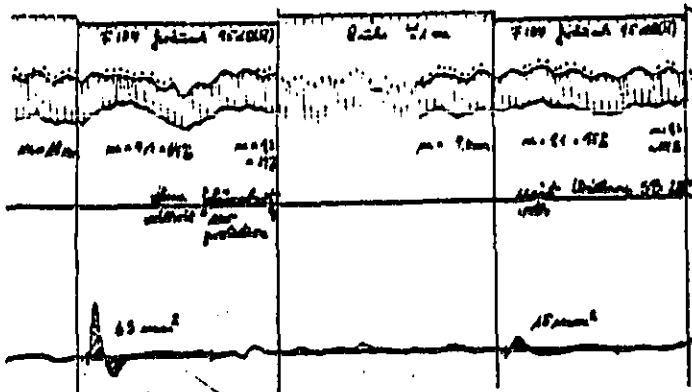


Fig. 3
Estimated noise rating for
autonomic responses as
proposed by JANSEN; spectrum
of F-104 noise and its
reduction by ear protectors.

The peripheral blood flow was determined by use of photoelectric transducers at the mid-phalanx of the right middle finger. From these plethysmographic traces we could also compute the pulse frequency (Fig. 4)



... powers of the resulting ITS were 10.5 and 27.5 dB respectively.

... aircraft's endurance record is 31 hours.

C-3

The electrodermal response (EDR) is the biphasic appearance of a voltage in response to an emotional stimulus. It reflects already minor changes in activity of the autonomic nervous system. The total areas of positive and negative phases were computed in mm^2 . All tests have been conducted in a room having normal reverberation at 22 - 24° Celsius between 14 - 1600 hours. In order to eliminate other sensory stimuli, the subjects had been blindfolded and possible random noises had been masked by a continuous 50 dB(A) white noise.

RESULTS

1. The Effects of Ear Protectors on Peripheral Vasoconstriction Caused by a 20 sec Jet Noise (Fig. 5)

Taking the average of the last 10 amplitudes before the sudden onset of noise as 100%, without ear protectors we have an amplitude reduction to 63.3% \pm 12.6% as initial response and towards the end of the stimulation a recovery to 84.4% \pm 13%. The amplitude reductions are considerably lower when ear protectors are used. With COM-FIT the amplitudes are reduced to 79% \pm 12% and recover to 99%. The responses with the MILLER SB EM are essentially the same: initial response 79% \pm 10% and 97% \pm 9% towards the end. The use of SELECTONE K offers less protection as is expressed by the more marked amplitude reductions to 72% \pm 12% as initial response and a recovery to 94% \pm 9% at the end of the 20 sec period. The differences of responses with and without the various ear protectors have been significant at the 0.001 level.

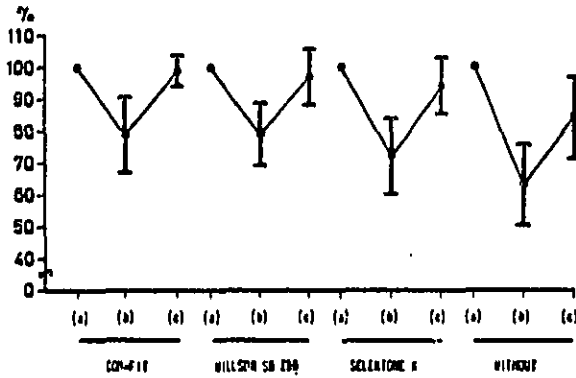


Fig. 5
Peripheral vasoconstriction caused by 95 dB(A) jet noise. (a) pre-exposure = 100%, (b) initial response and (c) recovery towards the end of exposure

2. Effects of Ear Protectors on Peripheral Vasoconstriction Caused by Impulsive Noise (Fig. 6)

The initial responses after the 130 dB pistol shots without ear protectors show marked amplitude reductions to 62% \pm 14%. With the use of COM-FIT there is only a small reduction to 86% \pm 9% and with SELECTONE K to 79% \pm 20%. In every intrasubject comparison, the protective

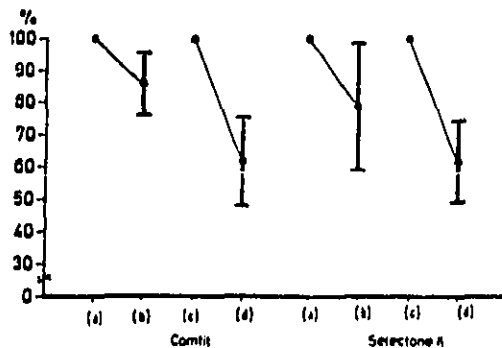


Fig. 6
Peripheral vasoconstriction caused by 13 dB impulsive noise. (a) pre-exposure = 100%, (b) with ear protectors, (c) pre-exposure and (d) without ear protector

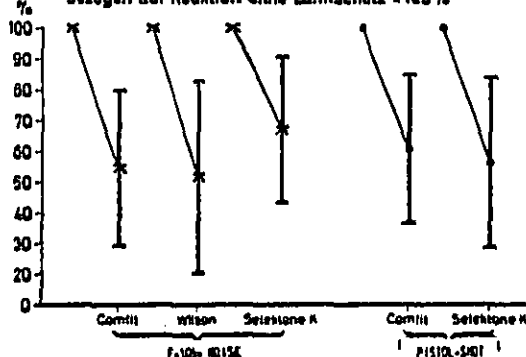
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3. The Effects of Ear Protectors on the Electrodermal Response (EDR) (Fig. 2)

If we take the EDR evoked by the F-104 noise without ear protectors as 100%, there is a considerable decrease of these responses when ear protectors are used. With COM-FIT in the external ear canal, the EDR is reduced to 53% ± 26%, with HILLSON to 51% ± 31% and with SELECTONE to 67% ± 24%. Thus, the effectiveness of the HILLSON earmuff and COM-FIT earplugs is the same, that of SELECTONE K somewhat less. When the 130 dB impulsive noise is presented, the COM-FIT earplug achieves an EDR reduction to 60% ± 25%, whereas SELECTONE K shows a reduction to 56% ± 28%. In this case, the SELECTONE K especially designed for protection against impulsive noise achieves the same effectiveness as COM-FIT, which otherwise has better protective properties.

Fig. 2

Erminderung der Galvanischen Hautreaktion bei F-104-Lärm (x) und Pistolen-schuss (o) bei Tragen von Lärmschutzgeräten, bezogen auf Reaktion ohne Lärm-schutz = 100%



Reduction of electrodermal response by wearing ear protectors. Response without ear protection = 100%

4. Heart Rate

As already mentioned by several authors, there are no significant changes of the heart rate under the stress of noise, as can be seen in the following table:

	COM-FIT	HILLSON	SELECTONE K	WITHOUT
TABLE I: Change of Heart Rates in % with 95 dB(A) noise	+ 1,8% ± 3,7%	+ 2,3% ± 3,7%	+ 2,4% ± 5,6%	- 1,7% ± 5,3%

	COM-FIT	WITHOUT	SELECTONE K	WITHOUT
TABLE II: Change of Heart Rate with 130 dB Impulsive Noise	+ 0,1% ± 5,5%	- 0,45% ± 6,6%	+ 0,55% ± 5,5%	+ 1,65% ± 4,2%

5. Subjects' Assessment of Ear Protectors Against F-104 Noise

Immediately after the tests, each of the 25 subjects was asked to assess the effectiveness of the three different ear protectors and to establish an order of rank:

EAR PROTECTOR	ORDER OF RANK			Average
	1.	2.	3.	
SELECTONE K	2	3	20	2,7
COM-FIT	12	8	2	1,3
HILLSON	8	14	3	1,8

The COM-FIT ear plug has been assessed as the most effective ear protector, closely followed by the HILLSON earmuff. In accordance with the results of objective measurements, the SELECTONE K earplug is assessed as considerably less effective.

For the three classifications of career flying time shown in Figure 1 (2500-4000 hours, 4000-5500 hours, and 5500 or more hours), $r = .043$, 1.45 and $.0032$ respectively (using Yates' correction for continuity).

CB-3

DISCUSSION

As shown by the frequency analysis in Fig. 3, marked autonomic responses had to be expected. They could be elicited for the peripheral blood flow as well as the electrodermal responses. Contrary to this, the heart rate showed no significant changes in terms of increase or decrease. The attenuation characteristics of the three ear protectors are quite different, so that at the eardrum of the subjects the intensities and qualities of the noise had been different too. By calculation, the protective effects of the WILSON earwuff is best because the preponderant frequencies about 1000 Hz are attenuated most effectively. The second best is the CCM-FIT earplug, whose attenuation at 1000 Hz is not as effective, but better in the lower frequency range. SELECTONE K has the least protective effect of all three in the lower and medium frequency range, whereas above 1000 Hz the attenuation properties are close for all three ear protectors. Thus, differences in autonomic responses must be due to the different noise levels in the lower and medium frequency range. While the exposure to F-104 noise without ear protectors is within the area of potential noxious effects (Fig. 3), the proper use of the ear protectors reduces the exposure to the area of only possible autonomic responses. All amplitude reductions as compared to pre-exposure amplitudes are significant at the 0.001 level as well as the differences without-with ear protectors. The protective effects of CCM-FIT and WILSON are essentially the same and both are significantly better than those of SELECTONE K. More important than the short-term initial effects are those towards the end of the 20 sec noise exposure period, since they indicate the constant response level for noise exposures of longer duration. Here we can safely state, that with CCM-FIT and WILSON the responses return to pre-exposure values during noise exposure, whereas with SELECTONE K a small vasoconstriction will still remain. The short-term initial response may be due to a certain startling effect at the rapid onset of noise. This opinion might be supported by the appearance of marked electrodermal response without ear protectors and a considerable decrease of this response by the use of WILSON, CCM-FIT and finally SELECTONE K. The standard deviations for the EDR are much larger, which makes the measurement of finger pulse amplitudes a more reliable parameter.

There was no fixed intraindividual relationship between the two test parameters inasmuch as under identical conditions strong responses in one parameter did not necessarily mean strong responses in the other one.

ACKNOWLEDGEMENT

Thanks are due to Dr. VITZ for compilation and evaluation of statistical data and to Mr. KIENER for his assistance and guidance in the preparation of the English version. Both are assigned to our Institute.

SECTION 7

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Prof. Sanford E. Gerber, Anthony Mulac and M. Elisa Lamb	<u>Institution and address where research was performed</u> Department of Speech, University of California, Santa Barbara, Calif. 93106
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> University of California
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Citation | Gerber, S.E. et al. The cardiovascular response to acoustic stimuli. *Audiology* 16: 1-10, 1977.

# of Ref.'s 18	# of Fig.'s 5	Language English
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<u>Type & duration of experiment</u> Laboratory experiment in sound-shielded room. short term.	<u>Purpose for study</u> To test the validity of the cardiovascular response to noise and to see if this response varies with the sound pressure level.
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Description of test groups (subjects, age, etc.)
 | group: 14 women, 21 - 23 years of age, all not menstruating at time of experiment. No separate control group. Subjects tested during chair-rest and requested to make no overt responses to the noise stimuli.

<u>Control of other stressors</u> laboratory conditions used - no other stressors present	<u>Statistical Methods</u> 3-way analysis of covariance for heart rate changes with sound level and under signal and non-signal events
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<u>Noise Stimulus</u> source: introduced through ear phones. spectral characteristics: Narrow band noise centered at 1000 Hz (graph included) noise level: 20, 40, 60, 80 dB-in short bursts (signals) length of exposure: 1 second per signal # of trials: Each trial: 4 bursts of noise (1 sec. each) 1 interval of quiet at a random point in the trial with 30 sec. inbetween.	<u>CVS Response Measured</u> heart rates-pre-and post-stimulus-Hewlett-Packard Heart Rate Finder Finger tip pulse-pulse vol. transducer <u>Nonauditory effects</u> finger-tip pulse-graph included; during noise signal, there was a significant CVS: deceleration in the heart rate and then a return to the pre-stimulus level; individual differences in response were significant Other: Not measured
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Author's conclusions
 The heart rate response is independent of the sound pressure level and is monophasic-deceleration than audiometric method.

Evaluation & comments |
 A separate control group would have made the results more meaningful, since normal heart rate changes during quiet could have been compared to that during exposure to short bursts of noise.

Gerber, S.E. et al. The cardiovascular response to acoustic stimuli. *Audiology*. 16: 1-10, 1977.

The use of a cardiovascular response (heart rate change) to noise as an audiometric method was evaluated in 14 nonmenstruating women with normal hearing, aged 21-23 years. The pre-stimulus heart rates of the subjects were compared to the post-stimulus rates, such that the subjects served as their own controls. The subjects were tested while seated quietly in a chair in a sound-shielded room. The noise stimuli consisted of bursts of narrow-band noise (signals) centered at 1000 Hz and dropping 22 dB/octave (a graph is included). Noise signals of 20, 40, 60, and 80 dB, lasting for 1 second each, were introduced through earphones. Each trial consisted of 4 one second bursts of noise (signals) at a given sound level interspersed randomly with 1-second intervals of quiet periods (non-signal), lasting for the same total time as the noise signals. The 4 bursts and 4 quiet periods were presented in 5 different arrangements at each sound pressure level consecutively, beginning with the 20 dB as follows:

- 1) 4 bursts 20 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules;
- 2) 4 bursts 40 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules;
- 3) 4 bursts 60 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules;
- 4) 4 bursts 80 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules.

The finger tip pulse volume was continuously monitored during the tests. The heart rates averaged over 3 pulse cycles were measured with a Hewlett-Packard heart rate finder for all signal and non-signal events. A three-way analysis of covariance with the pre-stimulus heart rate as the covariant was used to analyze the data. Significant heart rate differences were found between the signal (noise) and non-signal (quiet) conditions. The differences in heart rates were independent of the noise level (an all-or-none response). The heart rate response to the noise signals was monophasic--an early deceleration after the noise burst and a gradual return to the pre-stimulus rate. The pre-stimulus rates were from 53 to 110 beats/minute with a mean of 79.6 beats/minute and a standard deviation of 11.2 beats/minute. The mean decrease in heart rate following the noise bursts was -1.46 beats. Significant differences in the heart rate responses were also found between subjects due to noise. The authors conclude that the cardiovascular response to noise is a valid audiometric method, although significant individual differences in responses are encountered.

The Cardiovascular Response to Acoustic Stimuli¹

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University of California, Santa Barbara, Calif.

Key Words. Cardiovascular · Heart rate response · Autonomic response

Abstract. The purposes of this investigation were to determine whether adults display alterations of cardiac rate under acoustical stimulus conditions and whether such alterations are influenced by signal level. The stimulus consisted of a narrow band of noise centered at 1 000 Hz and presented at 20, 40, 60 and 80 dB SPL. The stimulus was found to produce alterations of heart rate significantly different from variation under non-stimulus conditions, indicating that cardiovascular responses occurred. However, the responses themselves were unaffected by differences of sound pressure level.

Electrophysiological audiometry employs responses to acoustic stimulation manifested by observable changes of some physiological property of the subject, while behavioral audiometry requires an overt bodily reaction. One would choose electrophysiological procedures for the difficult-to-test patient: an infant, a multiply handicapped person, a severely retarded person. Of the electrophysiological procedures, cardiovascular response audiometry may be preferable because (unlike some others) it does not require a noxious stimulus, nor does it necessarily assume an intact central nervous system. However, the properties of the cardiovascular response are not well understood; and, until they are, its utility as an audiometric method must be viewed with caution.

Attempts have been made to assess the cardiovascular response as a means of measuring hearing sensitivity, although heart rate has been only one of

¹ An abbreviated version of this paper was presented to the annual convention of the American Speech and Hearing Association meeting in Las Vegas, Nev., November 1974.

several autonomic responses investigated. Unfortunately, the heart rate response itself has been defined in a variety of ways, leading to discrepancies in the reported forms of the response. A definition is needed which accounts for the differences in cardiovascular behavior between stimulus and non-stimulus conditions since, in order for a change of cardiovascular rate to be considered a response to an environmental stimulus (rather than the normal variation of heart rate), it must be significantly different from those changes which occur in the absence of external stimulation. The difference between stimulus and non-stimulus conditions may be assessed by comparing heart rate changes under these two conditions when other variables are controlled. However, in investigations of the effects of signal intensity, this non-stimulus condition has rarely been included [e.g., DAVIS *et al.*, 1955; UNO and GRINGS, 1965] with the notable exception of ZEAMAN and WEGNER [1956] who claimed that they could distinguish responses to suprathreshold signals from responses to signals '48 dB below threshold' [sic]. The failure to include a non-stimulus condition makes it difficult to assess the discrepancy between UNO and GRINGS' [1965] finding that the magnitude and latency of the response varied with signal level and SMITH and STRAWBRIDGE's [1968] finding that no significant differences occurred for two signal levels 40 dB apart.

Similarly, disagreement about the nature of the heart rate response and its measurement has resulted in different findings regarding the form of the response.² DAVIS *et al.* [1955] measured pressure pulse, volume pulse, and interpulse interval as responses to acoustic and tactile stimuli, and found a biphasic response consisting of an initial decrease of interval (i.e., increase of rate) with an accompanying decrease of pressure, followed by increases of pressure and interval which exceeded the pre-stimulus values. However, HOGAN [1970] found a monophasic response having the form of deceleration of rate followed by recovery to pre-stimulus level, but not beyond. Such discrepancies may have been due to a failure to clearly state what stimuli were employed, what response intervals were used, what measurements were made, and what controls were employed.

Finally, the validity of much of this research is questionable in light of LACEY's [1956] work on quantifying the heart rate response through studying the influence of the pre-stimulus rate upon ensuing cardiovascular behavior. He adjusted for the law of initial values [WILDER, 1950] through regression

² The monophasic form of response is defined as a deviation in either direction without returning to or beyond the pre-stimulus rate; a biphasic response varies above and below the pre-stimulus value.

analysis, arguing that, since the response is dependent upon the pre-stimulus rate, comparison cannot properly be made among different response events without taking into account the pre-stimulus rate. It follows that a similar procedure is essential for the evaluation of response validity.

Clearly, controversy exists regarding the parameters of the heart rate response of adults to auditory stimulation. Studies which have incorporated control conditions of some type have seemed to indicate that the heart does respond to environmental acoustic events, but that the properties of this response as reported are as varied as the research designs which have purported to measure them. Especially confusing have been the results of the effects of signal intensity. The purposes of the present investigation were to assess the validity of the acoustic cardiovascular response and to determine whether that response is affected by sound pressure level.

Method

In an effort to control for the confounding variables discussed above, the following steps were taken: (1) changes of heart rate following signals were compared with heart rate changes following randomly placed periods of no stimulation; (2) subjects were included as a factor in the experimental design to assess individual differences of pattern of heart rate change; (3) to control for the law of initial values, pre-stimulus heart rate was used as a covariate to analyze post-stimulus heart rate change.

Subjects. 14 females between the ages of 21 and 23 years (mean = 22.4) served as subjects. All reported negative otological histories and passed a 15 dB HTL [ANSI, 1969] screening test on the experimental stimulus. Because of the possibility that physiological changes accompanying menstruation affect hearing [GAZEN, 1972], all subjects were scheduled at other times of the month.

Stimuli. Signals selected as test stimuli were bursts of narrow-band noise centered at 1000 Hz and dropping 22 dB/octave (fig. 1). These were presented diotically through earphones with each burst having a stimulus rise time of 0.5 ms and a duration of 1 s. 4 signal levels were used: 20, 40, 60, and 80 dB above 20 $\mu\text{N}/\text{m}^2$ (20 μPa). A 5th test condition was a 1-second interval in which no signal was presented. Hence, the stimulus conditions were 4 bursts of noise and 'blanks', arranged in 5 different presentation schedules in which the blank condition occurred as frequently as the combined signal conditions. The non-signal events were distributed randomly among the signal stimuli which occurred in a prescribed order; each condition was presented 5 consecutive times in order of increasing intensity. The test was begun with 5 presentations of 20 dB bursts, with blank events randomly intermixed. Furthermore, the random insertions of the non-signal events were different for each of the 5 presentation schedules assigned to subjects; all schedules except 1 were used 3 times.

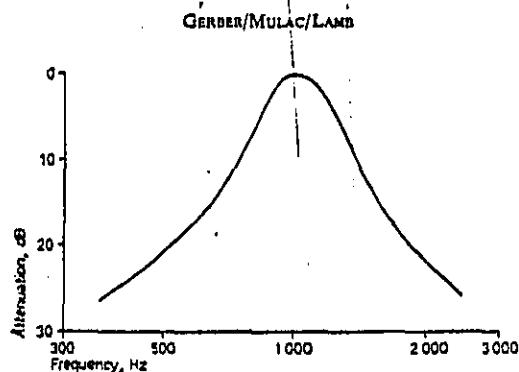


Fig. 1. Stimulus envelope.

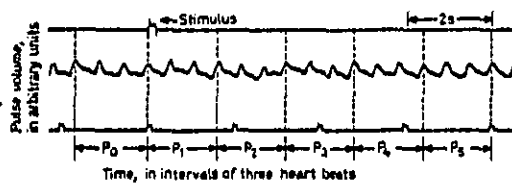


Fig. 2. Sample polygram of transduced pulse volume showing measurement intervals.

Recording. Testing was conducted in a darkened, sound-shielded room (General Acoustics Corp.). Subjects were seated with one wrist taped to a table. The finger tip pulse volume transducer (Gilson FP-6) and earphones (Telephonics TDH-39) were placed. Subjects were instructed to avoid overt responses to the signals and to refrain from unnecessary movement. During all phases of the testing, subjects remained awake.

The output from the pulse volume transducer was recorded on a polygraph (Gilson M5) at a writing speed of 25 mm/s. An event marker recorded signal and non-signal presentations on the polygraph paper (fig. 2).

Measurement. Heart rate measurement was done in the manner suggested by Lewis [1971] and used successfully in our laboratory [Goldstein, 1972; Gerber, 1973; Gerber, et al., in press]: the onset of each stimulus event marked the point from which the pre-

stimulus and 5 post-stimulus heart rate measures were taken. A Hewlett-Packard heart rate finder was employed to measure the heart rate averaged over 3 pulse cycles. (It should be noted that this method of analysis could obscure any biphasic response occurring during the first post-stimulus interval.) As figure 2 demonstrates, the pre-stimulus rate was determined by measuring backward from the pulse peak nearest signal onset. This defined the interval called P_0 . The 5 post-stimulus heart rates were determined in the same way measuring in beats per minute forward from signal onset, and were called P_1 - P_5 . These measures were made for every signal and non-signal event, with events separated by thirty seconds. Heart rates were then converted into change scores from pre-stimulus rate to each post-stimulus rate. In other words, the heart rate changes between P_0 and P_1 , P_0 and P_2 , ... P_0 and P_5 were computed for analysis.

Data analysis. Two separate data analyses were employed, each utilizing a three-way analysis of covariance with repeated measures [WINER, 1962] in which the covariate was pre-stimulus heart rate (P_0) before each signal or non-signal event. The efficacy of regression analysis to control for the law of initial values [WILDER, 1950] was suggested by LACEY [1956]. This statistical procedure assesses the prediction of post-stimulus rates from pre-stimulus rates. A practical application of this principle of controlling for the law of initial values is found in analysis of covariance. This permits the adjustment of post-stimulus rates to the extent they are related to pre-stimulus rates, and has been employed for this purpose in previous studies [BENJAMIN, 1963; GERBER *et al.*, in press].

Changes from P_0 to other time intervals (P_1 , P_2 , ... P_5) were represented as signed change scores in beats per minute. Subjects and post-stimulus measurement intervals were also evaluated as potential sources of variation. For each of the two covariance analyses, there were 14 subjects and 5 time intervals. In the first analysis, possible differences among the 4 intensity levels were investigated; in the second analysis, the 4 signal levels combined were compared to the non-signal events in order to assess the validity of the cardiovascular response to acoustic stimuli.

Results

Subject pre-stimulus heart rates ranged from 53 to 110 beats/min, with a mean rate of 79.6 and a standard deviation of 11.2 beats/min. These initial heart rates for each test event served both as P_0 for computation of change scores for that event and as the covariate score for that event.

Effect of Intensity

Results of the three-way analysis of covariance (14 subjects \times 4 intensities \times 5 time intervals) on data from the signals of 4 intensities (table I) indicated the following: (1) signal intensity was *not* found to be a significant source of variance; (2) the subjects themselves were found to be a source of variance ($p < 0.001$); (3) post-stimulus change across time intervals (response latency) was also seen to be a source of variation ($p < 0.001$); (4) no interactions were

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Table 1. Results of three-way analysis of covariance¹ of heart rate change scores for 4 signal intensity levels

Source of variation	SS	d.f.	MS	F
<i>Between subjects</i>				
A (subjects)	7 305.31	13	561.95	9.65*
Subj. w. groups	3 202.48	33	58.23	
<i>Within subjects</i>				
B (intensity)	190.90	3	63.63	1.03
AB	4 518.62	39	115.86	1.88
B × subj. w. groups	10 186.34	165	61.74	
C (time interval)	406.96	4	101.74	5.48*
AC	1 221.01	52	23.48	1.26
C × subj. w. groups	4 083.43	220	18.56	
BC	360.07	12	30.01	1.59
ABC	2 669.54	156	17.11	0.90
BC × subj. w. groups	12 487.08	660	18.92	

* $p < 0.001$.

¹ Pre-signal rate was used as the covariate.

found among the 3 independent variables. The lack of subject × time interval interaction indicated that the post-stimulus pattern of change did not differ among individuals over time; the lack of intensity × time interactions showed that the time of maximum heart rate change was unaffected by signal level.

The possibility that rapid habituation within each stimulus level might have masked a differential response to the 4 intensity levels was assessed through a separate two-way analysis of covariance with repeated measures. Heart rates following the first presentation of each intensity level were the data for this 4 (intensities) × 5 (time intervals) analysis. Results failed to show significant differences in heart rate changes for the 4 intensities ($p > 0.10$).

Response Validity

Since the first analysis of covariance revealed no difference as a function of signal intensity across signal events, these data were grouped for comparison with the non-signal events. In this second analysis of covariance (14 subjects × 2 signals × 5 time intervals), a statistically significant difference

Table II. Results of three-way analysis of covariance¹ of heart rate change scores for signal and non-signal events

Source of variation	SS	d.f.	MS	F
<i>Between subjects</i>				
A (subjects)	15 507.39	13	1 192.88	18.98*
Subj. w. groups	16 654.51	265	62.85	
<i>Within subjects</i>				
B (signal)	972.40	1	972.40	14.27*
AB	2 247.09	13	172.85	2.54
B × subj. w. groups	18 055.59	265	68.13	
C (time interval)	364.52	4	91.13	5.40*
AC	1 114.95	52	21.44	1.27
C × subj. w. groups	17 885.03	1 060	16.87	
BC	124.47	4	31.12	1.66
ABC	889.13	52	17.10	0.91
BC × subj. w. groups	19 851.93	1 060	18.73	

* p < 0.001.

¹ Pre-signal rate was used as the covariate.

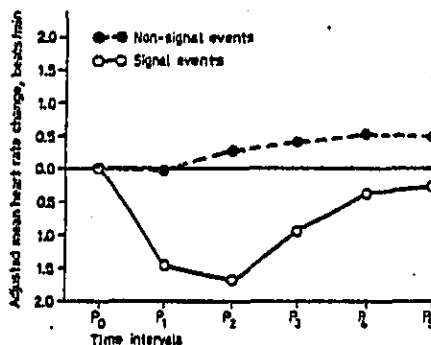


Fig. 3. Mean heart rate changes (adjusted for pre-stimulus rates) under signal and non-signal conditions.

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between these two signal conditions was taken as an indication of heart rate response to acoustic stimuli as distinguished from normal variation.

Results of this analysis (table II) indicated the following: (1) a significant difference was found between signal and non-signal events ($p < 0.001$). The signal response was monophasic, with deceleration followed by return toward the pre-signal rate (fig. 3); (2) subjects were again found to be a significant source of variance ($p < 0.001$); (3) time interval was again a significant source of variation ($p < 0.001$); (4) no interactions were found among the 3 independent variables.

Discussion

We set out to investigate the validity of the cardiovascular response to sound in adults and to assess the influence of signal level upon it. This study differed from previous research in that the validity of the response was itself a major issue. The data indicated that cardiovascular rate changes constitute a valid response to acoustic stimuli of the type tested here. Further, the response to the stimuli appears to be of the 'all-or-none' type since it was not differentially affected by signal level. Also, we observe that the response is monophasic, beginning with early deceleration after stimulus onset followed by gradual recovery toward the pre-stimulus rate. We pointed out earlier that the analysis of heart beats in groups of 3 could obscure a biphasic response occurring within the first post-stimulus interval. In light of the magnitude of the decelerative response observed during that time interval (Mean = -1.46 beats), it appears unlikely that an acceleration of sufficient size occurred during the first post-stimulus beat to make this hypothesis tenable.

These conclusions support the earlier results of SMITH and STRAWBRIDGE [1968] who found no differences of cardiovascular rate variation as a function of signal level. They employed a range of 40 dB, while we used a 60 dB range; however, in neither case was the customarily defined threshold of hearing assessed. While we have found that the cardiovascular response to sound is of the 'all-or-none' type, we do not yet know how close to threshold it may be elicited, but it must be less than 20 dB. In an earlier study at our center, AFFLERBAUGH [1971] was unable to evoke cardiovascular responses from adults at the previously determined audiometric threshold. Therefore, we conclude that the response may be evoked somewhere near to, but above, threshold; and that it is not altered by further increases of sensation level

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through 80 dB. Also, our results indicate that the form of the response at these levels is consistent with reports [e.g., CLIFTON and MEYERS, 1969] that the response of a subject past the age of early infancy takes a monophasic form of deceleration followed by return toward the pre-stimulus rate. However, this finding is contrary to the observations of DAVIS *et al.* [1955] that acceleration occurs first.

Our statistical analyses of the data lead us to believe that the test method employed herein may have audiometric utility; nevertheless, one must use caution in its clinical application. While we may have determined the form of the response in normal subjects as a group, we have no assurance that any given subject will display exactly the same response. However, our finding of no subject \times time interaction does suggest similarity of subjects' response patterns.

The present 'state of the art' is such that we have many unanswered questions. EISENBERG [1974] reported encouraging findings similar to our own. She hopes, as do we, that the cardiovascular response to sound may turn out to be independent of the age and state of the subject. However, we do not yet know this to be the case. For example, we are now addressing the question of the effect of intrathoracic pressure in adults upon cardiac responses to sound. Certainly, variations of intrathoracic pressure do influence heart rate; we want to determine whether they influence heart rate response to acoustic stimuli. We also want to know at what age the response assumes its apparent monophasic form. We have found that no such form exists in neonates [GERBER *et al.*, in press], but that it begins to emerge quite early during the first year of life [GOLDSTEIN, 1972].

The question of the efficacy of cardiovascular response audiometry is both clinically important and conceptually interesting. While we agree with EISENBERG [1974] that use of the cardiovascular response may lead to fool-proof measures of hearing adequacy, and are encouraged by our findings, we remain cautious as to their implications.

Résumé

Les buts de cette recherche étaient de déterminer si les adultes présentent des changements de la vitesse des battements cardiaques lorsqu'ils reçoivent des stimulus acoustiques, et si ces changements sont influencés par le niveau du stimulus. Le stimulus était un bruit à bande étroite, centré sur 1000 Hz, et il était présenté avec 20, 40, 60 et 80 dB de pression acoustique. On a pu observer que le son produit des changements dans la vitesse des battements cardiaques significativement différents, en comparaison des changements sur-

venant en dehors des stimulations sonores; ces résultats montrent l'existence de réponses cardio-vasculaires. Cependant les réponses n'étaient pas affectées par les différences de niveau de pression acoustique.

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SECTION 8

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Barbara Griefahn (Dr. Med.)	<u>Institution and address where research was performed</u> Arbeitsgruppe Exp. Arbeitsmed; Institut fuer Hygiene und Arbeitsmedizin im Universitaetsklinikum der Gesamthochschule Essen; D-4300 Essen Hufelandstr, 55 Federal Republic of Germany
<u>Investigator's Phone No.</u>	

Citation | Griefahn, B. Effects of sonic booms on fingerpulse amplitudes during sleep. Int. Arch. Occup. Environ. Health. 36:57-66, 1975

<u># of Ref.'s</u> 17	<u># of Fig.'s</u> 5	<u>Language</u> English
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<u>Type & duration of experiment</u> laboratory--in test chamber 2 parts to the experiment 1: 19 nights; 2: 53 nights	<u>Purpose for study</u> to test fingerpulse amplitude responses to sonic booms with respect to boom intensity, sleep stage, preboom pulse rate, temp., length of experiment, length of quiet time before booms
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Description of test groups (subjects, #, age, etc.)
 2 persons for Part 1 subjects served as their own controls during nights with no sonic booms, which were as follows: Part 1: 3 nights--quiet; 11 nights--booms; 6 nights, quiet. Part 2: 3 nights--quiet; 30 nights--booms; 10 nights--quiet; 4 nights--booms; 6 quiet

<u>Control of other stressors</u> laboratory conditions used temp. varied 17° C -23; 50 C	<u>Statistical Methods</u> standard deviation; correlation coefficients
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<u>Noise Stimulus</u> source: sonic boom--reproduced through sonic boom channel into test chamber spectral characteristics: not given noise level: 83.5 dBA average length of exposure: 300 msec. per boom between 10:30 PM and 3 AM # of trials: 2, 4, 8, or 16 booms per night	<u>CVS Response Measured</u> fingerpulse amplitude (recorded continuously) <u>Nonauditory effects</u> the fingerpulse amplitudes decreased within CVSt; 3 sec. after the onset of the sonic booms followed by a return to the pre boom value; the higher the preboom pulse rate, the smaller the pulse rate reaction Other: EEG used to monitor sleep stages
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Author's conclusions | The decrease in fingerpulse amplitude was not correlated with sonic boom intensity as is generally observed, probably due to the short rise time of the booms. The only variable for which a significant relationship was found was the preboom pulse rate. As the law of initial values states, the greater the preboom pulse rate, the lesser the change in pulse rate due to noise.

Evaluation & comments | The small number of subjects is a weak point. No description (age, sex, health) of test subjects is given.

Griefahn, B. Effects of sonic booms on fingerpulse amplitudes during sleep. Int. Arch. Occup. Environ. Hlth. 36:57-66, 1975.

The fingerpulse amplitudes during sleep of 4 human subjects were studied after sonic booms of varying intensities between 0.48-1.45 mbar (1 mbar or 83.5 dBA on the average). The experiments were conducted while the subjects slept in a test chamber between 10:30 p.m. and 3 a.m., during which the sonic booms were introduced through an opening in the chamber. The effects of the following variables on the fingerpulse amplitude response to noise were studied: preboom pulse rate, sleep stage (monitored by EEG), interval between booms, duration of experiment, and ambient temperature (varied from 17°C to 23.5°C). The experiment was divided into two parts with two subjects in each. Part 1 consisted of 19 nights as follows: 3 nights quiet; 11 nights with 2 or 4 sonic booms; 6 nights quiet. Part 2 consisted of 53 nights as follows: 3 nights quiet; 30 nights with 2 or 4 sonic booms; 10 nights quiet; 4 nights with 8 or 16 sonic booms; 6 nights quiet. The interval between booms varied with the number of booms: 2 booms -40 min. interval; 4 booms -20 min.; 8 booms -3.6 min.; 16 booms -4.6 min. Each sonic boom lasted for 300 msec. The fingerpulse amplitudes decreased within 3 sec. after the onset of the sonic boom, reached a minimum at 8 sec., and then increased toward the preboom value. The intensity of the sonic booms had no effect on the degree of fingerpulse amplitude response, which is contrary to previous experiments in which increased intensity of noise is correlated with a greater decrease in amplitude. The rise time of the sonic boom may have been too short to allow for this response. The only variable studied that affected the fingerpulse amplitude responses to the sonic booms was the preboom pulse rate. The greater the preboom pulse rate was, the smaller the decrease in amplitude (the law of initial values).

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Effects of Sonic Booms on Fingerpulse Amplitudes during Sleep

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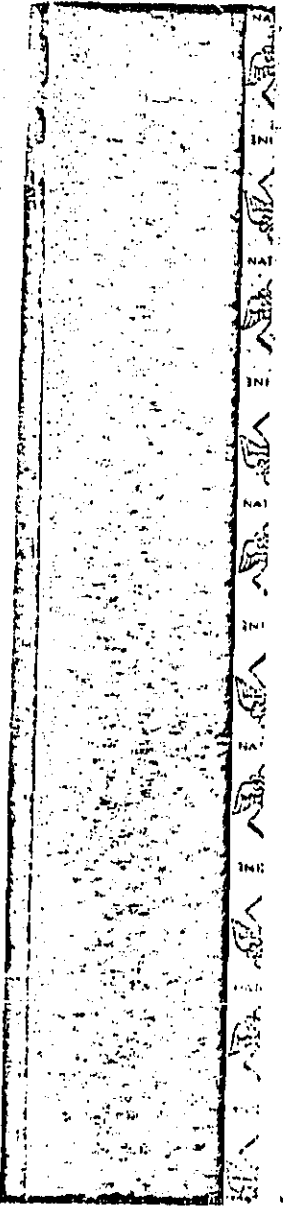
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Summary. In two experimental series (19 53 nights, respectively, in 2 different persons in each series; testtime 10.30 p.m. to 3.00 a.m.) fingerpulse amplitudes after sonic booms were recorded during sleep. In the first 3 nights the subjects slept undisturbed by noise. In the following 11 and 30 nights, respectively, sonic booms were applied alternately 2 or 4 times. In the main series, after 10 more nights without noise, 4 nights with 8 and 16 sonic booms alternately followed. The last 6 undisturbed nights in both series were used as a comparison phase. The interval between two sonic booms was 40 min in nights with 2 booms, 20 min in nights with 4 booms and in the nights with 8 and 16 sonic booms 8.6 and 4.6 min, respectively. Sound level of the sonic booms ranged from 0.48 to 1.45 mbar, 1 mbar (83.5 dB[A]) on the average. The first sonic boom was applied if one of the two subjects had entered the deepest stage of sleep.

Sonic booms induced a decrease in fingerpulse amplitudes, which begin 3 sec after the onset of the stimulus. The minimum occurs after 8 sec, followed by an increase towards the preboom value. The reduction of the fingerpulse amplitudes is very significant ($P < 1\%$) from the 4th to the 22nd sec. This reaction was analyzed with special regard to the following factors:

1. Intensity: Due to the very short rise time of noise intensity there was no significant correlation between the maximum intensity of each boom and the decrease of fingerpulse amplitudes.
2. Exogenic variables: There are no significant connections between post-boom fingerpulse amplitudes and noiseless time before the sonic boom, the duration of the test series, and the ambient temperature.
3. Endogenic variables: No correlation could be found between the stage of sleep and the reaction. On the contrary, a very significant correla-

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tion was found between the reduction of fingerpulse amplitudes and the pulse rate before boom. With increasing pulse rate the extent of reaction becomes smaller.

Key words: Sleep - Sonic booms - Moderator variables - Fingerpulse amplitudes.

INTRODUCTION

Different reactions within the human organism are induced by acoustical stimulation. The vegetative centers of the brain stem are stimulated via the vestibulocochlear nerve and the formatio reticularis. The resulting ergotropic reactions are, for instance: increase of respiration frequency, peripheral resistance, and metabolic rate, decrease of cardiac output, hand volume, and hand temperature as well as dilatation of the pupils. Thus, application of noise leads to numerous reactions of different vegetative functions. The extent of these reactions is dependent on intensity and band width (Corbeille & Baldes, 1929; Lehmann & Tamm, 1956; Oppliger & Grandjean, 1959; Jansen & Rey, 1962; Damsky, 1965).

One of the most simple indicators (in application and evaluation) of these ergotropic changes is the recording of fingerpulse amplitudes (FPA). Acoustical stimuli induce a great decrease of amplitudes ("on-reaction"), which is - when stimulation remains - followed by a steady value beneath the initial value. As a rule fingerpulse amplitudes begin to increase toward the initial value after the end of stimulation.

Because of the fact that acoustical stimuli cause alterations of the vegetative situation, these reactions consequently must be dependent on the vegetative situation immediately before stimulation. Due to the law of initial value (Wilder, 1931) the ergotropic reaction becomes smaller with increasing ergotropy. This law had been proved in several noise investigations such as, for instance, by Heinecker & Zipf (1960) as well as by Griefahn (1974), who recorded the changes of fingerpulse amplitudes.

Short noise exposures only induce on-reactions. In the investigation described here the alterations of fingerpulse amplitudes due to sonic booms (300 msec) had been proved as well as their dependency on the different moderator variables (intensity of sonic boom, quiet time before sonic boom, duration of test series, ambient temperature, sleep stage, and pulse rate).

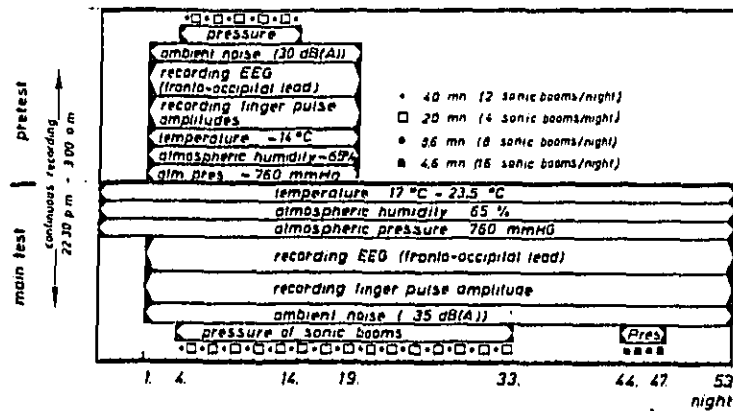


Fig.1. Experimental arrangement

evaluation of sleep stages

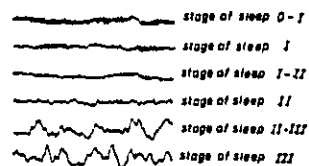


Fig.2. Evaluation scheme of sonic boom experiments

METHOD

Fig.1 presents a survey of the arrangement and the duration of both test series as well as of the recorded physiologic and physical parameters. Two subjects slept in the test room which had an opening to the sonic boom channel (ISL report). During the entire test time (10.30 p.m. - 3.00 a.m.) EEG and fingerpulse amplitudes (FPA) were recorded continuously. The first sonic boom was applied as soon as one of the two subjects had reached the deepest sleep stage and the other subject had reached at least stage I. The pressure of the sonic booms applied in this investigation had the typical N-shape, the duration was 300 msec, the intensity ranged from 0.48-1.45 mbar, 1 mbar on the average.



EVALUATION

Because of several artefacts in the curves (body movements, etc.) only 261 single tests were evaluated. This evaluation was done in the following way:

Sleep Stages. Evaluation of sleep stages according to Fig.2; the sleep stage within the minute before stimulation was considered an endogenic moderator.

Preboom Value of the FPA. Determination of the maximum amplitudes of the last 10 pulse waves preceding the sonic boom with an accuracy of 0.1 mm.

Postboom Value of the FPA. Until the 30th postboom sec the maximum amplitude of each single pulse wave and its distance to stimulus onset was measured. Related to the preboom average the maximum amplitudes were converted into percentage values. The distances from stimulus onset up to the maximum amplitude (measured in mm) had been converted into seconds. These distances had been classified into 0.2-sec-distances; each amplitude within the time 1.31 and 1.50 sec had been considered as if it had occurred at 1.4 sec.

STATISTICS

Calculation of averages and standard deviations in 0.2-sec- or 1.0-sec-distances.

Calculation of the FPA parameters out of each single curve (Table 1).

Calculation of the correlation coefficients between FPA parameters and intensity as well as all other exogenic and endogenic moderator variables.

Calculation of the partial correlation with exclusion of the five other influences.

Calculated values were considered as significant only if $P = 1\%$. Because of the very small number of subjects (2 in each test) it was necessary to prove these significances for several subgroups (each subject of the main test in nights with 2 and 4 booms, in nights with 8 and 16 booms, and for the subjects of the pilot study). Only if these values were considered significant, also was a value described as significant in this study.

RESULTS

Fig. 3 demonstrates the course of the fingerpulse amplitudes after a sonic boom. Three sec after stimulus onset amplitudes.

Table 1

Parameters calculated from each single curve and moderator variables

1) FPA parameters

- A = distance between onset of stimulus and minimum of fingerpulse amplitudes (sec)
- B = minimum of fingerpulse amplitudes (%)
- C = 14-sec-value of fingerpulse amplitude
- D = 18-sec-value of fingerpulse amplitude
- E = 22-sec-value of fingerpulse amplitude
- F = ascent between 3-sec-value and minimum
- G = ascent between minimum and 22-sec value
- R = angle above the minimum
- S = integral (beneath 100% value)
- T = average of fingerpulse amplitudes (4-22 sec in %)
- U = standard deviation (%)
- V = deviation from 100% value without consideration of the direction (%)

2) Moderator variables

- N = intensity of sonic boom (mbar)
- Q = quiet time before sonic boom (min)
- P = experimental night
- R = ambient temperature (°C)
- L = sleep stage (preboom)
- M = preboom pulse rate

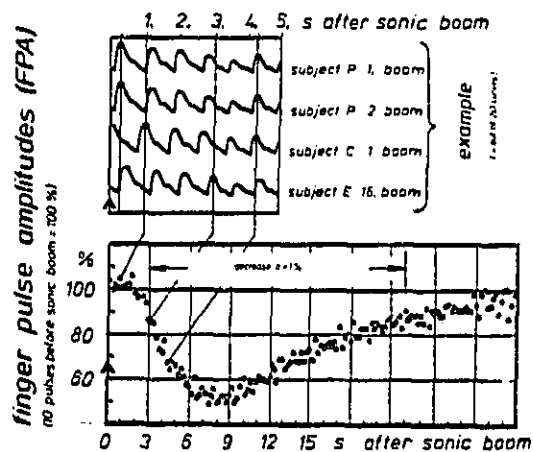
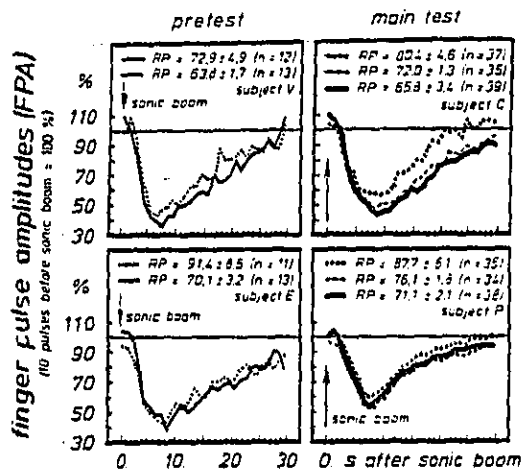


Fig. 3
Decrease of finger-pulse amplitudes after sonic booms during sleep

sonic boom intensity 0.48 - 1.45 mbar (2-1 mbar), duration 300 ms;
 2, 4, 8 or 16 sonic booms/night;
 interval between 2 sonic booms 40 min, 20 min, 8.6 min or 4.6 min;
 sleep stages 0-III (0:awake, III: deep sleep)
 4 subjects, 261 sonic booms.



sonic boom intensity 0.42 - 1.45 mbar (0-1 mbar), duration 200 ms.
 2, 4, 8 or 16 sonic booms / night.
 interval between 2 sonic booms 40 min, 20 min, 85 min or 1.5 min.
 sleep stages I-III (I awake, III and sleep)
 RP = average pulse rate (per minute) before stimulation

Fig. 4. Decrease of fingerpulse amplitudes after sonic booms due to different preboom values

begin to decrease; the minimum occurs after 8 sec followed by an increase towards the preboom value. After the 22nd sec the decrease of amplitudes is no longer significant ($P = 1\%$), so that all other calculations were done from the 4th until the 22nd sec.

The minimum duration of the pulse waves was 0.57 sec, so that at least 3 successive values of the demonstrated curve (distance 0.2 sec) were calculated from different experiments.

1. Intensity

Between FPA parameters and intensity of sonic booms (0.42-1.45 mbar) no correlation was found.

2. Exogenic Moderators

There was no significant connection between FPA parameters and exogenous influences [interval between sonic booms (quiet time before stimulus), duration of test series, ambient temperature]. For one single experimental night it was not possible to calculate significant correlations.

3. Endogenic Moderators

The sleep stages had no influence on the extent of the reaction.

Very significant was the connection between the preboom pulse rate and the FPA parameters C, D, and E [14-, 18-, and 22-sec values, coefficients (partial): 0.340, 0.351, 0.292, $df = 256$].

Therefore the curves of FPA after sonic booms were presented in Fig.4 due to the level of pulse rate before stimulation (RP). The decrease of amplitudes is greater with decreasing pulse rates.

DISCUSSION

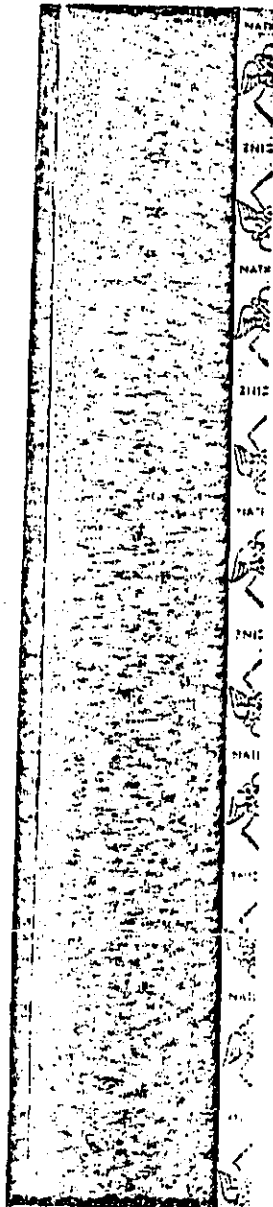
Three sec after the onset of sonic booms, fingerpulse amplitudes begin to decrease. The minimum occurs after 8 sec, followed by an increase towards the preboom value. The reduction of the amplitudes is significant ($P \leq 1\%$) from the 4th to the 22nd sec. The small deviation of the succeeding values gives evidence of the high reliability of these results.

1. Intensity

As pointed out by Jansen & Rey (1962) the decrease of fingerpulse amplitudes becomes greater with increasing intensity as well as with increasing band width. According to an increasing number of stimulated nerve fibers that means with an increasing number of impulses per second a greater number of impulses arrive at the vegetative centers in the brain stem causing a stronger reaction. The missed relation between the FPA parameters and the boom intensities in this study may be caused by different factors. As described by Miederhoff (1974) the on-reaction becomes greater and occurs earlier when the rise time of noise pressure decreases. The rise time of the sonic booms is extremely short (5 msec in average) so that the organism may be unable to respond adequately. On the other hand, the range of intensities (80-89 dB[A]) may be too small to cause different reactions (Ludlow & Morgan, 1972). As pointed out by Jansen (1967) the increase or reaction becomes smaller with increasing intensities; the minimum occurs within the range of intensities used in this study.

2. Exogenic Moderator Variables

Quiet Time before Sonic Booms. The relation between the quiet time before sonic booms and the extent of FPA decrease was not significant. This corresponds to the results of Jansen (1974), who applied noises with increasing intensity (ambient noise level - 105 dB[A] within 15 sec) and increasing frequency. These noises, applied at intervals of at least 8 min always



caused the same reaction, whereas an application in less than 8 min caused a smaller reaction, indicating that the initial value is not yet regained. In this study, however, sonic booms with intervals up to 4 min always caused the same reaction. That fact points out that recovery needs less time with increasing vagotony.

Duration of Test Series. Between the duration of the test series and the noise-induced decrease of fingerpulse amplitudes it was not possible to calculate any correlation. These results are in accordance to the results of McDonald et al. (1964) and Johnson et al. (1965), who described a decreasing ability of adaptation in drowsy and sleeping subjects. On the other hand, the intensities applied in this investigation are within a range which only causes defensive reactions, that is reactions independent from habituation (Sokoloff, 1963). This independence could be proved not only for several months but also for several years. Jansen (1971) tested 9 subjects within 2 years, whereas Lehmann & Tamm (1956) examined the reaction of noise workers and students. None of these investigations gave evidence of any adaptation.

Ambient Temperature. Examinations of noise-induced decrease of fingerpulse amplitudes during different levels of ambient temperature have been done by Jansen (1967). Increasing temperature causes a vasodilation so that the same noise becomes less effective. During an ambient level of 40°C and a 95 dB white noise, no reaction could be recorded. In the study described here the range of temperature was possibly too small (5.5°C in the pilot study 6.5°C in the main study) to cause different reactions.

3. Endogenic Moderator Variables

Sleep Stages. In spite of the fact that an increasing depth of sleep is accompanied by an increasing vagotony (Hess, 1933) no correlation could be calculated between the sleep stages and the FPA parameters. Williams et al. (1964), also, could not find any dependency of peripheral vasoconstriction on sleep depth.

The variation of the vegetative situation during sleep is probably not important enough to cause different reactions.

Preboom Pulse Rate. The connection between preboom pulse rate and FPA-parameters (C, D, and E 14-, 18-, and 22-sec value) was highly significant. With increasing preboom, pulse rate reaction decreases. These results are in accordance with the

law of initial value (Wilder, 1931). This law, which has been proved in several investigations demonstrate that the ergotrope reactions become smaller with increasing ergotropy.

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SECTION 9

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

Principal Investigator(s) W. Hawel	Institution and address where research was performed Institut fuer Arbeitsphysiologie an der Universitat Dortmund Abteilung Umwelphysiologie 4600 Dortmund, Ardeystrabe 67; West Germany
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Investigator's Phone No.	Sponsoring Organization Same
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Citation | Hawel, V. Research on the psychological and psychophysiological effects of repeated four-hour intermittent pink noise. Z. Exp. Angew. Psychol. 22(4):613-629, 1975.

# of Ref.'s 28	# of Fig.'s 8	Language Ger. (Eng. Translation)
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Type & duration of experiment 10 weeks: one 4 hour session per week	Purpose for study to study the relationship between performance, pulse rate, catecholamine excretion, and noise
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Description of test groups (subjects, #, age, etc.) | 10 male college students, who served as their own controls: sessions 1-4, 6-9 - intermittent noise given and Kraepelin-Pauli tests taken; sessions 5,10 - control sessions, no noise, no Kraepelin-Pauli tests

Control of other stressors Subjects were subjected to both noise and the performance test.	Statistical Methods Factor analysis; covariance analysis
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Noise Stimulus source: not specified spectral characteristics: pink noise noise level: 90 dB length of exposure: intermittent (30% or 70% quiet) # of trials: 10 (4 noise & quiet)	CVS Response Measured Pulse rate Nonauditory effects CVS: pulse rate - increased due to noise. Other: anxiety - decreased with noise, catecholamine excretion - increased due to noise, task performance - no different due to noise.
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Author's conclusions | No effects of noise on performance of the task was observed, due to the relative easiness.

Evaluation & comments |

1. Detailed experimental methods are given in a previous paper (Hawel and Stralinger, 1967, Int. Z. Angew. Physiol. 24:351-362).
2. Subjects were not studied with noise alone - but were given both a performance test and noise together (2 stressors).

Hawel, W. Research on the psychological and psychophysiological effects of repeated four-hour intermittent pink noise. *Z. Exp. Angew. Psychol.* 22(4):613-629, 1975.

The relationship between task performance, intermittent pink noise, pulse rate, catecholamine excretion (adrenalin, noradrenalin) and anxiety was studied in ten male college students. Each student was tested with various combinations of noise, task performance, and quiet during one four-hour session per week for ten weeks. Pulse rate, catecholamine excretion, and psychological state were monitored for each session. The subjects were given both intermittent noise and intellectual tests in four of the weekly sessions. In another four sessions, the subjects took the tests under quiet conditions. The control sessions in the 5th and 10th weeks, consisted of no noise and no test-taking during which the students could study on their own. The 90 dB pink noise was given intermittently 30-70% of the session. No effects of noise on the performance of the test, which consisted of simple computations, were noted. The effects of noise included increased pulse rate and catecholamine excretion, and decreased anxiety.

RESEARCH ON THE PSYCHOLOGICAL AND PSYCHOPHYSIOLOGICAL EFFECTS
OF REPEATED, FOUR-HOUR INTERMITTENT PINK NOISE.

Dedicated to the honor of Prof. Dr. med. Dr. med. h.c.
Gunther Lehmann.

Zeitschrift für experimentelle und angewandte Psychologie
1975, vol. XXII, numh.4, p. 613-629.

From the former Max-Planck Institute for Work Physiology
Dortmund. Former director: Prof. Dr. h.c. G. Lehmann.
Psychological Department. Former director: Prof. Dr. J.
Rutenfranz and the Institute for Work Physiology at the
University of Dortmund--Department of Environmental
Physiology--Director Prof. dr. med. H.G. Wenzel.

By Wolfgang Havel

Ten male test personnel (students of the Dortmund
institute) took part in a test series lasting for ten
weeks, one test per week, on four hour sittings. In each
session they could work freely at least two hours for
their study. In the sessions 1-4 and 6-9, they had to
carry out the Kraepelin-Pauli work test and alternating
from session to session, they were subjected to periodic-
ally interrupted pink noise (90 dB (lin), 5 sec, 30% on,
70 % off) and then to silence. The pulse frequency was
recorded during the Kraepelin Pauli test. Sessions 5 and
10 were control tests without noise and without Kraepelin-
Pauli tests. The personality variables in question and
the catecholamine precipitation were studied in all sessions.

The Kraepelin-Pauli work test alone gave no indica-
tion of a noise effect, which was interpreted as a result
of the slight difficulty of the tasks. During the free
activity, the test persons got the impression of being able
to work very fast, but with much less concentration than
without the noise. Both test conditions without noise
appeared much more similar to the test persons than each
of these of the noise condition. The influence of the
noise on disposition was considerable. There were less
anxiety symptoms with noise than during quiet.

There were indications for a relationship between
performance, error frequency, pulse frequency and noise.

		(1) Gewöhnung				(2) Versuch						
(3) SitzungNr.:		1	2	3	4	5	6	7	8	9	10	
Vp.Nr.:	1	-	*	-	*	0	-	*	-	*	0	DI (4)
	2	*	-	*	-	0	*	-	*	-	0	
	3	-	*	*	-	0	*	-	*	-	0	MI (5)
	4	-	-	-	*	0	-	*	-	-	0	
	5	*	-	*	-	0	*	-	*	-	0	Do (6)
	6	-	*	-	*	0	-	*	-	-	0	
	7	-	*	-	*	0	-	*	-	-	0	Fr (7)
	8	*	-	-	*	0	*	-	*	-	0	
	9	-	0	-	*	0	-	*	-	-	0	Mo (8)
	10	*	-	-	*	0	*	-	*	-	0	

0. Sid. (9) 2. Sid. (9) 4. Sid. (9) : Zeit (10)

HP-A → HP-A → P-F → "0" : Versuchsart (11)

HP-A → HP-K → P-F → "0" : "0" : "0"

(12) A=Arbeiten für Studium K=KRAEPELIN-PAULI
 F=Fragebogen P=Persönlichkeits-
 H=Horn I=Fragebogen

HAWEL	(13) Versuchsanordnung zur Bestimmung der Catecholaminausscheidung unter Lärm	MPI
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Fig. 1

(1) Acclimatization; (2) Test; (3) Session no.; (4) Tuesday; (5) Wednesday; (6) Thursday; (7) Friday; (8) Monday; (9) Hours; (10) Time; (11) Test type; (12) A = work for study; F=questionnaire; H=horn; K=Kraepelin-Pauli; P=personality questionnaire; (13) Test setup for the determination of catecholamine precipitation during noise.

* The study was carried out and promoted by the assistance of the German Research Society (AZ:Hz 400/6).

A. Korte and A. Hubert assisted in carrying out and evaluating the test.

The EDV was carried out on the IBM 360/44 of the Max Planck Institute for nutritional physiology (Director: Prof. Dr. Benno Hess).

Test setup for determining the catecholamine precipitation amid noise.

The scheme shows the test plan: left, from the top downward, the numbers of the test persons, to the right the days of the week on which they took part in the tests as a rule. Above, the numbers of the sessions, of which the first four can be called acclimatization, not included in the evaluation. The symbols 0, - and + in the fields correspond to the following: 0 = without the Kraepelin-Pauli test, without noise, - = with the Kraepelin-Pauli test, without noise and + = with the Kraepelin-Pauli test, but with noise. It is recognized at once that test type 0 was used in sessions 5 and 10, while both other test types were arranged in such a way that each test person went through each test type one time, and that each of the two test types took place five times in each session number. The course of the different test types is evident from the lower part of the scheme.

A report is made concerning the psychological results of a study whose physiological findings are given elsewhere (Hawel and Starlinger, 1967). A detailed foundation of the test and a thorough description of the test plan and of the test conditions took place there. For this reason the test arrangement is only briefly reported here:

Ten male test persons (students of the Dortmund technical school) took part in a test against a measured recovery in ten successive weeks, mostly on the same day of the week, at the same time of day, for four hours (see Fig. 1). The test conditions varied between "with noise" and "without noise" as well as between "with the Kraepelin-Pauli work test" and as a control condition "without the Kraepelin-Pauli test". These control conditions were without noise and always fell in sessions 5 and 10. In the other sessions, actually one test person was subjected to noise, while the other carried out the test in quiet. The noise was periodically interrupted pink noise of 90 dB (lin) (re $2 \cdot 10^{-4}$ μ b). In the first session 'with noise', there was street noise with decibel peaks from 85 dB (lin). At 5 second cycle time, the noise was interrupted by 30% (seating 2-4) or 70% (seating 6-9) quiet (with reference to the considerations of Teichner et al.). The first four sessions were not taken into account as acclimatization in the physiological study. For the last six sessions we determined an increased adrenalin precipitation during noise in the second half of the session with a 5% probability of error. Also, a tendency toward higher pulse frequency during noise had to be assumed during the Kraepelin-Pauli test.

In weighing a possible psychological effect of long-lasting noise, we posed four questions for ourselves, for the answering of which the study should bring information.

1. Is there an influence on carrying out a simple performance test?

This question was studied in tests of shorter length (among others by Sanders, 1961; Lienert and Jansen, 1964).

2. How will actual performance be judged with the usual intellectual work?

3. Do mood and/or self-image of the test personnel change?

4. Is there a tendency toward anxiety? To study this question we introduced the activation hypothesis discussed in connection with the effect of noise on man (Hörmann and Todt, 1960; Schönplug, 1969). According to it, it appeared not to be ruled out that long-lasting noise caused stimulation which can be accompanied by a tendency toward anxiety behavior (see also Lidberg and Levi, 1969).

1. Performance Test

The Kraepelin-Pauli work test was selected (Pauli-arnold, 1961) in order to obtain data concerning the objective performance capability under both test conditions "-" = "without" and "+" = "with noise". We were presented with a relatively simple task (to add actually two one digit numbers during one hour), which can be arbitrarily repeated and about which a large number of tests prevail (Ulich, 1958; other literature in the case of Christiansen, 1966). The control test without Kraepelin-Pauli and without noise received the symbol "o".

The commands for the Kraepelin-Pauli test ("attention, started!", every three minutes "line!" and at the end "attention" "pencils up") were spoken on a tape and were given over the loudspeaker into the booth. In the case of the sessions with noise, they were superimposed. The test persons were questioned before each test performance-- with a short specimen throughput--whether the commands arrived clearly. (The communication test leader-test person took place over a telephone installation). Their sameness over all the tests was guaranteed by the type of command giving. Each of both used tapes was played 40 times, and its length could be neglected at the end of the test series.

The evaluation of the data from this test (sessions 6, 7, 8 and 9) by means of the covariance analysis showed no difference. (The five values from the individual work curves 'without noise' "-" were taken as independent variables and brought into relationship with the corresponding values of the working curves 'with noise' "+"). Actually, two successive time curves were correlated).

The comparison of the five derived values, total, error percentage, improvement percent, climbing height and peak time--by means of discrimination analysis (Faber and Nollau) gave a difference at the 10% level in the acclimatization phase (sessions 1 to 4), which however could no longer be observed in sessions 6-9. Even a T² test for dependent random tests gave no difference. The mean value vectors are shown in Table 1. The "climbing height" proved to be a problematical measure, because occasionally it was clearly a "sinking depth".

Table 1

The table shows the mean value vectors of the Kraepelin-Pauli work test. To the far left the sessions and beside them the noise conditions about which we are actually reporting.

Sessions	Noise	Total	Error per cent	Improve-ment per cent	Climbing height	Peak time
1-4	with-	3196	4.8	8.0	46.6	8.7
	out					
	with	3186	6.1	5.2	35.3	10.4
	w.o.	3623	7.2	5.4	43.3	7.3
6-9	with	3640	5.7	4.9	48.7	8.3

2. Subjective Judgment of one's own Efficiency

As is thoroughly established in our report concerning the physiological results of our study, the test personnel could study during a part of the available test time. The idea of this was that for the test personnel, the participation in this test series became a part of their overall day and thus lost the character of an exceptional situation. In order to obtain information concerning the subjective judgment of one's own efficiency, at the end of this work, the test personnel filled out a questionnaire in which they entered what they had done, how long, and so on. They then judged their:

1. working tempo between counter pairs
fast oooooo slow
2. the intensity of their work between
concentrated oooooo unconcentrated;
3. their quality, between
good oooooo poor.

The activities named by the test persons were grouped according to appearance. The result was six main groups in which the other categories were classified. Table 2 shows the result of this last step. [Cand. phil. E. Rützel worked with the evaluation of this part].

Table 2 Total hours for the three test conditions among which the different works for the study were carried out.

	A	B	C	D	D	E	F	F
	Calc.	Tech.	Repe-	Liter.	Σ			Σ
		Lit.	tit-	from	A	personal	Tech.	A
			ions	rel.			signs	
				fields				
'4'	19.3	20.0	12.7	5.25	57.25	2.0	3.5	62.75
'-'	25.5	21.5	11.5	2.7	61.2	1.0		62.2
'o'	22.25	21.45	14.0	5.0	62.7	1.5		64.2
							Total:	189.15
							Pauli:	100.00
								<u>100.85</u>
								400.00

Test instructions, performance of the test,
urine emission, idle times.

The sum of the hours is actually entered in the cells during which a certain activity was carried out during a certain test condition. To the total of these times (= 189.15 hours) there are also 100 hours for the Kræpelin-Pauli test and 110.65 hours for the test instructions, performance of the test, urine emission and idle times.

Only the categories A, B, C and D were considered with the further evaluation, because category 'E' (= personal) did not fall in the area of questioning and category 'F' (=technical sign) required mental activity in another way than the other categories. It is noteworthy however that both these categories are occupied under the noise condition higher or alone.

The subjective judgment of our own activities during the test was therefore studied only for four categories. We proceeded in such a way that slow-fast, concentrated-unconcentrated, and good-bad were entered in each of the six classes (scale points) of the three evaluation scales in the number of hours in which the four activities (A, B, C and D) were subjectively carried out during the three test conditions with each of these (quality) classes.

Table 3

Quality Classes

		1	2	3	4	5	6	Summe (1)	
I. langsam (1)	+,	2,25	13,50	14,20	21,00	5,30	1,00	57,25	Best (2)
	o,	4,75	7,25	20,50	14,20	9,50	5,00	61,20	
	o,	5,50	10,10	26,00	9,85	8,25	3,00	62,70	
II. konzen- triert	+,	0,80	17,20	15,50	11,75	10,00	2,00	57,25	unkon-
	o,	12,50	20,25	8,95	14,50	5,00		61,20	zen-
	o,	11,50	22,70	17,50	3,00	3,00	5,00	62,70	triert
III. gut (1)	+,	1,00	19,75	15,20	11,50	5,00	4,00	57,25	schlecht (7)
	o,	12,50	17,50	20,20	7,00	4,00		61,20	
	o,	12,50	22,35	21,85	6,00			62,70	

(1) Total; (2) slow; (3) concentrated; (4) good; (5) fast; (6) unconcentrated; (7) poor.

Table 3 shows the compilation of these data: left the three scales or the three test conditions, above the six quality classifications, right the line totals.

In the following we now compare the distributions of the work hours in the quality classes of the three polarities, between the three test conditions by means of a 2x6 field chi-quadrant test. The result was thus three (I, II and III) times three ('o', "-". "+") chi quadrat values (Table 4), with actually five degrees of freedom.

The subjective judgment of one's own activity between the three test conditions was not different at the polarity I 'slow-fast'.

At polarity II there was a difference between all three test conditions 'concentrated-unconcentrated'. This difference was the slightest between both conditions 'without noise' ("o" and "-").

The polarity III, 'good-poor' gave no difference of the subjective judgment of one's own activity between the test condition 'with noise' ("+") and each of the two other conditions ("o" or "-"), but not between them (both were without noise).

Table 4

I. "slow-fast"

	"+"		"-"	
	χ^2	P	χ^2	P
o'	10,74	.10	2,52	.80
o'	8,96	.20		

II. "concentrated-unconcentrated"

	"+"		"-"	
	χ^2	P	χ^2	P
o'	19,93	.01	14,4	.025
o'	16,16	.01		

III. "good-poor"

	"+"		"-"	
	χ^2	P	χ^2	P
o'	19,76	.01	4,64	.50
o'	13,96	.025		

Table 5

	good/poor	conc/unconc.	rapid/slow
o'	1.8	1.4	0.9
o'	4.6	2.1	0.9
o'	9.4	4.7	0.5

Table 6.--Multidimensional scaling of the polarity polarity profiles.

		1	2
Control 'o'	Mood before the test	26	-1
	Self before the test	37	-11
	Mood after the test	37	4
	Situation after the test	-26	43
	Self after the test	31	-4
without noise '-'	Mood before the test	-20	2
	Self before the test	32	-7
	Mood after the test	-16	8
	Situation after the test	-43	44
	Self after the test	28	-6
with noise '+'	Mood before the test	-8	3
	Self before the test	21	-8
	Mood after the test	-46	-14
	Situation after the test	-89	-43
	Self after the test	34	-7

Table 7.--Factor analysis. The table shows the variables in their sequence during the tests. The numbers (1) and (2) mean the first and the second session halves. The second session half began with the KRAEPELIN-PAULI working test. The charges are shortened to two places and multiplied by one hundred. Charges < +0.4 are omitted for easier orientation "o" means 'without' noise and 'm' means 'with' noise. (1) Communalities; (2) Similarity; (3) Mean values; (4) scatterings.

	1		2		3		4		Kommunalitäten (1)	Ähnlichkeit (2)	Mittelwerte (3)		Streuungen (4)		
	o	m	o	m	o	m	o	m			o	m	o	m	
	Adrenalin (1)		43					83	82	72	91	86	10,0	11,0	3,1
Noradrenalin (1)			95	72					93	61	93	11,0	12,8	4,7	3,7
Pulse 4		-56			87	54			85	63	85	71,6	75,2	9,5	8,2
Pauli 5		90	63						94	58	91	17,9	17,9	3,2	3,3
Error percent					66	38			46	37	71	7,2	5,7	6,4	5,4
Peak time		92	48	43					91	64	77	7,3	8,3	6,7	7,1
Symptoms		-59	-56	52					52	73	89	9,2	8,0	2,0	2,4
Remarks					55	62			50	44	70	6,7	5,9	2,5	2,6
Adrenalin (2)							77	64	72	43	93	12,2	14,4	3,2	4,4
Noradrenalin (2)			87	82			-47		87	92	61	12,3	12,5	5,5	5,0
	2,3	1,9	2,1	1,9	1,6	1,6	1,5	0,8							

Column square totals

The differences were there the largest throughout between the conditions "+" (with noise) and "o" (without noise and without Pauli); on the other hand, they were smallest between conditions "-" (without noise) and "o" (without noise and without Pauli).

If one summarizes the evaluation categories 1-3 and 4-6 and if one divides the first by the second sums, then one obtains Table 5. The ratio figures of this table show the tendency of a subjective judgment of the test persons about their own working performance better than Table 3.

1.8 times more working hours were spent under the condition "+" with the subjective impression of good work performance than with poor working accomplishment. Under the conditions "-" and "o" the ratio increased to 4.6 and 9.5. The ratios in the case of 'concentrated-unconcentrated' were similar.

3. Self Judgment and Judgment of Mood

Since the polarity scheme according to Hofstätter proved itself to be a good usable method in several tests for the comprehension of situative components (Hawel, 1964, 1969, 1970), it was also used for the study of this question.

At the start of the session, the test personnel judged their momentary mood according to the polarity profile. The same judgments were also made at the end of each session and there was an additional judgment of the test situation.

The two dimensions given in Table 6 gave a multi-dimensional scaling of the profile data (Hawel, 1974), of which the first is immediately recognizable as the inner-outer dimension--relative to the subject. All self judgments and mood judgments of the control conditions ('o') lay here in the positive area, while all situation judgments and the mood judgments are under the both other test conditions in the negative area. The second dimension results from the position of the situation judgment, where both still conditions lie at the same place in the positive area and the noise conditions lie in the negative area. As is expected, the situation judgment 'with noise' lies next and the mood judgment at the end of the sessions 'with noise'.

Discrimination analyses of the five profile triples showed no differences for the three test conditions between the self judgments.

The mood positions at the start differed only between both test conditions 'without noise' ('o' and "-") with 2% coincidence probability.

The situational conditions corresponding to the three situations were judged in such a way that differences existed only between the test 'with noise' ("+") and both other test types, but not between these (0.1% coincidence probability).

At the end of the sessions, the mood judgment of the control condition ("o") differed very clearly from the judgments under both other conditions (0.1% coincidence probability); these differed from each other only relatively little (5% coincidence probability).

4. Tendency toward Anxiety

For the study of this question, we experimentally used a German formulation with five parallel forms of the Scheier and Cattellschen "Eight Parallel Form Objective Anxiety Scale" (1960) which was kindly made available to me by Prof. Dr. J. Fahrenbert (s.a. Beyme and Fahrenberg, 1966).

The questionnaires were answered by the test persons actually toward the end of sessions 5-9.

The form given in the fifth session was not taken into account in the evaluation. It was supposed to serve acclimatization to the method. Clearly less "fear symptoms" resulted from the effect of noise (such as "fear of bodily illness," "forgetfulness", "trouble with breathing", "itching on the skin", "cold arms and legs", "Nausea", etc) than without the effect of noise. If one uses the values of one comparison group to differentiate a difference (46 men, mean value 9.2; our mean values "+" = 7.9; "-" = 9.15) it is natural to assume that our test person found himself in a relatively tense state with the then existing increased general activity level which limited his tendency or even his capability for personal considerations compared with the tests without noise ("-") and the comparison group. One could also speak of a generalization of numbness.

Admittedly this is opposed to the findings referred to from Blau, who subjected his test persons with the performance of the psychological test to noise of 103 dB (lin) and found no effects other than "somatic complaints of specific anatomical location and description".

5. Relationship between psychological and physiological Data

Finally, two factor analyses were reckoned for the data from the tests 'without' and 'with noise' from sessions 6-9 (PAFADO). The following were included as variables: adrenalin and noradrenalin precipitation from both session halves; from the Pauli test the last (5th) value of the double smoothed working curve, the error percent and the peak time point (peak position) as well as the mean pulse frequency during the last quarter hour of the test; from the anxiety scale the 'anxiety symptoms' and the 'emotional nature of the remark'. The results of the analysis are shown in Table 7 in such a way that the sequence of the variables corresponds to its time sequence. Because of the slight occupation with the analyses, only charges up to ± 0.40 were interpreted. In order to make the differences of both analyses clearer, it is practical here to regard the corresponding factors from both test conditions in common. The analysis 'with noise' is maximally approached to the analysis 'without noise' according to the principle given by Fischer and Roppert (Febhardt). Because the catecholamine values from Vp were lacking, they were not taken into account with the evaluation of the variables relevant here, in order to make the results comparable.

Factor 1 without noise gives the negative relationship between the performance of the Kraepelin-Pauli test and the naming of anxiety symptoms. The later the peak time point lay, the higher the final performance and the less anxiety symptoms were named. (That can be interpreted that men under quiet conditions are less hypochondriac [fearful]). The performance maximum is reached more independently with noise and the level of performance seems to be related to the adrenalin precipitation in the first half of the session. Moreover, pulse frequency and naming of anxiety symptoms correlate here.

Factor 2 shows the high stability of the noradrenalin precipitation in both halves of the session under both test conditions. Under noise there is also a relationship between naming the anxiety symptoms and the peak time point.

Factor 3 exhibits only gradual differences under both test conditions. The relationship between "emotionality of the observations" pulse and mistakes exists clearly only without noise.

Factor 4 testifies of the proportionality of the adrenalin precipitation in both session halves and of the opposite tendency of the noradrenalin precipitation amid noise in the second session half.

Discussion of Results

1. In contrast to the studies of Sanders, but in agreement with Lienert and Jansen, we could find no influence of noise on the computing efficiency in the case of our performance test although a tendency to higher pulse frequency must be assumed during this test. The lack of performance difference is doubtlessly to be attributed to the small degree of difficulty of the task. The increased activation of the test persons during the noise sessions to be assumed on the basis of physiological findings could therefore only lead to another performance mode. Furthermore, it is to be considered that the test persons had absolved already before two four-hour sessions with noise and now in the sessions with noise, at the start of the performance test, were exposed to the same noise for two hours and supposedly had gotten used to the noise in this time.

In contrast to these considerations, the possibility moved into the background the same performance moves thus that acoustical environment is created by the quiet as well as by the noise phase in which the minimum values in which the minimum values of the known, inversely U-shaped performance curve lie.

2. Comparatively opened up test arrangements--with free theme selection (so to say test person-centered or non-directive test arrangements in the mode of C.R. Rogers) with noise as stress are not known in the pertinent literature.

The data concerning one's own working temp from which only slight differences result between the test conditions with noise and without noise, lie in the direction of the results from the Kraepelin-Pauli-test which is predominantly a speed test: hardly a finding, only a tendency to rapid work under noise.

Noise seems to have the greatest influence on the concentration, since the differences here are clearest compared with both other test conditions. But on the other hand, only the expectation of the Kraepelin-Pauli test appeared to have had a certain influence on the concentration of the test persons, for the tendency to unconcentrated work is noticeably greater under the concerned test conditions ("-") as over against the control sessions ("o"). Even the quality of the work is evaluated less amid noise than under both other test conditions.

In summary it can be said that in the subjective judgment under the noise conditions given here work was done with much less concentration and much worse than without noise, but that also a relatively short visit (as with the Kraepelin-Pauli test) had an influence on the concentration and quality of the performance.

3. The study of self image, mood and test situation confirms the results as they were found with the other questions (Hawel, 1964, 1969, 1970). So perhaps the constancy of the self-judgment and the agreement with mood judgment at the start of the tests with self-judgment or the influence of the test situation on mood judgment at the end of the sessions.

Profile comparisons show that under control (or quiet) conditions, the mood judgments lie completely with self judgments. Mood and self experience agree with each other; the situation was evaluated as something which is largely independent.

Under the condition with Kraepelin-Pauli, but without noise, the mood is largely influenced by the previous but already processed computation task.

Now under noise conditions, the test persons seemed to crawl into themselves at the start of the test, but later their mood is expressed by the computation task in connection with the noise, but very intensively by the situational conditions.

Both test conditions 'without noise' appear much more similar to the test persons than each of the two noise conditions.

4. Counter to our assumption that noise brings about a tendency to anxiety and to fear reactions, we found that there were no differences in the fear scale to be processed more cognitively and that during noise, less "fear symptoms" were named from the physical area. The assumption of a 'general numbness' used here as an interpretation aid in the sense of an irritation generalization presumably goes together with an ergotropic change of the vegetativum (Jansen, 1967).

5. The relationships existing here (factor 3) under both test conditions between error percent, emotionality of the remarks, and pulse frequency corresponds well to the Cattell-Scheier concept: "A refusal to deal seriously with the issue at hand." Admittedly, this relationship is more narrow without noise than with noise. Thus it can be said that during noise, with an increasing tendency of the pulse frequency, its relationship to the number of errors declines (and this itself), while a noticeable relationship to performance comes about, which is to be interpreted as the fact that at increased heart frequency, the performance maxima lie earlier and lower than with a slow heart frequency. Care is to be taken that the pulse frequency is registered only during the Kraepelin-Pauli work test. It is interesting that during noise, higher adding efficiency goes along with a tendency to low pulse frequency. In his studies, Rutenfranz found with the increasingly rapidly rotating Graf computer cylinder that the pulse frequency becomes higher and higher during the computations with more difficult tasks of the type $W-X+Y-Z$ with increasing time pressure, but in the case of an easy task under the same conditions it fluctuated only slightly around a solid value. Because the performance motivation was well recognizable among the test persons, we may assume that the ones who found adding difficult had been under more stress, which resulted in our findings.

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Summary

Ten male subjects (students) were required to take part in ten weekly sessions of four hours each. In the 5th and the 10th session, the subjects did their own study work without any noise disturbance. In the second half of these remaining experimental sessions, they were required to do adding work (Kraepelin-Pauli-Test); every other session under conditions of discontinuously presented noise of 90 db (lin). During the Kraepelin-Pauli-Test, the pulse rate was recorded. Relevant personality variables and catecholamine secretion were studied in every session.

No effect of noise could be recorded on the Kraepelin-Pauli-Test, probably because this test is too easy. While doing their own study work,

the subjects had the subjective impression of working more poorly and with less concentration, but more fluently under the conditions of noise than in silence. Subjects found both the experimental situations without noise much similar. Noise had a great effect on the mood of the subjects. The subjects reported more anxiety symptoms during the silence than during the noise.

Some relation between the noise and the performance, number of mistakes, and pulse rate could be determined.

L'économie. Les auteurs discutent quelques observations relatives à la tentent une explication de leurs résultats et font le point quant à l'utilité de l'adaptation allemande du questionnaire de Stumphauer.

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Research on the psychological and psychophysiological effects of repeated four-hour intermittent pink noise.

Aus dem ehemaligen Max-Planck-Institut für Arbeitsphysiologie Dortmund
Damaliger Direktor: Prof. Dr. Dr. h. c. G. Lehmann -
- Psychologische Abteilung -
Damaliger Leiter: Prof. Dr. J. Rutenfranz
und dem Institut für Arbeitsphysiologie an der Universität Dortmund
- Abteilung Umweltphysiologie -
Direktor Prof. Dr. med. H. G. Wenzel

Untersuchungen zur psychologischen und psychophysiologicalen Wirkung von wiederholtem vierstündigem, intermittierendem rosa Rauschen.¹

Wolfgang Hawel

Zehn männliche Versuchspersonen (Studenten des Technikums, Dortmund) nahmen in einer Versuchreihe, zehn Wochen lang, jede Woche einmal, an jeweils vierstündigen Sitzungen teil. In jeder Sitzung konnten sie wenigstens zwei Stunden frei für ihr Studium arbeiten. In den Sitzungen 'A' mit 'A' und 'B' mit 'B' hatten sie zudem den Kraepelin-Pauli-Arbeitsversuch auszuführen und wurden - von Sitzung zu Sitzung abwechselnd - periodisch unterbrochenem rosa Rauschen (90 db (lin), 5 sec, 30% ein, 70% aus) bzw. Stille ausgesetzt. Während des Kraepelin-Pauli-Versuches wurde die Pulsfrequenz registriert. Sitzungen 'A' und 'B' waren Kontrollversuche ohne Lärm und ohne Kraepelin-Pauli-Versuch. In allen Sitzungen wurden die in Frage stehenden Persönlichkeitsvariablen und die Catecholaminscheidung untersucht.

Der Kraepelin-Pauli-Arbeitsversuch alleine ergab keinen Hinweis auf eine Lärmwirkung, was als Folge der geringen Schwierigkeit der Aufgaben gedeutet wird. Während der frei gestellten Tätigkeit gewannen die Versuchspersonen den Eindruck, zwar eher flott, aber erheblich unkonzentrierter und viel schlechter arbeiten gekonnt zu haben als ohne Lärm. Den Versuchspersonen erschienen die beiden Versuchsbedingungen ohne Lärm sehr viel ähnlicher als jede von diesen der Lärmbedingung. Der Einfluss des Lärms auf die Stimmung war erheblich. Mit Lärm wurden weniger Angstsymptome genannt als bei Stille.

Es fanden sich Anzeichen für eine Beziehung zwischen Leistung, Fehlerhäufigkeit, Pulsfrequenz und Lärm.

¹) Herrn Prof. Dr. med. Dr. med. h. c. Gunther Lehmann zum Gedächtnis in Verachtung gewidmet.

POOR COPY

Es wird über die psychologischen Ergebnisse einer Untersuchung* berichtet, deren physiologische Befunde an anderer Stelle mitgeteilt worden sind

SitzungNr.:	Gewöhnung				Versuch						
	1	2	3	4	5	6	7	8	9	10	
Vp.Nr.: 1	-	-	-	-	o	-	-	-	-	o	Di
2	-	-	-	-	o	-	-	-	-	o	
3	-	-	-	-	o	-	-	-	-	o	MI
4	-	-	-	-	o	-	-	-	-	o	
5	-	-	-	-	o	-	-	-	-	o	Do
6	-	-	-	-	o	-	-	-	-	o	
7	-	-	-	-	o	-	-	-	-	o	Fr
8	-	-	-	-	o	-	-	-	-	o	
9	-	-	-	-	o	-	-	-	-	o	Mo
10	-	-	-	-	o	-	-	-	-	o	

0 Sid. 2 Sid. 4 Sid. :Zeit

:Versuchsart

A = Arbeiten für Studium K = KRAEPELIN-PAULI
 F = Fragebogen P = Persönlichkeitsfragebogen
 H = Hörn

HAWEL	Versuchsordnung zur Bestimmung der Catecholaminzuscheidung unter Lärm	MPI
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Abb. 1

* Die Untersuchung wurde durch eine Sachbeihilfe der Deutschen Forschungsgemeinschaft gefördert (AZ: Ha 400/6).
 Bei der Durchführung und Auswertung der Versuche haben A. Nörte und A. Hubbert mitgearbeitet.
 Die EDV wurde an der IBM 360/44 des Max-Planck-Instituts für Ernährungsphysiologie (Direktor: Prof. Dr. Benno Hess) durchgeführt.

(Hawel & Starlinger, 1967). Eine ausführliche Begründung des Versuches und die eingehende Beschreibung des Versuchsplanes und der Versuchsbedingungen erfolgten dort. Deswegen wird die Versuchsordnung hier nur kurz geschildert:

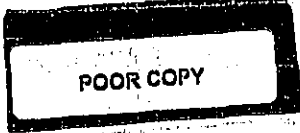
Zehn männliche Versuchspersonen (Studenten des Technikums Dortmund) nahmen gegen eine angemessene Vergütung in zehn aufeinanderfolgenden Wochen, vorwiegend am gleichen Wochentag, zur selben Tageszeit, für vier Stunden an einem Versuch teil (s. Abb. 1). Die Versuchsbedingungen variierten zwischen 'mit Lärm' und 'ohne Lärm' sowie zwischen 'mit Kraepelin-Pauli-Arbeitsversuch' und, als Kontrollbedingung, 'ohne Kraepelin-Pauli-Versuch'. Diese Kontrollbedingung war ohne Lärm und fiel immer auf die Sitzungen 5 und 10. In den übrigen Sitzungen wurde jeweils eine Versuchsperson dem Lärm ausgesetzt, während eine andere den Versuch in Ruhe ausführte. Der Lärm war periodisch unterbrochenes rosa Rauschen von 90 db (lin) (re 2·10⁻⁴ pb). In der ersten Sitzung 'mit Lärm' wurde Straßelärm mit Pegelspitzen von 85 db (lin) gegeben. Das Rauschen war, bei 5 sec Zykluszeit, mit je 30% (Sitzung 2—4) bzw. 70% (Sitzung 6—9) Stille unterbrochen (in Anlehnung an die Überlegungen von Teichner et al.). Bei der physiologischen Untersuchung wurden die ersten vier Sitzungen, als Gewöhnungsphase, nicht berücksichtigt. Für die letzten sechs Sitzungen wurde unter Lärm, in der zweiten Sitzungshälfte, eine erhöhte Adrenalinausscheidung mit 5% Irrtumswahrscheinlichkeit ermittelt; auch eine Tendenz zu höherer Pulsfrequenz während des Kraepelin-Pauli-Versuches unter Lärm muß angenommen werden.

Bei der Erwägung einer möglichen psychologischen Wirkung von lange dauerndem Lärm hatten wir uns vier Fragen gestellt, für deren Beantwortung uns die Untersuchung Information bringen sollte.

1. Besteht ein Einfluß auf die Ausführung eines einfachen Leistungsver-suches?

Abb. 1

Das Schema zeigt den Versuchsplan links, von oben nach unten, die Nummern der Versuchspersonen, rechts die Wochentage, an denen diese in der Regel an den Versuchen teilnahmen. Oben die Nummern der Sitzungen, davon die ersten vier als Gewöhnung, die nicht in die Auswertung einbezogen worden sind. Die Symbole o, - und + in den Feldern entsprechen: o = ohne Kraepelin-Pauli-Versuch, ohne Lärm, - = mit Kraepelin-Pauli-Versuch, ohne Lärm und + = mit Kraepelin-Pauli-Versuch, aber mit Lärm. Man erkennt ohne weiteres, daß in den Sitzungen 5 und 10 durchweg die Versuchsart o angewandt wurde, während die beiden anderen Versuchsarten so angeordnet wurden, daß jede Versuchsperson jede Versuchsart zweimal mitmachte und daß in jeder Sitzungsnummer jede der beiden Versuchsarten fünfmal vorkam. Aus dem unteren Teil des Schemas geht der Ablauf der verschiedenen Versuchsarten hervor.



Diese Frage war in Versuchen von kürzerer Dauer untersucht worden (u. a. von Sanders, 1961; Lienert & Jansen, 1964).

- Wie wird die eigene Leistung bei gewohnter geistiger Arbeit beurteilt?
- Ändern sich Stimmung und/oder Selbstbild der Versuchspersonen?
- Zeigt sich eine Neigung zu Ängstlichkeit? Zur Untersuchung dieser Frage führte uns die u. a. in Verbindung mit der Wirkung von Lärm auf den Menschen diskutierte Aktivierungshypothese (Hörmann & Todt, 1960; Schönplüg, 1969). Nach ihr erschien nicht auszuschließen, daß lange dauernder Lärm eine Erregung bewirke, die von einer Neigung zu ängstlichen Verhaltensweisen begleitet sein kann (s. auch Lidberg und Levi, 1969).

1. Leistungsversuch

Um Daten über die objektive Leistungsfähigkeit unter den beiden Versuchsbedingungen „—“ = „ohne“ und „+“ = „mit Lärm“ zu erhalten, wurde der Kraepelin-Pauli-Arbeitsversuch ausgewählt (Pauli—Arnold, 1961). Er stellt eine verhältnismäßig einfache Aufgabe dar (addieren jeweils zweier einstelliger Zahlen, während einer Stunde), die sich beliebig wiederholen läßt und über die schon eine große Anzahl von Untersuchungen vorliegen (Ulrich, 1958; weitere Literatur bei Christiansen, 1966). Der Kontroll-Versuch, ohne Kraepelin-Pauli und ohne Lärm, erhielt das Symbol „0“.

Die Kommandos für den Kraepelin-Pauli-Versuch („Achtung, — anfangen!“, alle 3 Minuten „Strich!“ und am Ende: „Achtung, — Meiststift weg!“) waren auf Band gesprochen worden und wurden über die Lautsprecher in die Kabine gegeben. Bei den Sitzungen mit Lärm wurden sie diesem überlagert. Die Versuchspersonen wurden jeweils vor jeder Durchführung des Tests — bei einer kurzen Probe-Durchgabe — befragt, ob die Kommandos auch deutlich ankämen. (Die Kommunikation Versuchsleiter—Versuchsperson erfolgte über eine Telefonanlage.) Durch diese Art der Kommando-Gabe wurde außerdem ihre Gleichheit über alle Sitzungen gewährleistet. Jedes der beiden verwendeten Bänder wurde 40mal gespielt, und ihre Dehnung konnte auch am Ende der Versuchsreihe vernachlässigt bleiben.

Die Auswertung der Daten aus diesem Versuch (Sitzungen 6, 7, 8 und 9) mittels Covarianzanalyse ergab keinen Unterschied. (Die fünf Werte aus den individuellen Arbeitskurven, ohne Lärm („—“) wurden als unabhängige Variablen genommen und in Beziehung zu den korrespondierenden Werten der Arbeitskurven „mit Lärm“ („+“) gebracht; dabei wurden jeweils zwei zeitlich aufeinanderfolgende Kurven korreliert.)

Der Vergleich der fünf abgeleiteten Werte — Summe, Fehlerprozent, Verbesserungs-Prozent, Steighöhe und Gipfelzeit — mittels Diskriminanzanalyse (Faber & Nollau) ergab in der Gewöhnungsphase (Sitzungen 1 mit 4) einen Unterschied auf dem 10%-Niveau, der aber in den Sitzungen 6 mit 9 nicht mehr beobachtet werden konnte. Auch ein T²-Test für abhängige Stichproben ergab keinen Unterschied. Die Mittelwert-

Tabelle 1

Die Tabelle zeigt Mittelwert-Vektoren der Kraepelin-Pauli-Arbeitsversuche. Ganz links die Sitzungen und daneben die Lärmbedingungen, über die jeweils gemittelt wurde.

Sitzungen	Lärm	Summe	Fehler-Prozent	Verbesserungs-Prozent	Steighöhe	Gipfelzeitpunkt
1—4	ohne	3196	4,8	8,0	46,6	0,7
	mit	3186	6,1	5,2	35,3	10,4
6—9	ohne	3623	7,2	5,4	43,3	7,3
	mit	3640	5,7	4,9	48,7	8,3

Vektoren sind in Tabelle 1 dargestellt. Die „Steighöhe“ erwies sich als problematisches Maß, weil sie gelegentlich eindeutig eine „Sinktiefe“ war.

2. Subjektive Beurteilung der eigenen Leistung

Wie in unserem Bericht über die physiologischen Ergebnisse dieser Untersuchung ausführlich begründet, konnten die Versuchspersonen während eines Teils der zur Verfügung stehenden Versuchszeit für ihr Studium arbeiten. Es sollte damit erreicht werden, daß für die Versuchspersonen die Teilnahme an dieser Versuchsreihe ein Teil ihres Alltags würde und damit den Charakter einer Ausnahme-Situation verlore. Um Information über die subjektive Beurteilung der eigenen Leistung zu erhalten, füllten die Versuchspersonen, nach Beendigung dieser Arbeiten, jeweils einen Fragebogen aus, in den sie eintrugen, womit sie sich, wie lange, beschäftigt hatten. Sie beurteilten dann:

- ihr Arbeitstempo, zwischen dem Gegensatzpaar
sollt o o o o o langsam;
- die Intensität ihrer Arbeit, zwischen
konzentriert o o o o o unkonzentriert;
- deren Qualität, zwischen
gut o o o o o schlecht.

Die von den Versuchspersonen genannten Tätigkeiten wurden nach Augenschein gruppiert. Dabei ergaben sich sechs Hauptgruppen, in die dann, nach Gutdünken, die restlichen Kategorien eingegliedert wurden. Das Ergebnis dieses letzten Schrittes zeigt Tabelle 2¹⁾.

Tabelle 2

Stunden-Summen für die drei Versuchsbedingungen, unter denen die verschiedenen Arbeiten für das Studium ausgeführt wurden.

	A	B	C	D	D	E	F	F
	Rechnen	Technische Literatur	Wiederholungen	Literatur Neben-sichern	Σ A	Persönliches	Techn. Zeichnen	Σ A
,+'	19,3	20,0	12,7	5,25	57,25	2,0	3,5	62,75
,-'	25,5	21,5	11,5	2,7	61,2	1,0		62,2
,o'	22,25	21,45	14,0	5,0	62,7	1,5		64,2

Summe: 189,15

Pauli: 100,00

Versuchsanweisungen, Durchführung von Tests, Harnabgabe,

Leerzeiten: 110,85

400,00

In den Zellen sind jeweils die Summen der Stunden eingetragen, während welcher unter der jeweiligen Versuchsbedingung eine bestimmte Tätigkeit ausgeführt wurde. Zu der Gesamtsumme dieser Zeiten (= 189,15 Stunden) kommen noch 100 Stunden für den Kraepelin-Pauli-Versuch und 110,65 Stunden für Versuchsanweisungen, Durchführung von Tests, Harnabgabe und Leerzeiten.

Bei der weiteren Auswertung wurden nur die Kategorien A, B, C und D berücksichtigt, weil die Kategorie 'E' (= Persönliches) nicht in den Bereich der Fragestellung fiel und die Kategorie 'F' (= Technisches Zeichnen) in anderer Weise gedankliche Tätigkeit erfordert als die übrigen Kategorien. Es ist aber bemerkenswert, daß diese beiden Kategorien unter der Lärmbedingung höher bzw. alleine besetzt sind.

Die subjektive Beurteilung der eigenen Tätigkeit während des Versuchs wurde also nur für vier Kategorien untersucht. Dabei wurde so ver-

1) Bei der Auswertung dieses Teils hat Herr cand. phil. E. Rützel mitgewirkt.

fahren, daß in jede der sechs Klassen (Skalen-Punkte) der drei Bewertungsskalen langsam—flott, konzentriert—unkonzentriert und gut—schlecht die Anzahl der Stunden eingetragen wurde, in denen die vier Tätigkeiten (A, B, C und D) unter den drei Versuchsbedingungen mit jeder dieser (Güte-)Klassen subjektiv ausgeführt worden war.

Tabelle 3

Güteklassen

		1	2	3	4	5	6	Summe	
I. langsam	,+'	2,25	13,50	14,20	21,00	5,30	1,00	57,25	flott
	,-'	4,75	7,25	20,50	14,20	9,50	5,00	61,20	
	,o'	5,50	10,10	26,00	9,85	8,25	3,00	62,70	
II. konzentriert	,+'	0,80	17,20	15,50	11,75	10,00	2,00	57,25	unkonzentriert
	,-'	12,50	20,25	8,95	14,50	5,00		61,20	zentriert
	,o'	11,50	22,70	17,50	3,00	3,00	5,00	62,70	
III. gut	,+'	1,80	19,75	15,20	11,50	5,00	4,00	57,25	schlecht
	,-'	12,50	17,50	20,20	7,00	4,00		61,20	
	,o'	12,50	22,35	21,85	6,00			62,70	

Tabelle 3 zeigt die Zusammenstellung dieser Daten: links die drei Skalen bzw. drei Versuchsbedingungen, oben die sechs Gütestufen, rechts die Zeilensummen.

Im folgenden wurden nun die Verteilungen der Arbeitsstunden, auf die Güteklassen der drei Polaritäten, zwischen den drei Versuchsbedingungen mittels eines 2x6-Felder Chi-Quadrat-Tests verglichen. Es ergaben sich somit drei (I, II und III) mal drei („o“, „-“, „+“) Chi-Quadrat-Werte (Tabelle 4), bei jeweils fünf Freiheitsgraden.

Auf der Polarität I, „langsam—flott“, war die subjektive Beurteilung der eigenen Tätigkeit zwischen den drei Versuchsbedingungen nicht unterschiedlich.

Auf der Polarität II, „konzentriert—unkonzentriert“, bestand zwischen allen drei Versuchsbedingungen ein Unterschied; dabei war dieser Unterschied zwischen den beiden Bedingungen „ohne Lärm“ („o“ und „-“) am geringsten.

Die Polarität III, „gut—schlecht“, ergab einen Unterschied der subjektiven Beurteilung der eigenen Tätigkeit zwischen der Versuchsbedingung „mit Lärm“ („+“) und jeder der beiden anderen Bedingungen („o“ bzw. „-“), nicht aber zwischen diesen (beide waren ohne Lärm).

Tabelle 4

I. „langsam—flott“				
	„+“		„-“	
	χ^2	p	χ^2	p
„o“	10,74	.10	2,52	.80
„-“	8,96	.20		
II. „konzentriert—unkonzentriert“				
	„+“		„-“	
	χ^2	p	χ^2	p
„o“	19,93	.01	14,4	.025
„-“	16,16	.01		
III. „gut—schlecht“				
	„+“		„-“	
	χ^2	p	χ^2	p
„o“	19,76	.01	4,64	.50
„-“	13,96	.025		

Die Unterschiede waren also durchweg am größten zwischen den Bedingungen „+“ (mit Lärm) und „o“ (ohne Lärm und ohne Pauli); dagegen am geringsten zwischen den Bedingungen „-“ (ohne Lärm) und „o“ (ohne Lärm und ohne Pauli).

Faßt man aus Tabelle 3 jeweils die Bewertungs-Kategorien „1“ mit „3“ und „4“ mit „6“ zusammen und dividiert die ersten durch die zweiten Summen, dann erhält man Tabelle 5. Die Verhältniszahlen dieser Tabelle zeigen noch übersichtlicher und knapper als Tabelle 3 die Tendenz der

Tabelle 5

	gut/schlecht	konz./unkonz.	flott/langsam
„+“	1,8	1,4	0,9
„o“	4,6	2,1	0,9
„-“	2,4	4,7	0,5

subjektiven Urteile der Versuchspersonen über ihre eigene Arbeitsleistung.

Unter der Bedingung „+“ wurden 1,8mal mehr Arbeitsstunden mit dem subjektiven Eindruck guter Arbeitsleistung verbracht als mit schlechter Arbeitsleistung; unter den Bedingungen „-“ und „o“ stieg das Verhältnis aber auf 4,6 bzw. 9,5 an. Ähnlich liegen die Verhältnisse bei „konzentriert—unkonzentriert“.

3. Selbstbeurteilung und Beurteilung der Stimmung

Da das Polaritäten-Schema nach Hofstätter sich schon in mehreren Untersuchungen als gut brauchbares Verfahren für das Erfassen situativer Komponenten erwiesen hatte (Havel, 1964, 1969, 1970), fand es auch für die Untersuchung dieser Frage Verwendung.

Zu Beginn jeder Sitzung hatten die Versuchspersonen sich selbst und ihre augenblickliche Stimmung nach dem Polaritäten-Profil zu beurteilen. Die gleichen Beurteilungen wurden auch am Ende jeder Sitzung vorgenommen und zusätzlich eine Beurteilung der Versuchssituation.

Tabelle 6

Mehrdimensionale Skalierung der Polaritätenprofile.

			1	2
Kontrolle	Stimmung vor dem Versuch	„o“	26	- 1
	Selbst	„-“	37	- 11
	Stimmung nach dem Versuch	„o“	37	4
	Situation	„-“	-26	43
ohne Lärm	Selbst	„o“	31	- 4
	Stimmung vor dem Versuch	„-“	-20	2
	Selbst	„o“	32	- 7
	Stimmung nach dem Versuch	„-“	-16	8
mit Lärm	Situation	„o“	-43	44
	Selbst	„-“	28	- 6
	Stimmung vor dem Versuch	„+“	-8	3
	Selbst	„o“	21	- 8
„+“	Stimmung nach dem Versuch	„-“	-46	-14
	Situation	„o“	-89	-43
	Selbst	„-“	34	- 7
	Selbst	„o“		

Eine mehrdimensionale Skalierung der Profildaten (Hawel, 1974) ergab die in Tabelle 6 wiedergegebenen zwei Dimensionen, von denen die erste ohne weiteres als die — auf das Subjekt bezogene — Innen-Außen-Dimension erkennbar ist: Alle Selbstbeurteilungen und die Stimmungsbeurteilungen der Kontrollbedingungen („o“) liegen hier im positiven Bereich, während alle Situationsbeurteilungen und die Stimmungsbeurteilungen unter den beiden übrigen Versuchsbedingungen im negativen Bereich sind. Die zweite Dimension ergibt sich aus der Lage der Situationsbeurteilung, wobei die beiden Stillebedingungen an gleicher Stelle im positiven Bereich und die Lärmbedingungen im negativen Bereich liegen. Der Situationsbeurteilung „mit Lärm“ am nächsten liegt erwartungsgemäß die Stimmungsbeurteilung am Ende der Sitzungen „mit Lärm“.

Diskriminanzanalysen der fünf Profil-Tripel für die drei Versuchsbedingungen ergaben zwischen den Selbstbeurteilungen keine Unterschiede.

Die Stimmungslagen zu Beginn unterscheiden sich lediglich zwischen den beiden Versuchsbedingungen „ohne Lärm“ („o“) und „—“) mit 2% Zufallswahrscheinlichkeit.

Die den drei Bedingungen entsprechenden situativen Gegebenheiten wurden so beurteilt, daß sich alleine zwischen dem Versuch „mit Lärm“ („+“) und den beiden anderen Versuchsarten, aber nicht zwischen diesen, Unterschiede ergaben (je 0,1% Zufallswahrscheinlichkeit).

Am Ende der Sitzungen unterschied sich die Stimmungsbeurteilung der Kontrollbedingung („o“) sehr deutlich von den Beurteilungen unter den beiden anderen Bedingungen (0,1% Zufallswahrscheinlichkeit), diese unterschieden sich voneinander aber nur relativ wenig (5% Zufallswahrscheinlichkeit).

4. Neigung zu Angstlichkeit

Für die Untersuchung dieser Frage wurde, versuchsweise, eine deutsche Fassung mit fünf Parallel-Formen der Scheier und Cattell'schen „Eight-Parallel-Form Objective Anxiety Scale“ (1960) benutzt, die mir freundlicherweise von Herrn Prof. Dr. J. Fahrenberg zur Verfügung gestellt wurde (s. a. Beyme & Fahrenberg, 1966).

Die Fragebogen wurden von den Versuchspersonen jeweils gegen Ende der Sitzungen „5“ bis „9“ beantwortet.

Die in der fünften Sitzung gegebene Form wurde bei der Auswertung nicht berücksichtigt; sie sollte der Gewöhnung an das Verfahren dienen. Es wurden unter Lärmwirkung deutlich weniger „Angst-Symptome“ (wie „Furcht vor körperlicher Krankheit“, „Vergesslichkeit“, „Mühe beim Atmen“, „Kribbeln in der Haut“, „Kalte Glieder“, „Übelkeit“ etc.) ange-

geben als ohne Lärmwirkung. (Fisher-Pitman $t_p \leq .015$, s. Liener). Wenn man zur Differenzierung dieses Unterschiedes Werte einer Vergleichsgruppe heranzieht (46 Männer, Mittelwert 9,2; unsere Mittelwerte „+“ = 7,9; „—“ = 9,15) liegt die Vermutung nahe, daß sich unsere Versuchspersonen während der Lärmversuche („+“), bei dem dann gegebenen erhöhten allgemeinen Aktivierungsniveau, in einem verhältnismäßig angespannten Zustand befanden, der ihre Neigung oder auch ihre Fähigkeit zur Selbstbesinnung — verglichen mit den Versuchen ohne Lärm („—“) und der Vergleichsgruppe — einschränkte. Man könnte auch von einer Generalisierung der Verdrückung sprechen.

Dies steht allerdings im Gegensatz zu den von Plutchik referierten Befunden Bla's, der seine Versuchspersonen, bei der Durchführung von psychologischen Tests, Lärm von 103 dB (lin) aussetzte und dabei keine Wirkungen fand als „somatic complaints of specific anatomical location and description“.

5. Beziehung zwischen psychologischen und physiologischen Daten

Schließlich wurden zwei Faktorenanalysen für die Daten aus den Versuchen „ohne“ bzw. „mit Lärm“ der Sitzungen „6“ mit „9“ gerechnet (PAFADO). Als Variablen gingen ein: Adrenalin- und Noradrenalinausscheidung von beiden Sitzungshälften; aus dem Pauli-Versuch der letzte (5.) Wert der doppelt geglätteten Arbeitskurve, die Fehler-Prozent und der Gipfelzeitpunkt (Gipfellage) sowie die mittlere Pulsfrequenz während der letzten Viertelstunde des Versuches; aus der Angst-Skala die „Angst-Symptome“ und die „Emotionalität des Bemerkens“. Die Ergebnisse der Analyse sind in Tabelle 7 so dargestellt, daß die Reihenfolge der Variablen ihrer zeitlichen Aufeinanderfolge entspricht. Wegen der geringen Besetzung der Analysen werden nur die Ladungen bis $\pm 0,40$ interpretiert. Um die Unterschiede beider Analysen deutlicher zu machen ist es hier zweckmäßig, korrespondierende Faktoren aus beiden Versuchsbedingungen jeweils gemeinsam zu betrachten. Die Analyse „mit Lärm“ ist der Analyse „ohne Lärm“ nach dem von Fischer und Roppert angegebenen Prinzip maximal angenähert (Gebhardt). Weil von Vp 2 die Catecholamin-Werte fehlten, blieb sie, um die Ergebnisse vergleichbar zu machen, bei der Auswertung der hier relevanten Variablen unberücksichtigt.

Faktor 1 gibt ohne Lärm die negative Beziehung zwischen der Leistung im Kraepelin-Pauli-Versuch und der Nennung von Angstsymptomen wieder. Je später der Gipfelzeitpunkt liegt, um so höher ist die Endleistung und um so weniger Angstsymptome werden genannt. (Das

Tabelle 7

Faktorenanalyse. In der Tabelle sind die Variablen in ihrer Aufeinanderfolge während der Versuche wieder gegeben. Die Ziffern (1) und (2) bedeuten erste bzw. zweite Sitzungshälfte. Die zweite Sitzungshälfte begann mit dem KRAEPELIN-PAULI-Arbeitsversuch. Die Ladungen sind auf zwei Stellen gekürzt und mit hundert multipliziert. Zur früheren Orientierung sind Ladungen $\leq \pm 0,4$ weggelassen. 'o' bedeutet 'ohne' Lärm und 'm' bedeutet 'mit' Lärm.

	1		2		3		4		Kommunikation	Abweichung	Mittelwerte		Streuung		
	o	m	o	m	o	m	o	m			o	m	o	m	o
Adrenalin (1)	43		95	72			83	82	72	91	86	10,0	11,0	3,1	3,6
Noradrenalin (1)									93	61	93	11,0	12,8	4,7	3,7
Puls 4		56			87	54			85	63	85	71,6	75,2	9,5	8,2
Pauli 5	20	63							94	58	91	17,9	17,9	3,2	3,3
Fehler-Prozent					66	38			46	37	71	7,2	5,7	6,4	5,4
Gipfzeit	92	48	43						91	64	77	7,3	8,3	6,7	7,1
Symptome		59	52						52	73	89	9,2	8,0	2,0	2,4
Bemerkungen					55	62			50	44	70	6,7	5,9	2,5	2,6
Adrenalin (2)							77	64	72	43	93	12,2	14,4	3,2	4,4
Noradrenalin (2)								47	87	92	81	12,3	12,5	5,5	5,0
Spalten-Quadratsummen	2,3	1,9	2,1	1,9	1,6	1,6	1,5	0,8							

kann gedeutet werden, daß Menschen unter Ruhebedingungen bei zunehmendem Leistungsanstieg (die etwa ihre Endleistung systematisch aufbauen) weniger hypochondrisch (ängstlich) sind.) Mit Lärm wird das Leistungsmaximum unabhängiger erreicht und die Höhe der Leistung scheint in Beziehung zur Adrenalin-Ausscheidung in der ersten Sitzungshälfte zu stehen. Zudem korrelieren hier Pulsfrequenz und Nennung von Angstsymptomen.

Faktor 2 zeigt die hohe Stabilität der Noradrenalin-Ausscheidung in beiden Sitzungshälften unter beiden Versuchsbedingungen. Unter Lärm besteht daneben noch eine Beziehung zur Nennung von Angstsymptomen und zum Gipfelzeitpunkt.

Faktor 3 weist unter beiden Versuchsbedingungen nur graduelle Unterschiede auf. Der Zusammenhang zwischen „Emotionalität der Bemerkungen“, Puls und Fehlern besteht deutlich nur ohne Lärm.

Faktor 4 zeugt von der Proportionalität der Adrenalin-Ausscheidung in beiden Sitzungshälften und von der entgegengesetzten Tendenz der Noradrenalin-Ausscheidung unter Lärm in der zweiten Sitzungshälfte.

Diskussion der Ergebnisse

1. Im Gegensatz zu den Untersuchungen von Sanders, aber in Übereinstimmung mit Lienert und Jansen konnten wir keinen Einfluß von Lärm auf die Rechenleistung bei unserem Leistungsversuch feststellen, obwohl eine Tendenz zu höherer Pulsfrequenz während dieses Versuches angenommen werden muß. Das Fehlen eines Leistungsunterschiedes ist zweifellos auf den geringen Schwierigkeitsgrad der Aufgabe zurückzuführen. Die auf Grund der physiologischen Befunde anzunehmende erhöhte Aktivierung der Versuchspersonen während der Lärm-Sitzungen konnte deshalb nur zu einem anderen Leistungsanstieg führen. Ferner ist zu berücksichtigen, daß die Versuchspersonen zuvor schon zwei vierstündige Sitzungen mit Lärm absolviert hatten und nun in den Sitzungen mit Lärm, zu Beginn des Leistungsversuches, schon jeweils zwei Stunden demselben Lärm ausgesetzt waren und sich in dieser Zeit vermutlich weiter an den Lärm gewöhnt hatten.

Gegenüber diesen Überlegungen tritt auch die Möglichkeit in den Hintergrund, die gleiche Leistung rühre daher, daß sowohl durch die Stille als auch durch die Lärmphase akustische Milieus geschaffen wurden, in denen die Minimalwerte der bekannten, umgekehrt U-förmigen Leistungskurve liegen.

2. Vergleichbar aufgelockerte Versuchsbedingungen — mit freier Themenwahl — (sozusagen Versuchspersonen-zentrierte oder nicht-direktive-

Versuchsarrangierungen *modo* C. R. Rogers) mit Lärm als Stressor sind in der einschlägigen Literatur nicht bekannt.

Die Angaben über das eigene Arbeitstempo, aus denen nur geringe Unterschiede zwischen den Versuchsbedingungen mit Lärm und ohne Lärm resultieren, liegen in Richtung der Ergebnisse aus dem Kraepelin-Pauli-Versuch, der ja vorwiegend ein Speed-Test ist: kaum ein Befund, lediglich Tendenzen zu flotterem Arbeiten unter Lärm.

Auf die Konzentration scheint Lärm den stärksten Einfluß zu haben, da die Unterschiede hier gegenüber den beiden anderen Versuchsbedingungen am deutlichsten sind. Andererseits scheint aber auch schon alleine die Erwartung des Kraepelin-Pauli-Versuches einen gewissen Einfluß auf die Konzentration der Versuchspersonen gehabt zu haben, denn die Tendenz zu unkonzentrierter Arbeit ist merklich größer unter den betreffenden Versuchsbedingungen („—“) gegenüber den Kontroll-Sitzungen („o“). Auch die Güte der Arbeit wird unter Lärm geringer bewertet als unter den beiden anderen Versuchsbedingungen.

Zusammenfassend läßt sich sagen, daß im subjektiven Urteil unter den hier gegebenen Lärmbedingungen eher flott aber erheblich unkonzentrierter und viel schlechter gearbeitet wurde als ohne Lärm, daß aber auch ein verhältnismäßig trister Versuch (wie der Kraepelin-Pauli-Test) Einfluß auf Konzentration und Güte der Leistung hatte.

J. Die Untersuchung von Selbstbild, Stimmung und Versuchssituation bestätigt Ergebnisse, wie sie bei anderen Fragestellungen gefunden worden sind (Hawel, 1964, 1969, 1970). So etwa die Konstanz der Selbstbeurteilung und die Übereinstimmung der Stimmungsbeurteilung zu Beginn der Versuche mit der Selbstbeurteilung bzw. den Einfluß der Versuchssituation auf die Stimmungsbeurteilung am Ende der Sitzungen.

Die Profilvergleiche zeigen, daß unter Kontroll- (oder Ruhe-)Bedingung die Stimmungsbeurteilungen ganz bei den Selbstbeurteilungen liegen. Stimmung und Selbsterleben stimmen überein; die Situation wird als etwas gewertet, das weitgehend unabhängig davon ist.

Unter der Bedingung mit Kraepelin-Pauli, aber ohne Lärm, ist die Stimmung anscheinend in hohem Maße von den noch bevorstehenden bzw. schon bearbeiteten Rechenaufgaben beeinflusst.

Unter Lärmbedingung nun scheinen sich die Versuchspersonen zu Beginn der Versuche in sich selbst zu verkriechen, aber später ist ihre Stimmung durch die Rechenaufgaben in Verbindung mit dem Lärm doch sehr intensiv von den situativen Gegebenheiten geprägt.

Die beiden Versuchsbedingungen „ohne Lärm“ erscheinen den Versuchspersonen einander sehr viel ähnlicher als jede von beiden der Lärmbedingung.

4. Entgegen unserer Annahme, daß Lärm eine Neigung zu Ängstlich-

keit bzw. zu ängstlichen Reaktionen bewirke, mußten wir feststellen, daß in den mehr kognitiv zu bearbeitenden Angst-Skalen sich keine Unterschiede ergaben und daß unter Lärm sogar weniger „Angst-Symptome“ aus dem körperlichen Bereich genannt werden. Die hier als Interpretationshilfe herangezogene Annahme einer „verallgemeinerten Verärbung“ im Sinne einer Reiz-Generalisierung geht vermutlich einher mit einer ergotropen Umstimmung des Vegetativums (Janzen, 1967).

5. Die hier (Faktor 3) unter beiden Versuchsbedingungen bestehende Beziehung zwischen Fehler-Prozent, „Emotionalität der Bemerkungen“ und Pulsfrequenz entspricht gut dem Castell-Scheiderschen Konzept: „A refusal to deal seriously with the issue at hand.“ Allerdings ist diese Beziehung ohne Lärm enger als mit Lärm. Man kann also sagen, daß unter Lärm, bei einer insgesamt ansteigenden Tendenz der Pulsfrequenz, sich ihre Beziehung zur Fehlerzahl (und diese selbst) verringert, während zur Leistung eine merklich negative Relation entsteht, die auch darin zu sehen ist, daß bei erhöhter Herzfrequenz die Leistungsmaxima früher und niedriger liegen als bei langsamer Herzfrequenz. Hier ist zu beachten, daß die Pulsfrequenz nur während des Kraepelin-Pauli-Arbeitsversuches registriert wurde. Interessant ist, daß unter Lärm höhere Addierleistung mit einer Tendenz zu geringerer Pulsfrequenz einhergeht. Rutenfranz fand ja bei seinen Untersuchungen mit der sich immer schneller drehenden Grafschen Rechenwalze, daß die Pulsfrequenz während des Rechenvorganges mit schwierigen Aufgaben vom Typ $W \cdot X + Y - Z$ unter zunehmendem Zeitdruck immer höher wurde, aber bei leichten Aufgaben unter denselben Bedingungen nur wenig um einen festen Wert schwankte. Weil die Leistungsmotivation unserer Versuchspersonen erkennbar gut war, dürfen wir annehmen, daß diejenigen, welchen das Addieren schwer fiel, sich mehr angestrengt haben, woraus dann unsere Befunde resultierten.

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LINE FOR PURPOSES OF STUDY OR RESEARCH IN LIEU OF OF LENDING THE ORIGINAL

SECTION 10

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Gerd Jansen (Prof. Dr. med. Dr. phil.) Universitätsklinikum Essen 43 Essen 1, Hufeland Str. 55 Federal Republic of Germany		<u>Institution and address where research was performed</u>
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Deutsche Forschungsgemeinschaft (DFG)	
<u>Citation</u> Jansen, G. Physiological responses due to noise in inhabitants around Munich airport. AGARD (Advis Group Aerosp Res Dev) Conf Proc 171: C13-1--C13-5, 1975		
<u># of Ref.'s</u> 0	<u># of Fig.'s</u> 0	<u>Language</u> English
<u>Type & duration of experiment</u> community survey, audiometric testing, psychological testing, pilot study--Hamburg airport area. main study--Munich airport area		<u>Purpose for study</u> The sociological, physiological, psychological impact of aircraft noise on the population surrounding Munich Airport.
<u>Description of test groups (subjects, #, age, etc.)</u> 660 persons, 15-70 years old, living in the vicinity of Munich airport in the first social survey. The subjects involved in the physiological and audiometric experiments and tests included 375 persons from 21 to 60 years.		
<u>Control of other stressors</u> no control--field studies	<u>Statistical Methods</u> curvilinear determination coefficients; regression analysis	
<u>Noise Stimulus</u> source: aircraft noise spectral characteristics: not given noise level: not given--varies length of exposure: varies # of trials: not applicable	<u>CVS Response Measured</u> heart rate blood pressure; fingerpulse amplitude <u>Nonauditory effects</u> most distinctive reaction to noise-- CVS: decreased fingerpulse amplitude reaction; linear relationship found between increasing noise (noise levels & # of flyovers) and reactions in humans--such as blood pressure (increased), heart rate (decreased), annoyance	
<u>Author's conclusions</u> In general, no adaptation to aircraft noise was found. Aircraft noise cannot be ruled out as a risk factor in hypertension.		
<u>Evaluation & comments</u> This is just a brief summary of results from earlier studies reported at the Dubrovnik congress in May 1973. (An interdisciplinary study on the effects of aircraft noise on man, B. Rohmann et al.)		

Jansen, G. Physiological responses due to noise in inhabitants around Munich airport. AGARD (Advis. Group Aerosp. Res. Dev.) Conf. Proc. 171:C13-1-C13-5, 1975.

The results of a series of surveys on the effects of aircraft noise on people who live near large airports are reported in brief summary form. A pilot study was done near Hamburg airport, after which the main study was conducted near Munich airport in East Germany. The studies gathered demographic, sociological, psychological, and physiological data on over 600 people from 15 to 70 years old. Cardiovascular responses to aircraft noise included decreased fingerpulse amplitude, decreased heart rate, and increased blood pressure. These reactions may be related to the annoyance caused by the aircraft noise. The author concluded that there was no evidence of adaptation and aircraft noise cannot be ruled out as a risk factor in cardiovascular disease. A more detailed report of the studies is published in the Dubrovnik congress (May 1973) proceedings by Rohrmann et al. and titled "An Interdisciplinary Study on the Effects of Aircraft Noise on Man."

Physiological Responses Due to Noise
in Inhabitants around Munich Airport

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Summary: The Deutsche Forschungsgemeinschaft (DFG) initiated and sponsored an inter-disciplinary research on aircraft noise effects on inhabitants around airports. A pilot study (around Hamburg airport) and a main study (around Munich airport) were conducted by acoustical, demographic, social scientific, psychological, physiological and medical sections of scientists. It was found out that, in general, there was no adaptation to aircraft noise. There is existing a linear relation between increasing noise stimuli (combined noise exposure measure of noise levels and number of flyovers) and human reactions esp. found in social scientific fields. The discussion of physiological results leads to the opinion that physiological reactions are more related to sound levels whereas the "whole reaction" (annoyance, blood pressure etc.) is more related to combined noise exposure measures.

1. Scope

The effects of aircraft noise on men living around airports were studied by an interdisciplinary team which was sponsored by the Deutsche Forschungsgemeinschaft (DFG). The main study was conducted around Munich airport and the pilot study was done around Hamburg airport.

There are existing some other 15 investigations in the world using survey techniques but they confined to mainly sociological aspects such as complaints or annoyance. Thus DFG initiated than an interdisciplinary research including not only sociological and psychological, but also physiological impact of aircraft noise.

2. Organization of the study

The team of the project was composed of 6 sections:

- "Acoustics" (H.-O. Finke, R. Martin; PTB Braunschweig)
- "Medicine" (A.W. v. Eiff, L. Horbach, H. Jürgens; Uniklinik Bonn)
- "Organization" (B. Rohrmann; Uni Mannheim)
- "Psychology" (H. Gusk, H. Hürmann; Uni Berlin, Uni Bochum)
- "Social-science" (M. Irle, R. Schümer, A. Schümer-Kohrs; Uni Mannheim)
- "Work-physiology" (G. Jansen, Uniklinik Essen)

Each section tested the same subjects to collect data for an interdisciplinary analysis.

Preparing the whole study the organizational section first selected the human beings living around Munich airport according to the exposure to the noise levels and secondly in accordance with demographic criteria. The whole area was divided into 32 areas with different noise levels; these "clusters" were combined to 4 "cluster-sets".

660 persons from 15 to 70 years were tested in a first social scientific step. Interviews based upon standardized questionnaires were taken at the respondents homes. This interview had a contacting function in order to ask the people to follow the second step of our investigation in psychological, physiological and audiometric experiments and tests. The examinations of 357 subjects in a separate test station took about 2 hours for each person.

The third step contained the medical case history, clinical examination and experiments at the test station. This step took another 2 hours for each person. The fourth step was a concomitant one, it consisted of acoustical measurements (one measuring point for each cluster). The tested subjects in the first and in the second step (psychophysiological and medical examinations) were restricted to 373 persons aging from 21 to 60 years.

The interviews of the sociological section were extended in a second part to 152 former inhabitants of the clusters who had moved during the last 12 months preceding the study.

3. Results

3.1. Former publications

The major results of the whole DFG-study were already reported at the congress at Dubrovnik in May 1973. Another survey of the results was given at Inter-Noise (Copenhagen 1973). Especially the relations between acoustic parameters and noise reactions in human beings were presented at the Transportation Noise Symposium (Southampton, July 22-23, 1974). The detailed DFG Forschungsbericht is in print and publication is expected on February 15, 1975.

1.2. Main results of the whole study

According to the assumption of the complex (multicausal instead of monocausal) system of interdependent variables these "moderator variables" were being attributed and regarded as decisive influences on the process of turning effecting stimuli into resulting reactions. This concept of moderators led to an interdisciplinary analysis and synthesis based on an analysis of the single sections. Thus, it is useful to know first the results of the single sections.

1.2.1 Social survey

By means of regression and correlation technics the social science section tried to clarify the relationship between stimulus moderators and reaction and between the relative contribution of stimulus and moderator variables to the prediction of reactions. It was found out that the relationship between stimulus and reaction variables are by no means perfect ones; the highest correlations ranked to $r = 0.36$.

This result means that only about 30 % of the variability in reactions can be predicted by means of one stimulus variable alone so that a considerable amount of the whole reaction remains unpredicted.

As in other aircraft noise studies "disturbance of communication (disturbance in conversation, in listening to radio, TV)" was the greatest one, whereas other ones like "perceived number of times of aircraft noise", "irritability by aircraft noise", "disturbance of tranquillity and relaxation", "the number of subjects spontaneously hearing aircraft noise when asked for interconveniences" etc. they all had smaller correlations.

All these relations are linear. Curvilinear determination coefficients led only to an insignificant increase as compared to linear determination coefficients. Even when correlating more than one stimulus variable no other result could be found.

1.2.2 Psychophysiological experiments

The psychophysiological laboratory experiments were done by the psychologists and work-physiologists together. On one side we had the hypothesis of "adaptive coping" with aircraft noise. On the contrary we tried to find out a "defensive blocking" which assumes an interruption of information processing and physiological state of defensive against noise, as a consequence of frequent and intense aircraft noise. Therefore the investigation was done under the aspect of the "general activation theory" and its possible splitting into "orienting" and "defensive" components.

Moreover it was assumed that damping or disturbance of the information input is in accordance to the "distraction theory"; further on it was expected, there were connections between aircraft noise stimuli and aircraft noise reaction by personal characteristics.

In order to find out the characteristic noise reaction of the people investigated, the psychologists used personality tests, recognition technics, memory tests, signal tracking test, and together with the physiological reaction they registered the behaviour of vasomotoric and muscular activity which were continuously recorded in experimental situations with quietness and noise interchanging.

It was not possible to confirm the hypothesis of "adaptive coping" with aircraft noise. The physiological responses due to noise increased in all cases. In detail, we saw a contraction of the bloodvessels at the finger and at the temple, an increase in the electrical muscle activity, and a decrease of the heart rate. This complex reaction was called "defensive reaction" following SOKOLOFF. One can conclude from this that there could be at least a blocking of information reception processes. These defensive reaction is correlated positively with the intensity and frequency of the aircraft ($r = 0.21$). It occurs especially with those persons who were characterized by a "low mobility", by "strong conservative tendencies" and by a "very high blood pressure".

Moreover we saw that the hearing acuity decreased with increasing aircraft noise exposure. But this result is statistically insignificant. Other respects of human psychophysiological behaviour especially psychological behaviour were not so much affected by aircraft noise.

1.2.3 Medical investigation

The medical examinations were done separately from that of psychophysiological sections after another two weeks. The people were assessed by means of anamnesis and examination of body containing the analysis of clinical status as well as experimental tests of vegetative functions.

The analysis of the medical data could not prove any cause of manifest illness which is due to aircraft noise. In physiological experiments systolic and diastolic blood pressure, heart rate, respiration rate and electrical muscle activity were recorded for 35 minutes. The subjects were submitted to quietness, mental arithmetics, continuous noise, and discontinuous noise. There was only a tendency of change in vegetative functions especially regarding the diastolic blood pressure. The medical scientists have the opinion that it cannot be excluded that aircraft noise is a "risk factor" for the generation of essential hypertonicity of the bloodvessels.

3.3. Interdisciplinary interpretations

The different data from the single sections were integrated (N = 357) to an interdisciplinary analysis which resulted only in low intercorrelations of the sociological, psychological, and physiological variables towards aircraft noise effects.

Using an interdisciplinary set of sociological, psychological and physiological moderators 1/3 of the variability of the "social-psychological" is determined by them whereas another third is determined by the stimulus variables. By using so called path models the scientists doing the interdisciplinary interpretations found chains within one path model containing the factors "indifference to noise", "age", "sex", "fear associations", "attention performance" connecting them with "annoyance and disturbance reaction", "defensive reaction", "diastolic blood pressure" and the dependence of all of them to the noise load.

With reference to the noise protection zones as they are defined in several countries like USA, Great Britain, West Germany, etc. the scientists doing the interdisciplinary analysis found out that outside of the areas confined by this protection values there is a considerable percentage of the population which is highly annoyed and influenced by aircraft noise.

But the regression lines of the "disturbance of communication", "disturbance of rest and recreation" and the "feeling of aircraft as a disturbing factor spontaneously mentioned" were linear regression lines. So there is no point which could be regarded as intolerable noise load. There is only an increasing number of people who feel annoyed and who are influenced physiologically by increasing of aircraft noise. So they conclude that the reduction of aircraft noise is a problem for those producing noise and also for those distributing noise. They feel that it is a problem involving aspects of engineering as well as of policy.

4. Discussion of results from physiological standpoint

Already the pilot study around Hamburg airport showed and proved that the results of former physiological noise research need no basic correction. The experimental physiology results of the Hamburg pilot study (aircraft noise, traffic noise and artificial white noise were applied), showed that the results were comparable to those expected from results of former noise research.

In the main study around Munich airport we tried to find out moderating factors of the physiological responses. These could give explanation of the value of the psychophysiological noise reaction within the total load of environmental factors of the human being. We stated already that the theory of "adaptive coping" had to be cancelled in favour of the "defensive reaction". The combined defensive reaction consisting of changes in finger and headskin blood volume, muscle activity and tracking test, were regularly influenced by single noise bursts. Though the whole defensive reaction is correlated in a linear regression to the combined noise measure FBI (which contains the number of movements and the noise levels of the single movements similar to the English NNI (noise number index)) we saw the most distinctive reaction in the finger pulse amplitudes. Comparing these results with former investigations done with approaching aircraft noise and with the noise reactions of people with different personality moderator variables we think that the physiological measuring parameters are closer correlated to noise intensity level whereas the combined reaction in the physical as well as in the psychical behaviour is more related to the combined noise exposure measure (number of movements and intensity level).

This leads us to the conclusion that for noise assessments around airports it is necessary to have first a combined measurement unit (as they used already internationally) and second (for realistic assessment and protection of the population) to have a maximum level for single noise events.

DISCUSSION

Q. (von Gierke) I agree with Dr. Jansen's statement that we have physiological responses to noise. We also heard in Dr. Cantrell's review about all of the physiological responses to noise which most of us think are very healthy and natural responses to our environment. Unless we have evidence that some of these transient physiological responses become chronic or somehow lead to chronic diseases and pathological effects it is really nothing to worry about. In all the research that I have followed over the past twenty years I have not been able to come up with any clear cut proof that there is a chronic health effect from the level of noise exposure that we are talking about. I am not saying that these effects don't exist, but the only study which shows such a correlation was Dr. Jansen's study, which was cited before, on noise in industry. This was done 15 or 20 years ago and hasn't been replicated since. This study showed a potential indication that noise exposure in industry might be correlated with some increase in cardiovascular disease. However, the same workers in noise that were studied had many other environmental factors associated with their work which might have been just as bad if not worse than the noise itself. Studies have been performed recently on mice and rats that show pathological effects to high level noises but I think we should really concentrate on studies in the real life situation that are made on man rather than on mice and rats. The stories we hear about malformations and reduced fertility in litters of rats and mice are open to some question. First, the noise levels are high. Second, it happens that there are not good controls used. When controls are handled the same way the experimental animals are handled these effects diminish.

A. (Jansen) You mentioned my study of twenty years ago. Yes, it should be replicated and we are just now undertaking experiments in order to find out the relevance of noise along with other factors in producing health effects on workers. We have a group of young men who are doing their research thesis just on this point. Perhaps it is possible within one or two years that we will have the results that you were asking about.

A. (Cantrell) I would like to answer Dr. von Gierke regarding the statement he just made. I hope that from the presentation I gave one didn't infer that there was an attempt to offer any clear-cut proof that there is a patho-physiological effect of noise, but rather that the indication is clear. It is clear in animals. Although we cannot necessarily apply animal studies to humans, nonetheless we do have the human studies of Dr. Jansen as well as studies done in Russia and Europe. Unfortunately, few studies have been done in the U. S. Part of my presentation was a plea for more activity in this problem area. I do feel, however, that prolonged exposure to noise, noise meaning unwanted sound, must act as a stressful stimulus. Theories of the effects of accumulated stress have been present for over forty years. Most people now agree that stress is a factor which causes follow-on patho-physiological problems. The stress of noise, if noise does cause stress, and I think it does, can very well lead to patho-physiological effects. We should study this. Whether we can ever say for certain that noise exposure for a given period of time at a given level is going to cause heart disease or biochemical abnormalities of certain kinds is not likely, but I think that certainly further studies need to be performed and that we agree on this point.

A. (von Gierke) I think it is simpler just to say that noise affects the quality of life and what we want to do is improve the quality of life.

A. (Ward) I disagree with Dr. von Gierke. Improving the quality of life is only one of the things we are after. More importantly, we are interested in the effects of noise on health. Not health as defined by the World Health Organization which includes such things as feelings of well-being. Let's talk about health as absence of pathology. True, we are interested in protecting the public's feeling of well-being in the long run but first let's concentrate on protecting the public from pathology.

A. (von Gierke) Noise is a stress, I agree. But we are exposed to many stresses during the day. Sitting on these chairs for eight hours is a stress and it just depends on how great the stress is. In all seriousness we once tried to follow-up some of Dr. Jansen's work and that of some other workers by obtaining vasoconstrictive responses to vibration stimuli. We exposed the arms of our subjects to vibration. We worked for hours, even days, until we had a nice response of vasoconstriction resulting from localized vibratory stimuli on the skin. We had this effect, finally, and suddenly the pointer went completely off the scale. It turned out that a young woman had walked through the room and our male subject was so stressed that the vaso-constriction from this stimulus was far more violent than from the laboratory vibratory stimulus.

Q. (Olson) How many people moved away from the Munich airport because the noise irritated them?

A. Those who moved did not do so because of noise nor were they found to be more sensitive to noise than was a control group.

Q. (Pardrie) I waited until all the papers were presented before asking any questions because I thought that one of the speakers might discuss the effect of noise on the visual system. For a number of years it is well known that exposure to high intensity noise for several minutes or for several hours can bring about changes in the visual function and thus might endanger flying safety. We have studied the effects of noise of 5,000 Hz, or a complex of 5,000 Hz, the intensity of which ranged between 85 and 105 dB, on the parameters of the visual system and we have found a decrease of about 20% in the night vision capability during exposure to noise. This was determined by measuring the mesopic night thresholds. These are objective values. Secondly, in certain subjects the time to perceive colored lights, as used in aviation, was greatly increased. Thirdly, the time needed to perceive ground relief features, or depth perception, was also greatly increased. It is

important to find out the anatomical location responsible for these interactions. We believe that the thalamus may be the responsible site for interaction between the auditory and visual functions. In fact an afferent activity of the sensory visual and auditory processes (pathways) does take place in the thalamus. Based on electro-cortical studies of the thalamus it may be assumed that an interference (disorder) occurs at this site leading to a diminished passage of the sensory visual messages when noise stimuli traverse the thalamus. Moreover, an inverse study carried out in Italy confirms to a degree these findings. If one exposes the eye to a strong light for several minutes one finds a decrease in the auditory threshold. This proves again an interaction between the auditory and visual sensory messages.

A. There have been many experiments performed in these areas. We have done work in our own laboratory on these problems. I think that it is not justified to generalize from these experiments to the real life situation, for one must consider man has motivation and has capability to compensate and these parameters must be taken into account. What one needs to do is to make experiments under real life conditions rather than laboratory conditions. From the experimental situation we know many things, but it is very dangerous to generalize from experiments to the real life situation.

SECTION 11

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Anders Jonsson; Volvo Inc.; Gothenburg, Sweden. Lennart Hansson; Dept. of Medicine 1; Sahlgren's Hospital; S-41345 Gothenburg, Sweden.	<u>Institution and address where research was performed</u> 1. Volvo Inc.; Gothenburg, Sweden
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> 1. Volvo Inc.; Gothenburg, Sweden 2. Bayer Farma; Sweden
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Citation
 Johnsson, A. and L. Hansson. Prolonged exposure to a stressful stimulus (noise) as a cause of raised blood pressure in man. The Lancet 1(8002):86-87, January 8, 1977.

# of Ref.'s 9	# of Fig.'s 2	Language English
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<u>Type & duration of experiment</u> Field survey - in auto factory Duration - not specified	<u>Purpose for study</u> to test whether noise-included irreversible hearing loss is associated with a permanent blood pressure (BP) increase.
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Description of test groups (subjects, #, age, etc.)
 196 male automobile factory workers divided into three groups:
 1. Tests - 44 with noise-induced hearing loss
 2. Controls - 74 with normal hearing (same age)
 3. 78-miscellaneous types of hearing damage (eliminated from survey).

<u>Control of other stressors</u> No control - factory conditions	<u>Statistical Methods</u> Students t-test - to compare BP in both groups. chi-square test - to compare no. of hypertensives
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<u>Noise Stimulus</u> source: auto factory machine noise spectral characteristics: not analyzed noise level: greater than 85 dB length of exposure: several years # of trials: not applicable	<u>CVS Response Measured</u> Blood pressure (BP) measured after 15 min. rest. <u>Nonauditory effects</u> CVS: The average BP was higher and significantly more workers had hypertension in the group with permanent hearing loss. Auditory effects: Audiograms showed irreversible hearing losses in 44 out of 196 auto workers. Degree of loss: 65 dB or more at 3000, 4000, or 6000 Hz.
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Author's conclusions Long exposure to stress, such as noise, may cause repeated increases in blood pressure that can lead to a permanent increase, due to a blood vessel adaptation. Noise-induced hearing loss was associated with hypertension in the auto workers, but no direct proof exists that the noise caused the blood pressure rise in this group.

Evaluation & comments
 Sample groups too small - less than 100 per group.

Jonsson, A. and L. Hansson. Prolonged exposure to a stressful stimulus (noise) as a cause of raised blood pressure in man. The Lancet 1 (8002): 86-87, Jan. 8, 1977.

A survey of 196 male automobile factory workers was conducted to see if there was a permanent blood pressure increase in people exposed to noise levels high enough to induce permanent hearing damage. During routine physical exams, audiograms were performed in a sound-proof room. Out of the 196 subjects, 44 had a noise-induced hearing loss (greater than 65 dB at 3000, 4000, or 6000 Hz) and 74 subjects of the same age had normal hearing. Graphs of the loss of hearing in dB at each frequency level tested are included. The other 78 subjects had miscellaneous degrees of hearing loss and were excluded from the blood-pressure study. Blood pressures were measured once after 15 minutes rest in the 74 subjects with normal hearing and in the 44 subjects with severe hearing loss. The average systolic and diastolic blood pressures were higher (significant at P less than 0.0001 using students t-test) in the subjects with hearing loss than in those with normal hearing. There was also a greater proportion of hypertensive subjects (significant at P less than 0.05 using chi square test) in the hearing-impaired group. The results indicate that prolonged exposure to noise, which is strong enough to cause permanent hearing damage, may be a cause of a permanent blood pressure increase due to blood vessel adaptation to repeated stress.

variably showed a significant increase in the amount of acetone, and in the case of adhesive inhalation a significant response also for toluene (fig. 4b).

DISCUSSION

During the last few years there has been growing concern in the west of Scotland over the increasing number of young people who inhale solvent-based materials "for kicks". The results presented show that males predominantly engage in the practice. The age-group involved is 12 to 19 with a mean of 14.9 years. More than half (54%) were between 14 and 16 years of age.

Although most (98%) cases were referred during the week these figures could be incomplete or even biased for two reasons. Firstly because "sniffing for kicks" is predominantly a group activity so that a clustering of cases in certain areas at certain times would almost certainly occur. Secondly, the source of referral in each case was the police who might well be fully occupied at weekends with accidents, alcoholics, &c., and therefore less likely to detect sniffers in a district. On the other hand the figures might represent a natural decline in sniffing practices at weekends, possibly because of the availability of other interesting and less deviant activities—e.g. football and youth clubs.

The observed clustering of cases which occurred during the morning, afternoon, and evening further supports our belief that this is, in the main, a group activity. At first glance, it seemed as if the absence of referrals at periods during the day might coincide with police meal times. However, this was found not to be the case. We can only assume that the drop in reported episodes reflected the children's meal hours, and that it represented not only an evening social activity but for almost half the cases an alternative to school attendance. It was not possible to determine from the information available whether truancy in these cases pre or post dated the sniffing history.

Although a wide variety of solvent-based materials in common domestic use is readily available to would-be sniffers, in practice 84% had chosen proprietary brands of adhesives. The method of inhalation showed even more conformity and undoubtedly reflected previous experience with solvents. It was interesting to note that the acute effects of these solvents most closely resembled alcoholic intoxication.

The analytical technique developed was rapid, reliable, and simple. Glues and other substances could be rapidly screened for suspected solvents thus confirming the formula stated by the manufacturer, although with respect to the wishes of certain manufacturers we have not listed the solvent formulations of the products investigated. The most popular adhesives involved were found to contain both acetone and toluene. The analysis of the blood-samples from sniffers readily detected these solvents.

Although this investigation has added to our current knowledge about the sniffers, the substances, the solvents, and the syndromes, it indicates a need for more factual information about the effects and toxicity of glues and other freely available solvent-containing materials whose vapours could be inhaled "for kicks".

We thank Miss Nasreen Usman for her technical assistance.

Requests for reprints should be addressed to J. S. O., Department of Forensic Medicine, The University, Glasgow G12 8QQ.

References at foot of next column

Occupational Health

PROLONGED EXPOSURE TO A STRESSFUL STIMULUS (NOISE) AS A CAUSE OF RAISED BLOOD-PRESSURE IN MAN

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Summary Systolic and diastolic blood-pressure was significantly higher in 44 male industrial workers with a noise-induced auditory impairment (>65 dB at 3000, 4000, or 6000 Hz) than in 74 males of the same age with normal hearing. Moreover, significantly more individuals with hypertension (resting recumbent blood-pressure $\geq 160/100$ mm Hg) were found in the group with noise-induced loss of hearing. It is suggested that repeated and prolonged exposure to a stressful stimulus (industrial noise severe and prolonged enough to cause a permanent loss of hearing at the relevant frequencies) may be a contributing factor to the rise in blood-pressure through a mechanism involving structural adaptation of blood-vessels in response to repeated peaks of raised blood-pressure.

INTRODUCTION

SEVERAL kinds of mental stress are associated with a temporary rise in blood-pressure.¹⁻³ In animal experiments it has been shown that repeated exposure to an alerting stimulus can cause a permanent rise in arterial pressure.⁴ Such a relationship between repeated stressful stimuli and a permanent rise in blood-pressure is obviously not so easy to establish in man, partly due to the fact that environmental stress is difficult to measure and quantitate.

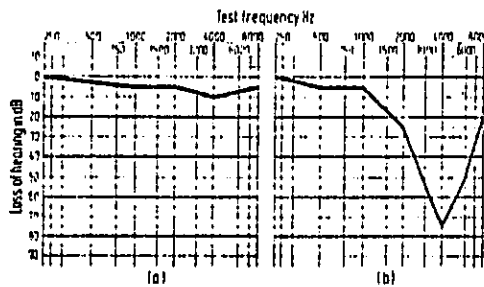
The purpose of the present investigation was to determine whether a permanent rise in blood-pressure had occurred in individuals exposed to noise severe and prolonged enough to cause an irreversible loss of hearing.

SUBJECTS AND METHODS

The subjects were 196 male industrial workers. At a routinely performed health examination an audiometry test was done for each subject. The audiometry test was performed in a sound-proof room with a standard tone audiometer connected to earphones. Hearing was tested at the following frequencies: 250, 500, 1000, 2000, 4000, 6000, and 8000 Hz. A normal audiogram was defined as a loss of acuity less than 20 dB at all frequencies tested. A severe noise-induced impairment of hearing was defined as a loss of acuity amounting to 65 dB or more at 3000, 4000, or 6000 Hz (see accompanying figure). Individuals with an impairment of hearing ≤ 65 dB but > 20 dB at these frequencies or a loss of hearing at other frequencies were excluded from the study.

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(A) A normal audiogram; (B) an audiogram showing a noise-induced auditory impairment with the characteristic "dip" at 4000 Hz.

Blood-pressure was measured in the recumbent position after the subject had rested for fifteen minutes. A mercury sphygmomanometer with a cuff containing a 13 x 30 cm rubber bag was used. Phase v (disappearance) of the Korotkoff sounds was taken as the diastolic blood-pressure. Blood-pressure was read to the nearest 5 or 10 mm Hg. The same nurse measured all the blood-pressures and an audiometry assistant did all the audiometry tests. They were both unaware of the purpose of the investigation.

Upon completion of these examinations 74 subjects were found to have normal hearing whereas 44 had a severe noise-induced loss of hearing. The remainder, 78 subjects, had various forms of impairment of hearing and constituted the "miscellaneous group" which was not further analysed.

Student's *t* test was used to compare systolic and diastolic blood-pressures in the two groups. The χ^2 test was used to compare the number of hypertensive individuals (defined as recumbent blood-pressure > 160/100 mm Hg after fifteen minutes of rest) in the two groups.

RESULTS

The average systolic and diastolic blood-pressures were significantly higher in subjects with a noise-induced loss of hearing than in subjects with normal hearing (see accompanying table). There were also significantly more hypertensive individuals in the group with impaired hearing than in the one with normal hearing (table).

DISCUSSION

There are three main findings in this study. First, out of 196 male industrial workers 44 had a severe loss of hearing at either 3000, 4000, or 6000 Hz but not at other frequencies indicating that this impairment was due to noise.⁵ Usually, prolonged exposure (several years) to severe noise (> 85 dB) is needed to cause

damage of this severity.⁶ Secondly, resting blood-pressure was significantly higher ($p < 0.0001$) in these men compared to 74 men of the same age but with normal hearing. Finally, the proportion of hypertensive subjects was significantly higher ($p < 0.05$) in the group with noise-induced loss of hearing as compared to the group with normal hearing.

Industrial noise can undoubtedly be regarded as a stressful stimulus, occasionally being powerful enough to cause pain. It is well known from animal studies that brief exposure to noise can cause a rise in blood-pressure.⁷ Exposure to noise has also been used as one of several means to raise blood-pressure acutely in normotensive and hypertensive subjects.⁸ However, little is known about prolonged exposure to noise and its effect upon blood-pressure in man. Noise exposure in these individuals had occurred during previous employment in, for example, shipyards or mechanical workshops. Obviously, it cannot be claimed with absolute certainty that exposure to noise caused the increased blood-pressure or the higher rate of "hypertension" observed in the group with noise-induced auditory impairment. On the other hand there were no other obvious differences between the two groups that could easily explain our findings.

Speculations that individuals with a genetic predisposition to develop hypertension are also more susceptible to noise or that such individuals seek out noisy or stressful jobs are not well founded and seem improbable. A more logical assumption is that repeated severe exposure to noise causes repeated rises in blood-pressure. This in turn could be expected to cause a permanent rise in blood-pressure in analogy with findings in animals repeatedly exposed to alerting stimuli.⁹ In addition individuals with a genetic predisposition to develop hypertension may "hyper-react" to stressful stimuli according to findings by Hallböök who described a more pronounced rise of blood-pressure in young "prehypertensive" spontaneously hypertensive rats as compared to normotensive rats.⁹ Exposure to repeated peaks of blood-pressure could then be expected to cause a permanent rise in blood-pressure due to structural adaptation of the heart and resistance vessels.

We therefore feel that the most reasonable explanation to the presented findings is that prolonged exposure to a stressful stimulus may have caused repeated rises in blood-pressure leading to circulatory adaptations and a permanent rise in blood-pressure. We intend to follow up these preliminary findings with detailed examinations of larger numbers of individuals exposed to industrial noise.

We thank Mr L. E. Petersson, department of statistics, University of Gothenburg, for the statistical analyses, and we gratefully acknowledge the skilful help of Ulla Josefsson and the support provided by Volvo Inc. and Haver Farms of Sweden.

Requests for reprints should be addressed to L. H., Department of Medicine I, Sahlgren's Hospital, S-413 45 Gothenburg, Sweden.

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COMPARISON BETWEEN SUBJECTS WITH NORMAL HEARING AND THOSE WITH A >65dB REDUCTION OF HEARING AT 3000, 4000, OR 6000 HZ

	Normal acuity (N 74)	Noise-induced impaired acuity (N 44)	Significance
Age (yr)	54 (41-66)	57 (41-66)	N.S.
Systolic blood-pressure*	132.6 ± 2.6	145.2 ± 1.3	$p < 0.0001$ †
Diastolic blood-pressure*	80.6 ± 0.8	88.6 ± 1.7	$p < 0.0001$ †
Number of hypertensives	6	10	$p < 0.05$ ‡

* Mean ± S.E.M.

‡ χ^2 test.

† Student's *t* test for paired observations.

N.S. = Not significant.

SECTION 12

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Dr. L.P. Lipsitt (Prof. of Psychology and Medical Science, Brown Univ., Providence, R.I. 02912) Mr. Steven Kittner	<u>Institution and address where research was performed</u> Brown University Providence, R.I.
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> U.S.P.H.S. (Public Health Service); Grant Foundation
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Citation | Kittner, S. and L.P. Lipsitt. Obstetric history and the heart-rate response of newborns to sound. Dev. Med. Child Neurol. 18(4):460-470, Aug., 1976

# of Ref.'s 3	# of Fig.'s 9	Language English
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<u>Type & duration of experiment</u> Laboratory--in sound-attenuated chamber; 18 min. per subject	<u>Purpose of study</u> to see if heart-rate response to noise can be used to identify neurological and behavioral problems in human infants
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Description of test groups (subjects, age, etc.)
 16 essentially normal newborns divided into 2 groups:
 (1) 4 males, 4 females--lesser number of non-optimal birth conditions (low-risk)
 (2) 4 males, 4 females--greater number of non-optimal birth conditions (high-risk)

<u>Control of other stressors; drugs given</u> the mother pre-delivery were recorded; obstetrical difficulties during delivery were recorded	<u>Statistical Methods</u> Mann-Whitney U-test; 2-tailed correlated t-test
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<u>Noise Stimulus</u> source: Hewlett-Packard Model 3300A audio os- cillator connected to an electro-voice speaker spectral characteristics: 700 Hz (habitua- tion); 300 Hz (dishabituation) noise level: background: 73 dB noise stimulus: 85 dB square wave length of exposure: 5 sec. tone # of trials: 20 trials per subject: 5 sec. tone; 25 sec. quiet	<u>CVS Response Measured</u> heart rate (ERG) heart rate acceleration and deceleration <u>Nonauditory effects</u> no significant differ- ence in heart rate response to noise over CVS; trials between groups; significant diff. was noted in averaged heart rate acceleration and deceleration: <u>high-risk group</u> --more heart rate acceleration; low-risk group-- more deceleration
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Author's conclusions | Heart rate responses to noise may be useful in identifying infants with psychological problems, but no conclusive evidence is available. Obstetric history bears some relation to neurological problems, which might be detected using this cardiovascular response.

Evaluation & comments | This demonstrates some of the vegetative-type responses to acoustical stimuli in infants.

Kittner, S. and L. P. Lipsitt. Obstetric history and the heart-rate response of newborns to sound. *Dev. Med. Child Neurol.* 18(4):460-470, Aug. 1976.

Heart rate responses to noise of normal infants with either high-risk or low-risk obstetrical histories were studied. High-risk infants had more adverse factors during their gestation and birth, such as abnormal intra-uterine position, a cord abnormality, caesarean birth, or adverse maternal factors (such as drugs taken). The experiment was designed to see if the heart rate response to noise could be used to identify potential neurological and developmental problems. The subjects were 16 newborns, of which 8 were a low-risk group and 8 were a high-risk group. There were 4 males and 4 females in each group. The infants were tested while in a crib in a quiescent state inside a sound-attenuated chamber. The sound source was a Hewlett-Packard Model 3300A audio oscillator connected to an Electro-voice speaker. A background noise of 73 dB was present, due to the ventilation system. The noise stimuli included a habituation tone at 700 Hz and a dishabituation tone at 300 Hz. The sound level was 85 dB and the duration of the noise stimulus was a 5 second tone. The heart rates were monitored by EKG during the 18 minute experimental session for each infant. Each session consisted of 2 minutes quiet, a 10 minute period of 18 habituation trials and 2 dishabituation trials, and 2 minutes quiet. Each trial included a 5 second tone and then a 25 second interstimulus interval. Values were recorded for the highest and lowest heart rates in the pre- and post-stimulus periods. Acceleration scores were determined by subtracting the highest pre-stimulus heart rate from the highest post-stimulus heart rate. Deceleration scores were computed by subtracting the lowest post-stimulus heart rate from the lowest pre-stimulus heart rate. The expected response for the high-risk infants was a delayed habituation, indicated by an accelerated heart rate (which may be a defensive reaction to noise). Low-risk infants were expected to show the opposite effect of habituation to the noise (possibly an orienting reflex). Neither group of infants was definitely habituated to the noise. No significant difference in heart rate response over trials between groups was found. When averaged acceleration and deceleration scores were considered, high-risk infants showed significantly more acceleration and less deceleration than the low risk group.

Obstetric History and the Heart-rate Response of Newborns to Sound

Steven Kittner Lewis P. Lipsitt

Introduction

It has been suggested that the inability to adequately evaluate the behavioral and neurological condition of a newborn infant is perhaps the greatest deficiency in perinatal medicine (Gluck 1974). Evaluative techniques are needed, not only by those concerned with the continuing welfare of the child, but also by the obstetrician and anesthesiologist who wish to know the consequences of their procedures.

Parmelee *et al.* (1974) developed a comprehensive approach to this problem, in which behavioral performances as well as pregnancy, perinatal, and neonatal biological events are included in the initial risk assessment of each newborn. The present study is an attempt to develop an additional behavioral measure for such a larger assessment battery.

The study was concerned with the relationship between obstetric history and newborn behavior. Its purpose was to determine whether two groups of infants differing in obstetric history would also differ in their heart-rate response to a series of auditory stimuli during the first few days of life.

The heart-rate response to auditory stimuli was selected as a test item for three reasons. First, the newborn response to

auditory stimulation may be an important factor in the mother-infant interaction. It has been reported that newborns can respond to their mother's voice by head-turning as early as the third day of life and can distinguish this voice from other voices (André-Thomas and Autgaerden 1966, Hammond 1970).

Secondly, studies employing other dependent measures have suggested that habituation may be delayed in newborns who have suffered perinatal complications (Bronstein *et al.* 1958, Eisenberg *et al.* 1966). Schulman (1970) compared the heart-rate response to auditory stimuli of low-risk and high-risk pre-term infants and found a difference in the latency of the response. Although a significant decrement in the accelerative heart-rate response between trials 1 to 5 and 26 to 30 was found for both groups, Schulman did not use a novel auditory stimulus at the end of the procedure and hence it cannot be determined whether both groups habituated according to the definition of Thompson and Spencer (1966), which requires a test of dishabituation. The design of the present study allows one to check for differential habituation of the heart-rate response between two groups.

Thirdly, the direction of the heart-rate

response is variable. D (1959) and sive review response in and Clifton tion is a co and is asse contrast, t gested to b reflex asso Much wo interpreted (Graham a Clifton 19 there is ev heart-rate stable indi period (Cl 1972. Th present ex be increa decelerati a difficult those with

The nu conditions was used each birth on the bas delivery a

Specific medicatio obtained, expected study of Aleksand know wh responsiv ante-part factors, variation

If the stimulati disparate drug eff

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response is also an important dependent variable. Drawing upon the work of Lacey (1959) and Sokolov (1963) and an extensive review of the research on heart-rate response in adult human subjects, Graham and Clifton (1966) suggested that deceleration is a component of the orienting reflex and is associated with stimulus intake. In contrast, cardiac acceleration was suggested to be a component of the defensive reflex associated with stimulus rejection. Much work with newborns has been interpreted in terms of these suppositions (Graham and Jackson 1970, Kearsley 1973, Clifton 1974, Porges 1974). In addition, there is evidence that the direction of the heart-rate response to stimulation is a stable individual difference in the newborn period (Clifton and Graham 1968, Jacklin 1972). Thus another hypothesis of the present experiment was that there might be increased acceleration and decreased deceleration responses in infants who had a difficult birth history compared with those with a relatively benign history.

The number of non-optimal obstetric conditions from the list of Prechtl (1968) was used as a measure of the difficulty of each birth. Prechtl selected these variables on the basis of the risk of mortality during delivery and the first two weeks of life.

Specific information on the pre-delivery medication of the mother of each child was obtained, since drug conditions might be expected to have extraordinary effects in a study of this type (Bowes *et al.* 1970, Aleksandrowicz 1974). It is important to know whether an infant's psychobiological responsivity is determined principally by ante-partum drugs, by multiple obstetric factors, or by individual genotypic variation.

If the heart-rate response to auditory stimulation differentiates newborns having disparate birth histories, independently of drug effects, strong support would be

given to the further study of this psychobiological measure as a newborn assessment item. Positive findings would also contribute towards an understanding of individual differences in newborn behavior.

Method

Sixteen newborns born at the Women and Infants' Hospital of Rhode Island were studied. The infants, all of them bottle-fed, were selected on the basis of the number of non-optimal obstetric conditions from Prechtl's list (Table 1) entered in the infants' hospital records.

The following categories of infants were excluded: (1) infants of less than 38 weeks gestational age or weighing less than 2495g (5.5lb) at birth; (2) infants whose clinical condition at birth was such that they were placed in special-care nurseries; and (3) infants whose parents were living outside the Providence area, because of inconvenience for possible follow-up studies. The first categories prevented testing of infants who were pre-term, small for gestational age, or who had any known or suspected abnormality. Therefore the study was of essentially normal newborns, with greater and lesser recorded indications of 'perinatal hazard'. No infant was accepted for study without the written consent of the mother and the child's pediatrician.

The selected newborns comprised two groups: a 'Low Prechtl' group (LPr) with three or fewer non-optimal conditions, and a 'High Prechtl' group (HPr) containing newborns with seven or more non-optimal conditions. There were eight infants in each group, four male and four female. Interestingly, the HPr and LPr groups had significantly different Apgar scores at 1 minute ($t = 2.38$, $df = 14$, $p < .05$), but this difference was reduced to non-significant effect at 5 minutes. Table II summarizes the characteristics of each group.

TABLE I
Non-optimal obstetric conditions*

<i>Maternal Factors</i>	
1.	Maternal age primipara <18 or >30, multipara <20 or >30 yrs
2.	Unmarried, divorced or widowed
3.	Parity 0 or >6
4.	Abortions >2
5.	Pelvic disproportion
6.	Luetic infection
7.	Rh antagonism
8.	Blood-group incompatibility
9.	Nutritional state poor
10.	Hemoglobin level <12g/l
11.	Bleeding during pregnancy
12.	Infection during pregnancy
13.	Abdomen X-rayed during pregnancy
14.	Toxemia, moderate or severe
15.	Blood pressure >135/90
16.	Albuminuria and edema
17.	Hyperemesis
18.	Psychological stress
19.	Unwanted sterility >2 years
20.	Maternal chronic diseases
<i>Parturition</i>	
21.	Twins or multiple birth
22.	Delivery induced or by caesarean section
23.	First stage duration <6 or >24hrs
24.	Second stage duration <10min or >2hrs
25.	Inadequate contractions
26.	Drugs other than O ₂ or local anesthetic given mother
27.	Meconium-stained amniotic fluid
28.	Membranes broken >6hrs
<i>Fetal Factors</i>	
29.	Intra-uterine position other than vertex
30.	Gestational age <38 or >41wks
31.	Fetal presentation other than vertex
32.	Cardiac irregularity
33.	Fetal heart-rate (second stage) <100 or >160
34.	Cord around neck other than loosely
35.	Cord prolapse
36.	Knot in cord
37.	Placental infarction, moderate or severe
38.	Respiration onset <1min
39.	Resuscitation given
40.	Drugs given
41.	Body temperature other than normal
42.	Birth weight <5.5 or >12.5 lb (<2495g or >5670g)

*Adapted from Prechtl (1968) pp. 306-307

Maternal Pre-delivery Medication

Table III shows the drug weighting system used in the present experiment. This scoring system is a modification of that used by Stechler (1964) and by Standley *et al.* (1974). For each drug administration, the dose weighting factor (d) is multiplied by the appropriate time weighting factor (t) to obtain the drug-by-

time (d x t) score. A narcotics (N) and barbiturate (B) subtotal (N + B subtotal) was obtained for the d x t score of each mother's drugs since, of the drugs found in this study, only narcotics and barbiturates have a documented effect on newborn behavior (Kron *et al.* 1966, 1968; Brackbill *et al.*, 1974a,b). A total drug score was obtained for each mother by

Prechtl score
Apgar score
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Birthweight
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Weighting factor

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TABLE II
Mean and standard deviations of the low and high Precht groups

Characteristic	Precht group			
	Low (n = 8)		High (n = 8)	
	M	SD	M	SD
Precht score	2.13	.99	10	2.27
Apgar score (1min/5min)	8.24/9.13	1.04/.35	7.25/8.75	.46/.46
Parity	1.38	.92	0	
Birthweight (lbs)	7.71	1.04	7.32	.68
Estimated gestational age (wks)	39.88	.35	39.93	1.17
Postnatal age at testing (hrs)	38.41	15.94	61.73	18.70

TABLE III
Maternal pre-delivery medication: drug and time weighting systems

Weighting factor	Drug dose weighting system (d score: d = dose) Drugs and dosage levels
	Narcotics and barbiturates
	Meperidine hydrochloride ('Demerol'), secobarbital ('Seconal'), pentobarbital ('Nembutal')
1	d < 100mg
2	d > 100mg
	Promethazine hydrochloride ('Phenergan')
	Promazine hydrochloride ('Sparine')
1	d < 50mg
2	d > 50mg
	Scopolamine hydrobromide ('Scopolamine')
1	d < .5mg
2	d > .5mg
1	Other drugs:
	Levallorphan tartrate ('Lorphan'), prochlorperazine ('Compazine'), diazepam ('Valium'), atropine sulfate ('Atropine')
	Time weighting system (t score: t = time before delivery)
	Time of administration before delivery
1	t > 8hrs
2	4hrs < t < 8hrs
3	1.5hrs < t < 4hrs
4	t < 1.5hrs

adding all the individual $d \times t$ scores. The range of total drug scores for the LPR group was 0 to 20 and for the HPR group it was 4 to 21.

The mothers of most infants received saddle anesthesia (five LPR and four HPR). The remainder received local (one), spinal (three), general (one) or no anesthesia (two). These sample sizes were too small for analysis of the relationship of anesthesia to the neonatal heart-rate response to auditory stimulation.

Apparatus

The infants were tested in a crib housed within a 6ft x 7ft x 11ft sound-attenuated chamber. While noise from a Grason Studler Model 901B noise generator, and background noise from the air ventilation system produced a constant sound intensity of 73dB measured at the infant's head.

Respiration was monitored by a Phipps and Bird infant pneumobelt strapped around the abdomen. An electrocardiogram

(EKG) was obtained by means of three Hewlett-Packard electrodes, two placed across the chest and one on the leg as a ground. A beat-by-beat measure of heart-rate was obtained using a Narco Bio-Systems Biotachometer (Model BT-1200). These three measures were continuously recorded on a Grass Model 5D polygraph.

The auditory stimulus for this study was produced by a Hewlett-Packard Model 3300A audio oscillator connected to an Electro-voice speaker (Model PA7).

Procedure

The infants were tested between 10.45 and 11.45 am, between the feedings which occurred at about 9.30 and 1.00. Each infant was brought from the nursery to the laboratory by a nurse, who attached EKG electrodes and the pneumobelt. The infant was then swaddled and placed on its left side to allow full view of the infant's face by the nurse and experimenter. Crying was the only behavioral state which excluded an infant from the experiment: for the most part the infants were in a quiescent state throughout but could have been awake or asleep, with eyes open or closed.

After calibration of the equipment there was a two-minute baseline recording period, a 10-minute auditory habituation procedure, and a second two-minute baseline period. The total time taken with each infant was approximately 18 minutes.

During the two baseline periods only heart-rate and respiration were recorded. The auditory habituation procedure consisted of 18 habituation trials and two dishabituation trials. Each trial consisted of a five-second tone followed by a 25-second interstimulus interval. The habituation tone was a 700Hz, 85dB square wave sound. The dishabituation tone was a 300Hz sound of the same dB level and wave form.

Data Analysis

The values used for this experiment are the highest instantaneous rate (the shortest interval between beats) and the lowest instantaneous rate (the longest interval between beats) in the 10-second periods preceding and following the initiation of the auditory stimulus. These values will be referred to as prestimulus-high, prestimulus-low, poststimulus-high and poststimulus-low (Fig. 1).

From these data, acceleration and deceleration scores in beats per min (bpm) were computed for each stimulus trial for each infant. Acceleration scores were calculated by subtracting the highest heart-rate in the 10-second prestimulus period from the highest heart-rate in the 10-second poststimulus period. Deceleration scores were computed by subtracting the lowest heart-rate in the 10-second poststimulus period from the lowest heart-rate in the 10-second prestimulus period. It is important to note that the acceleration and deceleration scores were computed from two different sets of data, hence it would be possible for both a positive acceleration score and a positive deceleration score to occur on a single trial.

The data were uncorrected for initial level effects (Lacey and Lacey 1962, Wilder 1967, Graham and Jackson 1970, Steinschneider 1971) because there were no significant differences between the groups in pre-experimental basal heart-rate (two-minute sample), nor in their prestimulus-low and prestimulus-high values (averaged across the first 18 trials). Using the Mann-Whitney U test (Siegel 1956) with eight infants in each group, the U values were 31, 25 and 26 respectively.

Results

Figures 2a and 2b show the group trends of acceleration and deceleration scores across the 20 stimulus trials. (The acceleration and deceleration values for the two



Fig. 1. Polygraph respiration, EKG and deceleration scores upon pre-an

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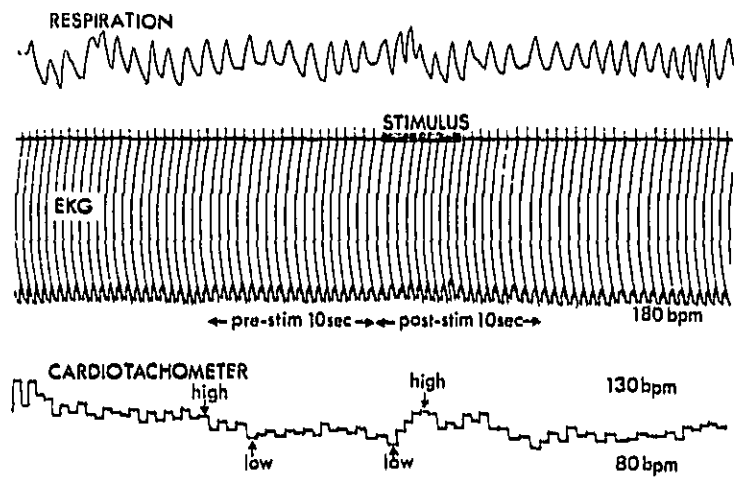


Fig. 1. Polygraphic record showing method of recording heart-rate response to sound. Top channel records respiration, second channel is electrocardiogram and third channel is cardiometer transformation of electrocardiogram inter-beat intervals into momentary (beat by beat) heart-rate. To obtain acceleration and deceleration scores, the 10-second pre-stimulus period is compared with the 10-second post-stimulus period. In this example the post-stimulus low heart-rate is 94 beats per minute, compared with a pre-stimulus low of 100 beats per minute; thus deceleration score is + 6. Acceleration score was + 4, based upon pre- and post-stimulus highs of 112 and 116, respectively.

groups of infants are presented in the same figures for purposes of comparison.) It can be seen that in comparison to the LPr group the HPr group shows consistently larger acceleration and smaller deceleration scores across the habituation trials.

In order to characterize these group differences further, each infant's acceleration and deceleration response was averaged across the first 18 trials. As expected, these group differences are highly significant (Table IV).

Table V shows the results of tests for a trials effect by comparing the average heart-rate acceleration and deceleration scores of trials 1 to 3 with those of trials 14 to 18 for each group. A two-tailed correlated *t* test showed no significant trend in acceleration scores for either

group. However, a significant decrement in deceleration scores was found for both the LPr group ($p < .001$) and the HPr group ($p < .02$).

Because the HPr group increased in its acceleration response from the early to the later trials, while the LPr group decreased, the suggestion of such an interaction was examined statistically by calculating a difference in each infant's acceleration response from early to later trials. The two distributions of differences were then compared by a Mann-Whitney *U*-test to determine whether these distributions were different for the two groups: they were not.

Although there was a significant decrement across trials for the deceleration scores, there was little recovery of the deceleration response to the novel stimulus

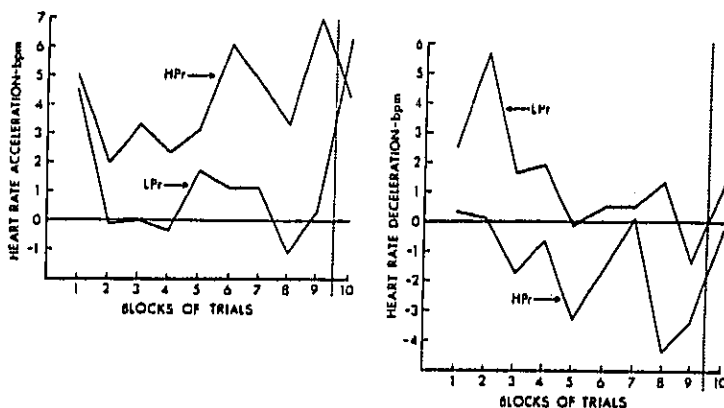


Fig. 2. Trends across trials of heart-rate acceleration and deceleration to auditory stimuli. (a) Left-hand graph shows average acceleration score in each block of two trials for Low Prechtl (LPr)

on trials 19 and 20 (see Figure 2).

Finally, the product moment correlations of heart-rate acceleration and deceleration scores with Prechtl score and other selected variables are shown in Table VI. Only the first two variables yielded significant correlations. The Prechtl score showed the strongest relationship to the deceleration scores, while maternal parity or the number of previous live births had the strongest relationship to the acceleration scores. It may be noted that the Apgar scores did not relate reliably to either

and High Prechtl (HPr) groups. (b) Right-hand graph shows deceleration scores in the same manner.

acceleration or deceleration. This finding, coupled with the fact that the Apgar scores were reliably different only at 1 minute, suggests that the Apgar rating is not as effective in separating infants at risk as is the Prechtl classification scheme, at least in essentially normal, surviving babies.

Discussion

Heart-rate response trends over trials. The HPr group was expected to show a delayed habituation compared with the LPr group. The results of this experiment

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TABLE IV
 Averaged heart-rate acceleration and deceleration scores and comparison of low and high Prechtl groups

	Averaged heart-rate response (beats/min)*			
	Acceleration Prechtl group		Deceleration Prechtl group	
	Low	High	Low	High
Mean	.81	4.06	1.29	1.68
Standard deviation	2.28	2.08	1.02	1.77
Mann-Whitney U test (two-tailed)	U = 8, p = .01		U = 5, p = .002	
t Test (two-tailed)	t = 2.97, p < .02		t = 4.13, p < .01	

*Averaged across trials 1 to 18.

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TABLE V
Comparison of trials 1 to 5 with trials 14 to 18 for heart-rate acceleration and deceleration scores

	Averaged heart-rate response (beats/min)		t correlated	p
	Trials 1-5	Trials 14-18		
Acceleration score				
Low Prechtl group	1.30	-.85	.998	N.S.
High Prechtl group	3.60	5.55	1.314	N.S.
Deceleration score				
Low Prechtl group	3.75	-.05	4.189	< .001
High Prechtl group	-.55	-3.00	2.295	< .02

do not clearly support a finding of habituation of heart-rate responses in either group. Although Table V does show a significant decrement in the deceleration scores for both groups, there was no significant recovery of the deceleration response on trials 19 and 20 for either group; thus the minimal criteria of habituation (Thompson and Spencer 1966) have not been satisfied.

Direction of the heart-rate response. Although there were no significant differences between the two groups across trials, there were significant differences between the two groups for both acceleration and deceleration. The LPR group had smaller acceleration and larger deceleration scores than the HPR group. The hypothesis that the Prechtl groups represent populations having differential receptivity to stimula-

tion is supported; the group having a relatively easy birth history showed more signs of orienting to auditory stimulation during the first few days of life. Studies testing different modalities with different response measures such as a visual fixation (Sigman *et al.* 1973) would be valuable for corroborating the significance of these results.

Relationship of heart-rate response to obstetric variables. Only Prechtl score and maternal parity showed a significant relationship to acceleration or deceleration scores (see Table VI). That parity should have a comparable correlation to the total Prechtl score is not surprising, since all the HPR infants were born of primiparous mothers, while all but one LPR subject were born of multiparous mothers. Being a

TABLE VI
Correlation of selected variables with heart-rate response to auditory stimuli for all infants

Variable	Heart-rate response scores	
	Acceleration	Deceleration
Prechtl score	-.480	-.63**
Maternal parity	-.52*	.49
Apgar score		
1 minute	-.12	.33
2 minute	-.35	-.03
Age at testing	.28	-.13
Maternal pre-delivery medication		
narcotics and barbiturates	.10	-.02
all drugs	.31	.08
time†	-.28	-.39

*p < .05, two-tailed test

**p < .01, two-tailed test

†Time between first pre-delivery medication and birth.

primiparous birth counts as a non-optimal obstetric condition on the Prechtl scale.

Parity has previously been suggested to co-vary both with the anxiety of the mother (Copans 1972) and with the use of general anesthesia for the mother (Moreau and Birch 1974). Friedman (1975) cites a study by Gemzell (1954) which reported that the level of 17-hydroxycorticosterone is higher in infants born of primiparous than of multiparous mothers. Paradoxically, Friedman (1975) found that infants born of high-parity mothers required significantly more trials on a visual habituation experiment than did infants born to low-parity mothers. This emphasizes the importance of using multiple response-measures in any attempt to assess newborn behavior.

The maternal predelivery medication variables assessed (see Tables III and VI) showed no significant relationship with heart-rate acceleration or deceleration scores.

Conclusions

While heart-rate response trends over trials did not discriminate well between groups scoring high or low on Prechtl's scale of non-optimal maternal, parturition and fetal conditions, significant differences between the 'high-risk' and 'low-risk' groups were found for the averaged heart-rate acceleration and deceleration scores. It is suggested that this difference may reflect a differential receptivity to stimulation in the two groups.

The reason for this study was that response measures which could differentiate

infants with a low number from those with a high number of non-optimal obstetric conditions would be worth further study, since such measures could prove valuable as an additional test item in a larger assessment battery. While it is known that obstetric history bears some relation to the manifestation of neurological abnormalities, this study demonstrates that even in ostensibly normal infants, non-optimal obstetric factors are responsible in some measure for psychobiological functioning at birth. The question remains as to whether behavioral measures such as heart-rate response to stimulation can enhance our ability to identify infants in jeopardy for later developmental problems. This could be extremely important in the search for methods of prevention and of effective remedial treatment.

Acknowledgements: We are greatly indebted to Bernice Kelly, R.N., for her very special expertise in all facets of newborn research, and without whose conscientious dedication the data for this study could not have been obtained. The assistance of Dr. Charles Crook, Dr. Patrick Burke, and Bonnie Zeigler during various phases of this study is also gratefully acknowledged. We are indebted to Prof. Leo Stern, Chairman of the Section of Reproductive and Developmental Medicine, Brown University, for a critical reading of an earlier manuscript. Support for the study came from USPHS Grant No. HD 03911 and a research grant from the Grant Foundation.

This study is a portion of an Honors Thesis conducted by Kiltner (1975) under the direction of the second author.

AUTHORS' APPOINTMENTS

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Mr. Steven Kiltner is at present a student in the University of Pennsylvania Medical School, Philadelphia.

SUMMARY

Two groups of clinically normal newborns, differing in the number of non-optimal factors in their obstetric history, were compared by measuring heart-rate response to a series of auditory stimuli. There was a significant difference between the groups in the direction of the average heart-rate response. The 'high risk' group showed more heart-rate acceleration and less deceleration compared with the 'low-risk' group. The heart-rate responses were significantly related to the number of non-optimal obstetric conditions and

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to parity, but not to the maternal pre-delivery medication score. The greater the 'risk' at birth, the less was the deceleration; the greater the maternal parity, the less did acceleration occur in response to auditory stimulation. There was no significant difference between the high-risk and low-risk groups in heart-rate response trends over trials. Both groups showed reliable diminution of deceleration heart-rate response over trials.

RÉSUMÉ

Histoire obstétricale et réponse de la fréquence cardiaque au son chez le nouveau-né

Deux groupes de nouveaux-nés cliniquement normaux mais différents par le nombre de facteurs non optimaux dans leur histoire obstétricale ont été comparés par la mesure de la fréquence cardiaque à une série de stimuli auditifs. Il a été noté une différence significative entre les groupes dans le sens de la réponse cardiaque moyenne. Le groupe à 'haut risque' a montré plus d'accélération et moins de décélérations par comparaison avec le groupe à 'bas risque'. Les réponses cardiaques ont été significativement reliées au nombre de conditions obstétricales non optimales et à la parité, mais non au score de médication maternelle avant la naissance. Plus grand est le 'risque' à la naissance et moindre est la décélération; plus grande est la parité maternelle, plus faible est l'accélération en réponse aux stimulations auditives. Il n'y a pas eu de différence significative entre les groupes à haut et bas risque dans la réponse cardiaque au cours de la répétition des essais. Les deux groupes ont montré une diminution réelle de la décélération dans la réponse de fréquence cardiaque au cours des essais successifs.

ZUSAMMENFASSUNG

Schwangerschaftsanamnese und das Ansprechen der Herzfrequenz auf Geräusche beim Neugeborenen

Zwei Gruppen klinisch gesunder Neugeborener, die sich durch einige nicht optimale Faktoren in der Schwangerschaftsanamnese unterschieden, wurden verglichen, indem die Reaktion der Herzfrequenz auf eine Serie akustischer Stimuli gemessen wurde. Es fiel eine signifikante Differenz unter den Gruppen in Bezug auf die Reaktion der Herzfrequenz auf. Die Reaktionen der Herzfrequenz waren signifikant korreliert zu der Anzahl der nicht optimalen geburtsstillischen Bedingungen und zum Geburtsverlauf und nicht zu der Geburtsprämedikation der Mutter. Je größer das 'Risiko' bei Geburt, desto weniger Pulsbeschleunigung trat als Reaktion auf akustische Stimuli auf. Es fand sich keine signifikante Differenz zwischen der Gruppe mit 'hohem' und der mit 'niedrigem Risiko' in der Reaktion der Herzfrequenz verglichen mit Kontrollen. Beide Gruppen zeigten verlässliche Decelerationsraten der Herzfrequenz gegenüber Kontrollen.

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SECTION 13

Koszarny, Z. et al. The effect of airplane noise on the inhabitants of areas near the Okęcie airport in Warsaw. Roczniki Panstwowego Zakladu Higieny 27(2):113-121, 1976. (partial English translation)

The psychological and physiological effects of aircraft noise were studied in residents of two areas near a large airport in Poland. A health questionnaire and a noise disturbance scale were used to survey 256 residents in area A (noise levels exceeding 100 dBA) and 255 residents in area B (noise levels of 80-90 dBA). The people surveyed were grouped according to sex, age, living conditions, education and socioeconomic level. The relative annoyance level for area A was 82% and for area B, 54%. The survey population had a normal distribution of psychological problems. The health questionnaires documented the frequency of complaints of various ailments. No statistically significant differences in complaints were found in groups of men living in the two areas. Significantly greater numbers of complaints related to the cardiovascular system, the digestive system, frequency of taking medication for heart problems or headaches, and nervousness were found in women living in the noisier area (A) than in women who lived in the lower noise level area (B). The results indicate that aircraft noise can have an effect on the general state of health. The authors note that many other stressful and adverse conditions besides noise may affect state of health, such as the work environments of the people studied.

Roczniki Państwowego Zakładu Higieny
27(2):113-121, 1976. Koszarny et al.
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EXPERIMENTAL METHODOLOGY AND MATERIALS

In our tests, we used a specific health questionnaire and a noise disturbance scale. The basics of working out the questionnaire and the method of collecting the material were discussed in an earlier article [8]. In the new tests, however, we made some changes in the way in which the disturbance scale was fashioned, so that it was possible to achieve better correlation between the replies and their order according to the importance of the question. In constructing the scale, we used the scale analysis method of Guttman [10], which is based on assigning each person being examined a degree of disturbance based on the level of his symptoms caused by airplane noise. We adopted a 5-degree evaluation scale. Any noise reaching a degree of at least 3 points, or 60%, was considered to be very harmful to the environment. This is the threshold above which systematic disorders in word perception occur. It was determined that airplane noise is the main cause of feelings of discomfort in persons found on the scale above this threshold.

A similar method of examining airplane noise disturbance has been used in England, France and the Benelux countries [2].

Our tests involved 256 residents of areas located in a region where the intensity level was higher than 100 dB (A) and 255 residents of an area with an intensity level of 80-90 dB (B). The tests were conducted during the winter months of 1974-75. The test groups were divided equally into men and women aged 20 to 70, of similar educational background, professional background, working conditions, but different social and residential conditions. Tables I, II and fig. 1 detail the professional and residential conditions of the populations of both regions examined.

The difference in the social and residential conditions could to a certain degree affect the validity of the results obtained in that the increased sensitivity to noise in persons having less favorable conditions of life; however, this is impossible to avoid, as it results from the very nature of the regions examined. In any case, this influence is not significant and will chiefly affect the residents of the areas with lower intensity levels [2,8].

Table I
Age of Persons Examined

Sex	Avg. age of subjects in various age groups						Avg. age of subjects	
	20-36		37-53		54-74		A	B
	A	B	A	B	A	B	A	B
Female	28	28	44	43	63	63	45	44
Male	28	27	45	44	62	64	45	44

A - region where 100 dB (A) 110
B - region where 80 dB (A) 90

Table II
Living Conditions

Evaluative factor	Indices	No. of persons or homes		t	P
		A	B		
Level of population	avg. no. of persons per room	1.39	1.31	0.08	n.i.
Ownership	owner	30.9%	18.9%	3.16	0.01
	renter	69.1%	81.1%		
Outfittings	bathroom	33.3%	77.6%	11.23	0.0001
	toilet	59.3%	85.8%	7.03	0.001
	running water and sewerage	69.9%	84.6%	4.02	0.001
Evaluation of living conditions	central heating	28.9%	75.6%	11.95	0.0001
	satisfied	30.5%	50.8%	4.77	0.001
	not satisfied	69.5%	49.2%		

A - region where 100 dB (A) 110
B - region where 80 dB (A) 90
t - value of t-Student test
P - level of significance
n.i. - statistically insignificant

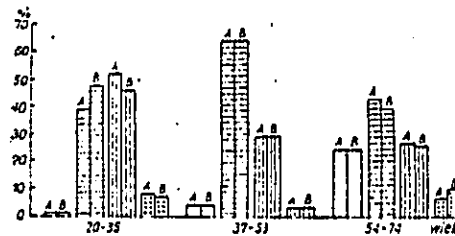


Fig. 1. Education; white areas - incomplete elementary education; horizontally striped - elementary; vertically striped - high school; dotted - higher education (college, university). A - region where $100 \text{ dB(A)} < 110$; B - region where $80 < \text{dB(A)} < 90$.

DISCUSSION OF RESULTS

The evaluation of acoustic conditions in the area of Okęcie involved, among other things, determining noise sources and their degree of bothersomeness. The data are detailed in Table III, in which the individual positions were arranged according to the average degree of disturbance for both of the regions discussed.

Analysis of the material shows that airplane noise plays a fundamental and significant role among the various types of noise that occur in the areas. The number of persons who complained of this type of noise was 91.1% in the over 100 dB (A) area and 63.4% in the 80-90 dB (A) area. The percentage of people who consider the noise to be very disturbing is significant, at 54.5% and 24.4% respectively.

Street noise was the object of complaints from about 38% of the respondents, and noise from neighboring houses or residences also represents a certain problem, but rather only for residents of areas with a lower airplane noise intensity level. It should also be pointed out that the number of people who indicated street and neighboring residence noise as very disturbing is small (not over 13%). The remaining types of noise sources are not a disturbance to the residents of the areas discussed.

In light of the differences in bothersomeness between the various types of noise occurring in both areas examined, only airplane noise

can, practically speaking, be taken as a basis for further analyses and comparisons. This concerns the goals of the tests, and above all determining the actual bothersomeness of airplane noise and its connection with physical parameters and the effect of the noise on the residents' health.

Table III
Evaluation of the acoustic climate in the area
of Okecie

1. Źródła hałasu	2. Liczba badanych (w %) określających hałas jako:											
	a. bardzo uciążliwy				b. średnio uciążliwy				c. nieuciążliwy			
	A	B	t	P	A	B	t	P	A	B	t	P
3. Urządzenia domowe	0,8	0,4	0,67	n.i.	0,8	5,1	2,88	0,01	98,4	94,5	2,23	0,08
4. Hałas kolejowy	0	1,0	2,03	0,05	0	0,8	3,25	0,01	100,0	88,8	4,71	0,001
5. Zakłady przemysłowe	1,6	0,5	2,37	0,05	1,2	12,2	5,07	0,001	97,2	82,2	3,60	0,001
6. Zakłady usługowe	10,3	2,1	6,22	0,001	8,1	10,2	0,61	n.i.	75,6	87,5	4,07	0,001
7. Sąsiednie mieszkania	8,1	8,3	0,08	n.i.	10,6	35,8	7,01	0,001	81,3	55,8	6,37	0,001
8. Hałas uliczny	13,0	7,0	1,80	n.i.	22,4	29,0	1,01	n.i.	64,6	62,2	0,66	n.i.
9. Hałas lotniczy	24,0	24,4	7,22	0,001	30,0	39,0	0,55	n.i.	5,0	30,6	7,88	0,001

A - region where $100 < \text{dB (A)} < 110$
 B - region where $80 < \text{dB (A)} < 90$
 t - values of t-Student test
 P - level of significance
 n.i. - statistically insignificant

Key:

1. noise source
2. number of subjects (in%) who indicated noise as:
 - a. very disturbing
 - b. somewhat disturbing
 - c. not disturbing
3. Household devices
4. Railroad noise
5. Factories
6. Service establishments
7. Neighboring residences
8. Street noise
9. Air traffic noise

The measuring scale used made it possible to determine the average degree of disturbance of air traffic and airplane noise, which is 4.1 points for the area with the highest intensity level, and 2.8 for the other. Expressing the above figures in relative values, we can say that the disturbance of airplane noise in the first area is 82%, and 54% in the second (fig. 2).

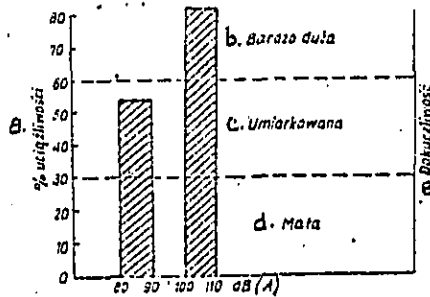


Fig. 2. Air traffic noise disturbance.
a. percent of disturbance. b. great c. moderate d. small
e. bothersomeness

The disturbance is generally worst during daylight hours, as night flights at the airport near Okecie are sporadic.

Based on the scale measurements and the comparative examinations (2), air traffic noise in the area of more than 100 dB (A) can be considered very disturbing, while the noise in the area of 80-90 dB (A) can be considered moderately disturbing noise, approaching the upper limit. However, it is highly probably that as the intensity of the air traffic increases in a given region, its percentage of disturbance will also rise. This increase, as the examinations showed, is approximately proportional to the logarithm of the frequency with which airplanes pass by [2, 12].

The examinations conducted also showed a close correlation between evaluation of the noise disturbance and the objective measure of its intensity. This connection is very strong, if we take into consideration the average noise disturbance of the region in question (the correlation coefficient is 0.86), but is insignificant if we consider the individual sensitivity of the persons examined (the correlation coefficient is 0.42). Thus, the level of noise intensity alone does not determine its degree of disturbance. Individual personality factors, social and living conditions, type of work done and even attitudes toward flying also affect the way in which one feels the disturbance of noise [8, 13]. Alexander [2] indicates that these factors can affect a change in the disturbance only to a certain degree. However, their effect is insignificant if the noise disrupts everyday activities.

Agreement between the evaluation of the noise disturbance level and the objective measure of its intensity indicates that personal bases for determining the bothersomeness of air traffic noise should be adopted. However, the fact that in areas recognized, on the basis of noise intensity levels, as being suitable for residence and school construction, up to 65% of the persons examined gave negative evaluations of the acoustic conditions is rather disturbing.

The percentage index of disturbance is also high, and though it is within the limits of so-called moderate noise, it indicates significant deterioration in the comforts of residence in the areas discussed.

Evaluations of the state of health and general well-being of the populations in the areas discussed were made on the basis of the frequency and intensity of symptoms of general ill-feeling and a poor state of health in groups of persons residing under varying acoustic conditions.

The following symptoms were observed:

1. In the overall evaluation of the state of health no real differences were noticed between the men of both areas examined (χ^2 square = 0.11), but there were marked differences among the women (χ^2 square = 4.114, $p = 0.04$).
2. No statistically significant differences in tests for neuroticism and extraversion were noted in either the men or the women. The distribution of the results relation to these characteristics is comparable to the distribution observed in the overall Polish population.
3. In analyzing the frequency of occurrence of specific symptoms of ill-feeling and a poor state of health, only in women living under worse acoustic conditions did we find a more frequent occurrence of general complaints related to the heart and the digestive tract, more marked feelings of fear and ill-ease, more frequent nervousness, and more numerous cases of taking medication for headaches or cardiac troubles.
4. Independent of the differences found between the areas discussed, frequent appearance among the subjects of complaints of chronic fatigue, troubled vision, relentless headaches, nervousness, trouble in hearing and frequent use of headache or cardiac medication should be emphasized. Numerical data are given in tables IV and V.

Based on these results, it is difficult to draw any final conclusions regarding the effect of acoustic conditions on the state of health of residents in areas near airports. The high percentage of people who complain of feelings of fatigue, headaches, trouble in hearing, frequent nervousness, cardiovascular troubles and digestive tract disorders, and the differences in the frequency with which symptoms of illness and general ill-feeling occur in women living under poorer acoustic conditions seem to testify to a relationship between air traffic noise and the state of health. On the other hand, we cannot exclude the possibility that the above indices are the result of other causes related to unsatisfactory environmental conditions, especially the working environment.

The observed differences in the effect of acoustic conditions in one's place of residence on the state of health of men as opposed to women seem to indicate that such an interpretation should indeed be adopted. For among women there was found a more numerous group of persons either not working (23% more than the men), or working under better acoustic conditions (13% compared to the men). The varying acoustic conditions in the place of residence could thus play a more decisive role in determining women's state of health than they do in men's, among whom acoustically unfavorable working conditions prevailed (60%). Specific and detailed explanation of this puzzle would require additional examinations and tests.

Table IV
Frequency of occurrence of symptoms of ill-feeling and illness under varying acoustic conditions

Health indices	Men		Women	
	A	B	A	B
Insomnia	20	15	28	30
Fatigue	71	65	85	82
Headaches	23	23	48	43
Fear or ill-ease	17	11	28	18
Nervousness	34	29	20	8
Taking headache medication	13	8	29	15
Taking sleeping pills	8	5	12	6
Taking heart medication	15	12	34	23
Troubled vision	53	48	61	54
Trouble in hearing	34	40	28	24
Digestive troubles	22	19	28	18
Cardiovascular troubles	24	20	48	43

A - $100 < dB(A) < 110$
B - $80 < dB(A) < 90$

Table V
Relation between objective evaluation of air traffic noise and the
selected indices of state of health

Health indices	Men		Women	
	X ²	P	X ²	P
Sleep	0.527	n.i.	0.157	n.i.
Fatigue	0.940	n.i.	0.343	n.i.
Headaches	0.084	n.i.	0.691	n.i.
Fear or ill-ease	2.016	n.i.	3.843	0.05
Nervousness	0.631	n.i.	5.121	0.03
Taking headache medication	1.734	n.i.	6.871	0.01
Taking sleeping pills	1.026	n.i.	3.641	n.i.
Taking heart medication	0.398	n.i.	3.830	0.05
Troubled vision	0.110	n.i.	1.090	n.i.
Troubled hearing	1.012	n.i.	0.542	n.i.
Digestive troubles	0.501	n.i.	3.843	0.05
"Heart" troubles	0.298	n.i.	4.416	0.04
Neuroticism	0.824	n.i.	0.046	n.i.

X² - value of chi square meter
P - level of significance
n.i. - statistically insignificant

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ZBIGNIEW KOSZARNY, STEFAN HAZIARKA, WANDA SZATA

WPLYW HALASU SAMOLOTOWEGO NA MIESZKAŃCÓW REJONÓW PRZYLOTNISKOWYCH LOTNISKA OKĘCIE W WARSZAWIE

Z Zakładu Higieny Komunalnej Państwowego Zakładu Higieny w Warszawie
Kierownik: doc. dr hab. S. Haziarka

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Dokonano oceny stanu zdrowia i dokuczliwości hałasu wśród osób zamieszkujących zróżnicowane pod względem akustycznym rejonu lotniska Okęcie. Stwierdzono wysoki stopień uciążliwości hałasu o poziomie natężenia ponad 110 dB (A) i wykazano związek hałasu lotniczego z niektórymi dolegliwościami zgłaszanymi przez mieszkańców.

WSTĘP

Zagadnienie wpływu hałasu lotniczego na mieszkańców obszarów przylotniskowych jest przedmiotem szczególnego zainteresowania licznych ośrodków naukowych.

Główny kierunek badań koncentruje się na ustaleniu rozmiarów i stopnia zagrożenia zdrowia ludności oraz określeniu uciążliwości hałasu lotniczego [1, 3, 6, 7, 8]. Wielu autorów zajmujących się powyższym problemem stwierdza u mieszkańców rejonów przylotniskowych nasilenie takich objawów jak bóle głowy, brak apetytu, trudności w koncentracji uwagi, trudności w zasypianiu, częste zakłócenia snu, uczucie zmęczenia oraz zaburzenia czynności układów sercowo-naczyniowego i oddechowego, zaburzenia nerwicowe i choroby psychiczne. Mieszkańcy tych rejonów skarżą się na niepokojące działanie hałasu, przeszkadzające w normalnej pracy i odpoczynku. Zjawiska te nasilają się wraz ze wzrostem poziomu hałasu na obszarach przylotniskowych i wzmożeniem tempa rozwoju ruchu lotniczego.

Innym niemniej istotnym zagadnieniem wpływu hałasu samolotowego jest próba znalezienia możliwych ścisłych powiązań między fizycznymi parametrami dźwięku a jego uciążliwością dla mieszkańców [5, 9, 11, 12]. Dotychczasowe wyniki badań wskazują na możliwość wykorzystania skal uciążliwości hałasu do opracowania bardziej dokładnych metod oceny hałasów lotniczych.

W badaniach oddziaływania hałasu lotniczego w rejonie Okęcia starano się uwzględnić oba wspomniane wyżej kierunki badań. Główny nacisk położono na ocenę stanu zdrowia mieszkańców i ustalenie uciążliwości hałasu w różnych strefach nagłośnienia. Podjęto również próbę oceny zgodności stosowanych metod pomiaru hałasów lotniczych z subiektywną oceną jego uciążliwości.

METODYKA BADAŃ I MATERIAŁ BADAWCZY

Dla realizacji wyżej sformułowanych celów zastosowano w badaniach specjalny kwestionariusz zdrowotny oraz skale uciążliwości hałasu. Zasady opracowania kwestionariusza i sposób zbierania materiału został omówiony w poprzedniej pracy [6]. W obecnych badaniach dokonano jednak zmian w sposobie opracowania skali uciążliwości, umożliwiając osiągnięcie większej spójności odpowiedzi i ich uporządkowanie według ważności pytań. Przy konstrukcji skali zastosowano metodę analizy skalogramów Guttmana [10] polegającą na przydzieleniu każdemu z badanych stopnia uciążliwości zależnie od poziomu zakłóceń spowodowanych działaniem hałasu samolotowego. Przyjęto 5 stopniową skalę oceny. Za hałas bardzo dokuczliwy dla odczucia uciążliwości hałasu o stopniu wynoszącym co najmniej 3 punkty czyli 60%. Jest to próg, powyżej którego występują systematyczne zakłócenia percepcji słownej. Stwierdzono, że dla osób umieszczonych na skali powyżej tego progu hałasu samolotowy stanowi główne źródło poczucia dyskomfortu.

Podobną metodę badań uciążliwości hałasu lotniczego zastosowano w Anglii, Francji i w krajach Beneluxu [2].

W ramach niniejszej pracy badaniami objęto 256 mieszkańców terenów położonych w strefie o poziomie natężenia powyżej 100 dB (A) oraz 255 mieszkańców rejonu o poziomie natężenia 80-90 dB (A). Badania przeprowadzono w miesiącach zimowych na przełomie 1974 i 1975 r. Grupy badanych osób reprezentowane są w równej mierze przez mężczyzn, jak przez kobiety w wieku 20-70 lat, o podobnym typie wykształcenia, zawadzie, warunkach pracy ale odmiennych warunkach mieszkaniowo-bytowych. Tabele I, II i rys. 1 charakteryzują bliżej strukturę zawodowo-społeczną i warunki mieszkaniowo-bytowe ludności badanych rejonów.

Odmienność warunków mieszkaniowo-bytowych może w pewnym stopniu wpływać na wiarygodność otrzymanych rezultatów przez podwyższenie wrażliwości na hałas osób z niekorzystnych warunków, jest jednak niemożliwa do uniknięcia, ponieważ wynika z samego charakteru badanych rejonów. Wpływ ten jednak jest niewielki i dotyczy głównie mieszkańców rejonów o niższych poziomach natężenia [2, 8].

Tabela I
Wiek badanych

Płeć	Średni wiek badanych w różnych grupach wiekowych						Przewidywany wiek badanych	
	20-29		37-43		64-74		A	B
	A	B	A	B	A	B	A	B
Kobiety	28	28	44	43	63	63	45	44
Mężczyźni	28	27	45	44	62	64	45	44

A — rejon 100<dB (A)<110

B — rejon 80<dB (A)<90

OMÓWIENIE WYNIKÓW BADAŃ

Ocena warunków akustycznych w rejonie Okęcia dotyczyła między innymi określenia źródeł hałasów i stopnia ich dokuczliwości. Szczegółowe zestawienie danych zostało podane w tabeli III, w której poszczególne pozycje ułożono według przeciętnego stopnia uciążliwości dla obu omawianych rejonów.

Tabela II
Warunki mieszkaniowe

Klasa oceny	Wskazniki	Liczba osób lub mieszkań		t	P
		A	B		
Stopień wyposażenia	Średnia liczba osób na łóżko	1,38	1,31	0,08	n. l.
Rusznik własności	własność	30,0%	18,9%	3,16	0,01
	lokatorska	40,1%	41,1%		
Wyposażenie	łazienka	33,3%	77,0%	11,23	0,0001
	ubikacja	59,3%	85,8%	7,00	0,001
	wodociąg i kanalizacja	68,0%	84,0%	4,02	0,001
Ocena mieszkania	centralne ogrzewanie	28,0%	75,0%	11,55	0,0001
	zakwaterunki niezadowoleni	30,5%	60,8%	4,77	0,001

A — rejon $100 < dB(A) < 110$ B — rejon $80 < dB(A) < 90$

t — wartość testu t-Studenta

P — poziom istotności

n.l. — niezgodny statystycznie



Fig. 1. Wykształcenie; pole białe — niepełne podstawowe, pole kreskowane pionowo — podstawowe, pole kreskowane pionowo — średnie, pole kropkowane — wyższe. A — rejon $100 < dB(A) < 110$; B — rejon $80 < dB(A) < 90$.

Z analizy materiału wynika, że spośród hałasów występujących na danym terenie zasadniczą rolę odgrywa hałas lotniczy. Liczba osób skarżących się na tego rodzaju hałas wynosi w strzale powyżej 100 dB(A) — $91,1\%$ mieszkańców, natomiast w strzale o natężeniu $80-90 \text{ dB(A)}$ — $63,4\%$. Udział osób oceniających hałas jako bardzo uciążliwy jest znaczny i wynosi odpowiednio $54,5\%$ i $24,4\%$.

Hałas uliczny stanowi przedmiot skargi około 38% ogółu respondentów, a hałas dochodzący z sąsiednich mieszkań przedstawia pewien problem, jedynie dla mieszkańców rejonu o mniejszym poziomie natężenia hałasu lotniczego. Należy przy tym zaznaczyć, że liczba osób oceniających hałas uliczny i z sąsiednich mieszkań jako bardzo uciążliwy jest niewielka

Tabela III
Ocena klimatu akustycznego w rejonie Dąbca

Źródła hałasu	Liczba badanych (w %) określających hałas jako:											
	bardzo uciążliwy				średnio uciążliwy				nieuciążliwy			
	A	B	t	P	A	B	t	P	A	B	t	P
Urzędzenia domowe	0,8	0,1	0,87	n.i.	0,8	0,1	2,98	0,01	98,4	94,8	2,33	0,05
Uluch kolejowy	0	1,8	2,03	0,05	0	0,8	3,25	0,01	100,0	88,0	8,71	0,001
Zakłady przemysłowe	1,8	0,8	2,37	0,05	1,2	12,2	6,07	0,001	97,9	82,3	6,60	0,001
Zakłady usługowe	10,3	2,1	0,22	0,001	8,1	10,2	0,81	n.i.	78,0	87,8	4,07	0,001
Stacje mieszkalne	8,1	8,3	0,08	n.i.	10,0	30,8	7,01	0,001	81,2	66,0	0,37	0,001
Uluch uliczny	12,0	7,0	1,80	n.i.	22,4	20,0	1,01	n.i.	84,0	82,3	0,88	n.i.
Hałas lotniczy	64,0	24,4	7,22	0,001	30,0	39,0	0,05	n.i.	8,0	38,0	7,88	0,001

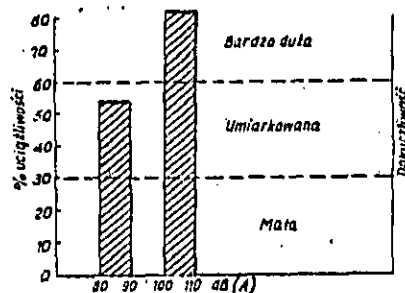
A — rejon 100<(dB (A)<110
B — rejon 80<(dB (A)<90
t — wartość testu t-Studenta
P — poziom istotności
n.i. — nieistotny statystycznie

(nie przekracza 13%). Pozostałe rodzaje źródeł hałasu nie stanowią doku-
czliwości dla mieszkańców omawianego rejonu.

Pomimo więc stwierdzonych różnic w dokuźliwości poszczególnych
rodzajów hałasów występujących w obu badanych strefach jedynie hałas
samolotowy może stanowić, praktycznie rzecz biorąc, podstawę do dal-
szych analiz i porównań. Dotyczy to zwłaszcza istotnych celów badań
a mianowicie określenia uciążliwości hałasu lotniczego i jej powiązań
z pomiarami fizycznymi oraz wpływem hałasu na zdrowie mieszkańców.

Zastosowana skala pomiaru pozwoliła na ustalenie przeciętnego stó-
pnia uciążliwości hałasu lotniczego, który wynosi dla strefy o najwyż-
szym poziomie natężenia 4,1 punktów, dla drugiej natomiast 2,8. Wyra-
żając powyższe liczby w wartościach względnych można powiedzieć, że
uciążliwość hałasu samolotowego w pierwszej z omawianych stref wy-
nosi 82%, w drugiej natomiast 54% (ryc. 2).

Wspomniana uciążliwość odnosi się zasadniczo do godzin dziennych,
ponieważ loty nocne związane z lotniskiem Okęcie występują tylko spo-
radycznie.



Ryc. 2. Uciążliwość hałasu lotniczego

Zgodnie z założeniami skali i badaniami porównawczymi (2) należy
uznać hałas lotniczy w strefie powyżej 100 dB (A) za bardzo uciążliwy,
natomiast hałas w strefie 80—90 dB (A) za hałas o umiarkowanej do-
kuczliwości, zbliżający się do jej górnej granicy. Jest jednak wysoce
prawdopodobne, że przy wzroście intensywności ruchu lotniczego w da-
nym rejonie wzrosnąć również wartość procentowa jego uciążliwości.
Wzrost ten, jak wykazały badania, jest w przybliżeniu proporcjonalny
do logarytmu częstości przelotu samolotów [2, 12].

Przeprowadzone badania wykazały ponadto występowanie ścisłej ko-
relacji między oceną uciążliwości hałasu a obiektywnym pomiarem jego
natężenia. Związek ten jest bardzo silny jeżeli bierze się pod uwagę śred-
nią uciążliwość hałasu badanego rejonu (współczynnik korelacji wynosi
0,88), jest natomiast niewielki, jeżeli uwzględni się indywidualną wra-
żliwość osób badanych (współczynnik korelacji wynosi 0,42). Tak więc
sam poziom natężenia hałasu nie decyduje o stopniu jego uciążliwości.

Określone czynniki osobnicze, warunki socjalnobytowe, rodzaj wykonywanej pracy a nawet postawy względem lotnictwa wpływają dodatkowo na kształtowanie odczucia uciążliwości hałasu [8, 13]. Alexander [2] zaznacza, że wymienione czynniki mogą tylko do pewnego stopnia wpływać na zmianę uciążliwości. Wpływ ich jest natomiast niewielki, jeżeli hałas zakłóca aktywność codziennego życia.

Zgodność między oceną uciążliwości hałasu a obiektywnym pomiarem jego natężenia wskazuje na przyjęcie właściwych zasad określania uciążliwości hałasów lotniczych. Niepokojący jest jednak fakt, że na terenach uznawanych pod względem intensywności hałasów za strefę nadającą się do zamieszkania i szkolnictwa aż wśród 65% badanych występują negatywne oceny warunków akustycznych.

Procentowy wskaźnik uciążliwości również jest wysoki i chociaż mieści się w granicach tzw. hałasu umiarkowanego, wskazuje na znaczne pogorszenie komfortu zamieszkania w omawianym rejonie.

Oceny stanu zdrowia i samopoczucia ludności badanych rejonów dokonano na podstawie częstości występowania i nasilenia objawów złego samopoczucia i złego stanu zdrowia w grupach osób zamieszkałych w różnych warunkach akustycznych.

↳ Zaobserwowano następujące zjawiska:

1. Przy kompleksowej ocenie stanu zdrowia nie stwierdzono istotnych różnic między mężczyznami obu badanych rejonów (chi kwadrat = 0,11), wystąpiły natomiast istotne różnice wśród badanych kobiet (chi kwadrat = 4,114, $p = 0,04$).

2. Zarówno wśród mężczyzn jak i wśród kobiet nie zanotowano znaczących statystycznie różnic w badaniach neurotyczności i ekstrawersji. Rozkład wyników wymienionych cech jest przy tym zgodny z rozkładem obserwowanym w populacji polskiej.

3. Analizując częstość występowania poszczególnych objawów złego samopoczucia i złego stanu zdrowia stwierdzono jedynie wśród kobiet zamieszkałych w gorszych warunkach akustycznych częstsze występowanie ogólnych dolegliwości ze strony serca i układu pokarmowego, nasilenie uczucia lęku i niepokoju, częstsze denerwowanie się, jak również liczniejsze przypadki przyjmowania leków od bólu głowy i leków nasercowych.

4. Niezależnie od różnic jakie wystąpiły między omawianymi rejonami należy podkreślić częste pojawianie się wśród badanych osób narzekania na chroniczne zmęczenie, zakłócenia wzroku, uporczywe bóle głowy, denerwowanie się, upośledzenie słuchu oraz częste zazywanie leków od bólu głowy i leków nasercowych. Szczegółowe dane liczbowe zestawiono w tabeli IV i V.

Na podstawie przedstawionych wyników badań trudno jest wyciągnąć ostateczne wnioski na temat wpływu warunków akustycznych na stan zdrowia mieszkańców rejonów przylotniskowych. Wysoki procent osób skarżących się na uczucie zmęczenia, bóle głowy, upośledzenie słuchu, częste denerwowanie, dolegliwości ze strony serca i układu pokarmowego, jak również różnice w częstości występowania objawów chorobowych i złego samopoczucia wśród kobiet z gorszych warunków akustycznych zdają się przemawiać za zależnością między hałasem lotniczym a stanem zdrowia. Z drugiej jednak strony nie można wykluczyć przypuszczenia, że wyżej wspomniane wskaźniki wynikają z innych przyczyn niekorzystnego oddziaływania środowiska, zwłaszcza środowiska pracy

Tabela IV

Częstość występowania objawów słabego samopoczucia i objawów chorobowych w różnych warunkach akustycznych

Wskazniki stanu zdrowia	Mężczyźni		Kobiety	
	A	B	A	B
	%		%	
Zakłócenia snu	20	18	28	30
Zmęczenie	31	25	45	42
Bóle głowy	23	23	48	42
Uczucie lęku lub niepokoju	17	11	26	18
Nerwowość	34	29	20	8
Przyjmowanie leków od bólu głowy	13	8	29	18
Przyjmowanie leków nasennych	8	5	12	6
Przyjmowanie leków nasercowych	18	12	24	22
Zakłócenia wariaku	43	44	41	44
Upośledzenie słuchu	34	40	26	24
Dolegliwości żołądkowe	22	19	28	18
Dolegliwości sercowe	24	20	48	43

A -- 100<dB (A)<110

B -- 50<dB (A)<90

Tabela V

Zależność między obiektywną oceną hałasu lotniczego a wybranymi wskaźnikami stanu zdrowia

Wskazniki stanu zdrowia	Mężczyźni		Kobiety	
	χ^2	P	χ^2	P
Sen	0,327	n.s.	0,127	n.s.
Zmęczenie	0,040	n.s.	0,343	n.s.
Bóle głowy	0,004	n.s.	0,001	n.s.
Uczucie lęku lub niepokoju	2,016	n.s.	3,843	0,05
Nerwowość	0,031	n.s.	5,121	0,03
Przyjmowanie leków od bólu głowy	1,734	n.s.	0,871	0,01
Przyjmowanie leków nasennych	1,020	n.s.	3,041	n.s.
Przyjmowanie leków nasercowych	0,308	n.s.	3,830	0,05
Zakłócenia wariaku	0,110	n.s.	1,080	n.s.
Upośledzenie słuchu	1,012	n.s.	0,542	n.s.
Dolegliwości żołądkowe	0,501	n.s.	3,843	0,05
Dolegliwości sercowe	0,296	n.s.	4,416	0,04
Nerwowość	0,821	n.s.	0,046	n.s.

 χ^2 -- wartość miernika chi-kwadrat

P -- poziom istotności

n.s. -- nieistotny statystycznie

Zaobserwowane różnice wpływu warunków akustycznych w miejscu zamieszkania na stan zdrowia mężczyzn i kobiet zdają się przemawiać za przyjęciem takiej właśnie interpretacji. Wśród kobiet bowiem znalazła się liczniejsza grupa osób bądź to niepracujących zawodowo (o 23% więcej aniżeli mężczyzn), bądź też pracujących w lepszych warunkach akustycznych (o 13% w stosunku do mężczyzn). Odmienne warunki akustyczne w miejscu zamieszkania mogły więc odgrywać bardziej decydującą rolę w określaniu stanu zdrowia kobiet aniżeli mężczyzn, u których przewżyły prawdopodobnie niekorzystne pod względem akustycznym warunki pracy (60%). Szczegółowe wyjaśnienie tego zagadnienia wymaga dodatkowych badań.

WNIOSKI

1. Hałas lotniczy o poziomie ponad 100 dB (A) stanowi znaczne obciążenie dla mieszkańców. Hałas o tym poziomie należy uznać za bardzo uciążliwy a teren ni nadający się do zabudowy mieszkaniowej.
2. Hałas lotniczy o poziomie 80-90 dB (A) przy dorychczasowym ruchu lotniczym stanowi umiarkowaną dokuczliwość dla mieszkańców. Należy się jednak liczyć z możliwością podniesienia jego uciążliwości w miarę wzrostu ruchu lotniczego. Jest to tym bardziej prawdopodobne, że procent uciążliwości w danym rejonie jest już obecnie bliski wartości granicznej.
3. Zależność między niektórymi objawami złego samopoczucia i objawami chorobowymi a warunkami akustycznymi w miejscu zamieszkania wskazuje na prawdopodobieństwo ujemnego wpływu hałasu lotniczego na stan zdrowia mieszkańców.

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ROCZNIKI
PAŃSTWOWEGO ZAKŁADU HIGIENY

POŚWIĘCONE NAUKOWEJ PROBLEMATYCE HIGIENY ŚRODOWISKA, A W SZCZEGÓL-
NOŚCI HIGIENIE ŻYWIENIA I ŻYWIENIA ORAZ PRZEDMIOTÓW UŻYTKU, HIGIENIE
SZKOLNEJ, TOKSYKOLOGII SANITARNEJ, HIGIENIE KOMUNALNEJ I INNYM DZIEDZI-
NOM POKREWNYM

DWUMIESIĘCZNIK

TOM XXVII

1976

Nr 2

Z. Kostarny, S. Maziarka, W. Szala

THE EFFECT OF AIRPLANE NOISE ON THE INHABITANTS
OF AREAS NEAR THE OKĘCIE AIRPORT IN WARSAW

Summary

Investigations were carried out of 311 inhabitants of areas near the airport who were exposed to various intensities of noise. Very high annoyance effects of airplane noise of intensities over 100 dB (A) were demonstrated and a moderate annoyance effect of noise of intensities below 80-90 dB (A).

A high proportion of subjects complained about a feeling of fatigue, headache, hearing impairment, cardiac and gastrointestinal symptoms. The association of acoustic conditions with certain symptoms of malaise and disease symptoms was demonstrated only in women.

ZESTAWIENIE WYNIKÓW I WNIOSKÓW Z BADAŃ NAD WYDZIAŁAMI ZDRAŃNIENIOWYMI, 21. WNIOSKI
dotyczące zjadania, sympozja (tj. h) wspomnienia polimerne.

3. Praca należy wydrukować w 2 egzemplarzach (oryginał i odbitka (drukowa))
wzajemnie na typowej maszynie biurowej, jednostronnie i z podwójną interlinią
(30 wierszy na stronie), z marginesem 4 cm z lewej strony znormalizowanego
arkusza A4. W maszynopiśmie nie należy wyodrębnić słów samymi dużymi literami
lub drukiem rozstrzelonym (można to zamoczyć słówkami przez podkreśle-
nie wyrazów odpowiednio — linią ciągłą lub przerywaną). Wszelkie inne pod-
kreślenia są niedopuszczalne.

4. Objętość pracy nie powinna przekraczać w zasadzie 12 stron maszynopisu,
zas doniesień tymczasowych — 4 stron maszynopisu, łącznie z piśmiennictwem,
tabelami, rycinami i streszczeniem.

5. Praca winna być opracowana wg zasad pisowni polskiej i pisana w formie
bezosobowej.

Przy użyciu nie przyjętych powszechnie skrótów, należy je objaśnić w miejscu,
gdzie pojawiają się po raz pierwszy. W przypadkach istotnych zmian w treści
pracy Redakcja odsyła autorowi i egz. pracy do poprawienia w myśl sugestii
Redakcji. Poprawiona i przepisana praca należy przesłać ponownie w 2 egzem-
plarzach w ciągu 10 dni. Jeżeli praca nie została przyjęta do druku, Redakcja
zwraca 1 egzemplarz (oryginał).

6. Na pierwszej stronie pracy, na wysokości 3/4 strony (wolne miejsce od
góry potrzebne jest na uwagi techniczno-wydawnicze) należy wymienić: pełne
imię i nazwisko autora lub autorów, tytuł pracy, nazwę zakładu skąd praca
pochodzi, tytuł naukowy, inicjały imienia i nazwisko kierownika zakładu, poniżej
2-3 zdaniowe streszczenie (synopsis) wprowadzające w temat pracy i dalej na
tej samej stronie — paragraf opisu pracy. Pożądanym jest, aby tekst pracy oryginalnej
był podzielony na rozdziały: Wstęp, Materiał i metodyka, Wyniki, Omówienie
wyników, Wnioski.

(c.d. III str. okładki)

SECTION 14

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> E. Kunitake (Dept. of Hygiene, Faculty of Medicine, Fukuoka University Fukuoka, Japan), N. Ishinashi, and Y. Kodama		Institution and address where research was performed Dept. of Hygiene Faculty of Medicine Kyushu University, Fukuoka, Japan	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> <p style="text-align: center;">same</p>		
<u>Citation</u> Kunitake, E. et al. Studies on the effects of aircraft noise causing mental fatigue during some intellectual performance. Nippon Eiseigaku Zasshi 30(3):417-429, 1975.			
<u># of Ref.'s</u> 24	<u># of Fig.'s</u> 28	Language Jpn (partial translation)	
<u>Type & duration of experiment</u> laboratory--in soundproof room 2 days tests per subject		<u>Purpose for study</u> to study effects of aircraft noise as a potential hazard to nearby residents	
<u>Description of test groups (subjects, #, age, etc.)</u> 9 female college students, average age 20.6, who did <u>not</u> live near an airport; (subjects were not menstruating at time of test)			
<u>Control of other stressors</u> laboratory conditions--constant temp. and background noise (30 dBA)		<u>Statistical Methods</u> (not translated)	
<u>Noise Stimulus</u> source: recorded aircraft noise spectral characteristics: high frequency-- 1000-2000 Hz noise level: average peak level=86.5 dBA; or 80 WECPNL (w/120 flyovers daily) length of exposure: continuously for 50 min. in AM, 100 min. in PM # of trials: 2 quiet & 2 noise periods per subject (2 days total)		<u>CVS Response Measured</u> pulse rate; Cornell Medical Index fingertip pulse amplitude--on middle finger <u>Nonauditory affects</u> noise increased pulse rate and decreased pulse amplitude; noise seemed CVS; to induce sustained tension after the intellectual tests were completed--amplitude did not return to control Other: performance on intellectual tasks was less due to noise	
<u>Author's conclusions</u> Both jet aircraft noise and highspeed train noise should be controlled to less than WECPNL 80, since this level reduced people's aptitudes on intellectual tests, causes mental fatigue, and may induce sustained tension of the sympathetic nervous system.			
<u>Evaluation & comments</u> The abstract was written on the partially translated article (parts translated were those dealing with the experimental design and effects on pulse rate and fingertip pulse amplitude).			

Kunitaka, E. et al. Studies on the effects of aircraft noise causing mental fatigue during some intellectual performance. Nippon Eiseigaku Zasshi 30(3):417-429, 1975. (Partial English translation)

The effect of jet aircraft noise on mental fatigue, intellectual performance, pulse rate, and fingertip pulse amplitude was studied in 9 female college students, of average age 20.6 plus or minus 0.89 years. The students were of good health and did not live near an airport, where they might have had prior frequent exposure to jet airplane noise. The experiments were conducted in soundproof rooms, with a background noise of 30 dBA and a temperature of 26°C plus or minus 1.13°C. The subjects took a card-sorting test and Kraepelin-Uchida's test under quiet conditions for 50 minutes in the morning and 100 minutes in the afternoon the first day. The second day, the same schedule was followed on the same subjects with the addition of continuous recorded jet aircraft noise equivalent to 120 flyovers (WECPNL 80) and an average peak level of 87 dBA. The noise was of high frequency having a peak of 1000 to 2000 Hz. Fingertip pulse amplitudes (of middle finger of left hand) and pulse rates were continuously monitored during the experiments. Degree of mental fatigue was measured before and after each session using the Cornell Medical Index of subjective psychophysiological complaints. Noise was associated with increased complaints related to hypertension. The intellectual tests alone acted as stressors, causing increased pulse rate and decreased amplitude, which returned to normal levels on completion of the tests. The fingertip pulse rate was greater before the subjects began the intellectual tests with the addition of jet noise. Noise also induced sustained tension of the sympathetic nervous system, since pulse amplitude decreased due to noise and did not return to normal levels after the intellectual tests were completed. The authors suggest that jet aircraft and highspeed train noise should be below WECPNL 80, due to the potential health hazards and effects on intellectual performance of this noise exposure level.

Nippon Eiseigaku Zasshi 30(3) : 417-429, 1975.

Partial Translation

STUDIES ON THE EFFECTS OF AIRCRAFT NOISE CAUSING MENTAL
FATIGUE DURING SOME INTELLECTUAL PERFORMANCE

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Noboru Ishinishi: Dept. of Hygiene, Faculty of Medicine,
Yasushi Kodama Kyushu University, Fukuoka

This is part 1 of a report of a study and investigation carried out in 1963 and 1964 by the Medical Faculty of Kyushu University relating to health hazards to residents who were living in the vicinity of Itatsuke Air Base of the U.S.A. in Fukuoka and were exposed to frequent aircraft noise of jet planes.

Test Procedure

1. Subjects of the Test

Nine female students from the same division of a certain public women's college were chosen as the subjects of the tests. These female students did not live in the specific air base vicinity and presented a healthy condition not being affected by the exposure to the jet noise. Their average ages were 20.6 ± 0.89 years old. A two-day continuous exposure test was scheduled for each subject avoiding her menstrual period.

2. Exposure Test Room (insulated room) Conditions

Two insulated rooms were used for the test, ie, one as a noise exposure room and another as a fatigue examination room. The insulated rooms consisted of: a 30cm thick outer wall, a suspended sound-absorbing inner wall, double doors for shutting the noise out and a non-echo type interior structure. The background noise of the room was 30dB(A), and the room temperature during the test periods was maintained at $26^{\circ} \pm 1.13^{\circ}\text{C}$ by controlling the air. The subjects were under surveillance from outside by monitoring TV. Communication between the subjects and the investigators was made by interphone, while fingertip pulse rates and pulse waves were measured by polygraph from outside the room.

3. Conditions for Noise and Intellectual Tasks

The noise of flying jets (F-102, F-105) of the U.S. Air Force taped around Itatsuke Air Base was edited to produce an environment of continuous flying noise. The maximum value of the flying noise was between 95dB(A) and 85dB(A) and the average value was 87dB(A), which was a high frequency range noise having its peak at 1000 and 2000 hertz. The time distribution of the noise exposure was 50 minutes in the morning and 100 minutes in the afternoon with a sufficient length of resting time inbetween. The each subject was committed to two days of intellectual tasks, ie, the first day under noise-proof conditions and the second day under jet flying noise. The intellectual tasks consisted of Kraepelin-Uchida's test using Uchida's method and a card sorting test.

Corneal Medical Index (C.M.I.) Questionnaire

Before and after the performance of the intellectual tasks, each subject was asked a total 81 questions relating to 9 items, C.I.J. and M - R out of the total questions regarding to 195 items of C.M.I., which was proposed by Fukamachi as the criteria to distinguish a neuropath.

According to the survey, the cases of complaints related to the cardiac blood vessel system, ie, questionnaire item C, were 0.4% higher under a sound-proof environment than under a noisy environment before performing the tasks, and the cases of complaints were reduced by 0.4% under a sound-proof environment and by 0.3% under a noisy environment after completion of the tasks.

Fingertip Pulse Waves and Pulse Rates

A pulsemeter was placed on the tip of the middle finger of the left hand before the start of the Kraepelin test until the completion of the test in order to continuously measure pulse waves and pulse rates. The measured values were those obtained during first 15 seconds of every 1st minute, and the values were multiplied by 4 for the pulse rate while the amplitude of the pulse waves was indicated by the average value of the total wave height.

The impulse rate during Kraepelin test under a sound-proof environment indicated an almost standardized fluctuation (Figure 4), where tension of the sympathetic nervous system during performing intellectual tasks and the relaxation of

the sympathetic nervous system during resting and after completion of the tasks were regularly observed. The maximum value was registered in 2-3 minutes after starting to perform the 1st series of tasks during the 50 minute test, while it was registered just before the completion of the tasks in the 2nd and 3rd series of tasks. Relating to the fingertip pulse rate under a noisy environment, the value before starting to perform the tasks was considerably large due to the noise, and the increase in pulse rate after starting the works, and the change in the pulse rate after completing the tasks were not as regular as seen under a sound-proof environment (Figure 5).

Figure (4) Key-1. 1st series of tasks
 2. 2nd series of tasks
 3. 3rd series of tasks
 4. before testing
 5. intermission
 6. aftertesting

Change in Pulse Rate During Kreapelin Test Under a Noisy Environment

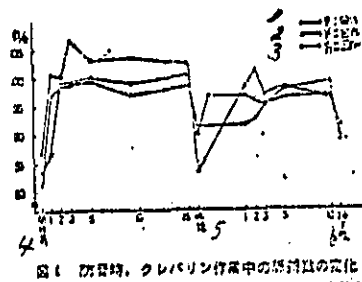
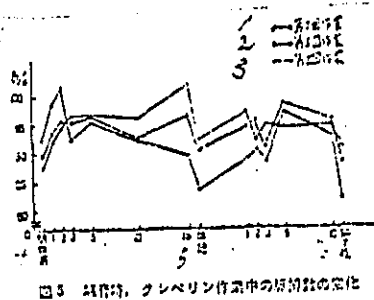


Figure (5) Change in Pulse Rate During Kreapelin Test Under A Sound-Proof Environment

Key-1. 1st series of tasks
 2. 2nd series of tasks
 3. 3rd series of tasks
 4. before testing
 5. intermission
 6. after testing



Referring to the fluctuations in amplitude of the pulse waves, they correspond to the pulse rate under the sound-proof environment (Figure 6), i.e; regular observation of tension and relaxation of the sympathetic nervous system as the tasks are being performed, minimum amplitude was registered during the first series of tasks and the maximum amplitude was registered during the last. Contrary to the above phenomena, under a noisy environment, a reduction in amplitude manifests strongly from before the beginning of the test (Figure 7), which remains all through the test allowing only slight increase of amplitude during resting and after completing the tasks.

Figure 6: Fingertip Pulse Wave Amplitude during Kreapelin test under sound-proof environment

- Key-1. 1st series of tasks
 2. 2nd series of tasks
 3. 3rd series of tasks
 4. before testing
 5. intermission
 6. after testing

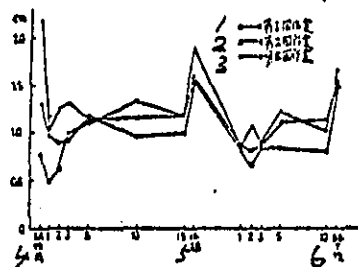


図6 静寂時、クレペリン作業中の指先脈波振幅

Figure 7: Fingertip Pulse Wave Amplitude during Kreapelin Test Under Noisy Environment

- Key-1. 1st series of tasks
 2. 2nd series of tasks
 3. 3rd series of tasks
 4. before testing
 5. intermission
 6. after testing

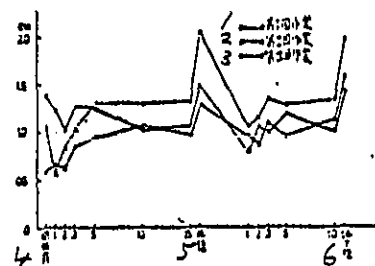


図7 騒音時、クレペリン作業中の指先脈波振幅

Also, the fluctuation in amplitude is not regulated. An interesting point here is the fluctuation during the third series of tasks under the noisy environment. The values of the amplitude of the pulse waves are larger than in the preceding two performances, which suggests that the effect

of the noise lowered the tension of the sympathetic nervous system.

Comparing the amount of fluctuation and the maximum and minimum values of the pulse rate and pulse waves during the performance of tasks (Table 19), under the sound-proof environment, the maximum value and the amount of fluctuation increased mutually, whereas under the noisy environment, both the amount of fluctuation and the maximum value reached a minimum during performing the third series of tasks, indicating that the noise changed the tension/relaxation rhythm of the sympathetic nervous system which corresponded to the duration of the test and created a sustaining tensed state.

COMMENTS

The amount of noise that the subjects were exposed to at this time had an average peak level power of 86.5dB(A) and flying frequency of 120 planes, which is equivalent to 80 of WECFNL per day. When the subjects were continuously exposed to this level of noise while performing intellectual tasks, cortical and mental fatigue increased. Also tension of the sympathetic nervous system and impairment of appetite for the tasks developed.

Conclusion

Among the subjective symptom of fatigue, "partial body pain" was most complained about under the noisy environment, all of which seemed to be related to the sympathetic nervous system.

It is appropriate for aircraft noise and super-express railway noise to be controlled under WECFNL 80, since this level of jet noise contributes to the increase in mental fatigue and the inhibition of appetite for performing tasks.

Table 19: Fluctuation of pulse wave amplitude during Kreapelin Test

- Key-1. 1st series of Kreapelin tasks
- 2. 2nd series of Kreapelin tasks
- 3. 3rd series of Kreapelin tasks
- 4. amplitude (cm)
- 5. under sound-proof environment
- 6. under noisy environment
- 7. maximum value

- 8. minimum value
- 9. difference
- 10. maximum value
- 11. minimum value
- 12. difference
- 13. maximum value
- 14. minimum value
- 15. difference

表19 クレベリン作製中の測定値の記録

	試料 (cm)	測定値	測定値
1	7 最大値	1.31	1.57
	クレベリン 測定値	0.66	0.58
	8 差	0.65	0.99
2	10 最大値	1.00	2.03
	クレベリン 測定値	0.49	0.37
	12 差	1.11	1.66
3	13 最大値	2.18	1.49
	クレベリン 測定値	0.66	0.79
	15 差	1.32	0.70

Nippon Eiseigaku Zasshi

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EFFECTS OF THE EFFECTS OF AIRCRAFT NOISE DURING MENTAL
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Studies of the Effects of Aircraft Noise causing Mental
Fatigue during some Intellectual Performance

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Studies of mental fatigue caused by exposure to the aircraft noise was carried out experimentally on 9 female students engaged in some intellectual performance. The tape-recorded noise of the flying jet engine was controlled from 65dB(A) to 95dB(A) in the peak level. The subjects were exposed continuously to the noise during 50 minutes and 100 minutes, respectively, in an insulated room protected from general outer noise.

Through the noise exposure period, the subjects performed intellectual tasks, that is, the Kraepelin-Uchida's test and card-sorting test. At the same time, pulse rates and pulse waves of their fingertips were measured. Before and after each exposure, the mental fatigue was measured by the critical fusion frequency (C.F.F.), the color naming test (C.N.) and the discriminative reaction of the multiple performance test (M.P.). And the information of subjective sensations of human fatigue were collected by the questionnaire. Central Medical Index (C.M.I.) were used for psychophysiological complaints. The control data of each subject were obtained by the same experiment except noise.

The following conclusions were derived from observations. The grade of mental fatigue increased in the case of the noise exposure rather than in the case of the noiseless situation. In C.F.F., fatigue increased 13% under noise exposure of 50 minutes, 37% under that of 100 minutes. In C.N. and M.P., as well as C.F.F., the same gradation between the noise exposure and fatigue were found. Furthermore, fatigue revealed itself more clearly in C.N. than in C.F.F., namely, the rate of prolongation of the time required for color identifying exceeded the critical level of mental fatigue, while the rate of mental fatigue in C.F.F. was within the normal or allowable range.

The results of the questionnaire of subjective sensations of fatigue and of the C.M.I. in the case of the noise exposure showed no remarkable differences compared with those of the noiseless situation. But, it was found that some complaints of sensations of fatigue increased slightly during noise exposure compared with those in the noiseless situation. These complaints were the symptoms probably caused by the sympathetic hypertension. Moreover, from the observation of the pulse rates and pulse waves, it was recognized that a biological basis of the relaxation of the sympathetic nerve system might occur when they were relieved from the intellectual tasks.

From the observation mentioned above, it is considered that there is a possibility of the inhibition of the learning effectiveness in school children when they are exposed to the aircraft noise such as

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the subjects were exposed to in this experiment. The exposed aircraft noise level used in this study, calculated from the peak levels and flying numbers, corresponds to 70 W.E.C.P.N.L. It can be said that mental fatigue induced markedly by the noise of 80 W.E.C.P.N.L. It is suggested that the noise level of 80 W.E.C.P.N.L. is not harmful to human being in the case of intellectual performance, especially during school hours.

I. 緒 言

騒音の影響に関しては多くの報告がある。そのなかで、生理的、心理的効果、公害騒音、うるささ等についての多くのもの¹⁾が発表のあることを認めているが、これを騒音に対する騒音として定量的にあらわすことは非常に困難らしい。近年、ジェット旅客機が、民間交通機関として一般化し、都市に飛来して騒音公害の多い空港周辺地域では、航空騒音による住民の被害が各所で訴えられた。また、国際航空機の全日への飛来に伴う沿線住民の被害の深刻化も大きな問題である。

これらの交通騒音を音源とする騒音は、その影響や環境騒音を定める場合、従来の騒音を主とした騒音と同一の考え方では申し渡すことが難しく、騒音公害の決められた等からみて、現在は騒音の“うるささ”を騒音公害の指標とし、“うるささ”の指標は、住民の主観的評価をもとにした社会心理学的評価法によって行なわれているのが現状である。“騒音公害”のアンチテーゼとして要求されるのは“騒かぬ環境”であり、その限りでは、“うるささ”を騒音公害の指標とすることに賛成はない。しかし、社会心理学的立場とは異なる科学的客観性をもって騒音をみる必要が一方では必要であろう。

昭和59年、当時の知事事務官(環境学)は、米原市立地として当時市立の使用下であり、其地周辺住民の健康被害を防止する目的で各自治体で実施された²⁾。若しくは、ジェット騒音(以下「騒音」とよぶ)の影響を中学生の健康被害から評価するために、飛行コース(下の中学校と上野(市立)区)の航空学学生を被験者として、昭和59年および60年の2回、2回間の同一被験者内で実験が行なわれた。第1回目は米原市立地(騒音)に、被験者(平均年齢)が騒音下にあったが、第2回目は米原市立地騒音下で実験が行なわれた。そのために、この2年間の調査結果が、同一被験者の「騒音曝露時」と「騒音時」を対照と比較した、実験的な条件をもつ研究となった。その結果、騒音曝露中の中学生の精神疲労は対照校生後より大きく、「騒音曝露」した環境では疲労度の差も消失し、学習成績に対する「騒音」の影響が、精神疲労

を指標として客観的に評価された。このことは、精神疲労を指標として取り立てている社会環境においても、精神疲労が騒音の影響の有効な指標となり得る環境であるといえる。

騒音の環境は騒音で、多因子性であるから、騒音曝露による実質的な騒音の発生、騒音曝露レベルとの関係および騒音に対するそれの測定法、精神疲労との関連の中で検討されなければならない。この研究はその第1回で、市立米原市立地騒音で騒音した「騒音」を曝露し、騒音曝露で精神疲労に罹患する被験者に対して、50分および100分間の騒音曝露を行って比較し、対照校生と比較した。騒音の曝露は、精神疲労状態によって評価するとともに、質問表検査、自律神経系の反応および精神生理学的立場からの観察も行なわれたものである。

II. 実験手続

1. 被験者

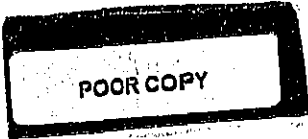
某公立女子大学の第一学年10名で、いずれも米原市立地周辺の住民を有しない者である。平均年齢20.61±0.59歳で、月曜日を避けて各被験者それぞれ2回2日間の実験日時を設定した。

2. 実験条件(騒音)条件

長さ30cmのコンクリート管内に騒音発生機および騒音計を置き、管内を騒音発生機とした騒音発生機をそれぞれ騒音発生機および騒音発生機として使用した。管内の騒音は90dB(A)、管内は騒音発生機により実験室中の騒音は26.0°±1.13°に維持された。被験者は実験からラウナーレビによって測定され、連絡電話はインターホンにより、騒音発生機(騒音)はシャッターによりすべて実験から退出した。

3. 騒音条件および精神疲労条件

市立米原市立地で騒音した米原ジェット機(F-102, F-103)飛行音を発生し、連続飛行音とした。飛行音の騒音は93dB(A)より95dB(A)の間にあり平均97dB(A)で、1600および2000ヘルツにピークをもつ高周波騒音である。騒音曝露時間は50分、午後100分の2回で、同じ十分な休息時間を取った。知覚作業負荷は、クレペリン内田法³⁾による前



半15分作題5分、後半10分計30分作題とい、5分休憩後カード整理作業1連を3回を行なわせ、100分作業時にはこの作業行程を繰り返した。カード整理作業は、よく混じた2組のカード計104枚を早く正確に分別整理する作業である。各被験者2日間の拘束で、第1日が練習時、第2日を1検査時とした。

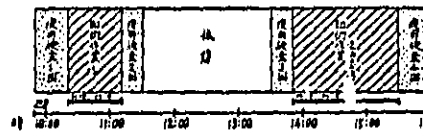


図1 実験のTime Table

4. 疲労検査項目

以下の各検査方法および装置により実施した。

- (1) フリッカー値 (Critical Fusion Frequency 以下 CFF という)：竹井機器フリッカー検査器
 - (2) 色名呼称時間 (Colour Naming Test, 以下 CN という)：分色式色名呼称器
 - (3) 識別作業反応時間 (Discriminative Reaction of Multiple Performance, 以下 MIP という)：竹井機器大脳式識別反応検査器
 - (4) 疲労感質問表：日本産業衛生協会請求発刊委員会「疲労自覚検査法しるべ」(1959)
 - (5) CMI (Cornell Medical Index) 質問表
 - (6) 脈博数および血圧測定：日本光電工業株式会社計測機製作所
 - (7) 聴力検査：リオン式スクールオーディオメーター CFF, CN, および MIP は30分および100分作業の前後に計4回検査した。CFF は下等法で連続5回測定し、最大値と最小値を平均して測定する。CN および MIP は一定の検出後検査を行ない、それぞれ100の色名呼称所要時間および100の作業反応時間測定である。3日の疲労検査法は、前に行なった中予12名員の疲労検査結果にもとづいて選択したものである。CFF は 中脳性疲労検査法として大脳皮質の興奮レベルとの相関性が認められ、CN はブロッキングによる作業反応時間の変化を所要時間の変化から検出出来るとされている。
- また、MIP は疲労反応における反応時間が精神活動の指標単位であり、反応時間には知覚—運動系の全般的レベルをあらわす¹⁾ものである。作業後測定しるべは1969年改訂版のものを使用し、CMI は全193問中質問法²⁾に

よって C. I. J. および M-R の9項目の間について、それぞれ全作業前後2回記入させた。疲労は、クレペリン作業時間を追って大脳皮質に生じた疲労感変化によって決定した。知的作業、疲労感および各検査の実験下表は(1)の通りである。

III. 成 績

- 1. クレペリン内用加算作業
下表にみられるように(1)(2)、第1回は50分作業時、第2回と第3回は100分作業時に順次行なったものである。(以下作業別に練習時-1, -2, -3, および検査時-1, -2, -3という)
- (1) 平均作業量
平均作業量は被験者によってかなりの差をみし、最低50.5、最高91.3であった。練習時、検査時の両方の平均値を比較すると(表1)、平均作業量はしだいに増加し、反復作業が作業熟練を促すことを示す。また、練習時作業量はすべて対応する検査時作業量を上回り、練習時間増加が作業量の増加を招き出した後々³⁾と一致する。ただし、検査時の作業量増加は常に小さくなるが、この原因としては、検査時の過剰な呼吸初期に強くあらわれ次第に弱まってゆくのか、あるいは練習が作業熟練を阻害するのかわいずれかが考えられる。
- (2) 休憩効果率
休憩効果率は正常値⁴⁾で1.10以上とされる⁵⁾。被験者の各作業時の値を見ると(表2)、検査時-1では全被

表1 各作業回数の平均作業量

	第1回作業	第2回作業	第3回作業	平均
練習時	65.3 ± 13.30	69.9 ± 16.86	71.2 ± 16.20	68.8 ± 15.97*
検査時	75.3 ± 16.59	76.3 ± 16.41	76.6 ± 16.16	76.1 ± 16.22
練習時—検査時	10.0	6.4	5.4	7.3

* 平均値 ± 標準偏差

が1.10以上であり、平均 1.17 ± 0.030 である。これに対して、昼番時-1では 1.09 ± 0.071 でわずかに1.10より低く、また、1.10以下を示すものが66.7% (6/9)で、初番時-1と比較すると有意に休憩効率の低下が注目されている。昼番時-2、および夜番時-3では、休憩効率率は初番時-2および初番時-3よりわずかに低いが有意ではない。また、夜番時、初番時として作業の反復によって休憩効率は次第に低下し、作業回数が（初めおよび上述）が小さくなる傾向を示し、その尤も夜番時-1と夜番時-2の間の低下を除いてすべて有意である。これらの比較の統計的有意性は、(表3)にみられる通りで、夜番の影響は第1回作業だけに明らかに認められる

が、以後は同らかの働きで精進されていると考えられる。

(3) 調整中

調整中は夜番時が初番時より低い(表4)。これまでの資料とは逆の結果であるが夜番による、交差神経緊張が、加算作業のような単純な型作業では誤りを減少する方向に働くとも考えられる。夜番時の調整中は作業反復により上昇し、また、全作業を通じて後半調整中は前半調整中より高く、後半調整中における割合は、夜番時と初番時の間に全く差がみられない(表5)。

2. カード管理作業

夜番時の作業所要時間は対応する初番時の作業時間より短く(表6)。これは作業法の傾向と同様に夜番が作業

表2 夜番時、調整時の休憩効率、作業回数別休憩効率

	H	S	Y. K.	Y. F.	M. T.	M. K.	Y. H.	N. K.	N. T.	平均	標準偏差
初 第1回作業	1.19	1.15	1.17	1.15	1.22	1.14	1.17	1.21	1.16	$1.17 \pm 0.030^*$	
初 第2回作業	1.21	1.09	0.99	1.11	1.10	1.03	1.07	1.04	1.07	1.08 ± 0.062	
初 第3回作業	1.03	1.04	0.91	1.04	1.02	1.00	1.04	1.01	1.02	1.01 ± 0.041	
昼 第1回作業	1.14	1.00	1.18	1.20	1.06	1.07	1.05	1.01	1.07	1.09 ± 0.071	
昼 第2回作業	1.15	1.00	1.11	1.00	1.09	1.09	1.00	1.04	1.08	1.07 ± 0.050	
夜 第3回作業	1.02	0.89	0.88	1.06	1.07	1.02	0.94	1.09	0.98	0.99 ± 0.077	

* 平均値±標準偏差

表3-1 夜番時、調整時の作業回数別休憩効率比較

	第1回作業		第2回作業		第3回作業	
	初番時	夜番時	初番時	夜番時	初番時	夜番時
休憩効率率	1.17	1.09	1.08	1.07	1.01	0.99
差	0.08		0.01		0.02	
有意性	有 t = 3.084		無		無	

表3-2 休憩効率率1.10以下の比較

	第1回作業		第2回作業		第3回作業	
	初番時	夜番時	初番時	夜番時	初番時	夜番時
1.10以下の割合、%	0/9, 0%	6/9, 66.7%	6/9, 66.7%	7/9, 77.8%	9/9, 100%	9/9, 100%
有意性	有 t = 0.102		無		無	

表3-3 作業反復による休憩効率率の低下

	初 番 時			夜 番 時		
	第1回作業	第2回作業	第3回作業	第1回作業	第2回作業	第3回作業
休憩効率率	1.17	1.00	1.01	1.09	1.07	0.99
作業回数の低下	—	0.09	0.07	—	0.02	0.08
有意性	—	有 t = 3.913	有 t = 2.885	—	無	有 t = 2.609

($t_{0.05}^{(8)} = 2.306$, $t_{0.01}^{(8)} = 2.898$)

表4 作業回別の前半、後半作業の所要時間

作業回	前半作業			後半作業		
	平均	標準偏差	標準誤差	平均	標準偏差	標準誤差
第1回作業	0.40±0.335	0.32±0.293	0.30±0.505	0.11±0.140	0.18±0.177	0.14±0.145*
第2回作業	0.17±0.147	0.35±0.271	0.25±0.412	0.13±0.172	0.22±0.151	0.17±0.094
第3回作業	0.32±0.304	0.33±0.297	0.33±0.289	0.23±0.183	0.31±0.208	0.29±0.176
計(1+2+3)	0.32±0.183	0.10±0.413	0.36±0.269	0.16±0.114	0.24±0.123	0.19±0.107

* 平均値±標準誤差

表5 後半所要時間が上昇する被験者の割合の作業回別比較

作業回	前半所要時間	後半所要時間
第1回作業	4/9 44.4%	8/9 88.9%
第2回作業	7/9 77.8%	7/9 77.8%
第3回作業	4/9 44.4%	8/9 88.9%

進行の速度を早める割合になったと考えられる。ただし、100分作業時の所要時間の変化を比較する際の作業所要時間比(後半所要時間-前半所要時間)/前半所要時間×100)で代用した(表7)。疲労時には明らかに比を示すものが多く、その差は有意である。

表6 作業回、飛行別のカード処理所要時間

作業回	第1飛行				第2飛行				
	平均	標準偏差	標準誤差	平均	標準偏差	標準誤差	平均	標準偏差	標準誤差
第1回作業	197.3	±32.81	±37.27	181.8	±31.82	±35.50	172.3	±32.05	±26.42
第2回作業	182.2	±26.54	±30.93	177.1	±34.33	±29.56	173.3	±33.15	±30.77
第3回作業	174.2	±27.29	±27.03	181.6	±34.13	±28.51	177.1	±40.77	±23.79

* 平均値±標準誤差

表7 100分作業時、作業回数によるカード処理所要時間延長率

被験者	S	Y.	K.	Y.	F.	M.	T.	M.	K.	Y.	H.	N.	K.	N.	T.	平均	標準偏差	標準誤差
初日	-2.62	1.06	3.81	3.35	-1.81	1.81	-5.70	-4.50	-2.81	-0.60	4.140*	4/9	44.4%					
2日目	-3.34	0.40	1.68	0.89	1.09	8.11	-1.74	2.09	5.06	1.70	±3.100	7/9	77.8%					

* 平均値±標準誤差

表8 作業前後の被験者別C.P.F.値

被験者	作業前	作業後										平均	標準偏差	標準誤差			
		S	Y.	K.	Y.	F.	M.	T.	M.	K.	Y.				H.	N.	K.
01	50分作業前	42.0	42.1	44.7	41.3	41.2	43.9	43.8	45.0	45.3	43.17±1.58*						
	50分作業後	39.3	40.1	43.3	40.5	40.4	45.2	42.1	45.9	44.1	42.32±2.407						
	100分作業後	41.4	41.1	41.3	40.7	43.0	45.7	44.7	46.0	44.1	43.44±2.095						
02	50分作業前	40.6	39.5	43.0	40.6	40.9	43.4	43.7	44.5	44.2	42.27±1.833						
	50分作業後	42.5	41.6	43.7	41.1	42.6	44.5	45.1	45.7	44.9	43.52±1.64						
	50分作業後	40.5	39.9	43.2	39.8	42.4	43.7	40.4	44.2	42.9	42.50±2.180						
03	50分作業前	40.9	41.4	43.3	40.7	43.2	40.0	47.2	44.7	45.3	43.64±2.333						
	50分作業後	39.8	40.2	43.9	39.2	43.6	44.6	39.8	43.5	43.6	42.04±2.213						

* 平均値±標準誤差

3. CFF

被験者の各 CFF 値 (表 8)、および平均値の変動 (図 2) では、閉音時、騒音時ともに作業前後の低下が起り、加算作業およびカード整理作業が顕微性疲労を生じることが認められる。作業後低下率および日間低下率を比較すると (表 9)、作業後低下率は騒音時が閉音時より、100 分作業が 50 分作業よりいずれも大である。閉音時 50 分作業後の低下率を 100 とした指数であると (表 10)、騒音時 113、作業後 130、騒音+作業後では 178 となり、騒音と作業後をいづれも、CFF を低下させ、作業を増加した場合の被害は、閉音時 50%、騒音時 58% の低下率増加であるから、騒音時には、作業原が 2 倍になると、約 2 倍の疲労増大を起す傾向にある。ただし、低下率 5% 以上の被験者は、騒音時 22.2%、閉音時 11.1% に過ぎず、平均低下率は閉音時 -2.48%、騒音時 -3.28% で統計的有意差といえず (表 9)、大抵に

よる精神作業の好悪傾向¹¹⁾を認めた CFF 値の低下も認められなかった。

4. 色名呼称時間 (CN)

呼称時間は第 1 作業日である閉音時に対して第 2 作業日の騒音時に、全般的な遅延がみられるが (表 11)、この傾向で、検出効果が大いことは著者らが以前にも認め

表 9 被験者別 C.F.F. 値の作業後および日間低下率 (%)

	50分作業後		100分作業後		日間低下率	
	閉音時	騒音時	閉音時	騒音時	閉音時	騒音時
H	-4.70	-6.43	-2.69	-1.93	-6.35	-3.33
S	-4.69	-4.73	-2.80	-3.89	-3.13	-6.18
Y. K.	-1.14	-3.13	-1.39	-2.93	+0.16	-3.80
Y. F.	-3.17	-2.41	-3.69	-0.25	-4.62	-2.17
M. T.	-0.47	-1.91	+0.09	-4.88	+2.28	-0.73
M. K.	-1.80	+2.56	-3.04	-4.16	+0.22	-1.14
Y. H.	+2.88	-1.64	-15.08	-2.31	-11.75	-1.40
N. K.	-3.28	+2.00	-2.68	-3.26	-4.81	-1.11
N. T.	-4.45	-2.65	-3.31	+0.23	-2.45	-2.43
平均	-2.25	-2.00	-3.55	-2.59	-3.35	-2.48
5%以上 ^a 低下者の割合	0/9	1/9	1/9	0/9	2/9	1/9

^a 大抵の基準

表 10 閉音時50分作業後低下率を100とした場合の騒音時100分、騒音時50分および100分作業後の低下率の指数

	閉音時	騒音時
50分作業	100	113 (100) ^a
100分作業	130	178 (158)

^a ()は騒音時50分作業後低下率を100とした場合の指数

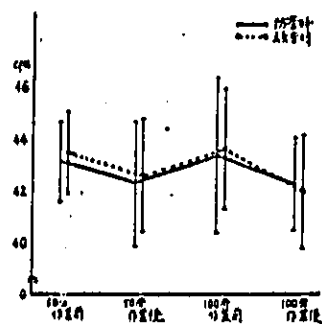


図 2 C.F.F. 平均値の変動

表 11 作業前後の色名呼称時間

	H	S	Y. K.	Y. F.	M. T.	M. K.	Y. H.	N. K.	N. T.	平均	標準偏差
閉音時	50分作業前	68.5	61.0	49.5	61.1	66.8	75.7	50.1	44.6	38.2	57.60 ± 11.083 ^a
閉音時	50分作業後	78.8	54.3	50.0	63.5	61.5	64.6	56.2	44.2	47.6	57.80 ± 9.989
閉音時	100分作業前	61.7	53.2	43.7	58.1	60.0	66.7	50.2	46.0	37.1	52.97 ± 9.467
閉音時	100分作業後	73.4	54.5	51.7	61.5	60.9	76.2	56.4	44.6	40.8	58.14 ± 11.682
騒音時	50分作業前	57.7	47.0	43.4	54.2	54.6	61.0	49.0	40.0	37.0	49.32 ± 7.956
騒音時	50分作業後	62.9	48.7	49.2	57.2	54.9	75.3	50.9	42.8	39.0	53.08 ± 11.140
騒音時	100分作業前	59.2	52.0	44.8	50.9	58.4	59.0	47.1	35.5	34.6	49.39 ± 8.962
騒音時	100分作業後	71.0	47.0	47.2	61.6	60.1	75.0	51.6	44.7	41.5	56.21 ± 12.460

^a 平均値 ± 標準偏差

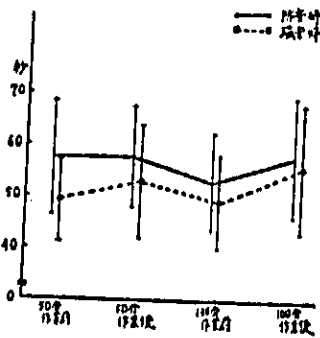


図3 色名呼称時間の変動

た通りである。ただし(表13)にみられるように、作業後には、明らかに呼称時間の延長がみられる。作業後および100分の呼称時間は単に延長率=(作業後呼称時間-作業前呼称時間)/作業前呼称時間×100であらわし(表12)、50分作業を100としてみると(表13)、作業増加は4.9倍、55分休息は4.0倍の延長率上昇を示し、騒音+作業増加は6.8倍で、CFFと比較して変動が非常に大きく、閉音時と騒音時の日間延長率の差は $1=8.378$ ($\log_{10} 1=0.013$) で極めて有意である。これはCNがブロッキング現象を現れやすいため、大聴覚野の生理的興奮レベルよりも作業時の心理的作業にむしろ強く影響されるのであろう。一方、閉音時50分

表12 被験者別色名呼称時間の作業後および日間延長率

被験者	50分作業		100分作業		50分休息		100分休息	
	延長率	延長率	延長率	延長率	延長率	延長率	延長率	延長率
H	9.01	15.01	10.93	18.96	23.03	7.15		
S	-2.77	-11.85	-9.62	3.01	0	-11.00		
Y. K.	13.36	1.01	5.36	23.17	8.76	10.91		
Y. F.	5.54	3.93	21.41	5.85	14.02	0.65		
M. T.	0.23	-10.61	13.18	1.50	21.06	-11.16		
M. K.	23.44	-11.66	27.12	14.21	22.95	0.66		
Y. H.	3.36	11.51	9.55	12.35	5.31	11.90		
N. K.	6.60	-0.90	16.10	-3.04	11.75	0		
N. T.	5.40	24.61	19.94	9.97	12.16	6.80		
平均	7.96	2.01	13.66	9.78	13.23	1.68		
10%以上の延長者の割合	2/9	3/9	6/9	4/9	6/9	2/9		
合計	22.2	33.3	66.7	44.9	66.7	22.2		

表13 閉音時50分作業後延長率を100とした場合の閉音時100分、騒音時50分および100分作業後の延長率の指数

	閉音時		騒音時	
	50分作業	100分作業	50分休息	100分休息
指数	100	487	306 (100)*	680 (172)

* () は騒音時50分作業後延長率を100とした場合の指数

作業の作業前傾が、他の各作業前傾よりもかなり大きいことが観察した結果のようにも思われる。100分作業時の延長率を騒音時と閉音時の比で示すと1.39:1であった。CFFの作業後低下率を同様に比で示した場合の1.37:1とはほぼ同じである。

また、騒音時の作業で作業増加の程度におよぼす差をみると、50分作業時:100分作業時の比は1:1.72である。CFFの場合の同じ比は1:1.58であるから、これも両被験者の結果はほぼ同じとみることが出来る。

ただし、色名呼称速度で疲労判定の基準として、10%以上の延長率は、各作業で被験者の20%以上に達し(表12)、日間延長率で騒音時は閉音時の3倍、作業後延長率で騒音時100分作業は50分作業の3倍の水準であり、平均延長率も10%を超える場合が多いことからみて、CNで評価される疲労は、やはりCFFによる疲労度よりも大きいといわなければならない。CNでは特に騒音や作業時間との相関を示さなかった。

5. 重複作業反応時間(MRP)

多種類の刺激に対して一旦、形成された認知-反応系を適切に動かせる複製反応は、反応時間そのものは個人差がかなり大きく、評価指標として採用されているが、被験者の平均反応時間は、正常人平均値0.775±0.093秒(表1)にして、閉音時最小0.619±0.104秒から最大0.638±0.080秒、騒音時最小0.573±0.071秒から最大0.621±0.083秒で反応系の働きは正常であった。この検査では、反応時間10%のあらわす疲労レベルが問題であり、疲労10%の反応時間からその精度度(e/m)を観察すると、作業後はつねに作業前より低く、知的作業進行の作業状態によって、作業後の反応系のレベルも高まっていると考えられる(表14)。作業前後の精度度を比較して、作業後精度度低下率[(作業後精度度-作業前精度度)/作業前精度度×100]を精度変化の指標とし、閉音時50分作業を100として指数化すると(表15)、閉音時100分作業では約1/3に減少し、55分は50分作業で

表14 作業前後のM.P.動脈圧平均値

	昼 時	夜 時
50分作業前	0.21±0.046	0.20±0.059*
50分作業後	0.18±0.041	0.17±0.049
100分作業前	0.19±0.033	0.22±0.050
100分作業後	0.18±0.076	0.16±0.034

* 平均値±標準偏差

表15 作業時50分作業後の動脈圧の低下を100とした場合の昼時100分、夜時30分および100分作業後の動脈圧の低下の指数

	昼 時	夜 時
50分作業	100	107 (100)*
100分作業	36	193 (160)

* () は昼時30分作業後の動脈圧の低下を100とした場合の指数

107、100分作業で193と増大する。昼時には、一定作業の反復から生じる作業疲労によって、興奮の過剰化と迷走神経の過剰活動、すなわち作業過剰が生じてゆくと考えられるのに対して、夜時には、興奮にうちかかって作業を遂行するために、自分の作業能力が要求され、作業後の認知-反応系の応答性を過剰に高めたとみることが出来る。このように考えれば、M.P.の動脈圧は、認知-反応系の応答性を支持する中枢神経系の興奮度の指標であり、作業後低下は、作業のための中枢の負荷度を示すもので、興奮は明らかに作業による負荷を増大するといえる。

Cの結果と、一般的な精神疲労検査法であるCFFやCNの結果とが、どのように対応するかはなお不明な点がある。昼時30分作業と100分作業を比較すると、動脈圧の低下は1:1.80となり、前に、CFFやCNでの同じ比1.58および1.72にはほぼ相当する値である。

6. 疲労感

日本産業衛生学会は、1970年に新しい疲労自覚度質問表を開発しているが、ここでは改定前の形式に採った。各症状群別の訴え率を、作業前後で比較すると(表16)、昼時には作業後訴え率が有意に上昇し、夜時には有意差を認めない。昼時の作業前訴え率は、昼時と比較すると有意に高く、また、両者の作業後訴え率には有意差が認められないことから、興奮は明らかに自覚疲労を増大したとはいえない。各症状群別にみると、身体的疲労、神経感覚的疲労の訴えでは昼時が高率

表16 作業前後の症状群別の平均訴え率

	昼 時		夜 時	
	作業前	作業後	作業前	作業後
A. 身体的疲労	5.6	23.9	13.3	21.1
B. 神経感覚的	12.2	21.4	22.2	26.7
C. 神経、感覚的疲労	4.4	20.0	12.2	15.6
計	7.4*	21.4	15.0**	21.1

*、**：1項目で相対度の存在を示す

で、逆の要因は身体的疲労では“肩がいたい”、“息がしんどい”の2項目、神経感覚的疲労では“目がしょぼい”、“手足がふるえる”の2項目にあり、1969年改訂で、削除された“目がしょぼい”を除き、前項成分(身体的疲労感)に分類されるものであり、いずれも自律失調因子と考えられる状態である。従って、興奮による交感神経緊張が、身体機能の不安として強く自覚されていることがわかる。

7. CMI質問表

198項目のCMI全質問のうち、探問が神経症者の判別基準として採用したC、I、JおよびM-Rの9項目の質問回答を作業前後に行なった。被験者は健康正常であるから、探問の基準による神経症傾向の判定にはあたらぬが、質問内容であるM不安感、N知うつ、O不安、P不安、Q怒り、R緊張の不安感傾向と自律神経系疲労、C心臓血管系、I筋力低下、J易感性的心身症傾向に対する探問の回答の可能性を調査した。項目別訴え率を比較すると(表17)、M-Rの訴え率は作業前では昼時

表17 C.M.I.質問表による項目別の作業前後の訴え率

	昼 時		夜 時	
	作業前	作業後	作業前	作業後
M	27.8	33.3	27.8	30.6
N	7.4	9.3	14.8	11.1
O	22.2	20.0	17.3	14.8
P	14.8	13.0	16.0	16.0
Q	23.5	24.7	20.0	24.7
R	24.7	23.5	20.0	23.9
精神的項目計	21.4	22.7	20.7	22.0
C	9.5	9.5	9.5	8.7
I	11.1	9.5	11.1	12.7
J	3.7	3.7	4.9	3.7
身体的項目計	8.1	7.8	8.5	8.1

C.I.J.	M-11										M-12									
	1	2	3	4	5	6	7	8	9	10	1	2	3	4	5	6	7	8	9	10
1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
4	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
5	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
6	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
7	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
8	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
9	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
10	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
11	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
12	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
13	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
14	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

図10 支那の主要な貿易商品の輸出入

支那の主要な貿易商品の輸出入は、1937年以降、急激な増減を繰り返している。これは、支那の経済状況と国際情勢の複雑な関係によるものである。特に、1937年の盧溝橋事件以降、支那の貿易は大きく影響を受けた。この表は、1937年から1945年までの主要な貿易商品の輸出入の推移を示している。

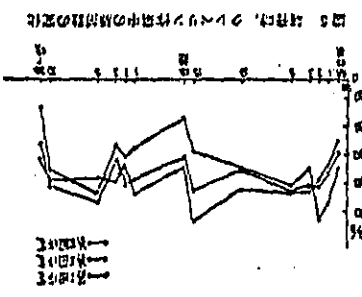


図11 支那の主要な貿易商品の輸出入の推移

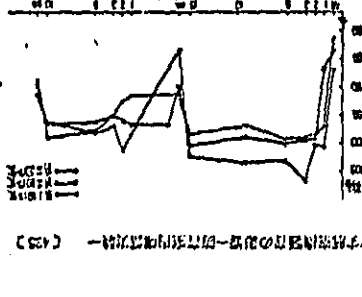


図12 支那の主要な貿易商品の輸出入の推移

支那の貿易は、1937年以降、急激な増減を繰り返している。これは、支那の経済状況と国際情勢の複雑な関係によるものである。特に、1937年の盧溝橋事件以降、支那の貿易は大きく影響を受けた。この表は、1937年から1945年までの主要な貿易商品の輸出入の推移を示している。

支那の貿易は、1937年以降、急激な増減を繰り返している。これは、支那の経済状況と国際情勢の複雑な関係によるものである。特に、1937年の盧溝橋事件以降、支那の貿易は大きく影響を受けた。この表は、1937年から1945年までの主要な貿易商品の輸出入の推移を示している。

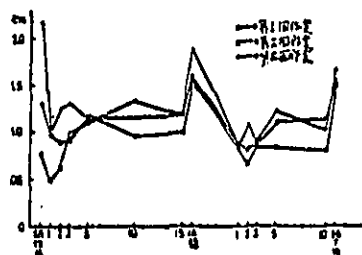


図6 騒音時、クレペリン作業中の脈拍数変動

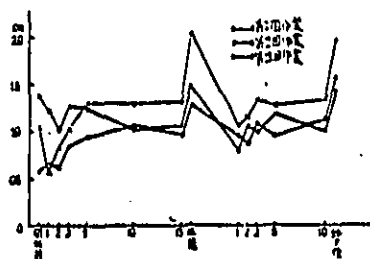


図7 騒音時、クレペリン作業中の脈拍数変動

表19 クレペリン作業中の脈拍数の変動

	測定項目	脈拍数 (b/min)	
		騒音時	騒音時
クレペリン 第1回作業	最大値	1.54	1.57
	最小値	0.66	0.53
	差	0.88	0.99
クレペリン 第2回作業	最大値	1.60	2.05
	最小値	0.49	0.57
	差	1.11	1.48
クレペリン 第3回作業	最大値	2.18	1.49
	最小値	0.86	0.79
	差	1.32	0.70

は不規則である。騒音時の変動で興味深いのは第3回作業のもので、安静時値は前2回よりも大きく、騒音による交感神経緊張は低下していることがわかる。しかし、休憩時の脈拍は第2回作業より小さく、終了後はさらに第1回より小となり、本来作業復旧によって起るはずの脈拍増加が抑制されていることを示している。作業中の

脈拍変動の最大値、最小値および変動量を比較すると(表19)、騒音時には最大値と変動量が相俟って増加してゆくが、騒音時は、第3回作業で最大値も変動量も最小となり、騒音が作業経過に対応する交感神経系の緊張と弛緩のリズムを変え、持続的な緊張状態を生じているように思われる。

なお、騒音で時に聞かされるパターン変化は、特に気配を認めない。

9. 聴力検査

50分および100分の騒音暴露前後に計4回の聴力検査を行ない、一過性聴力損失の有無を検査した。今回の騒音暴露時間は計150分で5時間以内であり、日本航空衛生学会の調査基準をかりに所定騒音暴露にまで拡大しても、適合することは無理であるが、一般に一過性聴力損失(TTS)の2分値(TTS-2)をもって10年後の永久聴力損失を推定する考え方が認められており、聴力損失の限界として15dBとするものが多い。被験者は、騒音暴露後直ちに聴力検査を行ない、TTSを測定したが、500、1000、2000、4000、および8000ヘルツにおいて5dB以上の聴力損失を認めず、今回行なった実験騒音で騒音性聴覚が生ずる可能性は考えられない。

IV. 考 察

学校騒音については、クレペリン作業結果、会話検査、うるさき等の研究がこれまでも報告されているが、学校騒音での騒音の影響として、精神疲労を増加することが、実態調査で明らかである。現在の社会が、知識階級を基盤として成り立っていることから考えると、職業衛生や公害の面から、騒音の影響の社会的側面として、精神疲労をとりあげることは妥当であると考えられる。ただし、そのためには、騒音と精神疲労の間の因果関係を明らかにすることが必要である。

若者の実態調査は、中学生集団の授業中の実態調査であって、疲労の要因としての他の影響を十分排除出来ないうちであった。今回の実態調査は、出来事だけでなく他の要因を除いた、同一条件の同一人の、騒音時と安静時を対比したもので、実態よりいくつてはるかに精度の高い結果であるといえる。実態調査は実態調査時の騒音条件に適合するピークレベル85dB A~95dB(A)で、飛行機騒音に制限した録音テープを使用し、午前50分、午後100分の2回、被験者に曝露した。これは、当時の騒音の時間的変遷を最大にしたもので、150分の曝露は、当時の時間的特性すなわち曝露時間中1/6に相当する飛行機騒音を除く15時間(午前7時から午後10

時まで) 続けた場合の自覚的疲労に等しい。

現在、航空機騒音の環境基準に使用されている WECPNL は、飛行の開始を問題とせず、飛行開始と飛行時間を考慮して決定するが、今回の実験騒音騒音量は、ピークレベルのパワー平均 86.5 dB(A)、飛行高度 120 度で、WECPNL は 80 に相当する。

文献的には、騒音と知的作業に関する報告は数多く、平均 70 dB(A) の道路騒音で中学生の集中力の低下が Karandorf¹¹⁾ により、62~71 dB(A) の学校で授業後の試験の上昇が Flynn¹²⁾ により報告され、また Broadbent¹³⁾ は単純計算作業所要時間が、100 dB(A) の騒音環境では対照時 70 dB(A) に比べ上述の半の消費を示すことをのべている。鈴木¹⁴⁾ は、高校生に騒音を負荷して学習の成績の増加を報告し、一方、最近、丹ら¹⁵⁾ は、騒音は学習効果のみには影響されずとして騒音を否定している。いずれにしても、これらは作業効率を問題とした調査で、負荷騒音は逆効果であり、航空機騒音や同音帯については授業時間内の飛行音の不快感¹⁶⁾ や、実地的な騒音と知能に関する研究¹⁷⁾ 等以外に、知的作業の精神疲労を問題とし、騒音を実験的に負荷した比較研究はこれまでみられない。

今回の実地的騒音負荷によって著者は、騒音の知的作業、作業中の疲労、騒音および精神疲労に対する影響を、同一被験者について、同一手続による対照実験と対で行なうことによって、騒音と精神疲労の因果関係を追究した。

クレベリン作業成績からみると、平均作業量は 3 回のクレベリン作業でいずれも、騒音時が大きいが、作業量の作業量増加傾向は初音時の方が大きい。ただし、実験手続上、騒音時が第 2 回の成績であることから、この結果を騒音による上進効果の現れであると結論出来ない。

休憩効果率では、影響は第 1 回作業のみに認められ、騒音環境が持続すると、休憩効果に対する影響は消失される。従って、内田のいう作業阻害¹⁸⁾ に対する騒音の影響は、休憩初期だけにみられる一時的なものといえるが、作業内容が単純増加の過程と異なり、多様な精神活動が必要とするような知的作業の場合、同じ様に、作業量が補償されるとは断言出来ず、クレベリン作業は、作業負荷としては適当であっても、作業効率の変化からこれを全精神活動の能力にまで拡大して解釈するのは難しい。

作業負荷の現れは、騒音と関係なく休憩効果を著しく低下させ、第 3 回作業では、初音時 1.01、騒音時 0.99 で、休憩効果自体がほとんど認められなくなる。

休憩率は騒音時がほぼ低く、特に第 1 回作業の差は

大きく統計的に有意である。しかし、騒音時には、作業反復によって誤差はほぼ規則的に増加し、練習効果による誤差の減少¹⁹⁾ とは逆の傾向、すなわち騒音の上進傾向がうかがえる。カード整理作業で、騒音時に作業時間の延長が有り、内音時には練習効果による作業時間短縮傾向が明らかであることも、同じ様に騒音の作業は逆に対する傾向を思わせるものである。

疲労指数の結果を総合すると、騒音が疲労を助長する傾向が認められた。CFF の日間低下率は初音時 -2.48%、騒音時 -3.33% で、騒音時の値は 1.35 倍大きく、知的作業が人体疲労性疲労を生じ、騒音はさらに疲労を増強する。また、作業後低下率の指数化から (表 10)、作業量が増加した場合の疲労の増加は、初音時 30%、騒音時 58% で約 2 倍であり、騒音による疲労の増加が明らかである。

CN 所要時間の日間低下率は、初音時 1.68%、騒音時 13.23% で、7.88 倍であり、作業後低下率は、初音時 50 分作業と比較して、作業時間増加で 4.9 倍、騒音負荷で 4.0 倍の増加を示し、前者の範囲による疲労の増大が著しい。騒音+作業増加では 6.8 倍、騒音時の作業増加は 72% の疲労度上昇である。CN は心的疲労を評価するもので、心理的影響が大きく、疲労感とその範囲の時間的大きさが必ずしも算術的に比例しないであろう。CN の疲労判定基準²⁰⁾ によると、騒音時の日間低下率では、10% 以上の延長が 0.7% にみられ、初音時を合めて、ほとんどが 25% 以上であり、CFF の日間低下率がほぼ限界以内であるのと比較して、CN の場合には、より大きな疲労度が判定される。さらに、MP では同程度の作業低下後を指標として、騒音の影響が明らかにもられた。知的作業の量に対しては、作業を行なうための集中力が要求される。この作業中の緊張が、作業後に作業前よりも高い興奮性のレベルを示す理由で、作業後の安静低下は、作業進行による中枢の負荷を示す尺度であり、作業遅延は、この負荷を軽減する方向に働くと思われる。(表 15) をみると、初音時には、作業開始によって、100 分作業時にはむしろ精神疲労の低下がみられ、騒音時は、騒音による安静低下が強い緊張の持続を要求するために、安静度の作業後低下が 100 分作業より、50 分作業よりも大きくあらわれたことを理解出来る。

各機能負荷の結果から、騒音時の 50 分作業と 100 分作業の疲労度を比較してみると、CFF、CN および MP でそれぞれ 1.58、1.72 および 1.80 の倍率となり、騒音時の作業増加による疲労の増加は、ほとんど変わらない。しかし、初音時と比較した場合、50 分作業と 100 分作業によって、各検査法の疲労度はかなりの差を示し、騒音の

有差は色覚検査時に強く影響し、検査の結果は MIP の知覚性に大きく影響を及ぼす。したがって、知的作業時の検査の結果は、検査の結果には心的疲労として、検査が維持される場合は作業負担の増大として強くあらわれるようである。

MIP のあらわすものは脳波変化現象ではないにしても、検査の結果による自律神経系の機能変化として CFF や CN よりむしろ大きく表示されることは注目する必要がある。

飛行および操縦時の反応をみると、クレベリン作業に伴う交感神経系の興奮と抑制が、飛行時には規則的にみられるが、検査時には不規則となり、特に第3回作業で作業負担の増大と作業負担の減少が強い。

検査による交感神経系との関係、つまり「動れ」の現象についてはかなり研究され¹⁰⁾、「動れ」を認めるものが多いようである。一般に知的作業を遂行する場合の交感神経系は、作業時にみられるように、作業を重ねる毎に次第に興奮し、作業遂行とみられる興奮を示すものと思われる。これに対して、検査時は、作業中の興奮は作業前に始まり、作業後の興奮状態は次第に減弱して、作業遂行として期待される傾向と逆の結果を示した。このことは、検査結果の交感神経系の変化(表19)に明らかのように、検査中交感神経系の持続的な興奮状態をひきだし、作業負担と操作のリズムを変え、作業遂行を阻害し、過剰な興奮があらわれることを物証している。これは、MIP 検査時の低下と状態を一にした、自律神経系の生理リズムに対する検査の干渉と思われ、知的作業時の自律神経系の状態からみれば、「動れ」とは逆の現象である。

自覚疲労の測定法は、日本航空衛生学会の刊行のものを使用した。クレベリン作業と自覚疲労では、女子学生の場合¹¹⁾で作業中の疲労および身体各部のいたみの訴えが多いことが報告されており、また、女子一般事務作業の訴え半分の基準が得られたこと¹²⁾が示されているが¹³⁾、今回の検査者の訴え率は、作業後で検査前、検査時ともに一般事務より高く、女子学生の検査後より低い。これは、検査者の知的作業の性質が、一般事務よりも強度が高いが、検査後の疲労時間数でないことから考えて妥当であろう。検査時と検査後の作業後訴え半分の間には有意差はないが、検査時に多く訴えられた質問項目は「頭がいたい」「息が短い」「目がしょぼい」および「手足がふるえる」であって、1969年改訂で部成分として「身体」部成分のため、異相態に分類される症状であり、交感神経興奮状態を指向するといえる。なお、CMI による訴え

率および疲労判定基準から、検査の不安水準に対する影響は特に認められなかった。

以上の結果から、知的作業時の検査結果は、検査時と比較して、大なり度で検査および心的疲労を増大し、また、交感神経系と作業遂行を促すことが明らかである。

今回の検査は、80 WECPNL に相当するものであるが、航空機操縦者検査が 85 WECPNL であることから考えると、現在、飛行場周辺地区住民の検査検査負担は相当大きく、環境基準の達成される10年あるいはそれ以上後まで、精神疲労を避ける、かなりの検査を受けるものといわねばならない。

また、知的作業遂行に対する興奮と考えられる結果から、学習効果の興奮や疲労を生ずる可能性も指摘されるため、今後の、より詳細な研究が必要である。

これは、検査と自律神経系変化の報告で、Harris¹⁴⁾が Emergency Reaction として考え、「動れ」によって興奮がなくなるとのべているのとは異なった結果であるが、より長期間の検査条件下で、知的作業に対する作業遂行、および精神疲労に、「動れ」の現象すなわち検査の興奮の消失が起り得るかという問題も重要である。

航空機検査の検査基準は、社会生活によって、住民のうるささについての苦情の訴え20ないし30名で認められているが、精神疲労および知的作業遂行からの影響も、特に学校保健を中心にして測定されることが望ましいといえる。

V. 結 論

検査時間内で、検査者に一定の知的作業を遂行させ、作業中に「検査の身体的学習を行なって、作業遂行、作業中の交感神経系および精神疲労を抑制した」、「検査は、飛行場周辺地区で検査した航空用シート検査飛行をターフ検査したものを併用した。検査者に対する検査レベルは、飛行場周辺地区の検査時間内検査レベルとし、飛行速度を最大とした航空飛行で午前中60分、10分の休息時間において午後100分の検査を行なった。検査者は、同一検査者の検査結果である検査時との比較である。検査した「検査は1日版として80 WECPNL に相当する。

検査者は、性、年齢、知的能力、健康状態などは一定の航空機検査大に検査結果生り者で、「検査時間生活様式のないものである。

検査結果を総括して、次のように結論された。

- 1) CFF, 色覚検査結果, および自律神経系反応検査の結果から、検査によって知的作業時の精神疲労が増大す

る傾向が示された。疲労程度での、作業的加と疲労度の増加は、各検査法においてはほぼ同一の割合を示したが、疲労時と比較すると、疲労の初期には強い心的疲労としてあらわれ、疲労の後半によって、作業量も著しく減り、作業速度による誤差も増加される傾向が観察された。

日間低下および夜間低下からみると、CNの疲労度(疲労基準)を越えており、CFEでは経過観察内であるので、単純作業のような加的工作の場合、心的疲労すなわち作業量の変化の傾向が大きくあらわれると考えられた。

2) クレバリン加工作業の場合、加的工作に対する疲労の影響は、疲労の初期を過ぎ加減されるように思われた。しかし、カード作業を含めて、加的工作の種別上はどの程度かがわかれ、また、作業中の交感神経活動から、作業速度のリズムの乱れ、持続的な交感神経緊張がみとめられた。このことはMFの結果からも示行されることで、疲労が作業効率を阻害し、学習効果に悪影響する可能性を示唆するものであり、特に学校成績と疲労の関係として重要であろう。

3) 疲労自覚能で検査時に強く訴えられたのは、「身体の一部がいたむ」に相当する症状で、すべて交感神経緊張と関連するものであった。

4) WECPL80の「騒音で実際に精神疲労大をみとめ、作業速度の低下も考えられることから、騒音は疲労あるいは精神疲労は少なくともこれを下回るのが妥当である。

3) 加的工作を目的または手段として成立する環境では、作業によって生じる精神疲労および作業速度の低下が、騒音の影響を測る一つの示標となり得ると思われた。(この研究は、九州大学工学部心理学教室で、原田名洋教授の指導により行なわれたものである。)

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SECTION 15

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> same as above
<u>Citation</u> Marinyako, A.Z. and V.V. Lipovoy. An estimate of the total time of individual noise effects in hygienic evaluation of intermittent noises. Gig. Truda i Prof. Zabol (8):15-18, 1972	
<u># of Ref.'s</u> 1	<u># of Fig.'s</u> 1
<u>Language</u> Russian (Eng. transl.)	
<u>Type & duration of experiment</u> laboratory--acoustically isolated chamber	<u>Purpose for study</u> to compare the effects of continuous v. intermittent noise on fingerpulse amplitude, pulse rate, brain function, and auditory threshold
<u>Description of test groups (subjects, #, age, etc.)</u> 20 healthy 18-25 year old men	
<u>Control of other stressors</u> laboratory conditions	<u>Statistical Methods</u> used but not specified
<u>Noise Stimulus</u> source: not specified spectral characteristics: low frequency--250 cycles/sec. (average) noise level: 110 dB (low frequency) 105 dB (high frequency) length of exposure: 1 hour (total time equal for continuous and intermittent noise) # of trials: 4 series; 20 observations each (1 hr. noise, 1 or 2 hrs. quiet per series)	<u>CVS Response Measured</u> pulse rate fingerpulse amplitude (index finger) <u>Nonauditory effects</u> fingerpulse amplitude--greater decrease with CVS; intermittent noise than with continuous noise. pulse rate--decreased pulse rate w/ both continuous & intermittent noise Other: reaction time and accuracy on a simple task decreased due to noise to a greater degree with intermittent noise.
<u>Author's conclusions</u> Continuous noise decreased hearing sensitivity to a greater degree than intermittent noise. Greater effects on the vascular and nervous systems were produced by intermittent noise. The authors feel that the main variable is the combination of noise and quiet used, not the total noise exposure time.	
<u>Evaluation & comments</u> the number of subjects in each of the 4 parts of the experiment was not defined.	

Marinyako, A. Z. and V. V. Lipovoy. An estimate of the total time of individual noise effects in hygienic evaluation of intermittent noises. Gig. truda i. Prof. Zabol. (8):15-18, 1972. English translation.

The effects of intermittent and continuous noise on hearing sensitivity, the nervous system, and the cardiovascular system were studied in 20 healthy men, 18 to 25 years old. The nervous system effects were tested using the subjects' reaction time to a simple task. Cardiovascular functioning was measured using the pulse rate and the fingerpulse amplitude. The experiment included 4 parts, all of which were done in a sound-isolated room. The first 2 parts studied low frequency (medium 250 cycles per second) noise at a level of 110 dB for a total of 1 hour noise exposure and 2 hours quiet. Part 1 involved a 1 hour continuous noise dose, and part 2 consisted of 2½ min. noise and 5 min. quiet alternately for 3 hours. Parts three and four of the experiment used a noise level of 105 dB at a high frequency (medium frequency 200 cycles per second) for a total time of 1 hour noise and 1 hour quiet. A continuous noise dose was used in part three; ½ minute noise was alternated with ½ minute of quiet in part four. Continuous noise had a greater effect on hearing sensitivity, whereas intermittent noise had a stronger effect on the nervous system and cardiovascular functioning. Subjects made more errors on the simple task during the intermittent noise of high frequency than with high frequency continuous noise; no difference in number of errors was found with low frequency noise. Intermittent noise, especially of high frequency, affected the fingerpulse amplitude response to noise. The decrease in amplitude was greater with 50 minutes of intermittent noise (52% of the original amplitude) than with the same exposure to continuous noise (72% of the original amplitude). The pulse rate decreased by 2 or 3 beats with both continuous and intermittent noise. The total noise exposure time may not be as important as the combinations of noise and quiet in producing certain nonauditory physiological effects.

Gig. Tr. Prot. Z. obel. (5): 15-18, 1972
(Amoy)

AN ESTIMATE OF THE TOTAL TIME OF INDIVIDUAL NOISE EFFECTS
IN HYGIENIC EVALUATION OF INTERMITTENT NOISES

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(Arrived at the editor July 20, 1971).

An analysis of the literature concerning the study of the influence of intermittent acting noises on the organism shows that in one case the investigators observed a more expressed influence in the case of an intermittent effect, and in the other case, the intermittent effect of noise caused the same effect or to a lesser degree a biological effect compared with continuous noise. Some authors (Poth and Weinberg) believe that the degree of harmfulness of intermittent noises together with the calculation of the level and of the spectral makeup is determined by producing the general time of their effect on acoustical energy, that is to say, in the opinion of the indicated investigators, the [illegible] amount of effective acoustical energy is harmful to the same extent depending on how this energy is distributed in time. Meanwhile, according to the data of Schröder and Rempt, stable noise causes greater shifts on the part of some physiological functions, in particular hearing, than intermittent noise (other conditions being equal), if the time of effect of the stable noise and the total time of the intermittent noise are the same.

In connection with what has been stated, we are confronted with the problem of studying the characteristics of the influence of some forms of intermittent noise compared with stable noise with the same total effect time of them. The studies were carried out in a sound-insulated chamber with 20 practically healthy people of the male sex aged 18-25. There was a series of 4 with 20 observations in each. In all the series, the people being studied were subjected to the effect of noise for the course of 1 hour.

In the first series, we studied the influence of low frequency (maximum acoustical energy in the octave band with a medium geometric frequency of 250 cycles per second) stable noise with a level of 110 decibels. In the second series, we established the influence of noise with the same level and spectrum, but acting intermittently: the noise period of effect of 2 1/2 min alternated with 5 minute intervals. Such a character of the noise exists in some operational regimes of a vibrational area according to the condensation of the concrete. The general time of the experiment in this series of investigations amounted to 3 hours (total time of the noise 1 h and the time of the pause 2 h). In the IIIrd series we studied

the influence of high frequency (maximum acoustical energy in the octave band with medium geometric frequency 200 cycles per second) stable noise with a level of 105 decibels. In the IVth series, we evaluated the influence of intermittent noise, where the half minute high frequency sounds with a level of 105 decibels alternated with half minute intervals. Noise with such parameters is noted with the hydraulic study of pipes. The time of the experiment in the given series of investigations is 2 h (period of the noise 1 h and the time of the pause 1 h).

With the subjects, we studied the functional state of the auditory equipment, the cortex of the brain and some functions of the heart-vessel system (tone of the vessels, frequency of the pulse). The auditory sensitivity was determined by an audiometer at tones of 500, 1000, 2000, 4000 and 6000 cycles per second by studying the sound conductivity of the air.

The study of the functional state of the brain cortex was carried out with the aid of an apparatus for studying advanced nerve activity, the principle of effect of which is based on presenting stimulation to the subject with a different wave characteristic and obtaining the corresponding response to it.

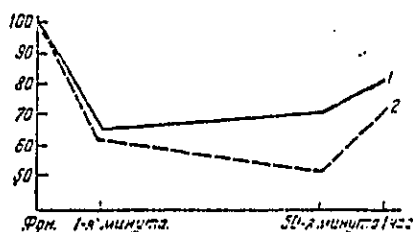
On the front panel of the instrument there are two rows of buttons (8 buttons in each) of different colors and a screen on which colored signals appear in a determined sequence at a rate of 22 to 110 per minute which light up and go out automatically. With the appearance on the screen of the same or different colored signal-stimulation, the subject must press the button of the corresponding color. With a correct and well-timed reaction (up to the appearance of the following stimulation), a signal confirmation is given along with the signal stimulation. The sequence of the presentation of stimulations is determined experimentally with the aid of 4 programs (according to 8 colored signals in each). The maximum rhythm of the delivered signals is established for each subject at which his responses contain the minimum number of errors. For the majority of those studied, 60 presentations per minute is the maximum rhythm. The tone of the vessels is determined with the aid of a twin-channel, plethysmograph. The plethysmogram is recorded with the index finger. When analyzed, the plethysmograph is considered to be the amplitude of the volumetric changes and the amplitude of the pulse strokes.

The results of audiometric studies showed that despite the similar total amount of acoustical energy, the stable noises caused a more expressed decline of hearing sensitivity, than intermittent noise. So, after the effect of low-high frequency noises with levels of 110 and 105 decibels, a decline of hearing in the frequency of 4000 cycles per second was noted, corresponding to 24 and 26 decibels. After the effect of the intermittent noises, a drop of 15 and 14 decibels was noted. It should be noted that after the effect of the stable noises, we noted a decline of hearing not only in the frequency of 4000 cycles per second, but at adjacent frequencies---3000 and 6000 cycles per second (on the average of 20 and 17 decibels). After the effect of intermittent noises, the decline of auditory sensitivity at the indicated adjacent frequencies was less expressed (10-13 decibels). The fact that the time reduction of hearing was noted at tones adjacent to the tone 4000 cycles per second is evidence of the great fatigue of the auditory analyzing equipment with the effect of stable noise compared with intermittent noise.

The results of the study of advanced nerve activity show that in the case of the effect of stable as well as of intermittent noises there was a decline in the rate of the nerve processes in the case of those studied, certainly increasing the amount of erroneous responses to the colored stimulations. However, a more expressed tendency to a worsening of the indicated characteristics was observed after the effect of intermittent noises. So, after the effect of high-frequency, intermittent noises with a level of 105 decibels, the subjects made 2.7 ± 0.26 errors where the original amount was 1.9 ± 0.18 errors. Stable noise caused an increase of the erroneous responses up to 2.5 ± 0.27 errors compared with an original value of 2.0 ± 0.17 errors.

After the effect of the low frequency, stable and intermittent noises with a level of 110 decibels, the amount of erroneous responses increased to the same degree. in the case of stable noise from 3.8 ± 0.21 to 5.4 ± 0.43 and with intermittent noise from 3.0 ± 0.27 to 4.6 ± 0.29 .

A rather notable difference in the effect of stable and intermittent noises (especially with high frequency components) was observed in the study of the tone of vessels (see figure). It is seen on the drawing that with a 1 minute effect of stable and intermittent noises, the amplitude of the plethysmogram amounted to 65 and 62% correspondingly of the original level. In a 50 min exposure to stable noise, which constitutes the same time of the general total effect of intermittent noise, the difference in the amplitude changes of the plethysmogram was more noticeable: in the case of a stable noise, its value amounted to 72%, in the case of an intermittent noise, it amounted to 52%. The observed difference in amplitudes is statistically reliable.



Background of the 1st min. 50 min/1 h

Change in the amplitude of the finger plethysmograph

According to the axis ordinate--amplitude of the plethysmograph (in %); according to the axis of the abscissa, effect time of the noises; 1--stable noise; 2- intermittent noise.

After a one hour effect of the stable and intermittent noises, we observed a tendency toward reducing the frequency of the pulses on the average of 2-3 per minute. It is not thought possible to explain the dependence between the character of the effective noises and the degree of reduction of the pulse.

Thus, the investigations which were carried out provide a basis for thinking that the intermittent effect of noises causes a different reaction compared with stable, despite the similar amount of acoustical energy, perceived by the hearing organ. So, for the auditory analyzer, the distribution of acoustical energy in time with an intermittent noise effect turns out to be positive. Evidently, the comparatively long pauses between noise facilitate auditory sensitivity and an increase in the adaptation processes. The fact that after the effect of intermittent noises, the functional state of the brain cortex is changed to a large degree is probably explained by the fact that intermittently the active noises are subjectively perceived as more unpleasant, and due to this may exert a greater effect on the mental activities of the man, which in turn reflects the condition of the higher nerve activity.

The more expressed vessel reaction to the effect of intermittent noise evidently may explain the difficulty of working out an adaptation of the vessel system to individual intermittent sounds.

Taking into account the results of the present studies, and also the studies carried out by us and other fellow workers in our laboratory in recent times (A.A. Men'shov et al.), it can be expected that with the hygienic characteristic of the intermittent effective noises in each concrete case, one must mainly take into account the time combinations of the "noise-pause" complex, and not the total time of effect of the noise.

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AN ESTIMATE OF THE TOTAL TIME OF INDIVIDUAL NOISE EFFECTS IN
HYGIENIC EVALUATION OF INTERMITTENT NOISES

A. Z. Marinyako, V. V. L'povoy

Summary

The effect on the human organism of stable noise (low-frequency of 110 db and high-frequency of 105 db) and of the same, but intermittent noise was studied in a sound-proof chamber. Low-frequency soundings of 2 1/2 minutes duration alternated with 3 minute long intervals. Half-minute long high-frequency soundings alternated with half-minute long intervals. In all cases the sum-total of the exposure to the effect of sound lasted one hour. In persons undergoing examination the authors determine time shifts of the auditory thresholds, functional state of the brain cortex, vascular tone and pulse rate. The effect of stable noise was found to produce more pronounced changes in the auditory function, whereas at the level of the higher nervous and vascular systems the shifts proved greater under the effect of intermittent noise.

The work of the emergency medical aid physicians entails a considerable mental and emotional stress, a high sense of responsibility and is distinguished by the complexity of the functions to be performed. The authors have set the task to study the working conditions of the emergency aid physicians, to elucidate changes of their performance capacity throughout the 24 hour long stay on duty. The functions of the central, vegetative and cardiovascular systems were investigated to measure the degree of fatigue. An analysis of the resultant information enabled it to establish the presence of fatigue and emotional stress in physicians of the emergency medical aid consequent to performance of their duties and also to clear up certain regularities in the dynamics of these processes taking place in each system under study. A subjective sensation of fatigue made itself felt simultaneously with initial signs of fatigue, but, while the fatigue in some systems of the organism somewhat decreased by the end of the work-day under the effect of some rest, the sensation of general fatigue steadily increased. In emergency medical aid physicians with a long service record no manifestations of overstrain were observed.

УЧЕТ СУММАРНОГО ВРЕМЕНИ ОТДЕЛЬНЫХ
ШУМОВЫХ ВОЗДЕЙСТВИЙ ПРИ ГИГИЕНИЧЕСКОЙ ОЦЕНКЕ
ПРЕРЫВИСТЫХ ШУМОВ

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Анализ литературы, касающейся изучения влияния на организм прерывисто действующих шумов, показывает, что в одном случае исследователи наблюдали более выраженное влияние при прерывистом воздействии, в другом — прерывистое воздействие шума вызвало одинаковый или в меньшей степени биологический эффект по сравнению с непрерывным. Некоторые авторы (Roth и Weinberg) считают, что степень вредности прерывистых шумов наряду с учетом уровня и спектрального состава определяются произведением общего времени их действия на акустическую энергию, т. е., по мнению указанных исследователей, равно количеству воздействующей акустической энергии разным образом вредны независимо от того, как эта энергия распределяется во времени. Между тем, по данным Schröder и Rentsch, стабильный шум вызывает большие сдвиги со стороны некоторых физиологических функций, в частности слуховой, чем прерывистый (при прочих равных условиях), если время воздействия стабильного шума и суммарное время прерывистого одинаково.

В связи с изложенным перед нами была поставлена задача изучить особенности влияния некоторых видов прерывистых шумов по сравнению со стабильным при одинаковой суммарной длительности действия их. Исследования проводились в звукоизолированной камере с 20 практически здоровыми лицами мужского пола в возрасте 18—25 лет, всего 4 серии исследований по 20 наблюдений в каждой. Во всех сериях исследуемые лица подвергались воздействию шума в течение 1 часа.

В I серии изучали влияние низкочастотного (максимум звуковой энергии в октавной полосе со среднегеометрической частотой 250 гц) стабильного шума уровнем 110 дб. Во II серии выявляли влияние шума с таким же уровнем и спектром, но действовавшего прерывисто: шумовые воздействия продолжительностью $2\frac{1}{2}$ мин. чередовались с 5-минутными перерывами. Такой характер шума бывает при некоторых режимах работы вибрационной площадки по уплотнению бетона. Общее время опыта в этой серии исследований составляло 3 часа (суммарное время шума 1 час и время пауз 2 часа). В III серии исследовали влияние высокочастотного (максимум звуковой энергии в октавной полосе со среднегеометрической частотой 2000 гц) стабильного шума уровнем 105 дб. В IV серии оценивали влияние прерывистого шума, при котором полуминутные высокочастотные звучания уровнем 105 дб чередовались полуминутными паузами. Шум с такими параметрами отмечается при гидравлическом испытании труб. Время эксперимента в данной серии исследований 2 часа (длительность шума 1 час и время пауз 1 час).

У исследуемых лиц изучали функциональное состояние слухового анализатора, коры головного мозга и некоторые функции сердечно-сосудистой системы (тонус сосудов, частота пульса). Слуховую чувствительность определяли аудиометром на тонах 500, 1000, 2000, 4000 и 6000 гц путем исследования воздушной звукопроводимости.

Изучение функционального состояния коры головного мозга проводили с помощью прибора для исследования высшей нервной деятельности, принцип действия которого основан на предъявлении исследуемому раздражителей с различной волновой характеристикой и получение соответствующего ответа на них.

На передней панели прибора находятся два ряда кнопок (по 8 кнопок в каждом) различного цвета и экран, на котором в определенной последовательности и со скоростью от 22 до 110 в минуту появляются цветовые сигналы, которые загораются и гаснут автоматически. При появлении на экране того или иного цветового сигнала-раздражителя исследуемый должен нажать кнопку соответствующего цвета. При правильной и своевременной реакции (до появления следующего раздражителя) рядом с сигналом-раздражителем дается сигнал-подтверждение. Последовательность предъявления раздражителей определяется экспериментатором при помощи 4 программ (по 8 цветовых сигналов в каждой). Для каждого исследуемого устанавливали максимальный ритм подаваемых раздражителей, при котором его ответы содержат минимальное количество ошибок. Для большинства исследуемых 60 предъявлений в минуту являлись максимальным ритмом. Тонус сосудов определяли с помощью двухканального чернильно-пишущего плетизмографа. Плетизмограмму записывали с указательного пальца. При анализе плетизмограмм учитывали амплитуду объемных изменений и частоту пульсовых ударов.

Результаты аудиометрических исследований показали, что несмотря на одинаковое суммарное количество звуковой энергии, стабильные шумы вызывали более выраженное снижение слуховой чувствительности, чем прерывистые. Так, после воздействия низко-высокочастотных шумов с уровнями 110 и 105 дб снижение слуха на частоте 4000 гц отмечалось соответственно на 24 и 26 дб; после воздействия прерывистых — на 15 и 14 дб. Следует отметить, что после воздействия стабильных шумов отме-

чается понижение слуха не только на частоте 4000 гц, но и на смежных частотах — 3000 и 6000 гц (в среднем 20 и 17 дб). После действия прерывистых шумов понижение слуховой чувствительности на указанных смежных частотах было менее выраженным (10—13 дб). Тот факт, что временное снижение слуха отмечалось на тонах, смежных с тоном 4000 гц, свидетельствует о большей утомляемости слухового анализатора при воздействии стабильного шума по сравнению с прерывистым.

Результаты исследования высшей нервной деятельности показали, что при воздействии как стабильных, так и прерывистых шумов у исследуемых происходило снижение подвижности нервных процессов, достоверно увеличивалось количество ошибочных ответов на цветовые раздражители. Однако более выраженная тенденция к ухудшению указанных показателей наблюдалась после воздействия прерывистых шумов. Так, после воздействия высокочастотного прерывистого шума уровнем 105 дб исследуемые делали $2,7 \pm 0,26$ ошибки при исходном количестве $1,9 \pm 0,18$ ошибки. Стабильный шум вызывал увеличение ошибочных ответов до $2,5 \pm 0,27$ ошибки по сравнению с исходным значением $2,0 \pm 0,17$ ошибки.

После воздействия низкочастотного стабильного и прерывистого шумов уровнем 110 дб количество ошибочных ответов возросло в одинаковой степени: при стабильном — с $3,8 \pm 0,21$ до $5,4 \pm 0,43$ и при прерывистом — с $3,0 \pm 0,27$ до $4,6 \pm 0,29$.

Довольно заметная разница в действии стабильных и прерывистых шумов (особенно с высокочастотными составляющими) наблюдалась при исследовании тонуса сосудов (см. рисунок). На рисунке видно, что на 1-й минуте действия стабильного и прерывистого шумов амплитуда плетизмограмм составляла соответственно 65 и 62% исходного уровня. На 50-й минуте экспозиции стабильного шума, что составляет такое же время общего суммарного действия прерывистого шума, разница в изменении амплитуды плетизмограмм была более значительна: при стабильном шуме ее величина составляла 72%, при прерывистом — 52%. Наблюдаемое различие в амплитудах статистически достоверно. После одночасового воздействия стабильных и прерывистых шумов наблюдалась тенденция к урежению частоты пульса в среднем на 2—3 в минуту. Выяснить зависимость между характером воздействующих шумов и степенью урежения пульса не представлялось возможным.

Таким образом, проведенные исследования дают основания считать, что прерывистое действие шума вызывает несколько иные реакции по сравнению со стабильным, несмотря на одинаковое количество звуковой энергии, воспринимаемой органом слуха. Так, для слухового анализатора распределение звуковой энергии во времени при прерывистом шумовом воздействии сказывается положительно. По-видимому, относительно длительные межшумовые паузы способствуют восстановлению слуховой чувствительности и улучшению процессов адаптации. Тот факт, что после воздействия прерывистых шумов функциональное состояние коры головного мозга изменялось в большей степени, вероятно, объясняется тем, что прерывистые действующие шумы субъективно воспринимаются как более неприятные, а отсюда могут оказывать большее воздействие на психическую деятель-



Изменение амплитуды пальцевой плетизмографии.
По оси ординат — амплитуда плетизмограмм (%); по оси абсцисс — время действия шумов;
1 — стабильный шум; 2 — прерывистый шум.

(I)

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ность человека, которая в свою очередь отражает состояние высшей нервной деятельности.

Более выраженную сосудистую реакцию на воздействие прерывистого шума, по-видимому, можно объяснить трудностью выработки адаптации сосудистой системы к отдельным прерывистым звучаниям.

Принимая во внимание результаты настоящих исследований, а также исследования, проведенные нами и другими сотрудниками нашей лаборатории в последнее время (А. А. Меньшов и соавт.), можно полагать, что при гигиенической характеристике прерывисто действующих шумов в каждом конкретном случае следует учитывать главным образом временные комбинации комплекса «шум — пауза», а не суммарное время воздействия шума.

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SECTION 16

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> T. Matoba (Dept. of Medicine, Kurume University School of Medicine, Kurume 830), H. Kusumoto, H. Omura, T. Kotorii, H. Kuwahara, M. Takamatsu		<u>Institution and address where research was performed</u> Laboratory of Clinical Physiology Yufuin Kosei Nenkin Hospital	
<u>Investigator's Phone No.</u>		<u>Sponsoring Organization</u> same as above	
<u>Citation</u> Matoba, T. et al. Digital plethysmographic responses to auditory stimuli with vibration disease. Tohoku J. Exp. Med 115(4):385-392, 1975			
<u># of Ref.'s</u> 11		<u># of Fig.'s</u> 9	<u>Language</u> English
<u>Type & duration of experiment</u> type: laboratory--in sound-proof room		<u>Purpose for study</u> to study effects of noise on sympathetic nervous system in workers with vibration disease	
<u>Description of test groups (subjects, #, age, etc.)</u> control group: 15 healthy men aged 43.8 ± 3.1 years; average blood pressures 123.9 ± 12.5 over 74.9 ± 8.8 mm. mercury. test group: 82 male patients with vibration disease who worked as chain-saw operators; average age 48.6 ± 7.3 yrs.; average blood pressure 129.3/77.5			
<u>Control of other stressors</u> the chain-saw workers were exposed to cold temp., noise and vibration stressors in their work		<u>Statistical Methods</u> test for significance used	
<u>Noise Stimulus</u> source: chain-saw noise recordings introduced through headphones spectral characteristics: not given noise level: 98-102 dB length of exposure: 10 sec. # of trials: not specified		<u>CVS Response Measured</u> digital plethysmogram (finger pulse amplitude)	
		<u>Nonauditory effects</u> healthy subjects--immediate decrease in CVS; digital plethysmographic amplitude due to noise; recovery to normal amplitude in 30 sec. men w/vibration disease: slower response and recovery times Other: not studied	
<u>Author's conclusions</u> Vibration disease is associated with impaired autonomic nervous system responses. Digital plethysmographic changes due to auditory stimuli can serve as indicators of autonomic activity, as a diagnostic tool.			
<u>Evaluation & comments</u> The finger pulse amplitude data for the healthy controls illustrates the effects of noise on blood vessels (vasoconstriction) very clearly--graphs are included.			

Matoba, T. et al. Digital plethysmographic responses to auditory stimuli with vibration disease. *Tohoku J. Exp. Med.* 115(4):385-392, 1975 (in English).

Finger pulse amplitude responses were compared in patients with vibration disease and in healthy subjects. The healthy subjects were 15 males from the Japanese Self-Defense Force aged 43.8 plus or minus 3.1 years and with average blood pressures of 123.9 plus or minus 12.5 over 74.9 plus or minus 8.8 mm mercury. The group with vibration disease consisted of 82 male chain-saw operators aged 48.6 plus or minus 7.3 years and with average blood pressures of 129.3 plus or minus 12.5 over 77.5 plus or minus 10.5 mm. mercury. Healthy subjects were selected using the Cornell Medical Index. Digital plethysmograms were obtained in response to recorded chain-saw noise introduced through headphones in a sound-proof room. The noise dose was from 98 to 102 dB for 10 seconds. Post-stimulus recovery was monitored by digital plethysmogram for up to 60 seconds from the beginning of the noise. The noise produced an immediate decrease in amplitude of the plethysmograms in the healthy subjects. The amplitude usually returned to normal within 30 seconds in these subjects. The patients with vibration disease had both poor responses to the noise and slower recovery times--they were still abnormal at 60 seconds. Individual differences in the plethysmograms were considerable among all the subjects. The response of the small arteries in the fingers to noise is under control of the autonomic nervous system. When a drug (tolazoline hydrochloride) that blocks autonomic nervous system receptors was given, the finger pulse responses to noise were poor. Digital plethysmograms in response to noise can be used as indicators of the functioning of the autonomic nervous system.

Digital Plethysmographic Responses to Auditory Stimuli in Patients with Vibration Disease¹

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MATOBA, T., KUSUMOTO, H., OMURA, H., KOTORII, T., KUWAHARA, H. and TAKAMATSU, M. *Digital Plethysmographic Responses to Auditory Stimuli in Patients with Vibration Disease.* Tohoku J. exp. Med., 1975, 115 (4), 385-392 — Digital plethysmographic responses to auditory stimuli in 15 healthy men and 82 patients with vibration disease were analyzed in order to clarify the functional conditions of autonomic nervous system in this disease. The auditory stimuli given to healthy men caused a rapid decrease in the amplitude of the plethysmograms. After cessation of the auditory stimuli the decreased amplitude recovered to the control value within 30 sec. In the patients with vibration disease, however, the recovery of the decreased amplitude was delayed. The plethysmographic changes in the patients with vibration disease were divided into 4 types: normal (N), intermediate (I), delayed (D) and poor response (P) types. Each type of I, D and P was altered to type N by treatments consisting of therapeutic exercises, hot spring cures and so on. All healthy men showed type N. There were no significant differences between the time courses of the recovery of the plethysmographic changes and the amplitudes of the plethysmograms before the auditory stimuli. The results obtained seem to indicate that the autonomic nervous system in the patient with vibration disease is in disorder, and that the digital plethysmography with auditory stimuli is instrumental to detect the functional changes in the autonomic nervous system. — plethysmogram; auditory stimuli; vibration disease; activity of autonomic nervous system

A digital plethysmography has been reported to be able to detect the condition of the sympathetic activity in man (Ackner 1956). Several investigators have described that the auditory stimuli given to a human body stimulate the hypothalamus and the limbic lobe of cortex in the brain (Kluge and Friedel 1933; Lehman 1957; Sakamoto 1957). The higher centers of autonomic nervous system are situated in the hypothalamus and the limbic lobe of the cerebral cortex (Green 1972).

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Vibration disease induced by the mechanical vibration has been reported to impair not only the functions of the peripheral nervous system and circulatory system and mobility in the upper extremities, but also the function of the central nervous system (Matoba et al. 1974). The main causes of this disease are presumably noises and vibrations generated by a vibrating tool and coldness in the environment. The noises, vibrations and coldness would be regarded as the so-called stressors against a human body. Therefore, it is of interest to know the changes in the function of the autonomic nervous system in the patients with vibration disease.

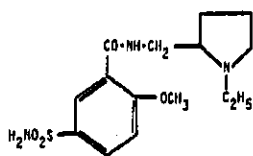
The purpose of the present paper is to clarify the function of the autonomic nervous system in patients with vibration disease as expressed by a digital plethysmography in response to the auditory stimuli.

SUBJECTS AND METHODS

Fifteen healthy men with averaged age of 43.8 ± 3.1 were selected from 37 of the members of Japanese Self-Defense Force after the examination of blood pressure in order to exclude those with hypertension and after the test of Cornell Medical Index for the purpose of screening of the body condition. Their blood pressures were 123.9 ± 12.5 mmHg in systole and 74.0 ± 8.8 mmHg in diastole. Eighty two male patients with vibration disease had been engaged in forestry as chain-saw operators. Their averaged age was 48.0 ± 7.3 . The mean blood pressures were 120.3 ± 12.5 mmHg in systole and 77.5 ± 10.5 mmHg in diastole. The severity of the disease was the third degree according to Andreeva-Gulanina's criteria (Morawa 1973).

The digital plethysmograms and their responses to auditory stimuli were recorded by means of a plethysmograph (Takachiho Instrument Co., PG-102) in a sound-proof room. Noises given as auditory stimuli were those recorded from a chain-saw and given by a headphones for 10 sec. The intensity of noises was 98 to 102dB.

The drugs used were tolazoline hydrochloride (Yamanouchi Pharm. Co.), one of the imidazoline derivatives with alpha-adrenergic blocking properties (Carrier 1972), and sulphiride (Fujiwara Pharm. Co.) which has an inhibitory action against hypothalamus (Fukuda and Takaori 1980). The chemical structure of sulphiride is shown below.



N-(1-ethyl-2-pyrrolidinyl)methyl-
2-methoxy-5-sulfamoylbenzamide

All observations were performed at room temperature (23-24°C).

RESULTS

Digital plethysmographic responses to the auditory stimuli in healthy men and patients with vibration disease

Observations were made on the changes of digital plethysmograms induced

by auditory stimuli. A typical pattern of the digital plethysmographic response was shown in Fig. 1.

In healthy men the auditory stimuli caused a prompt and marked decrease in amplitude of the plethysmograms. After cessation of the auditory stimuli, the amplitude tended to recover toward the control level within 30 sec (Fig. 1-A). In the patients with vibration disease, on the contrary, there were poor responses to the auditory stimuli (Fig. 1-B). The recovery of the amplitudes was also incomplete, less than 80% of the control value even at 60 sec.

Fig. 2 shows the analysis of the digital plethysmograms taken from 15 healthy men and 82 patients with vibration disease. In the patients with vibration disease the recovery of the amplitude of the plethysmogram once reduced by the auditory stimuli was retarded as compared with that of the healthy subjects. The time course of the recovery varied considerably from patient to patient.

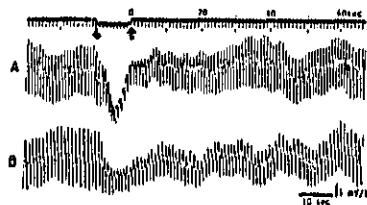


Fig. 1. A typical pattern of the digital plethysmogram responded to auditory stimuli in a healthy man and a patient with vibration disease. In a healthy man, there is a good response to the auditory stimuli and the excellent recovery of the reduced amplitudes (A). On the other hand, the response and the recovery are poor in a patient with vibration disease (B). Arrow marks indicate the auditory stimuli for 10 sec.

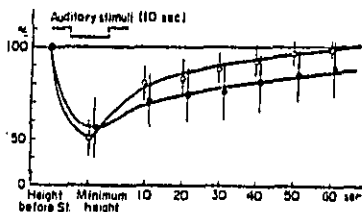


Fig. 2. A diagram of digital plethysmograms taken from 15 of the healthy men and 82 of the patients with vibration disease. \circ , healthy men ($n=15$); \bullet , vibration disease ($n=82$).

Classification of the digital plethysmographic responses to the auditory stimuli in the patients with vibration disease

The patterns of plethysmographic responses obtained in 82 patients with vibration disease could be divided into 4 types as shown in Fig. 3: normal (N), intermediate (I), delayed (D) and poor response (P) types. The number of the subjects was 24, 17, 26 and 15 in the types of N, I, D and P, respectively. These types were classified on the basis of the following criteria: In type N, the reduced amplitude elicited by the auditory stimuli recovered to the control value within 30 sec. In type I, the reduced amplitude was recovered more than 80% of the control value at 60 sec. In type D, it was less than 80% of the control value at 60 sec. In type P, the response to the auditory stimuli was poor; the reduction of amplitude

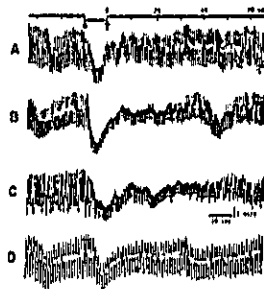


Fig. 3. Typical four types of the digital plethysmographic responses to auditory stimuli in patients with vibration disease. The types of normal (N), intermediate (I), delayed (D) and poor response (P) are shown in A, B, C and D, respectively. The reduced amplitude due to auditory stimuli recovers promptly to the control value in N type (A). In type I, the reduced amplitude is recovered more than 80% of the control value in 60 sec (B). In type D, it is less than 80% (C). In type P, the response to auditory stimuli is poor (D).

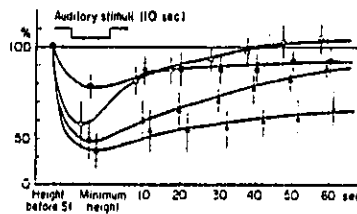


Fig. 4. A diagram of 4 types of the digital plethysmograms in the patients with vibration disease. \circ , N type ($n=24$); \bullet , P type ($n=15$); \blacktriangle , I type ($n=17$); \triangle , D type ($n=26$).

TABLE I. Changes of the digital plethysmographic types by the treatments

Type		Number of patients
Before	After	
N	N	6/6 (100%)
I	N	4/7 (57.3%)
	I	4/7 (42.8%)
D	N	7/13 (53.8%)
	I	1/13 (7.7%)
	D	5/13 (38.4%)
P	N	3/9 (33.4%)
	I	5/9 (55.6%)
	D	1/9 (11.0%)

was less than 30% of the control value. The time courses of responses of four types were illustrated in Fig. 4.

Fifteen healthy subjects revealed type N response in the plethysmograms.

Each type in the patients with vibration disease was shifted to the other types by the treatments, consisting of therapeutic exercises, hot spring cures and drugs of vasodilators. These data were summarized in Table I. Namely, type I altered to type N. Type D altered to type I or N. Type P changed to type I or N.

Mechanism of the digital plethysmographic responses to the auditory stimuli in the patient with vibration disease

The experiments were designed to find out the principal cause of the altered response to the auditory stimuli and the delayed recovery of the plethysmogram with a particular interest in knowing its central or peripheral origin.

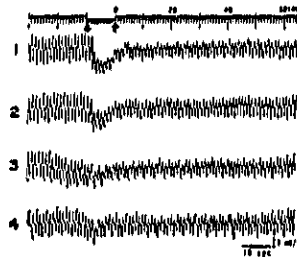


Fig. 5. A digital plethysmographic response to auditory stimuli in the patient with type D under the influence of alpha blockade. The auditory stimuli decrease the amplitude (1). The intramuscular injection of tolazoline hydrochloride, 20 mg, results in poor responses to auditory stimuli (2 to 4). The phenomena at 15, 40 and 50 min after the administration are shown in the figures of 2, 3 and 4, respectively.

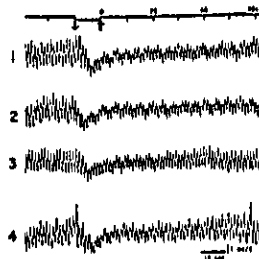


Fig. 6. A digital plethysmogram in the patient with type D under the influence of sulpiride. The good responses to the auditory stimuli are observed (1 to 4). The reduced amplitudes elicited by auditory stimuli can not recover to the control value within 60 sec (1). Sulpiride leads to become faster recovery in amplitudes after cessation of the auditory stimuli (2 to 4). The digital plethysmograms at 15, 40 and 50 min after the administration are shown in the figures of 2, 3 and 4, respectively.

A patient with type D was intramuscularly administered with 20 mg of tolazoline hydrochloride. After 50 min, the responses to the auditory stimuli became poor without changes in the time course of the recovery (Fig. 5). This indicates that tolazoline blocks alpha-adrenergic receptors of the blood vessels in the fingers, and that the responses to the auditory stimuli are manifested after the transmission of autonomic nerve impulses to the blood vessels in the fingers.

Intramuscular injection of 50 mg sulpiride, given to the same patient, caused no changes in plethysmographic responses to the auditory stimuli as compared with those of control even after 50 min as shown in Fig. 6. The recovery of the reduced amplitudes, however, tended to become faster. The time course of the recovery became similar to that of type N. This finding clearly suggests that the time course of the recovery after cessation of the auditory stimuli shows the continuity of vasoconstriction of small arteries. In other words, it represents the functional condition of the autonomic nervous center. The condition of the autonomic activity in type D seems to be hyperreactive.

Without tolazoline or sulpiride, there were no significant changes of patterns in the digital plethysmograms responded to the auditory stimuli within at least 60 min.

Relationship between the amplitude of the digital plethysmogram before the auditory stimuli and the digital plethysmographic response to the auditory stimuli

The mean amplitudes of the plethysmograms before the auditory stimuli in 16 healthy men and 82 patients with vibration disease were 4.5 ± 0.9 and 3.8 ± 1.3 mV/V, respectively, the difference being statistically significant ($p < 0.05$). The 82 patients were divided into three groups by the degree of amplitudes before auditory stimuli. Forty eight patients were included in group B, having the amplitudes

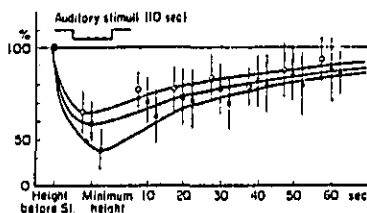


Fig. 7. Correlation between the amplitudes before the auditory stimuli and the patterns responded to the auditory stimuli. The subjects with mean \pm s.d. of the amplitude are included in group B. The patterns responded to the auditory stimuli are indicated to be almost the same in each group. o, A ($h \leq 2.4$ mV/V) ($n=16$); •, B ($2.4 < h < 5.1$ mV/V) ($n=48$); ▲, C ($h \geq 5.1$ mV/V) ($n=8$).

from 2.4 to 5.1 mV/V. Group A with amplitudes of less than 2.4 mV/V and group C with more than 5.1 mV/V had 16 and 8 patients, respectively. As shown in Fig. 7, there were no significant differences among those three groups. The mean amplitudes of the plethysmograms in types N, I, D and P were 4.1 ± 1.5 , 3.7 ± 1.0 , 3.9 ± 1.2 and 3.4 ± 1.0 mV/V, respectively. There were no significant differences among them.

DISCUSSION

Small arteries in the fingers are innervated with autonomic nerve fibers. The sympathetic vasoconstrictor nerves apparently exert their action on smooth muscle fibers with so-called alpha-receptor sites (Rushmer 1970). The degree of vasoconstrictions is directly proportional to the quantity of the nerve impulse of sympathetic nervous system (Yamagishi 1974).

Noises given as auditory stimuli, on the other hand, is known to produce the excitation of the limbic system in the brain (Kluge and Friedel 1933; Lehman 1957; Sakamoto 1957).

As demonstrated in the present study, the digital plethysmographic responses to the auditory stimuli and the time courses of the recovery of the response in patients with vibration disease are different from those in healthy subjects. The reduction of amplitudes induced by auditory stimuli manifests the transmission of autonomic nerve impulses to the blood vessels, and the time course of the recovery of the response shows the continuity of vasoconstriction of arteries. These digital plethysmographic patterns are divided into 4 types. Each type has been shifted from one type to another by the treatments. One of the treatments has been therapeutic exercises. A proper amount of physical exercises results in vagotony. Thus, these types would represent the level of the activity of autonomic nervous system. Namely, types I and D may be of hyperreactive, and type P of hyporeactive. In fact, a patient with vibration

disease complains of the disturbance of sound sleep, forgetfulness, the abnormal increase of palmar sweating, the disturbance of circulation and so on (Matoba et al. 1974).

It is concluded that the patients with vibration disease are associated with the disorder of autonomic nervous system. It would be also accepted that the digital plethysmography combined with auditory stimuli is one of the excellent indicators of the level of the activity of autonomic nervous system.

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SECTION 17

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> C. Ohkubo K. Miyazaki Y. Ozada	<u>Institution and address where research was performed</u> Department of Physiological Hygiene Institute of Public Health Tokyo, Japan
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Same
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Citation Ohkubo, C. et al. Response of finger pulse amplitude to intermittent noise. Bull. Inst. Publ. Health 25(1):1-8, 1976.

<u># of Ref.'s</u> 10	<u># of Fig.'s</u> 8	<u>Language</u> Jpn (Engl. translation)
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<u>Type & duration of experiment</u> laboratory - soundproof room 2 3/4 hours AM, 1 3/4 hours in PM per subject	<u>Purpose for study</u> to study the effect of intermittent noise on finger pulse amplitude and pulse rate.
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Description of test groups (subjects, age, etc.)
 6 healthy male students 19-24 years old
 6 healthy female students 19-23 years old
 Subjects tested during chair-rest

<u>Control of other stressors</u> laboratory test - constant temp. background noise - 25dBA	<u>Statistical Methods</u> factor analysis
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<u>Noise Stimulus</u> source: recorded white noise and train noise by Bruel & Kjaer type 1402 oscillator spectral characteristics: white noise (broad band)-rectangle; train noise- trapezoid noise level: 65-85 dBA length of exposure: intermittent - 7 sec. every 5 min. for 1 3/4 hours # of trials: 2 per subject	<u>CVS Response Measured</u> finger pulse amplitude (index finger and middle finger) pulse rate <u>Nonauditory effects</u> Pulse rate - slight increase due to noise CVS: <u>finger pulse amplitude</u> - decreased markedly due to noise; greater decrease observed due to the white noise; response slower in male subjects Other: annoyance - greater with white noise
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Author's conclusions Reduced fingertip pulse amplitude in response to noise is an accurate physiological index of noise effects and indicates noise-induced sympathetic nervous tension. The finger pulse amplitude response is non-specific.

Evaluation & comments

This study confirms some of Gerd Jansen's earlier work.

Ohkubo, C. et al. Response of finger pulse amplitude to intermittent noise. Bull. Inst. Publ. Health 25(1):1-8, 1976.

The effect of train noise and white noise on pulse rate and fingerpulse amplitude was studied in 6 healthy male and 6 healthy female students, 19-24 years old. Each subject was tested during chair-rest in a soundproof room with a background noise level of 25 dBA. The subjects were exposed to recorded intermittent train noise and white noise at varying levels of 65-85 dBA, arranged in random order. Each subject was exposed to noise for 7 seconds every 5 minutes for 1 3/4 hours from 10:30 - 11:15 a.m. and 2:00 - 3:45 p.m. in one day. The white noise had a rectangular pattern (broadband), whereas the train noise had a trapezoidal pattern, the rise time being 10 dB per 0.08 seconds. Pulse rates and fingerpulse amplitudes were measured during noise and quiet by a photoelectric plathysmograph. The pulse rates increased significantly due to noise; however, no significant variations were observed with the different noise types or levels. After a latency period of 1-2 seconds, fingerpulse amplitudes were reduced due to noise to an average of 60% of the amplitude during quiet. White noise produced a greater decrease in amplitude than train noise at the same level, possibly due to its broader band. The amplitude response increased with the noise level. The minimum amplitude response was reached later in the male than in the female subjects. The authors conclude that fingerpulse amplitude is a reliable index of the effects of noise, although the response is nonspecific.

ORIGINAL WORK

RESPONSE OF FINGER PULSE AMPLITUDE TO INTERMITTENT NOISE

BY

Chiyoji Ohkubo, Kuratoshi Miyazaki and Yasutaka Osada
(Department of Physiological Hygiene, National Institute of
Public Health)

PREFACE

The annoyance aspect of noise is affected by various factors such as quality, level, duration and rising & falling time of the noise. Therefore, studies are conducted to make primarily a psycho-physiological evaluation of the noise exposing combinations of these factors to subjects who were assigned to judge the "annoyance" of the noise. On the other hand, however, there is some room for questioning as to the validity of evaluating noise based upon the physiological index of the noise effect. It has been known for a long time that peripheral blood vessels are induced to constrict by the effect of noise. However, it was Lehmann and Jansen 1)-5) who started to conduct various experiments using fingertip pulse waves. They investigated primarily the effect of pure tone and band noise. Therefore, we have used white noise and train noise as actual noise to study the effect of noise on fingertip photoelectric pulse waves.

METHODS

Six healthy male students (19-24 years of age, average 21 years old) and six female students (19-23 years of age, average 20.8 years old) were chosen as test subjects. The breakdown of the students whose present residence was in a quiet, normal and noisy neighborhood was 5, 5 and 2 respectively. However, the environmental difference of the subject's residence did not influence the test results.

The tests were performed in a sound proofed room of this institute. The back ground noise of the room was controlled at approximately 25dB(A), and the room temperature was maintained at 25 ± 1.5°C. Due to the limited availability of the measuring instruments, tests were carried out at the rate of 2 subjects per day. The subjects entered the room at 9.30am, and the instruments were attached to them. From 10.30 to 12.15, they were exposed to the noise while they rested on a chair. After lunch and recess, they came back in the room at one o'clock and were exposed to the afternoon noise from 2.00 to 3.45pm.

The noise exposed was white noise and train noise. The former was transmitted by an Bruel & Kjaer type 1402 oscillator, and the latter was the noise from the Tokaido line super-express trains passing on the rails built upon raised ground. Both of these noises were recorded on magnetic tape. The recorded changes in the level of the noise exposure are indicated in Figure 1.

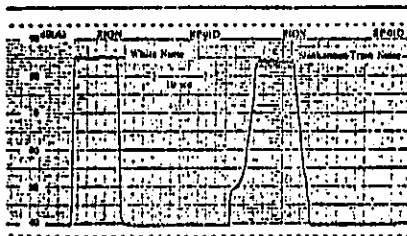


Fig. 1. Paper record of white noise and train noise used in the experiment.

White noise indicated an almost perfect rectangle without any ascending or descending time, while the train noise indicated an trapezoid with an ascending and descending rate of approximately 0.08sec/10dB. Both noises indicated a peak level for 7 seconds. In the experiment, these two different noises were exposed at the levels of 65-85dB(A) by increments of 5dB. Ten sets of noises were prepared by arranging the types and levels of the noises, and each set of noises was arranged to appear twice in random sampling, and recorded on magnetic tape. The random arranged order of the noises are indicated in Figure 2.

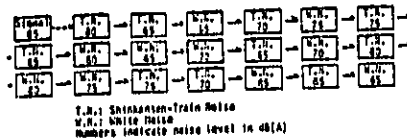


Fig. 2. Random arrangement of noise exposure.

Each noise was applied twice within one run of exposure. Noise was exposed every five minutes.

The noise was exposed once in every five minutes allowing an equal interval. The initial signal, 85dB(A), of the random samples in the figure was white noise, which was placed to adjust the level of the noise exposure and was not the test noise. The level of the exposure was determined *in the vicinity* of the ears of the subjects. The subjects were exposed to the noise for 7 seconds

every 5 minutes and allowed to read light literature inbetween the noise exposure so that they would not fall asleep. However, they were instructed to stop reading one minute before the starting of the noise and not to move their body but sit still. Further, they were instructed to remain seated and keep the same posture for approximately one minute after the noise terminated.

Photoelectric plethysmograph and impedance plethysmograph of finger-tip pulse waves were taken. The former was taken by attaching a reflecting type pick-up (MPP-2 model by Nihon Kodan Co.) to the back side of the index finger phalanx of left hand, and the latter was taken by attaching an electrode (AD3-2 model by Nihon Kodan Co.) to the both sides of the middle finger phalanx of the left hand. Both inputs were drawn on recording paper through the medium of a DC Amp (AD3-2 model by Nihon Kodan Co.). An example of the record of both plethysmographies are indicated in Figure 3. However, only photoelectric plethysmographic records were measured due to various problems relating to the interpretation of the impedance plethysmography.

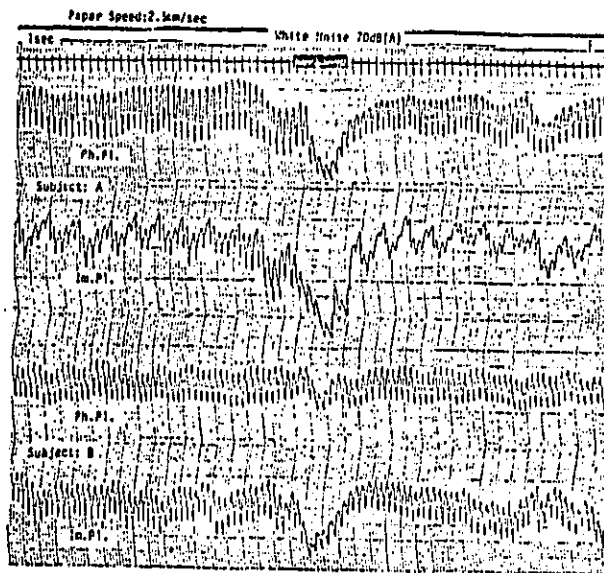


Fig. 3. A case of paper record of photoelectric and impedance plethysmographies. Two subjects were tested in a single experiment.

RESULTS

1. CHANGES IN PULSE RATE

Relative changes in pulse rate were measured from the photoelectric plethysmograph. First, from the paper recordings, the pulse rate of the subjects while sitting still and stabilized during the one minute before the noise exposure was counted, and this rate was designated as an initial value. Next, the pulse waves every 10 seconds including 7 seconds of noise exposure time was read a total of 4 times, and the value which changed most among the 4 was designated as the value after exposure. The relative value(%) of this value to the initial value was calculated. Each subject was exposed to the same noise (for example, 65dB(A) of white noise) a total of 4 times, ie, two times each in the morning and in the afternoon, and the values obtained at the above 4 readings was averaged and designated as a response value of the subject to that particular noise. Table 1 indicates the average value obtained by the above described method and the standard error of each 6 subjects according to the types and levels of the noises and the sex of the subjects. The value designated as control indicates the changes in the pulse rate during the period of time one minute before exposure to the noise when the subjects were instructed to remain in a resting position. This value is almost 100%, which indicates that when the subjects obediently followed the above instructions, the pulse rate hardly changed. However, when the subjects were exposed to the noise, the value increased almost all the time to above 100%. Although the rate of increase might have been small, it is obvious that the pulse rate increased by the noise. According to the results of the factor analysis, there was a significant difference between the pulse rate during the noise exposure and the control, there was, however, no significant difference detected among the pulse rates taken under different types and levels of noise.

2. CHANGES IN PULSE WAVE AMPLITUDE

As clearly seen in Figure 3, pulse waves of photoelectric plethysmograph reduced their amplitude as an effect of the noise, which indicates the occurrence of vaso-constriction. Now, designating the average value of the pulse wave amplitude during the stabilized state before the noise exposure as 100%, the relative value of the amplitude immediately before and after the exposure was calculated for each pulse beat and is shown in figure 4. This diagram is an example of the response of a certain subject to white noise of 80 and 85dB(A). Each beat is drawn in an equal interval, thus the horizontal axis indicating time is not equally spaced. As revealed in figures 3 and 4, the amplitude of the pulse waves after the starting of the noise exposure radically decreased after a 1-2 second latent period, and once it reached

Table 1. Changes in Pulse Rate by Noise Exposure
 (a) Percent of initial value \pm S.E.

Noise level	Shinkansen-train noise			White noise		
	Male n=6	Female n=6	Total n=12	Male n=6	Female n=6	Total n=12
65 dBA	103 \pm 3.1	103 \pm 1.2	103 \pm 1.6	104 \pm 1.7	100 \pm 1.1	102 \pm 1.2
70 dBA	105 \pm 3.0	101 \pm 1.4	103 \pm 1.7	102 \pm 1.5	99 \pm 1.5	100 \pm 1.1
75 dBA	106 \pm 2.2	100 \pm 0.8	103 \pm 1.4	102 \pm 2.7	104 \pm 1.0	103 \pm 1.4
80 dBA	104 \pm 1.7	102 \pm 0.9	103 \pm 1.0	103 \pm 2.0	101 \pm 1.0	102 \pm 1.1
85 dBA	105 \pm 2.5	102 \pm 0.8	103 \pm 1.4	104 \pm 1.4	101 \pm 1.0	102 \pm 0.9
[Control]	100 \pm 1.3	99 \pm 0.6	100 \pm 0.7]			

(b) Factor analysis

Factor	ss	df	v
Type of noise [N]	32.1	1	32.1
Level of noise [L]	200.5	5	40.1*
Sex [S]	186.8	1	186.8
Subject [D(S)]	787.2	10	78.7**
N \times S	6.2	1	6.2
N \times L	28.3	5	5.7
L \times S	35.6	5	7.1
N \times S \times L	98.2	5	19.6
Error	1789.1	110	16.3
Total	3164.0	143	

(c) Significant difference (**p<.01, *p<.05)

dBA	Total				Shinkansen-train noise					White noise							
	Control	65	70	75	80	dBA	Control	65	70	75	80	dBA	Control	65	70	75	80
85	**					85	*					85					
80	*					80	*					80					
75	**					75						75	*				
70						70						70					
65	**					65	*					65					

the minimum value it gradually recovered. However, amplitude greatly changed for each beat, so it cannot be necessarily concluded that the minimum value in this diagram indicates the state of amplitude during the minimum blood flow. Consequently, just as done with the pulse rate, the relative values of amplitude at every 10 seconds after the start of exposure were averaged, and the average values and standard errors of the 6 subjects pertaining to its minimum value are shown in Table 2 and in Figure 5(A). In Table 2, the results of the factor analysis are also indicated. The control in the table, same as in the case of the pulse rate, was the value obtained during the time when the subjects were instructed to rest. Even by this instruction alone, amplitude average lowered to 90% indicating a slight vasoconstriction. However, when exposed to actual noise, even a

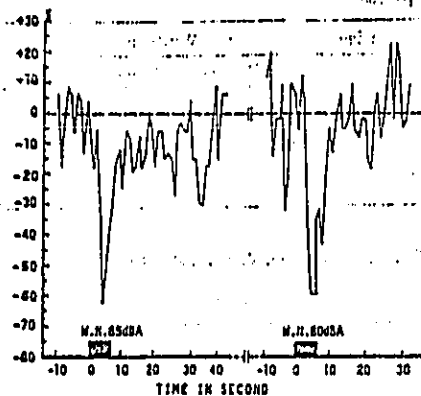


Fig. 4. Two cases of relative changes in finger pulse wave amplitude. Relative amplitude is expressed in percentage of its initial value. In this graph, interval between pulses is fixed and thus the time scale is not equally spaced.

train noise of 65dB(A), the average amplitude was reduced below 60%, and amplitude reduced radically corresponding to the elevation of the noise level. According to the results of the factor analysis, significant changes are seen in the amplitude of the pulse waves not only by the noise level but also by the type of the noise, and it revealed that pulse amplitude was more affected by the white noise than by the train noise. According to an examination of the differences in amplitudes by the noise levels, among the control; 65-80dB(A); 85dB(A) significant differences were observed.

Table 2. Changes in Amplitude of Pulse Waves
[a] Percent of initial value \pm S.E.

Noise level	Shinkansen-train noise			White noise		
	Male n=6	Female, n=6	Total n=12	Male n=6	Female n=6	Total n=12
65 dBA	58 \pm 3.8	57 \pm 4.7	57 \pm 2.9	45 \pm 3.4	48 \pm 4.0	47 \pm 2.5
70 dBA	51 \pm 6.0	53 \pm 4.0	52 \pm 4.0	43 \pm 4.1	45 \pm 3.5	44 \pm 2.8
75 dBA	48 \pm 3.9	51 \pm 4.0	50 \pm 2.7	47 \pm 3.2	48 \pm 4.3	47 \pm 2.6
80 dBA	53 \pm 5.8	53 \pm 5.4	53 \pm 3.8	40 \pm 2.6	44 \pm 4.6	42 \pm 2.6
85 dBA	45 \pm 5.8	43 \pm 5.7	44 \pm 3.8	38 \pm 3.6	43 \pm 5.2	40 \pm 3.1
(Control)	90 \pm 4.3	90 \pm 3.2	90 \pm 2.6]			

[b] Factor analysis

Factor	ss	df	v
Type of noise [N]	1201.8	1	1201.8**
Level of noise [L]	36804.4	5	7360.9**
Sex [S]	78.0	1	78.0
Subject [I(S)]	9021.5	10	902.2**
N x L	241.0	5	48.2
N x S	46.7	1	46.7
L x S	9.9	5	2.0
N x S x L	428.2	5	85.6
Error	8332.5	110	75.75
Total	50164.0	143	

[c] Significant difference (**p < .01, *p < .05)

Total					Shinkansen-Train noise					White noise							
dBA	Control	65	70	75	80	dBA	Control	65	70	75	80	dBA	Control	65	70	75	80
85	**	**	*	*	*	85	**	**	*	*	*	85	**	**	**	**	*
80	**					80	**					80	**				
75	**					75	**	*				75	**				
70	**					70	**					70	**				
65	**					65	**					65	**				

3. TIME TO THE MINIMUM AMPLITUDE

After initiating noise exposure, time required to reach the minimum amplitude was read from the recording paper, and the average values and the standard errors calculated according to the types and levels of the noise are indicated in Figure 5 (B). Also, its factor analytic results are listed in the Tm column of Table 3. There was no significant difference detected in time required to reach the minimum amplitude regardless of whether the train noise or the white noise was exposed.

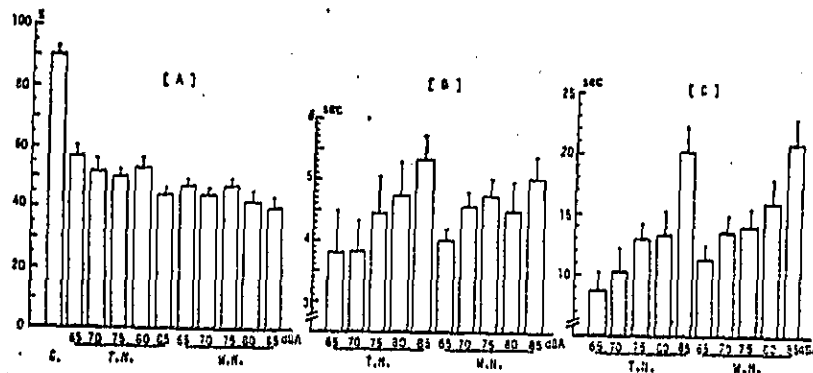


Fig. 5. Changes in photoelectric pulse wave amplitude by noise exposure.
 (A) Relative amplitude obtained at the time of maximum vaso-constriction.
 (B) Time from the onset of noise taken for the maximum vaso-constriction to appear.
 (C) Time from the onset of noise taken for the amplitude to recover to its initial level.
 Averages and standard errors for 12 subjects are illustrated.

However, it was greatly affected by the level of the noise, ie, the time required to reach the minimum amplitude extended corresponding to the increase of the noise level. As for the differences according to the levels, among three groups, 65; 70-75; 80-85dB(A), significant differences were indicated. Significant differences were also detected according to the sex

Table 3. Factor Analysis of the Changes in Time of Maximum Vaso-constriction [Tm] and of its Recovery [Tr]

(a) Factor analysis (**p<.01, *p.05)

Factor	df	Fo(Tm)	Fo(Tr)
Type of noise [N]	1	—	**
Level of noise [L]	4	**	**
Sex[S]	1	*	—
Subject [B(S)]	10	**	**
N x L	4	—	—
N x S	1	—	—
L x S	4	—	—
N x L x S	4	—	—
Error	90		

(b) Significant difference (**p<.01, *p<.05)

Tm		Total				Shinkansen noise				White noise					
dBA		65	70	75	80	dBA	65	70	75	80	dBA	65	70	75	80
85	**	**	**	**	**	85	**	*	*	*	85	**	*	*	*
80	**					80	**	*	*	*	80	**			
75	*					75	*				75				
70	*					70					70				

Tr		Total				Shinkansen noise				White noise					
dBA		65	70	75	80	dBA	65	70	75	80	dBA	65	70	75	80
85	**	**	**	**	**	85	**	**	**	**	85	**	**	**	**
80	**	*	*	*	*	80	**	*	*	*	80	**	*	*	*
75	**					75	**				75				
70						70					70				

of the subjects, and the time required to reach the minimum amplitude was larger in male than in female.

4. TIME TO RECOVERY

The time required for the reduced pulse wave amplitude to recover to its initial value (100%) was measured from the point of time of initiation of the noise exposure, and the average values and standard errors calculated according to the types and levels of noise are indicated in Figure 5(C). The results of the factor analysis are listed in the Tr column of Table 3. This time to recovery was longer when a subject was exposed to the white noise than to the train noise, and further, it extended with the increase of the level of the noise. Among the groups of 56-70;75-80;85dB(A) significant differences were observed. However, sex differences of the subjects did not play an important role for this matter.

COMMENTS

The following is a summary of the test results. When exposed to noise, the amplitude of fingertip photoelectric pulse waves critically decreased after one to two second latent period, and it recovered gradually after reaching the minimum value. This response was intensified in proportion to the increase of the levels of noise resulting in the rapid reduction of the amplitude and the extension of the time required for reaching the minimum value and for recovery. The amplitude responded more strongly to the white noise than the train noise although the level of the noises were identical, and the white noise reduced the amplitude and extended the recovery time in great deal. Also, in male subjects, the reduced amplitude took a longer time to recover to its initial level than in female subjects. Pulse rate increased by the effect of the noise, but there was no significant differences caused by the types and levels of the noise in particular. Viewing the above described results, although the changes in pulse rate may not be much, response of the pulse wave amplitude to the noise detected in the tests can be a considerably accurate physiological index of the effect of noise.

As mentioned in the "preface", it was Lehmann and Jansen who conducted many studies using fingertip pulse waves. They first studied them using ballistocardiographies and confirmed that the resistance to the peripheral blood flow was incremented by the effect of noise 1), and then, they continued their studies using a sort of impedance plethysmographies 2) resulting in the achievement of the following convictions: Fingertip pulse wave amplitude decreased by the noise, which was not clearly detectible in infants but manifested better in children of 8-11 years old and presented a normal adult level of decrease in youngsters of 19 years old 4) 5); Louder the noise the greater the response became 5); As for frequency, the response of the fingertip pulse waves did not change by the frequency when white noise was exposed but it became greater with the increase of the frequency when a 1/3 octave belt noise was exposed 5); Also, even with the same center frequency, the wider the belt of the noise expanded, i.e., 1/3 octave to one octave to broadband noise, the larger the response became 3). Around the same time as Lehmann and Jansen, Grandjean and his group 6), 7) proved peripheral vaso-constrictions due to noise from the changes in plethysmography and the skin surface temperatures. In comparatively recent years, Fuchs-Schmuck 8) investigated the reduction in the amplitude of fingertip volume pulse waves due to the broadband noise using the same method as Jansen used. Also, Conrad 9) studied the decrease of fingertip pulse wave amplitude based upon photoelectric plethysmographies.

According to the above described data made available up to now, it is quite certain that the noise induces peripheral

vaso-contractions and thus blood flow reduction-pulse wave amplitude reduction. This response was confirmed at the noise level of dB 5) according to Jansen and his group's data, and 60 or 70dB 8) according to Fuchs-Schmuck's data. Even in this experiment, as seen in Figure 5(A), 65dB(A) of noise greatly contributed to the response of the subjects compared to the control. Based upon Jansen's data, amplitude was hardly decreased by a 50dB level of noise 5). It is for a future study to find out the lower limit of the potential detection of the response by the plethysmographies applied in this experiment. It was found that the response induced by the white noise was greater than the response by the train noise when the level of both noise was identical, which, for one, may be attributable to what Jansen found, i.e. white noise has a broader belt than train noise and is annoying to the ears. This point was agreeable with the subjective judgement of the test subjects. Another reason for this is assumed to be the abruptness of the occurrence of the white noise, which was manifested in a rectangular shape in the paper record of the noise. It is a future problem to investigate the relationship between the response and the ascending time of the noise. Incidentally, sexual differences affected the time required to reach the minimum amplitude value, and the results of the test indicated that a much longer time was required in male subjects than in female ones. Nevertheless, it is not disclosed yet why it should be so.

Compared to the response manifested in the reduction of the pulse wave amplitude, an increment of the pulse rate was not so ideal an index. Although the pulse rate increased slightly by noise, differences in types and levels of noise did not affect the pulse rate.

Well, peripheral vaso-contractions and pulse rate increase by noise, the same as salivation reduction and stomach motor inhibition, are the results of a tensed sympathetic nervous system caused by noise which worked as a mental and psychological stimula. Lehmann stated 10) that response manifested in fingertip pulse wave amplitude reduction was a response specifically related to noise different from other non-specific tensions of the sympathetic nervous system, which, however, has not been substantiated. Considering that vaso-contractions occur after a considerably long latent period of 1-2 seconds, and that the constrictions to a small degree occurred even only by the instructions to remain still, it should be considered that this type of response is a non-specific response which involves mental tensions. However, since the response of fingertip amplitude was detected to vary according to the quality and level of the noise, although it may be a non-specific response, it can be regarded as an accurate index of noise effect. In the future, response to noise in relation to quality, ascending time and duration will be investigated.

SUMMARY

The effect of intermittent noise on the fingertip photoelectric pulse waves was studied. Six male and six female students were chosen and subjected to exposure to train noise and white noise which had a peak level duration of 7 seconds once every 5 minutes for 105 minutes twice a day, once in the morning and the other in the afternoon. Timing of the train noise indicated a trapezoid with approximately 0.08sec/10dB for both the ascending and descending noise while the timing of the white noise indicated a rectangle. Peak level was exposed to the subjects in 5 phases from 65-85dB(A) measured at the ears using increments of 5dB. Each noise was exposed in random order.

Pulse rate slightly increased by the effect of the noise but was not detected to be influenced by the differences in the types and levels of the noise.

The pulse wave amplitude taken by photoelectric plethysmographies decreased radically after 1 to 2 seconds of the latent period subsequent to the commencement of the noise exposure. The rate of decrease grew larger proportionately to the increase of the exposure level, and also the white noise affected the amplitude more than the train noise. The time required to reach the minimum amplitude became longer with the higher levels of the noise but the differences in the types of noise did not affect the time. However, it took longer in males than in females. The time required to recover to the initial level was also found to be influenced more by white noise than by the train noise. The response to white noise which was stronger than the train noise may be due to the broadness of the band and the abruptness of the occurrence of the noise, which coincided with the "annoyance" that the subjects complained about concerning the white noise.

The reduction of the fingertip pulse wave amplitude is responsible for the non-specific tension of the sympathetic nervous system. Based upon the results of the tests described above, this response to the noise can be considered a quite accurate physiological index of noise effect.

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原 著

指先脈波振幅に対する間欠的騒音の影響^{*1)}

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Response of Finger Pulse Amplitude to Intermittent Noise

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C. OHKUBO, K. MIYAZAKI, and Y. OSADA *Response of finger pulse amplitude to intermittent noise.* Bull. Inst. Publ. Health, 25 (1): 1-3, 1976.—Six male and six female students were subjected in this study and two of them were tested in a single experiment. They were sitting in a sound-proofed and air-conditioned room and were exposed to white noise (W.N.) or train noise (T.N.) every five minutes for 105 minutes. W.N. and T.N. were reproduced through tape-recorder and speaker at peak levels of 65-85 dB (A) at the seats of the subjects. Duration of peak level of noise was fixed for 7 seconds (Fig. 1). The exposure was so arranged that each noise appeared twice but in random order (Fig. 2). The experiment was performed twice a day, in the morning and afternoon, under the same schedule and condition.

Finger pulse waves were recorded by photoelectric inducer attached at the index finger phalanx of left hand and their amplitude and rate were measured (Fig. 3). Impedance plethysmograph was also taken from the middle finger of the same hand but was not served for measurement because of some uncertainties in physiological explanation of its amplitude changes.

Pulse rate counted from paper record of pulse waves slightly increased by noise but there was no significant difference of the increment according to types and levels of noise (Table 1).

Amplitude of pulse wave was remarkably decreased by noise with a 1 to 2 seconds latency and recovered gradually to its initial level. This response indicated peripheral vaso-constriction at finger induced by noise. For quantitative analysis of the response, changes in pulse wave amplitude were expressed in percentages of its initial value (Fig. 4). Minimum value of relative amplitude thus obtained became smaller according as the noise level increased. W.N. produced smaller values than T.N. did, even when the noise level was identical (Table 2 and Fig. 4). The time taken for the pulse wave amplitude to reach to its maximum constriction (Tm) and to return to its initial level (Tr) was measured from the onset of noise exposure. The higher the noise level, the more both of Tm and Tr were elongated (Table 3 and Fig. 4). W. N. elongated Tr more than T. N. did, while such difference was not observed with Tm. Sexual difference was noticed only with Tm; longer in males than in females, but the reason was not clear. Above findings indicated that the higher the level of noise, the larger the vaso-constrictor response. W. N. had a more potent effect than T. N. because of its broadband characteristics

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^{**1)} 専攻生

and of promptness of occurrence. Thus, the response of finger pulse wave amplitude was concluded to be sensitive enough to be used as a psychophysiological index of noise effect.

背景

騒音のやかましき noisiness は騒音の音質、レベル、持続時間、立上り立下り時間など種々の要因の影響をうける。そのため、これらの要因を組合せて被験者にやかましさを判定させ、専ら感覚心理学的に評価する研究がおこなわれている。しかし一方、生理学的な示標をもって評価できないかという要求もある。そこでこの研究では、指先の光電脈波を用いて騒音に対する反応の鋭敏度をしらべることとした。騒音によって末梢血管の収縮がおこることは古くから知られていたが、指先脈波を用いて多数の実験をおこなったのは、Lehmann や Jansen ら⁽¹⁾である。彼らはこの方法で主として純音や帯域雑音の影響をしらべた。そこで私達は、白色雑音と現実の音としての列車騒音とを用い、指先光電脈波に対する影響を調べ、この反応が生理学的示標としてどの程度有効であるかを検討した。

方法

被験者は健康な男子学生 6 名 (19~24 才, 平均 21 才) と女子学生 6 名 (19~23 才, 平均 20.8 才) である。自宅での環境が静かであるというもの、普通というもの、騒がしいというものはそれぞれ 5, 5, 2 名であったが、この差は実験結果に影響しなかった。

実験は本院の聴音室でおこなわれた。室内の暗騒音は約 25 dB(A)、室温は 25±1.5°C に調節された。器具の都合上、1 日に 2 名ずつ実験した。被験者は午前 9 時 30 分に入室し、器具を装着され、10 時 30 分

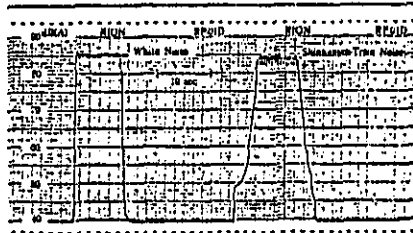


Fig. 1. Paper record of white noise and train noise used in the experiment.

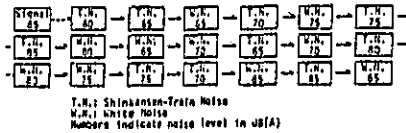


Fig. 2. Random arrangement of noise exposure.

Each noise was applied twice within one run of exposure. Noise was exposed every five minutes.

から 12 時 15 分まで椅坐安静位で騒音曝露をうけた。そのうち午後 1 時までは昼食と休憩とし、午後 1 時に再び入室し、2 時から 3 時 45 分まで、午後の騒音曝露をうけた。

曝露した騒音は白色雑音と列車音とである。前者は Brüel & Kjaer Type 1402 発振器によるもの、後者は東海道新幹線列車の盛土軌道通過時の騒音で、それぞれ磁気テープに録音したものである。そのレベル変化記録を Fig. 1 に示す。白色雑音はほとんど矩形で立上り、立下り時間 0 であるが、列車騒音の方は台形状の時間経過で立上り、立下りとも約 0.08 秒/10 dB である。両者ともピークレベルの持続時間は 7 秒である。実験では、この 2 種の音を 5 dB ステップで 65~85 dB(A) で曝露した。音の種類とレベルを組合せた 10 個の音が乱数表を用いて順序ランダムに 2 回ずつ配列され、磁気テープに録音されたものが用いられた。配列順序を Fig. 2 に示す。音の曝露は 5 分に 1 回ずつ、等間隔とした。図の最初の 85dB(A) の signal (信号音) は白色雑音で、曝露レベル調整用であり、試験音ではない。曝露レベルは被験者の耳の位置で決定した。被験者は 5 分に 1 回ずつ 7 秒の音をきくわけであるが、その間は眠気防止のため軽い読物がゆるされた。ただし、音のはじまる 1 分前に読書を止め、安静にして体を動かさぬよう命令され、さらに音が止んでから約 1 分はそのままの姿勢を保つよう命ぜられた。

測定したのは指先の光電プレチスモグラフィとインピーダンスプレチスモグラフィで、前者は左手示指第 1 節股側に反射型ピックアップ(日本光電 MPP-2 型)を固

定して採取、後者は本光電 IMP-26 型) プ(日本光電 AD3-た。両者の記録の 1ンピーダンスプレチ問題があるため、記のみにしておこな

1. 脈拍数の変化

Fig. 3 に示した光の相対的变化を計測し、前 1 分間のうち体動がなかつたものを採用し、これを前値と 10 秒ごと 4 回の脈動も変化の大きい値を相対する相対値 (%) を dB(A) の白色雑音) の 2 回ずつ、計 4 回脈動を平均して、その

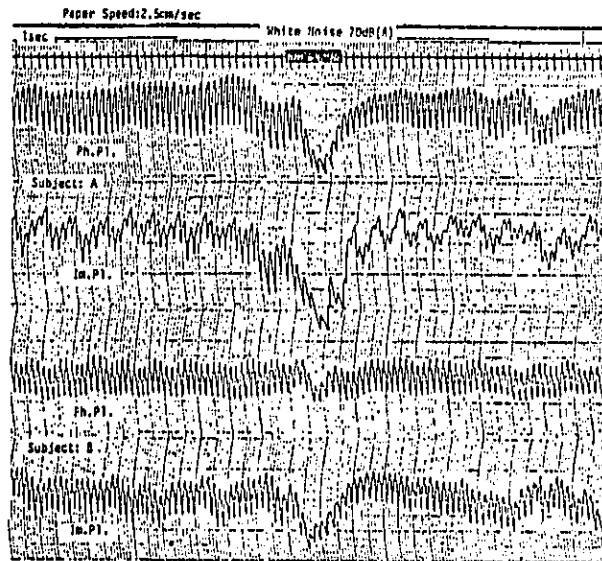


Fig. 3. A case of paper record of photoelectric and impedance plethysmographies. Two subjects were tested in a single experiment.

定して採取、後者は左手中指第1部の両側に電極(日本光電IMP-26型)を固定して採取、ともに直流アンプ(日本光電AD8-2型)を介して記録紙上に描記した。両者の記録の1例をFig. 3に示した。但し、インピーダンスプレサモグラフィはその解釈上、種々の問題があるため、記録の計測は光電プレサモグラフィのみについておこなった。

結 果

1. 脈拍数の変化

Fig. 3に示した光電プレサモグラフィから、脈拍数の相対的变化を計測した。まず記録紙から、騒音曝露前1分間のうち体動のない安定した状態での脈拍数をかぞえ、これを前値とした。ついで騒音7秒をふくむ10秒ごと4回の脈波の数をよそとり、そのうちの最も変化の大きい値を曝露後の値とし、その値の前値に対する相対値(%)を計算した。同じ音(たとえば65dB(A)の白色雑音)に各被験者とも午前、午後のおおの2回ずつ、計4回曝露されているので、この4回の値を平均して、その被験者のその音に対する反応値と

した。Table 1はかくして得られた騒音の種類別、レベル別、男女別6名ずつの平均値と標準誤差である。対照として示した値は、騒音曝露1分前に、安静な姿勢を保つように指示された時の脈拍数変化を示す。この値がほぼ100%であるのは、上の指示のみでは脈拍数にほとんど変化がないことを意味する。騒音をきいたときの値は、ほとんど100%以上になっており、増加率は小さいとはいえず、脈拍が増大することがわかる。要因分析の結果によると、対照との差が有意で、騒音の種類、レベルの間には有意の差はみられなかった。

2. 脈波振幅の変化

Fig. 3でも明らかのように、光電プレサモグラフィの脈波は、騒音によって振幅を減らし、血管収縮反応がおこったことを示す。そこで騒音曝露前の安定した状態の脈波振幅の平均値を100%とし、曝露直前および直後の振幅の相対値を、脈拍ごとに計算して図示するとFig. 4のようになる。この図は、ある被験者での白色雑音80および85dB(A)に対する反応を例示したものである。脈拍ごと等間隔に描いてあるので、横

Table 1. Changes in Pulse Rate by Noise Exposure
[a] Percent of initial value \pm S.E.

Noise level	Shinkansen-train noise			White noise		
	Male n=6	Female n=6	Total n=12	Male n=6	Female n=6	Total n=12
65 dBA	103 \pm 3.1	103 \pm 1.2	103 \pm 1.6	104 \pm 1.7	100 \pm 1.1	102 \pm 1.2
70 dBA	105 \pm 3.0	101 \pm 1.4	103 \pm 1.7	102 \pm 1.5	99 \pm 1.5	100 \pm 1.1
75 dBA	106 \pm 2.2	100 \pm 0.8	103 \pm 1.4	102 \pm 2.7	104 \pm 1.0	103 \pm 1.4
80 dBA	104 \pm 1.7	102 \pm 0.9	103 \pm 1.0	103 \pm 2.0	101 \pm 1.0	102 \pm 1.1
85 dBA	105 \pm 2.5	102 \pm 0.8	103 \pm 1.4	104 \pm 1.4	101 \pm 1.0	102 \pm 0.9
[Control]	100 \pm 1.3	99 \pm 0.6	100 \pm 0.7]			

[b] Factor analysis

Factor	ss	df	v
Type of noise [N]	32.1	1	32.1
Level of noise [L]	200.5	5	40.1*
Sex [S]	186.8	1	186.8
Subject [B(S)]	787.2	10	78.7**
N \times S	6.2	1	6.2
N \times L	28.3	5	5.7
L \times S	35.6	5	7.1
N \times S \times L	98.2	5	19.6
Error	1789.1	110	16.3
Total	3164.0	143	

[c] Significant difference (**p<.01, *p<.05)

Total					Shinkansen-train noise					White noise							
dBA	Control	65	70	75	80	dBA	Control	65	70	75	80	dBA	Control	65	70	75	80
85	**					85	*					85					
80	*					80	*					80					
75	**					75						75	*				
70						70						70					
65	**					65	*					65					

軸の時間は等間隔になっていない。Figs. 3, 4 からわかるように、脈波振幅は騒音開始後、1~2 秒の潜伏期をおいて急に減少し、最小値に達してから徐々に回復に向う。ただし、振幅は1 拍ごとに大きく変化するので、この図の最小値が必ずしも血液最少時の状態を示すとは限らない。そこで脈拍数のときと同様に、騒音曝露開始後、10 秒ごとの振幅相対値の平均値をとり、その最小値について6名の平均値と標準誤差を示したのが Table 2 と、Fig. 5 [A] である。Table 2 には要因分析の結果も掲げた。表中の対照は、脈拍数のときと同じく、安静の指示を受けた時の値で、この指示

のみでも振幅の平均は 90% となっており、僅かながら血管収縮がおこる。しかし騒音をきかせると、65 dB (A) の列車音でも 60% 以下になり、騒音レベル上昇とともに振幅減少が著しい。要因分析の結果によると、騒音レベルによる差のほか、騒音の種類による差も有意で、列車音よりも白色雑音による振幅減少の方が強い。レベル間の差の検定では、対照; 65~80 dB (A); 85 dB (A) の間が有意である。

3. 最小振幅到達時間

騒音開始後、最小振幅に到達するまでの時間を記録紙上で読みとり、騒音の種類別、レベル別の平均値と

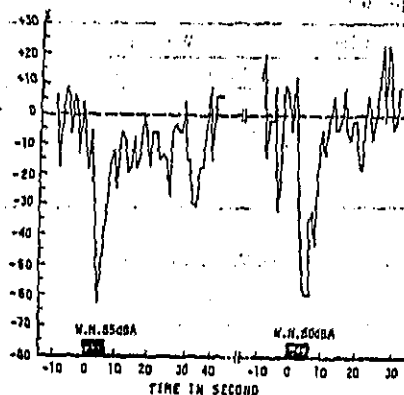


Fig. 4. Two cases of relative changes in finger pulse wave amplitude. Relative amplitude is expressed in percentage of its initial value. In this graph, interval between pulses is fixed and thus the time scale is not equally spaced.

標準誤差を計算したものを Fig. 5 の [B] に示す。またその要因分析の結果を Table 3 の T_m 欄に掲げた。白色雑音と列車音との間の差は有意ではないが、騒音のレベルによる差は有意であって、レベル上昇とともに最小振幅到達時間は延長する。レベル間の差をみると、65; 70・75; 80・85 dB(A) の3群間の差が有意である。男女の間にも有意差があり、女子よりも男子の方が大であった。

4. 振幅回復時間

騒音によって減少した脈波振幅が前値のレベル(100%)にまで回復する時間を、騒音開始の時点から測定し、騒音別、レベル別に平均値と標準誤差を示したのが Fig. 5 [C] である。要因分析の結果は Table 3 の T_r の欄に掲げた。この回復時間は、白色雑音の方が列車音より長く、かつ騒音のレベル上昇とともに長くなる。65・70; 75・80; 85 dB(A) の間に有意差がある。男女の間には有意差がなかった。

考 察

実験結果を要約すると次のようになる。騒音に曝露

Table 2. Changes in Amplitude of Pulse Waves

[a] Percent of initial value \pm S.E.

Noise level	Shinkansen-train noise			White noise		
	Male n=6	Female n=6	Total n=12	Male n=6	Female n=6	Total n=12
65 dBA	58 \pm 3.8	57 \pm 4.7	57 \pm 2.9	45 \pm 3.4	48 \pm 4.0	47 \pm 2.5
70 dBA	51 \pm 6.0	53 \pm 4.0	52 \pm 4.0	43 \pm 4.1	45 \pm 3.5	44 \pm 2.6
75 dBA	48 \pm 3.9	51 \pm 4.0	50 \pm 2.7	47 \pm 3.2	48 \pm 4.3	47 \pm 2.8
80 dBA	53 \pm 5.8	53 \pm 5.4	53 \pm 3.8	40 \pm 2.6	44 \pm 4.6	42 \pm 2.6
85 dBA	45 \pm 5.5	43 \pm 5.7	44 \pm 3.8	38 \pm 3.6	43 \pm 5.2	40 \pm 3.1
[Control]	90 \pm 4.3	90 \pm 3.2	90 \pm 2.6]			

[b] Factor analysis

Factor	ss	df	F
Type of noise [N]	1201.8	1	1201.8**
Level of noise [L]	36804.4	5	7360.9**
Sex [S]	78.0	1	78.0
Subject [B(S)]	9021.5	10	902.2**
N x L	241.0	5	48.2
N x S	46.7	1	46.7
L x S	9.9	5	2.0
N x S x L	423.2	5	85.8
Error	8332.6	110	75.75
Total	56164.0	143	

[c] Significant difference (**p < .01, *p < .05)

Total					Shinkansen-Train noise					White noise							
dBA	Control	65	70	75	80	dBA	Control	65	70	75	80	dBA	Control	65	70	75	80
85	**	**	*	*	*	85	**	**	*	*	*	85	**	**	**	**	*
80	**					80	**					80	**				
75	**					75	**	*				75	**				
70	**					70	**					70	**				
65	**					65	**					65	**				

Table 3. Factor Analysis of the Changes in Time of Maximum Vaso-constriction [Tm] and of its Recovery [Tr]

[a] Factor analysis (**p < .01, *p < .05)

Factor	df	Fo(Tm)	Fo(Tr)
Type of noise [N]	1	—	**
Level of noise [L]	4	**	**
Sex[S]	1	*	—
Subject [B(S)]	10	**	**
N × L	4	—	—
N × S	1	—	—
L × S	4	—	—
N × L × S	4	—	—
Error	90		

[b] Significant difference (**p < .01, *p < .05)

Tm					Shinkansen noise					White noise				
dBA	65	70	75	80	dBA	65	70	75	80	dBA	65	70	75	80
85	**	**	**		85	**	*			85	**		*	
80	**				80	**	*			80	**			
75	*				75	*				75				
70	*				70					70				

Tr					Shinkansen noise					White noise				
dBA	65	70	75	80	dBA	65	70	75	80	dBA	65	70	75	80
85	**	**	**	**	85	**	**	**	**	85	**	**	**	**
80	**	*			80	**	*			80	**			
75	**				75	**				75				
70					70					70				

されると指先の光電脈波の振幅は 1~2 秒の潜時を経て急激に減少し、最小値に達したあと徐々に回復する。騒音のレベルが高いほど、この反応が強く、振幅減少が著しく、最小値到達時間と回復時間は長くなる。列車騒音より白色雑音の方が同じレベルであって

も反応が強く、振幅減少と回復時間延長が著しい。また最小値到達時間は女子より男子の方が良い。脈拍数は騒音によって増加するが、騒音の種類やレベルによる差は有意でなかった。以上の結果からみると、脈拍数の変化はとも角、脈波振幅減少反応はかなり鋭敏な

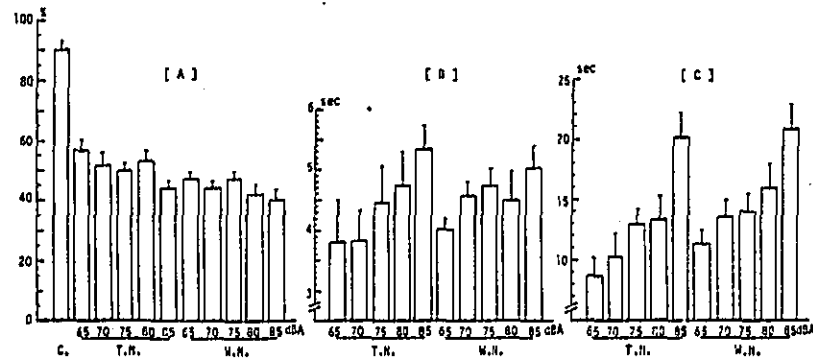


Fig. 5. Changes in photoelectric pulse wave amplitude by noise exposure.
 (A) Relative amplitude obtained at the time of maximum vaso-constriction.
 (B) Time from the onset of noise taken for the maximum vaso-constriction to appear.
 (C) Time from the onset of noise taken for the amplitude to recover to its initial level.
 Averages and standard errors for 12 subjects are illustrated.

生理的示標であるといえる。

「まえがき」にのべたように、指先の脈波を用いて多くの研究をおこなったのは Lehmann や Jansen であった。彼らは最初、バリストカルディオグラフを用いて研究し、騒音によって末梢血流抵抗が高まることを確めたが¹⁾、ついで1種のインピーダンスプレサモグラフを用いて研究し²⁾、指先の脈波の振幅が騒音によって減少すること、この反応は幼児では著明でなく、8-11才でよく反応するようになり、19才ごろ成人なみの反応に達すること³⁾、音が大きいほど反応が大であること⁴⁾、周波数については、純音では周波数による反応の差を見出し得ないが、1/3 オクターブ帯域音では周波数の高い程反応が大きくなり、また同じ中心周波数を持っていても、純音、1/3 オクターブ、オクターブ、広帯域音と帯域がひろがるにつれて反応が大きいこと⁵⁾を見出した。同じ頃、Grandjean ら⁶⁾は手のプレサモグラフと皮膚表面温度の変化とから、騒音による末梢血管の収縮を証明した。比較的最近になって Fuchs-Schmuck ら⁷⁾は Jansen と同じ方法で広帯域騒音による指先容積脈波の振幅減少をしらべ、また Conrad⁸⁾は光電プレサモグラフによって指先脈波振幅の減少をしらべている。

以上のようなこれまでのデータによって、騒音が末

梢血管の収縮をおこし、血流減少—脈波振幅減少をおこすことは間違いない。この反応は、Jansen らのデータでは 60 dB¹⁾、Fuchs-Schmuck らのデータでは 60 ないし 70 dB⁷⁾ から証明されているが、本実験でも Fig. 5 [A] にみるように、65 dB (A) でも対照と大きな差が出ており、かなり鋭敏な反応といえよう。Jansen のデータでは 60 dB ではほとんど振幅減少がおこっていないが¹⁾、本実験に用いた光電プレサモグラフでどこまで反応が検出できるか、今後の問題である。本実験では、列車音よりも白色雑音の方が、同じレベルの音でも反応が大きい結果を得たが、Jansen らの知見からみると、列車音よりも白色雑音の方が広い帯域を持ち、より耳ざわりであることに1つの原因があろう。この点は被験者の主観的判断とも一致した。また白色雑音の時間経過がほぼ矩形で、列車音より断片的であることも原因の1つになっていると思われるが、立上り時間との関係も今後の問題である。なお、振幅最小値に到達するまでの時間に性差があり、女子よりも男子に有意に長いという結果を得たが、この理由は不明である。

脈波振幅減少反応に比べると、脈拍増加反応は良い示標とはいえない。騒音で僅かながら増加するとはいえ、騒音の種類やレベルの差を検出できなかった。

さて、騒音による末梢血管の収縮と脈拍増加は、唾液分泌減少、胃運動の抑制などと同様に、騒音が精神的心理的刺激となっておこる交感神経の緊張の結果である。Lehmann は、指先脈波振幅減少反応を、他の非特異的な交感神経緊張反応とは異なる騒音特異の反応であると述べているが¹⁰⁾、その証拠はない。1~2秒という、かなり長い潜時をおいて血管収縮がおこること、姿勢を保つよう指示されただけでも多少の収縮がおこること等からみて、精神的緊張を介する非特異反応と考えるべきであろう。ただ、音質やレベルの差を抽出しうる点で、非特異反応とはいえず、鋭敏な示標であると言えよう。今後、音質、立上り、持続時間などとの関係を追及したい。

要 約

指先の光電脈波に及ぼす間欠騒音の影響を調べた。男子および女子学生、おのおの6名ずつを被験者とし、午前と午後の2回、105分間のあいだに5分に1回ずつ、ピークレベル持続時間が7秒の列車音と白色雑音をきかせた。列車音の時間経過は立上り、立下りが約0.08/秒10dBの台形状、白色雑音のそれは矩形状である。ピークレベルは被験者の耳元の位置で、5dBステップで65から85dB(A)の5段階とした。音の曝露順序はランダムとした。

騒音によって脈拍数は僅かながら増加するが、音のレベルの差は検出されなかった。

光電プレナムモググラフの脈波振幅は、騒音開始後1~2秒の潜時をおいて急速に減少する。減少度は曝露レベルが高い程大きく、列車音よりも白色雑音の方が大きい。最小振幅に到達するまでの時間は、レベルが高いほど長いが騒音の種類による差は見られなかった。女子より男子の方が長かった。振幅が元の値にまで回復するに要する時間も、レベル上昇とともに延長し、かつ列車音より白色雑音の方が長かった。白色雑音の方が列車騒音より反応が強いのは、その広域性の音質と、衝動性によるおもわれ、被験者の感じに

「やかましき」とも一致した。

指先の光電脈波の振幅減少反応は、非特異的な交感神経緊張によるものであるが、以上の結果から、かなり鋭敏な生理的示標であるといえる。

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SECTION 18

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Daryoush Parvizpoor M.D., D.I.H.		<u>Institution and address where research was performed</u> Dept. of Occupational Health University of Tehran, School of Public Health Tehran, Iran	
<u>Investigator's Phone No.</u>		<u>Sponsoring Organization</u> University of Tehran, Iranian government	
<u>Citation</u> Parvizpoor, D. Noise exposure and prevalence of high blood pressure among weavers in Iran. <u>Journal of Occupational Medicine</u> 18(11);730-731, Nov., 1976.			
<u># of Ref.'s</u> 10	<u># of Fig.'s</u> 3	<u>Language</u> English	
<u>Type & duration of experiment</u> Epidemiological survey of occupational exposure to noise in three textile mills in Iran duration of study - not given		<u>Purpose for study</u> To see if a greater percentage of people occupationally exposed to high noise levels were hypertensive compared to similar controls of same age.	
<u>Description of test groups (subjects, #, age, etc.)</u> Test group: 821 male weavers (aged 19-59, with 9-20 years in the mills) in three textile mills in Iran. Control group: 412 people (aged 19-59, unspecified length of employment) of similar socio-economic backgrounds who worked in light industries with no high occupational exposure to noise (selected at random from same geographic area).			
<u>Control of other stressors</u> Other stressors besides noise included cotton dust (7.8 mg/M ³), heat, high humidity.		<u>Statistical Methods</u> Methods not reported. Statistical results: difference between tests & controls significant at P < 0.01 level.	
<u>Noise Stimulus</u> source: machinery in textile mill spectral characteristics: not given noise level: 96 dBA average length of exposure: daily for 9 - 20+ yr. (duration of employment) # of trials: not applicable		<u>CVS Response Measured</u> Blood pressure (BP) measured on right arm while subject was seated after 5-10 min. of quiet rest before work - 1 measurement per subject. <u>Nonauditory effects:</u> CVS: 8.5% of the weavers were hypertensive 12.4% of the weavers were borderline Significantly higher than in controls who worked in quieter environments. Other: Not measured	
<u>Author's conclusions</u> A higher percentage (statistically significant) of males occupationally exposed to noise were hypertensive than in the control group. Hypertension also occurred at a younger age in the 96 dBA noise-exposed group. There appeared to be a direct correlation between length of exposure to 96 dBA noise and hypertension.			
<u>Evaluation & comments</u> 1. The blood pressures were taken only once per subject. 2. The large number of subjects and generally good design of study compensate for these points.			

Parvizpoor, D. Noise exposure and prevalence of high blood pressure among weavers in Iran. *Journal of Occupational Medicine* 18(11):730-731, Nov. 1976.

The occurrence of high blood pressure was studied in 821 male weavers in three textile mills in Iran. The average noise level in the mills was 96 dBA, with no major variations. The weavers in the study were screened with a medical exam and a questionnaire to exclude those with family histories of cardiovascular, renal, or thyroid diseases. The blood pressures of the weavers were compared to those of a randomly selected control group of 412 people from the same geographic area and from similar socioeconomic backgrounds. The subjects in the control group were employed in light industries, with no occupational exposure to high noise levels. Blood pressure readings were taken after 5-10 minutes rest under quiet conditions before work in the morning. Tables are included which show the percentage of subjects who are hypertensive (160/95 mm mercury or more), normotensive (below 140/90 mm mercury), and borderline by age and length of employment. There was a moderate increase in the prevalence of hypertension with age in the control group. The increase was greater in the weavers occupationally exposed to 96 dBA, and hypertension occurred in younger age groups. The hypertension rates for each age group were as follows:

<u>Age</u>	<u>Weavers</u>	<u>Controls</u>
20-29	1%	0%
30-39	7.3%	1.2%
40-49	12.1%	6.5%
50-59	27.1%	8.6%
Total	8.5%	2.4%

A higher percentage of hypertension was associated with longer occupational exposure to high noise levels-26.1% of the weavers with over 20 years experience were hypertensive. The study shows that occupational noise levels above 90 dBA can produce nonauditory physiological effects. Other stressors in textile mills that could contribute to hypertension include cotton dust, high temperature and humidity, and other adverse environmental conditions.

Noise Exposure and Prevalence of High Blood Pressure Among Weavers in Iran

Daryoush Parvizpoor, M.D., D.I.H.

821 weavers working in three textile mills were examined in this survey. It was found that 8.5% of the workers were hypertensive and 12.4% borderline. All these rates are significantly different from those of the control groups. Also there is a clear increase in the prevalence of hypertensive and borderline cases in the weavers in relation to the length of employment. These findings emphasize the need for, at least annually, periodic examination of the entire work population in order to detect early such adverse health effects and to consider suitable medical care.

Several studies, carried out on animals and man, have described nonauditory effects of noise.

In animals: Medoff and Bongiovanni¹ provided objective evidence on the influence of audiogenic stimulation on systolic blood pressure by exposing rats to a constant air blast for 5 to 10 minutes per day for several weeks. McCann et al² demonstrated, by adrenalectomy in noise induced hypertensive rats, that the adrenal cortex mediated the elevation of blood pressure occurring during prolonged auditory stimulation. Smirk³ reported that in rats exposed to a long period of intense noise stimulation the rise in blood pressure persisted for at least several months after the audiogenic stimulation ended.

In man, under experimental conditions: Arguelles et al⁴ studied 26 men exposed to pure sounds of 125, 2,000, 5,000 and 10,000 cps and intensities between 65 and 93 dB for 1-hour periods, and observed substantial elevations in the free plasma 17-OHCS and urinary 17-Ketogenic steroids. Fuchs and Vogel⁵ examined pulse volume, blood pressure and pulse frequency in 13 subjects exposed to noise of 55, 65, 75 and 85 dB for 10 minutes and observed significant vasoconstrictory effects, decreased systolic and increased diastolic blood pressure and tachycardia.

In field studies: Meinhart and Menker⁶ detected a significantly higher frequency of cardiovascular abnormalities, including hypertension, in noise injured persons in the District of Halle as compared to the cardiovascular morbidity of the general population of the same area. Kavoussi⁷ reported that among people occupationally exposed to noise of about 110 dB at a silo in Tehran, the prevalence of high blood pressure increased considerably with length of employment independently of the prevalence increase due to age.

These data indicate adverse health effects of noise occurring in other systems besides the auditory one. In order to investigate the effect of long-term occupational exposure to noise on blood pressure in a generally steady population, a study was carried out in industrial plants providing such conditions of exposure in the

Isfahan area of Iran. This report presents the findings of that investigation.

Materials and Methods

Three textile mills were randomly chosen from the 20 mills with more than 200 employees in the area. 821 weavers were selected for this study after excluding, by means of a questionnaire and medical examination, seven employees with a family history of hypertension, hyperthyroidism, renal and cardiovascular diseases.

As the number of female workers in the investigated plants was very low, the study was carried out on males only.

The average noise level at the three textile mills was 96 dBA without significant variations and the cotton dust concentration in the workrooms' air was about 7.8 mg/M³.

Blood pressure was measured on the right arm in the sitting position using the spring sphygmomanometer, after 5 to 10 minutes rest in a quiet room before beginning the day's work. The blood pressure findings were classified according to the recommendations of the WHO Expert Committee⁸ into three groups: (a) Normotensive: Systolic blood pressure below 140 mm Hg and diastolic blood pressure below 90 mm Hg. (b) Hypertensive: Systolic blood pressure 160 mm Hg or more or diastolic blood pressure 95 mm Hg or more. (c) Borderline: In between these figures.

A control group of 412 persons of similar socio-economic conditions as the weavers who were working in light industries without occupational exposure to intensive noise were randomly selected from the population of the same area.

Findings

Table 1 shows the distribution of the 412 persons in the control group by age and blood pressure level. Ninety three per cent were considered to be normotensive, 2.4% hypertensive and 4.6% borderline. There is a significant association between age and hypertension starting at the 40-49 age group.

Table 2 shows the distribution of the 821 weavers by age and blood pressure level: Only 79.1% of them were considered to be normotensive, while 8.5% were hypertensive and 12.4% borderline. All these rates are significantly different from those of the control group ($P < 0.01$). An association between age and hypertension may also be observed among the weavers but the significant increase in the hypertension rates starts at a lower age group (30-39) as compared to the control group (40-49).

Since the three textile mills studied have a very low turnover of workers, length of employment and therefore noise exposure are closely related to age. Table 3 shows the distribution of the 821 workers by length of employment and blood pressure level. A clear increase in prevalence of hypertension and borderline cases may be observed in relation to length of employment.

From the Department of Occupational Health, University of Tehran School of Public Health, Tehran.

Table 1. — Distribution of the Control Group by Age and Blood Pressure Level.

Age Groups	No. of Persons Examined	Normotensive		Borderline		Hypertensive	
		No.	%	No.	%	No.	%
<19	98	98	100	0	0	0	0
20-29	108	105	97.2	3	2.8	0	0
30-39	88	81	92.2	4	4.6	1	1.2
40-49	62	50	80.6	8	12.9	4	6.5
50-59	58	49	84.5	4	6.9	5	8.6
Total	412	383	93.0	19	4.6	10	2.4

Discussion

The findings for the control group do not differ significantly from those described by Nadim et al⁹ who reported that hypertension is becoming a frequent condition in rural areas of Iran.*

The findings among those men employed at the textile mills show that they have a significantly greater risk of developing hypertension and that this difference appears already at relatively young ages (30-39) and increases with length of employment.

In many industrial activities workers are exposed to much higher noise levels than the 96 dBA observed at the textile mills studied. The findings reported suggest that occupational exposure to noise that slightly exceeds the permissible levels of 85-90 dBA does produce nonauditory effects. In this respect it should be mentioned that Martin et al¹⁰ reported an increased risk of noise-induced hearing loss at noise levels between 85-90 dBA, from 4.0% to 22.5% above the 10% normally impaired due to presbycusis, for those subjects 50 to 65 years of age.

This last fact suggests that the people investigated in this survey should again be examined, both for assessing the development of the nonauditory effects as well as the state of their hearing capacity.

Since other pathogenic factors, besides noise, such as high temperature, high humidity, cotton dust, etc. may play a role in the etiology of high blood pressure among weavers, further studies in plants where the environmental conditions, rather than noise, are different are required to learn more about the long-term noise effects on the blood pressure of occupationally exposed people.

The data reported are in keeping with Kavoussi's findings regarding the harmful effects of noise on blood pressure in occupationally exposed people and call for provision of suitable protective devices against noise exposure.

Conclusions

1. People not exposed to intensive noise in their working environment

Table 2. — Distribution of 821 Weavers by Age and Blood Pressure Level.

Age Groups	No. of Workers Examined	Normotensive		Borderline		Hypertensive	
		No.	%	No.	%	No.	%
<19	148	148	100	0	0	0	0
20-29	202	198	97.0	4	2.0	2	1.0
30-39	191	181	79.1	26	13.6	14	7.3
40-49	149	97	65.1	34	22.8	18	12.1
50-59	133	59	44.3	38	28.6	36	27.1
Total	821	649	79.1	102	12.4	70	8.5

Table 3. — Distribution of 821 Weavers by Length of Employment and Blood Pressure Level.

Length of Employment (Years)	No. of Workers Examined	Normotensive		Borderline		Hypertensive	
		No.	%	No.	%	No.	%
<9	388	382	98.5	6	1.5	0	0
10-19	268	194	72.4	47	17.5	27	10.1
20+	165	73	44.2	49	29.7	43	26.1
Total	821	649	79.1	102	12.4	70	8.5

in the Isfahan area showed a moderate increase in hypertension prevalence with age: Up to 29 years, 0%, 30-39 years, 1.2%, 40-49 years, 6.5% and 50-59 years, 8.6%.

2. Textile mill workers of the same area, exposed to an average noise level of 96 dBA, showed in comparison with the control group, an increase in hypertension prevalence with age, characterized by its occurrence in younger age groups and at higher rates: Up to 19 years, 0%, 20-29 years, 1%, 30-39 years, 7.3%, 40-49 years, 12.1% and 50-59 years, 27.1%.

3. The fact that 26.1% of the weavers with more than 20 years of exposure are hypertensive, suggests a direct association between length of exposure to a noise level of about 96 dBA and hypertension. However, the fact that only 26.1% of them are hypertensive also suggests a particular role played by individual reactivity.

4. These findings emphasize the need for, at least annually, periodic examinations of the entire work population in order to detect early such adverse health effects and to consider suitable medical care.

5. A minimum preventive programme will consist of providing protective devices, educating the people regarding the need for them and supervision in their use throughout working hours. This could provide a reasonable basis for assessing, in prospective studies, the efficiency of such protective devices in preventing adverse health effects of industrial noise.

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SECTION 19

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> E.A. Peterson (Chief, Div. of Auditory Research) et al.	Institution and address where research was performed University of Miami School of Medicine Dept. of Otolaryngology Division of Auditory Research Miami, Florida	
<u>Investigator's Phone No.</u> (305) 547-6413	<u>Sponsoring Organization</u> University of Miami School of Medicine EPA/ONAC	
<u>Citation</u> Peterson, E.A. et al. Noise and cardiovascular function in Rhesus monkeys. The Journal of Auditory Research 15: 234-251, 1975		
<u># of Ref.'s</u> 104	<u># of Fig.'s</u> 9	<u>Language</u> English
<u>Type & duration of experiment</u> in acoustically isolated booths - 3 experiments (1) 60 days--signalled noise bursts (2) 66 days (36 d. quiet & 30 d. noise) (3) 190 days--quiet		<u>Purpose for study</u> to study the cardiovascular effects of noise in a species closely related to man under controlled conditions
<u>Description of test groups (subjects, #, age, etc.)</u> 3 4-kg. female Rhesus monkeys: 1 for each of the 3 experiments. Each monkey had an implanted blood-pressure catheter in the femoral artery		
<u>Control of other stressors</u> controlled laboratory (effect of chair restraint studied alone)	<u>Statistical Methods</u> not specified	
<u>Noise Stimulus</u> (1) source: speaker (2) recorded urban noise (3) none spectral characteristics: spectra given (1) broadband noise level: (1) 112 dBA; (2) L_{eq} = 78 dBA (3) no noise-chair restraint only. length of exposure: (1) 9 sec. (2) 12 hr./day-30 days (3) none # of trials: (1) 8 per day-30 days (2) 1 continuous for 12 hrs. daily (3) none	<u>CVS Response Measured</u> heart rate blood pressure (BP)	
	<u>Nonauditory effects</u> (1) both heart rate and BP increased due to CVS noise; baseline BP increased by an average of 28%, significantly (2) major increase in heart rate and BP in 1st few hrs. (AM) of stimulus & 1 hr. before (BP) (3) no sustained changes in heart rate Other: behavioral activity	
<u>Author's conclusions</u> Unexpected (intermittent) noise and continuous noise can cause sustained cardiovascular changes, which can become independent of a noise stimulus. Noise may be a contribution to the long-term development of cardiovascular disease.		
<u>Evaluation & comments</u> The use of only 1 monkey per experiment is a weak point--individual variations in responses were not studied.		

Peterson, E. A. et al. Noise and cardiovascular function in Rhesus monkeys. The Journal of Auditory Research 15:234-251, 1975.

Three experiments on the effects of noise on heart rate and blood pressure in Rhesus monkeys are reported. A different 4 kg. female with a blood pressure catheter implanted in the femoral artery, served as the subject for each experiment. The monkeys were placed in restraining chairs in double-walled acoustically isolated booths for the duration of the studies. The first experiment used a classical conditioning procedure with a red light as the conditioned stimulus (CS) and 112 dBA broadband noise at the monkey's ear as the unconditioned stimulus (UCS). The most common schedule was as follows: the CS was presented first for 6 sec., then the UCS (noise) for 9 sec. Baseline values were recorded for 10-15 sec. before the CS. The conditioning trials were presented up to 8 times per day for 30 days. During the final 30 day period, the stimulus schedule was held constant. Evidence of some adaptation to the noise stimulus was obtained, since during the final 12 days of the experiment, the magnitude of the responses (heart rate and blood pressure increases) decreased. An apparently conflicting finding was an overall average increase in the baseline blood pressure of 28% over the 30 day interval, which is not an adaptive response. The authors explain the apparent contradiction in terms of two different mechanisms operating: a neurological short-term reaction (adaptation) and a hormonal long-term reaction (upward shift in baseline level). The second experiment consisted of a 30 day control period and then a 30 day test period, during which the monkey was exposed to noise continuously for 12 hours per day. The noise stimulus was a recording of urban traffic noise with an average equivalent noise level (L_{eq}) of 78 dBA. Major increases in heart rate and blood pressure due to noise occurred in the early morning hours and declined throughout the day for the 30 day period. The blood pressure increase began 1 hour before the noise stimulus was turned on, possibly an anticipatory response. The third experiment examined the effect of chair restraint alone on a monkey for 190 days. No sustained effect on heart rate and blood pressure was observed. The authors conclude that both intermittent and continuous noise stimuli can cause sustained changes in cardiovascular responses, which can be independent of the noise. Noise may contribute to the development of cardiovascular diseases in humans.

NOISE AND CARDIOVASCULAR FUNCTION IN RHESUS MONKEYS¹

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I. INTRODUCTION

For the predictable future, urban societies will continue to be noisy. (1) Diverse and pervasive activities, including those associated with transportation, (2,3) work, (4) and even leisure (5-10) contribute to the contemporary din. It has been estimated that some seventy million people in this country alone are exposed to average noise levels greater than 60 dbA, and six hundred thousand more are exposed to average noise levels greater than 80 dbA. (11) It is not surprising, therefore, that in recent years the question of whether or not such levels eventually become injurious to health has developed into an important scientific and medical issue which has been recognized not only by scientists and physicians, (12-16) but by public administrators (17) and private citizens as well. (18)

Much work has been published which bears on the relationship between noise and various neurological, hormonal and behavioral responses in both animals and humans. (13, 19-22)

Acute responses to sudden noise are well-known and were summarized some time ago by Rosen in the following way: "...the blood vessels constrict, the skin pales, the pupils dilate, the eyes close, one winces, holds one's breath and the voluntary and involuntary muscles tense. Adrenalin is suddenly injected into the blood stream which increases neuromuscular tension, nervousness, irritability and anxiety." (23)

In addition to producing acoustic trauma, prolonged exposure to noise is claimed to foster a wide range of non-auditory physiological changes, such as: increased adrenal cortical activity (24, 25) with associated complicated changes in hormone balance (26, 27, 28) and blood-urine composition, (29-32) gastric ulceration, (33, 34) adrenal hypertrophy, (34) thymus hypotrophy, (32) sexual-reproductive, developmental and neurological anomalies, (21, 35, 36) decreased resistance to viral diseases, (37) decreased immunological reactivity, (38) decreased fetal osteogenesis, (39) increased cholesterol deposition andtherosclerosis (31) and increased susceptibility to chemically-induced cancer. (40)

¹A portion of this report was read before a Joint Acoustical Society of America-Internoise '76 Technical Session, "Noise: Its Significance for the Individual and Society," on April 7, 1976, Washington, D.C.

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³Department of Psychology

Changes in salivary gland secretions, (41) blood sedimentation rate, (42) blood-brain barrier permeability, (43) cerebral flow, (44) transketolase enzyme activity, (45) brain metabolism (46) and function, (47) respiration, (48) and the kidneys (49) have also been noted.

Many of these changes are said to be protective reactions associated with the development of the classic General Adaptation Syndrome. (50)

Because of the obvious implications for health related to malfunctioning of the heart and blood vessels, the cardiovascular system has been singled out for extensive study and numerous experiments have been performed under a variety of noise exposure conditions.

Cardiovascular studies can be conveniently classified as those in which animals are used as models for understanding human reaction to noise and those in which humans are used. Human studies can further be divided into those which are carried out under field conditions or retrospectively and those which are carried out under laboratory conditions.

Animal studies originating in this country predominately have used rodents and lagomorphs. (51, 52) In the typical paradigm, rats or rabbits are subjected to intense noises of varying character and duration. Small to significant increases in blood pressure and heart rate, as well as certain changes in heart structure have been reported. (53, 54)

Studies in which these species are used to specify noise effects have been roundly criticized. (12, 13, 55) The most commonly voiced complaint centers about the special susceptibility of numerous rodent species to noise-induced convulsive behavior. (56, 57) Another distinctive reaction of rats is the increase in blood pressure associated with moving these animals to unusually quiet surroundings. This phenomenon has recently been labeled "sound withdrawal hypertension." (58, 59) By contrast, blood pressure in humans goes down under conditions of acoustic isolation. (60, 61) Also, rodents and rabbits often respond to stressful stimuli, including noise, with immobility or freezing. The physiology underlying such behavior, particularly the cardiovascular aspects, differs importantly from that associated with the classic "fight or flight" behavior (62) prominent in anthropoid species.

A final criticism relates to selection of inappropriate stimulus parameters. Firstly, the sound pressure levels used are usually well above present or even projected environmental values, and secondly, the major energy of these intense stimuli often lies below the range of frequencies to which rats, at least, are most sensitive. (63)

In short, the value of research in which acoustically aberrant species are bombarded with unrealistically intense "infra-sound" in order to infer human reactions to lifelong noise exposure seems slight indeed.

In human studies based on field data, changes in cardiovascular function have been noted mainly in the industrial setting because, despite the fact that urban noise levels have generally been increasing over the years, still higher levels are to be found in the work place. (64-74) Many investigators in eastern and western Europe and Russia have noted the relation between cardiovascular anomalies

and noise exposure occurring during work hours. These studies can generally be described as retrospective in the sense that noise-induced changes are not traced as they occur but are reviewed after the workers have been exposed for a number of years.

In this form of research, factors other than noise cannot be ruled out as causative agents of the effects noted. Nevertheless, there does appear to be a consensus that the noisier the industrial environment within which a worker must perform, the more likely he is to develop cardiovascular diseases, including waveform disorders, peripheral circulatory disorders and hypertension.

The criticism most frequently leveled against the bulk of field-industrial studies is that the often slight differences in cardiovascular function between exposed and non-exposed workers may be accounted for by factors not directly associated with noise. These include the anxiety of working with dangerous machines under conditions of poor ventilation and excessive heat and vibration, as well as problems associated with job security and personnel selection. (12, 55) A second major criticism relates to stimulus control. Off-the-job noise exposure has generally not been accounted for and thus, not controlled. Indeed, even on-the-job exposure has not yet been precisely specified because of the continuously changing relationship between the worker and various noise sources. Because of these shortcomings, the results of field-industrial studies have to be considered at this time provocative rather than definitive.

Better control of stimulus-response parameters is possible in the laboratory setting. Work by Jensen and his colleagues over the past 15 years has provided many of the details of human cardiovascular adjustment to sounds. (70-72) Included among their many significant findings is the observation that little adaptation of peripheral vasoconstriction responses to noise occurs with repeated testing over a span of 6 mo to 3 yrs. Other, mainly short term, laboratory studies (75-80) have, with one exception, (81) noted changes in heart rate, blood flow and blood pressure resulting from exposure to noises ranging from "muffled rustling" to sonic booms.

Most laboratory studies performed to date can be legitimately criticized for their failure to control extra-experimental noise exposure and for their short duration.

II. EXPERIMENTATION

We have undertaken a long-term study of the relationship between noise and cardiovascular function in our laboratory which is designed to overcome, at least partially, the deficiencies inherent in much of the previous work: by studying cardiovascular responses in a species closely related to man and by maintaining rigorous and continuous control of the acoustic environment of the subject. Work described below represents the first three studies carried out under this program.

EXPERIMENT I

In our first experiment, we attempted to trace the course of cardiovascular responses to noise over an extended period. We made no attempt to emulate life-like stimulus conditions.

Methods and Procedures.

Our subject was a young, female Rhesus monkey of about 4 kg weight. A blood pressure catheter was surgically implanted in the abdominal aorta via the femoral artery. During recovery, the monkey was continuously restrained in a specially designed chair. It remained in this chair for the duration of the experiment and, in keeping with previous observations, it did so without apparent ill effects. The blood pressure catheter was continuously infused with a saline and heparin solution in order to prevent clotting and to maintain catheter patency.

The restraining chair and auxiliary equipment were placed in a double-walled, acoustically isolated booth. Responses measured included behavioral activity, blood pressure and heart rate. Signals from the appropriate transducers located within the booth were amplified and fed into a polygraph which provided a graphic record of the raw data.

In this experiment, a classic conditioning procedure was used. A broad band noise measuring 112 dbA at the monkey's ear served as the unconditioned stimulus (UCS). It was obtained by driving a speaker with square wave pulses at a rate and pulse duration which by trial and error had proven to be most disruptive to the ongoing behavior of this particular animal. A narrow band analysis of the UCS is shown in Fig. 1. Major energy extended from about 0.4-4 kc/s.

In order to distinguish differences which might occur in the learned and unlearned responses associated with this potentially stressful stimulus, a signal for the noise (CS) was also used in most of the trials. CS was a red light placed well within the animal's field of vision.

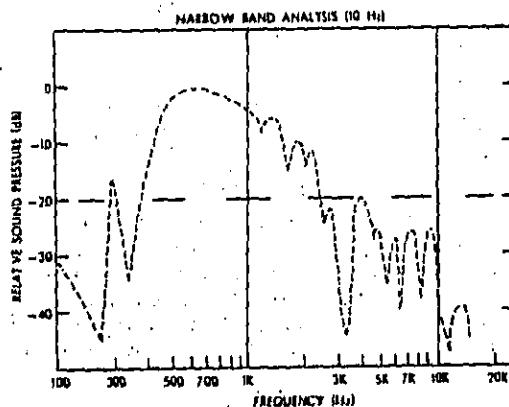


Fig. 1. Narrow band (10 c/s) analysis of noise stimulus used in Exper. 1. "0" db equals 112 dbA. Only peaks are shown. Frequency in kc/s.

Various conditioning trials were presented up to 8 times/day. A baseline (control level) for heart rate and blood pressure was recorded for 10 to 15 sec immediately prior to CS, UCS, or CS-UCS presentations. In the most common schedule, the CS was presented first for 6 sec and then the UCS was presented for 9 sec directly upon termination of the CS. Responses were monitored during CS and UCS presentation and for 5 min after termination of the UCS.

Results.

Heart Rate, Acute Responses: Over the final 30-day interval of this experiment, during which time stimulus conditions were held constant, a slight but consistent decrease in heart rate occurred within the CS interval. By contrast, a large elevation in heart rate occurred within the UCS interval. On most trials, heart rate approached baseline levels sometime during the 5-min post-stimulus interval, although these post-stimulus responses became quite variable. With repeated daily exposure, the peak change in heart rate to the UCS became slightly smaller indicating possible adaptation to the noise. Fig. 2 summarizes these data. Heart rate adjustments in UCS alone trials were not significantly greater than those in CS-UCS trials and the trend toward smaller, acute responses was similar under both conditions.

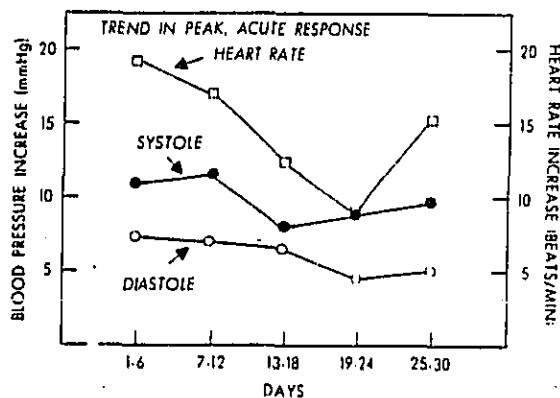


Fig. 2. Exper. I: 30-day trends in acute cardiovascular responses of Rhesus monkeys to signaled noise bursts. Conditioned stimulus was a red light. Unconditioned stimulus was noise burst at 112 dbA. Values of response change are shown on the ordinates and relevant measurement periods are shown on the abscissa. Changes are not statistically significant.

Blood Pressure, Acute Responses: Blood pressure also exhibited a slight decrease during the CS interval and then an increase associated with the UCS. This response, however, was uniformly delayed until about 10 sec after termination of the UCS. Toward the end of the experimental period, the magnitude of blood pressure adjustments became very small during presentation of either CS or UCS. Peak changes, that is, those occurring 10 sec after the stimulus was terminated, however, were not significantly reduced (see Fig. 2).

Baseline Trends: Perhaps the most important finding associated with this first experiment is that baseline blood pressure (sampled before either CS or UCS presentation) increased progressively over the 30-day interval (see Fig. 3). The overall averaged blood pressure increase was 28% ($p < .001$).

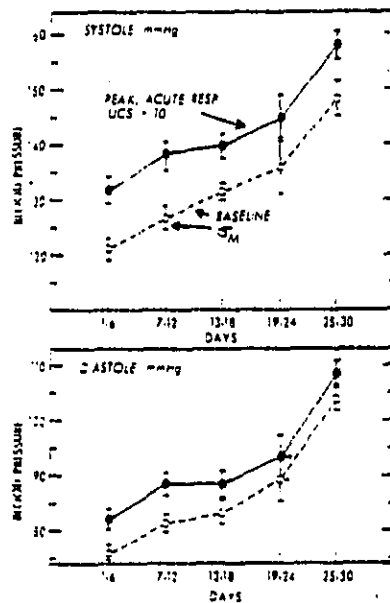


Fig. 3. Exper. I: Chronic trends in cardiovascular functioning: solid lines represent absolute value of peak, acute response to UCS, while dashed lines represent absolute value of "quiet time" responses. Increases in systolic and diastolic blood pressure over a 30-day period are significant ($p < .001$).

Unlike blood pressure, baseline heart rate did not continue to increase during the same period, despite a precipitous change in the first half of the experiment. Baseline heart rate trends, however, hint at a form of cyclicality in which there is a rhythmic rise and fall of responses coupled with a failure to return to earlier

baseline levels (see Fig. 4).

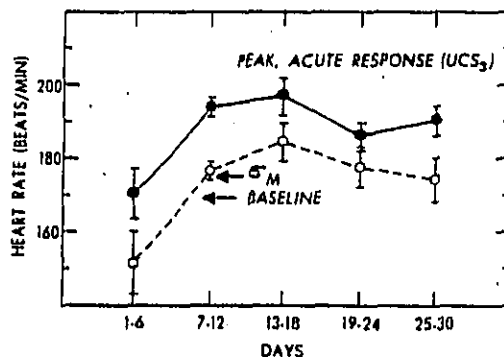


Fig. 4. Exper. 1: Chronic trends in cardiovascular functioning; over a 30-day period, no significant changes in heart rate occurred during the final 12 days of the exposure.

Discussion.

Our baseline results are consistent with those of Forsythe (82) who found that, using long-term intermittent stress involving shock avoidance, blood pressure remained elevated even at times when the stress was not present. In general, previous work in other laboratories has established that the short-term elevation in blood pressure which occurs in response to repeated presentation of aversive stimuli come to be associated with long-term elevations in baseline blood pressure. Such shifts in cardiovascular functioning are typically seen in experiments which use peripheral electric shock as aversive stimulus. (83-85) While shock is an excellent laboratory tool, it is hardly a widespread environmental pollutant. Our findings are of interest because changes similar to those which have been produced by shock have now been produced by noise, an aversive stimulus to which we are all exposed.

Our results also bear upon the significant environmental question of whether or not physiological adaptation eventually occurs to noises which initially produce startle reaction or discomfort. We found, for example, a generalized reduction in the magnitude of change in acute responses during UCS presentation as the experiment progressed. Averaged blood pressure responses taken over the final 12 days of the experimental period, in fact, showed very little or no elevation during the UCS interval. Such data might be interpreted as upholding the position that physiological adaptation does occur even to severe acoustic stimulation. Nevertheless, the perseveration of elevations in immediate post-stimulus blood pressure and the large increase in the absolute levels of baseline blood pressure seem to provide an effective counter-argument to this position.

It may also be speculated that these trends are not necessarily contradictory. Each may have been mediated by a different control mechanism. Adaptation during the relatively brief poststimulatory interval, for example, may reflect fast acting, neurogenic influences while post-stimulus perseveration and baseline sensitization may reflect slower acting, but longer lasting, hormonal influences. Whatever their basis, the important point is that the sustained adjustments did occur which apparently shifted the operational characteristics of the cardiovascular system upward. Whether ultimately adaptive or mal-adaptive, they represent an attempt by the animal to cope with a new and possibly threatening environmental factor.

EXPERIMENT II

Few individuals would be exposed to the level and character of the stimulus we used in the first experiment. We therefore initiated a second experiment designed to trace over an extended period cardiovascular responses to a noise recording which emulates more closely actual environmental conditions.

Methods and Procedures.

As in Exper. I, our subject was a young, female Rhesus monkey of about 4 kg weight. The proximal sensor was again a cannula placed in the abdominal aorta via the femoral artery. S was restrained and placed in the sound proof booth immediately after surgery. Surgical recovery took place within this context.

Baseline data was accumulated for 36 days until stable blood pressure and heart responses were achieved over a 9-day period. A protracted acclimation period was necessary because an obviously painful breakdown and infection of this monkey's ischial callosities was associated with a steady rise in blood pressure which lasted for almost 4 weeks until we were able to instigate healing.

After healing progressed, blood pressure and heart rate returned to the desired stable levels. A high level of cleanliness coupled with the placement of sanitary napkin pads beneath the ischial callosities undoubtedly contributed to the rapidity with which a recovery was made in this monkey.

Once stable baseline responses were established, the monkey was exposed to noise for 12 hrs each day for 30 days.

We carefully specified the stimulus for this experiment. It consisted of a 20-min endless loop recording of daytime exterior noise made in the vicinity of Jackson Memorial Hospital, Miami, Florida. The sample was meant to represent, with regard to both source and level, typical inner-city, urban noise. Major noise sources were 154 ground vehicle passbys (autos, trucks and buses) and 6 aircraft flyovers, as well as several nearby conversations, whistles, horns and tire squeals.

For experimental purposes, the level of the recording was adjusted upward about 5 dbA so that it exceeded 68 dbA 90% of the time, 76 dbA 50% of the time, and 84 dbA 10% of the time (see Fig. 5). The average equivalent noise level (L_{eq}) was 78 dbA. The total noise environment of the animal was such that a large majority of the humans so exposed would express dissatisfaction.

The best evidence available, however, indicates that these levels will generally not lead to irreversible changes in *auditory* function in humans.

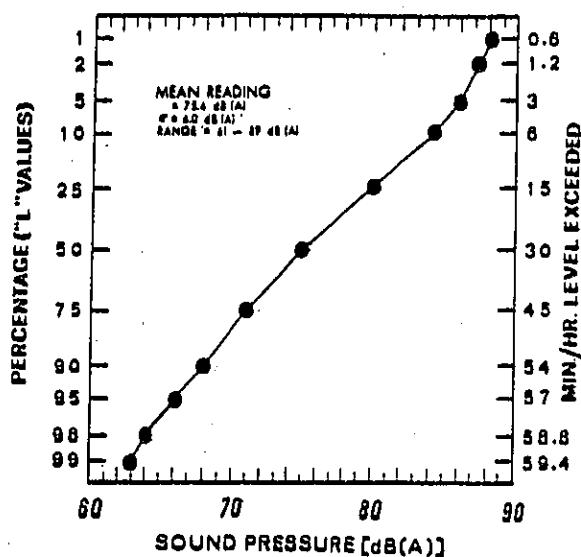


Fig. 5. Exper. II: Analysis of community noise stimulus: probability that sound pressure i is exceeded j per cent of the time (i.e., "L" values) dbA. Loq (average equivalent energy level) = 78 dbA.

The above analysis was performed in the sound attenuating booth with the sound level meter placed at the level of the monkey's ear.

Results.

Fig. 6 shows the blood pressure responses for the last 39 days of Exper. II.

As in the Exper. I, the data present arguments for and against cardiovascular system adaptation to noise. For example, subsequent to the commencement of daily stimulation, blood pressures became elevated well above control levels. During the first 10 days of the exposure period, nighttime (i.e., quiet time) blood pressure remained about as high as daytime blood pressure. Thereafter, however, the former declined and hovered close to baseline levels for the remainder of the experiment. Daytime levels by contrast remained elevated for the rest of the experiment.

Since measurements were taken on an hourly basis, we have been able to assess diurnal variation in cardiovascular function during the pre- and per-exposure periods. In the "natural" rhythm of heart rate and systolic blood pressure in this particular animal, both parameters reached maximum values around mid-

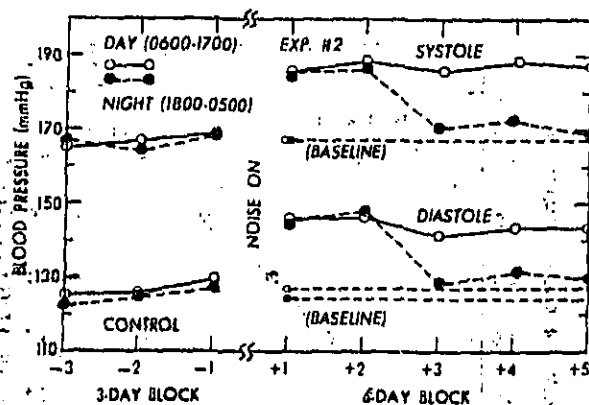


Fig. 6. Exper. II: 30-day trends in blood pressure responses to 12 hr per day community noise stimulation. Note initial increase in both daytime and nighttime blood pressure followed by a return of nighttime level to baseline and continued elevation of daytime levels.

morning and minimum values occurred during the early morning hours. The diurnal rhythm during the period of noise exposure was grossly similar to that for the control period, except that, hour for hour, the absolute values of systolic blood pressure were always higher.

By subtracting the hourly values associated with the pre-exposure period from the hourly values associated with the per-exposure period, the effects of noise, independent of diurnal rhythm, can be determined.

As Fig. 7 indicates, the peak noise effects on systolic blood pressure began around 0500. It is important to note that this is 1 hr before the noise was turned on each day. The effects lasted until roughly 1400, and thereafter, fell to a minimum around midnight. A similar pattern was obtained for heart rate, although the major peak occurred at about 0800 to 0900 with a secondary one again at around 0500.

The far more rapid buildup of both systolic blood pressure and heart rate during the early morning hours, 0200 to 0500, in the per-exposure period compared to the static or opposite trend during the pre-exposure period, indicates the possibility of an anticipatory response on the part of the animal. Such responses are said to enhance the perception of noise as noxious or annoying by humans.

Finally, as seen in Fig. 7, both heart rate and systolic blood pressure appear to be less affected as the noise continued throughout the day. Systolic blood pressure adjustments began to decline after 1400, and heart rate adjustments

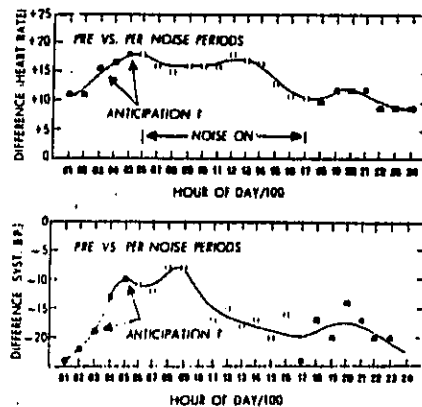


Fig. 7. Exper. II: Hourly effects of noise with diurnal influences parcelled out: (a) systolic blood pressure and (b) heart rate. Note that during the pre-noise, early morning hours an anticipatory response is apparent for both heart rate and systolic blood pressure. The effect of the noise at 0500 is at or near the maximum for the day.

declined after 0900. It is possible that this particular decrease in noise effect reflects the daily buildup of an increase in auditory threshold. The low effective intensity of the stimulus makes it seem unlikely that the adaptation of cardiovascular adjustments is based on a decline of sensory function, however.

EXPERIMENT III

Forsythe, (84) Augenstein (83) and others have demonstrated that chair restraint *per se* has little sustained effect on gross cardiovascular function in the Rhesus monkey. However, in order to demonstrate the innocuousness of our particular procedures, we chair-restrained an animal for 190 days. It was planned that the animal would be exposed to no unusual conditions, including no unusual noise.

In Fig. 8, the daily average heart rate for the period of restraint is shown: 80% of the daily averages fall between 150 and 170 beats/min. Interestingly, the two peaks in heart rate, which occurred about Days 110 and 150, were associated with heavy construction noise and vibration within our building. Other than these two changes, no sustained trend was apparent.

After 190 days, the catheter was removed and the monkey returned to its cage where it remains active and healthy as of this writing.

III. CONCLUSIONS

Our preliminary experiments demonstrated that both unexpected and continuous noise when presented repeatedly or chronically bring about sustained alterations in the balance of cardiovascular function which may become stimulus independent. The results are not definitive, but they do suggest to us that noise

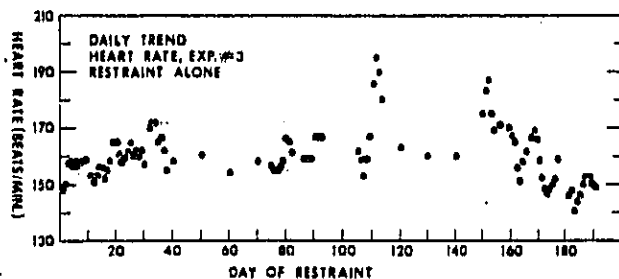


Fig. 8. Exper. III.: Long-term trends of chair restraint alone (Control animal): heart rate for 190 days of restraint; 80% of averaged daily heart rate fell between 150 and 170 beats/min; no trend is evident.

may be one of the factors contributing to the long-term development of cardiovascular pathology in man. Of particular interest in this regard, are the sustained blood pressure elevations which may be induced by moderate but protracted noise stimulation.

Today, "cardiovascular diseases constitute the main underlying cause of mortality and morbidity in the United States." (86) One form of cardiovascular disease, hypertension, is the most common chronic disease. It is said to afflict between 10 and 15% of all Americans (87) and fully one-quarter of those above the age of 55. (88) Still, the pathogenesis remains unknown in 80-90% of these cases.

There are many lines of conflicting and contrasting evidence surrounding the mystery of "essential" hypertension, but perhaps the least contested fact is that the elevated blood pressure associated with hypertensive disease contributes in a striking way to disease and death. (89)

Despite universal agreement that hypertension is a widespread and serious - even life-threatening - problem, confusion remains as to whether "it" is a single disease entity (90) or various diseases subsumed under a single label. (91) It is not surprising then that there is also considerable disagreement regarding the underlying causes of hypertension. (91,92) Traditionally, a variety of somatic mechanisms (93, 94) has been proposed as etiologic factors, but more recently, the importance of certain situational, psychological and environmental effects have been amply documented. (95-98)

Perhaps the studies most pertinent to our present research have established that the etiology of hypertension can also be viewed from a larger, socio-environmental perspective. There is now "abundant evidence that social stress plays an important role in accelerating the progress of hypertensive illness." (99) Take as a case in point the often noted observation that essential hypertension is more prevalent and more severe in blacks than in whites. American black men under 50 have a death rate some 6 to 7 times greater than American white men of the same age. (100) The relation between skin color and blood pressure level, the

common patterns of blood pressure in certain isolated populations and in the descendants of African tribes and the frequent occurrence of hypertension in both maternal twins have been offered as substantiation for a genetic basis of hypertension. (90) That such a position is probably not entirely accurate is made clear by recent observations that:

a. poor blacks and poor whites of low socio-economic status, living in inner-city areas, have higher blood pressure on the average than do their richer, high status, suburban counterparts;

b. blacks who were born in the inner city have higher blood pressure on the average than do blacks who migrated into these areas; and

c. black adolescents attending suburban schools have lower pressure than do those who attend inner-city schools. (101-103)

Such findings suggest at the very least an environmental overlay on the pathogenesis of essential hypertension.

There are many ways, both obvious and subtle, in which inner-city areas differ from suburban and rural areas. The distinguishing characteristic which is most germane to the issues raised by our study, however, relates to the fact that excessive noise seems to be an ever-present by-product of urban activity. It has been shown that downtown areas are about 10 to 15 dbA noisier than quiet suburban areas. (104)

High blood pressure appears to co-exist with higher noise in the inner city and with noisier occupations. This fact, together with evidence from many studies, including our own, strongly suggest that noise has profound effects on cardiovascular function, particularly in aspects relating to blood pressure regulation. Unfortunately, present lines of evidence do not specify the nature of these effects conclusively. Therefore, we believe further inquiry is vitally important.

The procedures developed in our preliminary studies should enable us to examine cardiovascular adjustments to long periods of noise exposure in both semi-restrained and unrestrained monkeys. We believe the course and site of these adjustments, if they occur, can now be described in detail in a species closely related to man.

IV. SUMMARY

Considerable evidence has accumulated which bears on the non-auditory aspects of noise effects. Because of the serious and pervasive nature of cardiovascular disease, including hypertension, the relation between noise and cardiovascular function has received particular attention. For a variety of reasons, however, past efforts in this area have not provided a satisfactory understanding of man's long-term reaction to noise.

In the preliminary studies reported here, an attempt was made to overcome some of the deficiencies inherent in previous work: first, by studying cardiovascular responses in a species closely related to man, and second, by maintaining rigorous and continuous control of the acoustic environment of the subject.

We exposed one semi-restrained Rhesus monkey to unpredictable, intense noise bursts and another to recordings of exterior community noise. Cardio-

vascular adjustments, measured by an indwelling catheter, persisted or became sensitized to certain aspects of the experimental situation and adapted to other aspects. In Exper. I, acute blood pressure adjustments to the noise bursts became slightly smaller as the stimuli were repeated over a 30-day period. Baseline blood pressure measured during quiet times rose by about 30% over the same period; however. In Exper. II, perstimulation blood pressure remained elevated for more than 30 days to recorded community noise at an Leq of 78 dbA, while non-stimulation blood pressure fell to baseline after 10 days. Exper. III explored the long-term effects of our restraining techniques alone; no discernible trends in cardiovascular responses were noted over a 190-day period.

These results offer suggestive evidence that noise may have a profound effect on cardiovascular function, particularly blood pressure regulation.

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SECTION 20

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> E.A. Peterson (Chief, Div. of Auditory Research) et al.	<u>Institution and address where research was performed</u> University of Miami School of Medicine Dept. of Otolaryngology Division of Auditory Research Miami, Florida
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Citation Peterson, E.A. et al. Continuing studies of noise and cardiovascular function. Unpublished paper, 1976, 13 p.

# of Ref.'s 20	# of Fig.'s 6	Language English
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<u>Type & duration of experiment</u> controlled laboratory. 4 mo.--control period--quiet 7 weeks--test period--noise 4.5 mo.--control--quiet	<u>Purpose for study</u> to measure the effects and after-effects of noise on heart rate of an unrestrained animal
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Description of test groups (subjects, #, age, etc.)
 1 female Rhesus monkey, 4.5 kg. with no prior exposure to loud noise, unrestrained

<u>Control of other stressors</u> laboratory conditions	<u>Statistical Methods</u> average daily standard error of the mean
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<u>Noise Stimulus</u> source: recorded urban community & traffic noise spectral characteristics: described in previous paper noise level: Leq=79.4 dBA length of exposure: 12 hour per day (6 AM-6 PM) # of trials: 1 daily for 7 weeks	<u>CVS Response Measured</u> heart rate--EKG <u>Nonauditory effects</u> heart rate increased significantly due to the chair restraint alone in a previous experiment; the heart rate increased significantly due to noise; heart rate did not return to normal even 4.5 mo. after the noise was ended.
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Author's conclusions Prolonged exposure to moderate noise levels affects heart responses both during stimulation and for a period of several months afterward. Pathological cardiovascular change could thus begin prior to significant acoustic trauma.

Evaluation & comments

- (1) It is curious that blood pressure, which showed the greatest response to noise in the first experiments (published in the J. Auditory Research 15:234-251, 1975) was not monitored in this follow-up study.
- (2) the monkey had a cardiac conduction defect resulting in missed beats, which may have affected the results

Peterson, E. A. Continuing studies of noise and cardiovascular function. Unpublished paper. 1976.

The effect of a moderate noise stimulus on heart rate was studied in a 4.5 kg. female Rhesus monkey. The animal had electrodes and a radio transmitter for heart rate monitoring implanted with a vest and a backpack assembly to allow free movement (restraint alone was found to cause significant overall changes in heart rate in a previous study). A four month quiet control period in the acoustic test chamber was included to establish baseline heart rate levels in the monkey. The monkey was then exposed to a noise stimulus of recorded urban noise with an average energy level, L_{eq} , of 79.4 dBA (over 68 dBA 90% of the time) for 12 hours per day for 7 weeks. This noise level is below that which could cause acoustic injury in the Rhesus monkey. The noise exposure began at 6 a.m. each morning. Daily heart rate rhythms (diurnal rhythms) were altered in the early morning hours by the noise. Heart rate increased significantly during this time period. An anticipatory response to noise was indicated by the presence of near peak effects on heart rate one hour before the noise began. Neither heart rate nor the early morning rhythms returned to normal baseline levels in a 4.5 month post-stimulus period. Moderate noise levels can cause significant cardiovascular changes prior to any acoustic changes.

CONTINUING STUDIES OF NOISE AND CARDIOVASCULAR FUNCTION

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A substantial amount of evidence derived from both animal and human experimentation indicates that prolonged exposure to high levels of noise may be associated with various forms of cardiovascular dysfunction (Anticaglia and Cohen, 1970; Welch, 1973; Miller, 1974; Hattis et al., 1976). Nevertheless, a clear and unequivocal relationship between noise per se and cardiovascular function has yet to be established. Certain deficiencies which characterize work in this important area contribute to the unsatisfactory state of our knowledge. Among them are selection of inappropriate animal models, relatively short-term measurement periods and inadequate specification and control of noise exposure (Smookler, Goebel and Siegel, 1973; Lockett and Marwood, 1973; Rosecrans, Watzman and Buckley, 1966; Yeakel et al., 1948; Cartwright and Thompson, 1974; diCantogno et al., 1976; Smookler and Buckley, 1969; Jonsson and Hansson, 1977; Parvizpoor, 1976; Andriukin, 1971; Kavoussi, 1973).

Recently, my colleagues and I performed a series of experiments which overcame some of the problems encountered in earlier work. For example, we chose as an animal model a species closely related to humans and we carefully specified the noise conditions to which each subject was exposed (Peterson et al., 1975). We demonstrated, with chair-restrained rhesus monkeys, that changes in heart rate and blood pressure can occur in association with protracted exposure to both short bursts of intense noise, presented randomly throughout working hours, and to moderate levels of community noise, presented for 12 hours daily.

Despite provocative findings, these pilot studies were themselves open to criticism. For example, although it improved stimulus control, partial restraint imposed unnatural limits on the animal's freedom to move in space and, therefore, may have caused additional, uncontrolled stress. Further, no attempt was made to follow the course of cardiovascular function after the period of exposure had been completed. We did not, in other words, study the long-term after-effects of noise exposure.

We are presently conducting an experiment which deals with these specific criticisms; that is, we are monitoring an index of cardiovascular function (heart rate) in an unrestrained animal and are continuing the measurements beyond the period of noise exposure. We hope, thereby, to provide a clearer, though still incomplete, understanding of the relationship between noise and cardiovascular function. This report is a summary of our progress to date.

METHOD

Subject

The subject for the experiment is a young, 4.5 kg female rhesus monkey named Jean. Although used in an earlier experiment as a control animal, she had not been exposed to any unusual noise conditions prior to serving in this study.

Stimulus conditions

The stimulus has been described in detail elsewhere (Peterson, Augenstein and Hosek, 1976). Briefly, noise was recorded for 30 minutes on a sidewalk adjacent to Jackson Memorial Hospital, a major community hospital in Miami, Florida. Included in the sample are about 150 bus, truck and automobile pass-bys, a half-dozen aircraft fly-overs, numerous conversations, shouts, horns, whistles and brake squeals, as well as incidental noise. The sample was designed to be illustrative of inner city, daytime community noise.

Actual recorded levels are increased by about 5 dB to a level just below

that which might eventually cause acoustic trauma in the rhesus monkey (Luz et al., 1973). As can be seen in Figure 1, levels exceed 84 dB(A) 10% of the time, 75 dB(A) 50 % of the time and 68 dB(A) 90% of the time. Average energy level, L_{eq} , is 79.4 dB(A). These values are accurate only if there is a constant relationship between subject and source.

INSERT FIGURE 1 HERE

Because in this experiment the monkey is allowed full freedom of movement within the 1m x 1m x 2.5m test chamber, its actual exposure to the noise can only be approximated. The problem is further complicated by the use of highly reflective surfaces which are necessary to maintain hygienic conditions within the chamber. Thus L_{eq} measured at 8 different points within the chamber was found to vary by about ± 4.0 dB(A).

During the noise exposure portion of the experiment, the animal was subjected continuously to the community noise sample for 12 hours, from 0600 to 1800, each day. Steady operation of a ventilating fan provided a "noise floor" of about 52 dB(A). Its operation also provided masking for extraneous sounds.

Procedure

The animal is lightly anesthetized with Sernylan^R (0.11 ml/kg I.M.). Prophylactic Cephadyl^R is also administered (0.5 gms I.M.). Two strands of 00 stainless steel suture material, sewn into the skin above the ascending aorta and the cardiac apex, serve as electrodes. The electrode leads are connected to a radio transmitter¹ mounted within the rear pocket of a protective vest. The vest and backpack assembly is light enough to allow almost complete freedom of movement and when fitted properly, causes no apparent discomfort to the monkey. Upon recovery from anesthesia, the

R- Sernylan is a registered trademark of Parke, Davis and Company.

R- Cephadyl is a registered trademark of Bristol Laboratories.

1 "Saturn 3," Space Labs, Inc., Los Angeles, Cal.

animal is placed in the test chamber which is furnished with various perches and swings to encourage a normal amount of gross bodily movement.

Analog EKG information is telemetered to a processing receiver¹ that is connected to a second monitor² which provides a hard copy print-out of both EKG waveforms and continuous heart rate trends for off-line analysis.

Transmitter batteries last about 30 days. When it becomes necessary to replace them, the animal is again anesthetized, the vest is removed and washed, the electrodes are inspected and the monkey is washed and examined for skin lesions.

RESULTS

Trends in daily heart rate

Our findings are summarized in Table I and Figures 2 through 5.

Baseline heart rate levels in this animal were established daily for 4 months prior to the noise exposure period. Since, as noted above, she had previously served as a chair-restrained control subject, it became possible to compare, in the same animal, long-term heart rate trends under conditions of quiet and restraint with those under conditions of quiet and non-restraint.

When chair-restrained, average daily heart rate for this animal was 150.1 Beats Per Minute [BPM]. An index of variability, the average of daily standard errors of the mean, was 1.24 BPM. As shown in Table I, when free-moving, average daily heart rate was 140.8 ± 2.85 BPM. The difference is significant ($p < .01$). Contrary to earlier reports (Forsythe, 1969; Augenstein, 1974), then, chair-restraint does indeed seem to affect overall heart rate levels. In keeping with the findings of the above reports, however, no discernible trend is evident either during the 6-month restraint or the 4-month non-restraint period. This is clearly shown for

1 "Saturn 3,"Space Labs, Inc.,Los Angeles, Cal.

2 "Cardio-Care EBD,"American Optical Co.,Bedford, Mass.

the latter period in Figure 2.

INSERT FIGURE 2 HERE

Following the establishment of a baseline, the animal was exposed 12 hours per day to the noise sample. Over the next 24 days, daily heart rate increased significantly ($p < .01$) to an average 157.0 ± 2.10 BPM.

At this point, the radio transmitter malfunctioned. While it was being repaired, the subject was maintained on the same 12-hour per day exposure schedule. Within a week, the backpack had been re-installed and the entire system re-calibrated. Measurements then resumed on a continuous 24-hour basis. During this hiatus, however, heart rate had dropped significantly ($p < .01$) and for the following 21 days it remained at an average 113.3 ± 3.00 BPM.

At the end of the exposure period, the vest and transmitter were removed and the animal allowed to move about in the chamber unencumbered for approximately 6 weeks.

At the end of that period, heart rate measurements under quiet, non-restraint conditions were resumed and continued for the next 3 months. During this time, only a gradual recovery in heart rate has been observed. In fact, at 4.5 months, post-exposure heart rate still has not returned to baseline levels.¹

This particular monkey suffers from a cardiac conduction defect which is manifested as an occasional generation of P-waves without subsequent generation of QRS complexes. Such a pattern appears to be consistent with a second degree atrio-ventricular block in which "... all QRS complexes are preceded by P-waves, but not all P-waves are followed by QRS complexes" (Berne and Levy, 1972). We have labeled this conduction defect simply as "missed beats." The percentage of missed beats

¹ Note on the right of Figure 2 that during the course of recovery, the animal developed a fibrous growth on her arm which appeared to cause her considerable discomfort. While the lesion was growing and immediately subsequent to its removal, heart rate increased significantly.

throughout the experiment is indicated in Table I. Note that the percentage is inversely related to overall heart rate. Fewer missed beats are generated with increased heart rate. Note also that for the pre-exposure baseline period, missed beats averaged about 11% of total beats during the daytime hours. Within the first half of the noise exposure period, when heart rate was significantly higher than baseline levels, missed beats accounted for only about 2% of the total. During the second half of the noise exposure period, when heart rate was significantly lower than baseline levels, missed beats increased significantly to 24%. As recovery proceeded, missed beats decreased to 16% during January and February and to about 13% throughout the month of March. This is close to pre-exposure baseline levels.

Trends in diurnal rhythm

It should be evident from the heart rate and missed beat trends described above that in this experiment, noise exposure after-effects are not short-lived. Perhaps the most striking demonstration of this fact, however, is derived from changes in the diurnal rhythm of heart rate. An illustration of how noise affects this aspect of rhythm is shown in Figure 3. These data are derived from an experiment in which a different chair-restrained monkey, but the same community noise sample, was used. In the top half of the figure, diurnal rhythm for heart rate before and during noise exposure is indicated.

INSERT FIGURE 3 HERE

Throughout the pre-exposure period, heart rate remained relatively constant during the early morning hours, 0100 to 0600. During the exposure period, it increased steeply.

By subtracting hourly heart rate values associated with the exposure period from those associated with the pre-exposure period, it became possible to determine the effect of noise independent of diurnal rhythm. In the bottom half of Figure 3, it can be seen that near-peak noise effects were reached at 0500, one hour

before onset of daily noise. This rapid build up resembled what would, in humans, be called anticipation. Although in the present experiment we did not initially measure heart rate each hour, confirmation of this "anticipatory" response eventually became possible.

Clearly, heart rate varied systematically throughout the day. As shown in Figure 4, heart rate for this animal is generally lower during the late evening and early morning hours than it is during midday under all the conditions we have studied, including pre-exposure restraint, per-exposure non-restraint and post-exposure non-restraint. There are, nevertheless, significant differences among the various conditions. These are illustrated in Figure 5.

INSERT FIGURES 4 & 5 HERE

Return to a diurnal pattern similar to that which characterized the pre-exposure era for this animal would seem to be a reasonable indication that noise exposure after-effects have subsided. While this animal was in quiet and restrained (in 1975), the daily heart rate cycle reached a low point between 0500 and 0600. Slope of a line fitted to data points is steeply negative for the hours 2400 to 0600. In contrast, while this animal was exposed to noise and was unrestrained, quite a different trend was manifested; that is, a heart rate minimum was reached near 2400 hours and slope of the line fitted to data points is steeply positive for the hours 2400 to 0600. This trend reinforces the notion that some sort of anticipatory response is associated with daily onset of the noise. Six to ten weeks after the noise exposure period (labeled "JAN" in Figure 5), slope of the fitted line is still positive. After 10-14 weeks (FEB.), slope of the fitted line is about zero, and after 14-18 weeks (MAR.), it has become slightly negative.

DISCUSSION

The noise-induced changes in early morning diurnal rhythm observed in the present experiment are consistent with those changes observed in a previous experiment in which the animal was restrained.

The course of changes manifested in overall heart rate, proportion of missed beats and diurnal rhythm clearly indicates that prolonged exposure to moderate noise levels not only influences cardiac function during the period of stimulation but also that these influences dissipate slowly after the exposure has ended. The protracted recovery process shown thus far for this monkey may in fact never be completed.

A failure of cardiac function to return to baseline levels may provide the opportunity for resolving a paradox implied in recent studies. It has been a common assumption that if noise cannot be heard, it cannot have significant physiological effects. Investigators consistently report, however, that industrial workers with greater hearing loss appear to have a higher incidence of cardiovascular dysfunction. Based on our present findings, the possibility arises that pathological cardiovascular adjustments are begun before audiological significant acoustic trauma occurs. These adjustments may in fact contribute to hearing loss usually ascribed solely to noise.

Some comments should be made regarding the puzzling downward shift in heart rate which took place during the final three weeks of the noise exposure period. Physical discomfort on the part of the monkey, malfunction of the monitor and changes in feeding or cleaning schedules have all been eliminated as possible sources for this drastic alteration in cardiac dynamics. It is difficult to specify the physiological factors responsible for the abrupt change, however, in the absence of other measures of cardiovascular response. Since under present circumstances these

are impossible to obtain, it can only be speculated that perhaps during the first weeks of noise exposure, sympathetic-adrenergic influences governing the greater heart rate were severely diminished, thereby inducing parasympathetic-cholinergic dominance. This was manifested, in turn, by lowered heart rate.

A second possibility is that heart rate and blood pressure were elevated simultaneously. At some point, in order to maintain cardiovascular homeostasis, it became necessary to compensate for the increase in blood pressure.

SUMMARY

Long-term trends in heart rate were observed in a free-moving rhesus monkey.

Initially, normal heart rate for the animal was defined over a span of 4 months. She was then exposed to a community noise recording 12 hours per day for 7 weeks. Per- and post-exposure changes were noted in several aspects of cardiac function.

During the exposure period, heart rate and proportion of aberrant EKG responses rose significantly above, and then, fell significantly below baseline levels. Patterns of diurnal rhythm for early morning heart rate were also altered by noise exposure. These last results are consistent with those of an earlier study in which a restrained animal had been subjected to similar stimulus conditions.

After-effects of the single, prolonged noise exposure episode have dissipated gradually. During the course of a 4-month post-exposure period, heart rate and proportion of aberrant EKG responses have slowly returned to near-baseline levels.

Early morning diurnal rhythm for heart rate has not yet returned to its pre-exposure pattern.

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FIGURE CAPTIONS

- FIGURE 1: Community Noise Experiment II: Statistical characteristics (L values) of stimulus.
- FIGURE 2: Community Noise Experiment II: Daily average heart rate for unrestrained monkey, Jean. Heart rate trends after 11-3-76 were measured 24 hours per day. Note the precipitous increase then decrease in heart rate during 7-week exposure period.
- FIGURE 3: Community Noise Experiment I: Restrained monkey, "Mars".
Top: Diurnal rhythm for heart rate during pre- and per-exposure periods.
Bottom: Diurnal noise effects on heart rate. Near-maximum effects were exhibited 1 hour prior to onset of noise each day (adapted from Peterson *et al.*, 1975).
- FIGURE 4: Community Noise Experiment II: Trends in diurnal rhythm of heart rate during early morning hours. Recovery from 6-week exposure to community noise sample may be manifested by gradual return to negative slope of curve for these hours.
- FIGURE 5: Community Noise Experiment II: Diurnal rhythm of heart rate. Absolute values have been converted to percentage of maximum daily heart rate for normalization. Note truncated dynamic range of heart rate under restraint, no-noise conditions and increased dynamic range of non-restraint, noise exposure conditions (thick, solid line). Generally, the course of diurnal rhythm follows a bell-shaped curve. Peaks during the day are associated with feeding and other activity near the test chamber.

T A B L E I

Heart Rate Trends in
Unrestrained Monkey, Jean

PRE-EXPOSURE

(6/9/76 - 10/1/76)

\bar{X} = 140.8

$s_{\bar{X}}^*$ = 2.85

Missed Beats** = 11%

PER-EXPOSURE

I (10/2/76 - 10/26/76)

\bar{X} = 157.0

$s_{\bar{X}}$ = 2.10

Missed Beats = 2%

II (11/3/76 - 11/23/76)

\bar{X} = 113.3

$s_{\bar{X}}$ = 3.00

Missed Beats = 24%

POST-EXPOSURE

I (1/6 - 2/1/77)

\bar{X} = 129.8

$s_{\bar{X}}$ = 3.54

Missed Beats = 16%

II (2/3 - 2/10)

\bar{X} = 134.6

$s_{\bar{X}}$ = 2.91

Missed Beats = ?

III (2/10 - 2/21) Fibroma
growth and removal

\bar{X} = 150.8

$s_{\bar{X}}$ = 2.54

Missed Beats = 7%

IV (2/22 - 3/13)

\bar{X} = 133.6

$s_{\bar{X}}$ = 2.89

Missed Beats = 13%

V. (3/29 - 4/4)

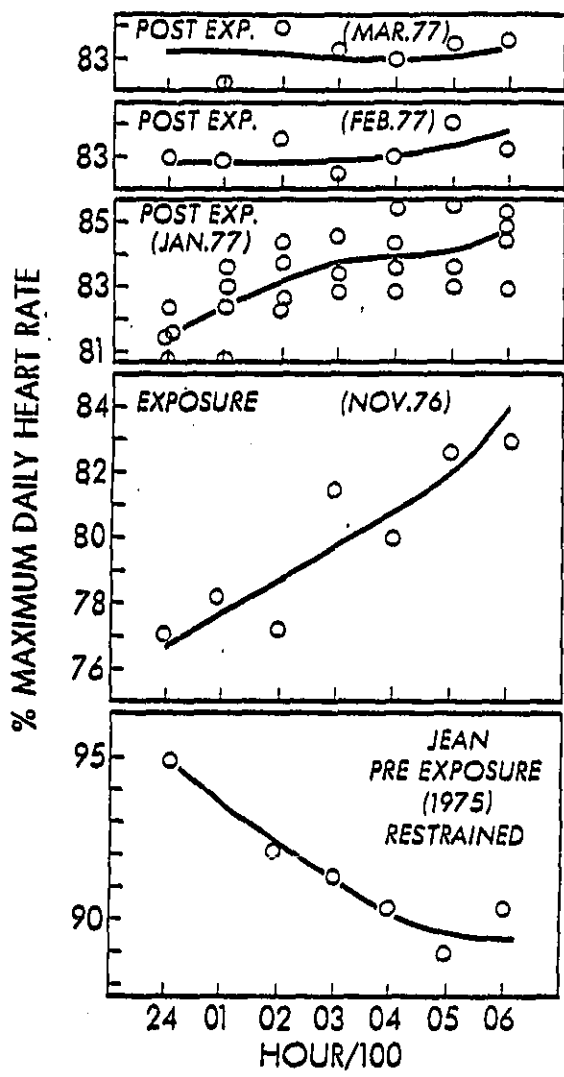
\bar{X} = 131.0

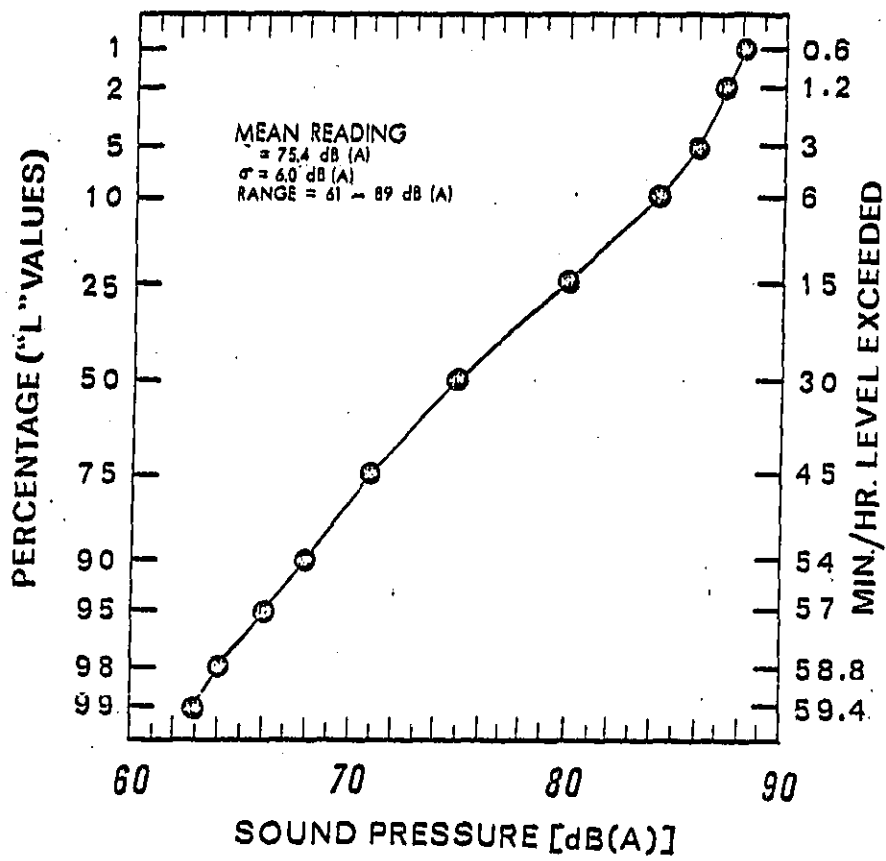
$s_{\bar{X}}$ = 3.11

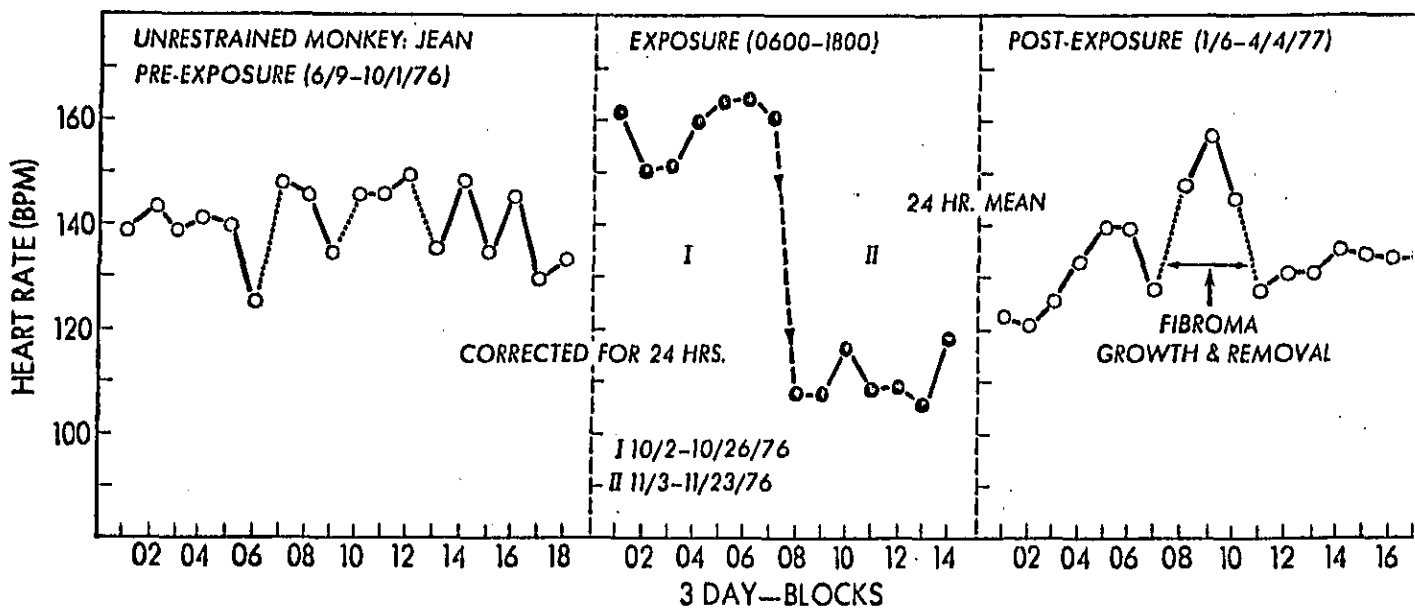
Missed Beats = ?

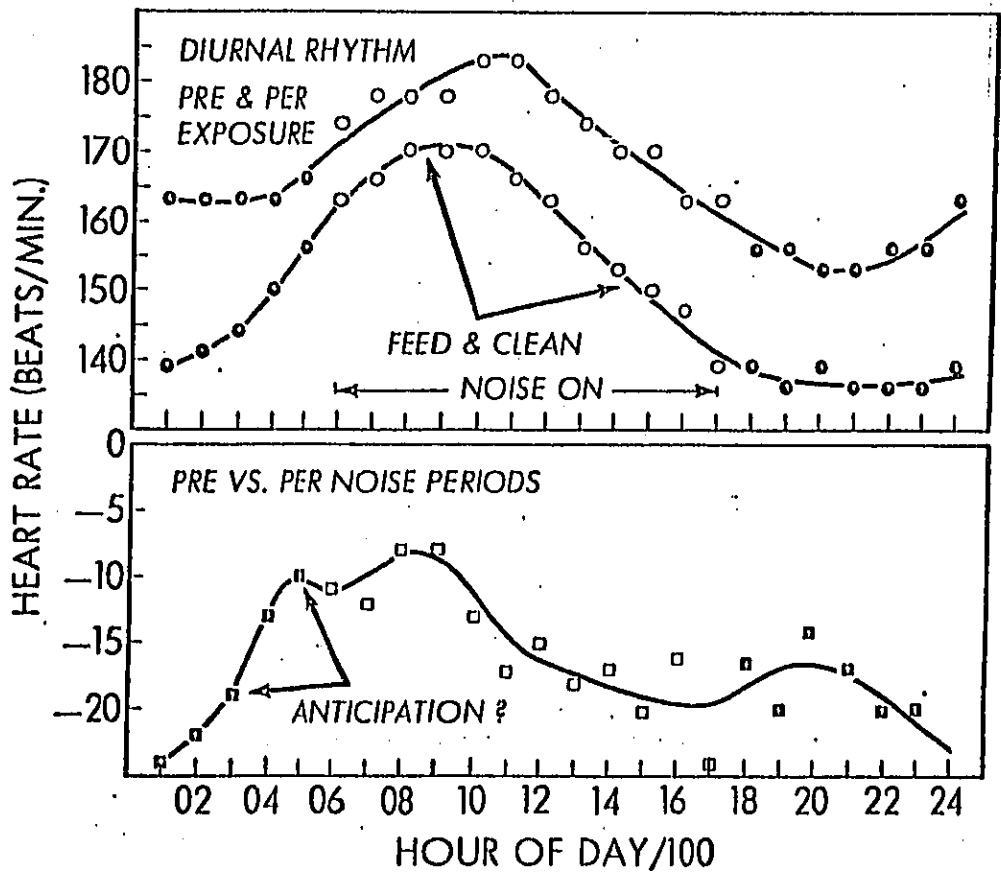
** EKG waveform which may indicate a second degree A-V block.
Measured during period 0900-1700 hours.

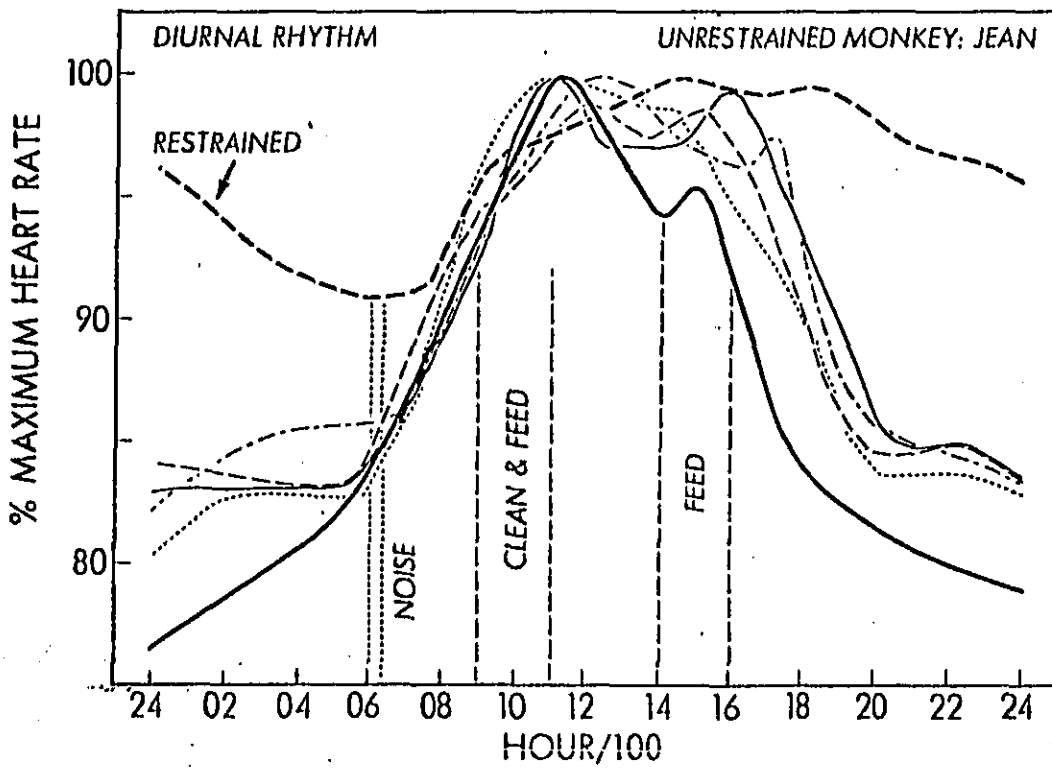
* Average daily standard error of the mean.











SECTION 21

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> G.S. Zvereva M.V. Ratner A.V. Kolganov		<u>Institution and address where research was performed</u> Scientific research Institute of Labor Hygiene and Professional Illnesses Donetsk, USSR
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> same as above	
<u>Citation</u> Zvereva, G.S. et al. Rolling mill noise and its effect on workers. Gig. Sanit. 1975 (11): 104-105, 1975 English translation.		
<u># of Ref.'s</u> 2	<u># of Fig.'s</u> 2	<u>Language</u> Russian (Eng. trans.)
<u>Type & duration of experiment</u> survey of workers at various types of factories having rolling mill noise		<u>Purpose for study</u> to study the effects of rolling mill type noises (intermittent, percussive, impact noises) on workers
<u>Description of test groups (subjects, #, age, etc.)</u> no test and control groups--a general survey of groups of workers was done in various factories; all workers were 30-40 years old and had 5-15 years of service (in this type of factory, presumably)		
<u>Control of other stressors</u> no control		<u>Statistical Methods</u> used to compare significance of differences in noise effects between groups of workers
<u>Noise Stimulus</u> source: pipe and sheet rolling plants--various types of machinery spectral characteristics: low-med. frequency; no graph is given noise level: variable--high noise levels of 122 dB were present intermittently length of exposure: intermittently during working day for 5-15 years # of trials: not applicable		<u>CVS Response Measured</u> pulse blood pressure <u>Nonauditory effects</u> CVS: blood pressure was altered--elevated in many cases--exact changes not reported pulse--fluctuations noted due to noise Other: memory and attention; physiological complaints were listed
<u>Author's conclusions</u> Physiological changes, such as increased or fluctuating pulse rate and blood pressure, decline in pain sensitivity, dizziness, as well as a decline in memory and attention, were observed.		
<u>Evaluation & comments</u> The cardiovascular changes were not reported quantitatively, so no real conclusions can be drawn. The one interesting finding was that non-auditory physiological effects were observed prior to the development of auditory impairment.		

Zvereva, G. S. et al. Rolling mill noise and its effect on workers. Gig. Sanit. 1975 (11):104-105, 1975. English translation.

Workers in various types of factories in the U.S.S.R. that have intermittent rolling mill noise were surveyed for auditory and non-auditory physiological effects, as well as psychological effects. Various trends in types of physical ailments in this industry (metallurgical plants) were reported, although very little quantitative data is included. Some very high intermittent noise levels were recorded, some as high as 122 dB. The 340 workers surveyed were 30 to 40 years old and had been working in the factories for up to 15 years. Hearing impairments were found in 168 (50%) of the workers surveyed. Physiological changes included neurological problems such as dizziness, numbness, and headaches and cardiovascular effects such as altered and often increased pulse rate and blood pressure. The psychological effects included a decrease in memory and attention. Many of the changes were observed even in the first year of work, before the development of any hearing impairment.

ROLLING MILL NOISE AND ITS EFFECT ON WORKERS

By G.S. Zvereva, M.V. Ratner and A.V. Kolganov

Gig. Sanit; (11), 1975

Scientific-research Institute of Labor Hygiene and
Professional Illnesses, Donetsk.

According to the observations of a series of authors, the influence of intermittent noise on the human organism is different from the effect of stable noise. The intensity, the spectral make-up and the time parameters of the former in areas where cutting is going on, where they are trimming, stacking and where they are transporting different types of rolled stock are determined with the aid of a pre-calibrated loop oscillograph N-117 and an NIV-1 noise and vibration gauge. Before the start of a shift of 340 workers, subjected to the effect of intermittent noise, we measured the audibility threshold, the arterial pressure and the pulse, we determined the condition of pain, vibration and temperature sensitivity, and we actively identified their complaints. We also studied the function of memory and attention.

In pipe and sheet rolling plants in the areas around the pilger mills and cross-cutting aggregates, the noise is typically percussive, with almost instantaneous increase of the front part of the impulse (40 ms), with short interval vibrations (0.2 sec) and with high maximum levels (122 decibels). According to the intensity and the character of the intermittent noise generated by the stamping presses and the presses of the Pels type in a rail structuring mill and in rail-splicing plants, it is close

to that which has been described, but it differs in the lower frequency of the impulses (20 imp/min), it is somewhat greater, close to 100 msec, in the increase time of the front as well as the long length of the vibrations and pauses (within 1-2 sec). In section rolling plants, the noise emerging in the work of cutting cold and hot metal, may be referred to the noise of friction. It is characterized by a relatively long increase time of the front (more than 100 msec), comparatively long vibrations and pauses (several seconds), and consequently a low frequency (2 imp/min). In the case of the friction of rolled profiles around the cooling rack of the coolers while they are being transported with rolling skids, the noise of friction also emerges, but with a higher medium intensity of impulse (115 decibels). At the Communist metallurgical plant, the construction of rolling skids has been changed so that the metal does not touch the cooling rack of the coolers when it is moving; this did away with the friction of slip, thanks to which the intensity of the noise dropped ~~to~~ 12 decibels in impulse and 20 decibels in average magnitude.

The noise generated in thick sheet rolling mills with disk and guillotine cutters, and also with the movement of metal sheets along roller conveyors is a combination of percussive noise and the noise of friction and is characterized by non-periodic, inconstant parameters. In some samplings, separate realizations are very essential to distinguish one from the other (100-150 times). Momentary values fluctuate from extremely unsatisfactory (intensiveness in the impulse up to

142 decibels, increase time 20 msec, pause period 0.1 sec) to relatively satisfactory.

Although an opinion exists concerning the great aggressiveness of non-periodic noises (G.A. Suvorov and L.A. Marakushkin), we did not observe this. Evidently, the reason for this is that in production, as opposed to office conditions in the case of the experiment, the alternating of impulses of a different intensity and a pause is caused by the course of the technological process and the appearance of an alternate, high-intensity stimulus (impulse) was not unexpected for the workers. At the same time, the amount of high intensity impulses is known to be less than for example in the case of percussive noise of comparable force. Evidently, this determined the lower activity of the non-periodic noise. Thus, among the 37 workers under conditions of non-periodic noise where the guillotine knives and disk knives are located we found a hearing loss of an initial and slight degree, 43% according to the classification of V.E. Ostapkovich and N.I. Ponomarevoi. In the case of 25 workers at the pilger mills and cross cutting aggregates under the influence of percussive noise, the hearing changes were more profound: we found not only initial and slight, but also moderate impaired hearing and impaired hearing of a considerable degree (20%). The general number of those with impaired hearing in this group (84%) was also substantially greater than in the preceding group ($P < 0.01$). The stable noise, with an intensity of 105 decibels, equivalent to the average force of non-periodic and impact noise, brought about a

corresponding change in 68% of those examined (25 people). But the age and the work stage of all the comparison groups were similar: for the workers chosen to be examined 30-40 years old and a service life of 5-15 years.

Influence of the production stage and the hearing condition on the function of memory and attention (in % of the number surveyed) Table 1

Condition of the function of memory and attention	Service time (years)				Coefficient of ranked correlation with a hearing loss at stage		
	Up to 1	1-2	3-5	6-9	10 and more years	Up to 10 years	More than 10 years
Memory decline:							
hearing:	41.7	61.1	40.7	50.0	65.0	0	0.9±0.01
visual	8.4	11.1	14.8	10.7	42.7	0	0.9±0.01
Drop in attention concentration	16.7	22.2	33.3	32.1	60.9	0.6±0.03	0.9±0.01

Table 2.--Influence of the character of labor on the function of memory and attention (in % of the number surveyed)

Condition of the function of memory and attention	Predominant component of labor		
	Physical	Nerve-tension	Not expressed
Memory decline:			
hearing:	50.0	54.8	46.1
visual	20.0	38.7	7.7
Drop in attention concentr.	53.0	54.8	11.5
			63.8+
			30.0
			76.9+

Note: the + sign on the figure is the presence of a direct, intimate correlation dependence on hearing loss (coefficient of the correlation $r > 0.6$).

168 men were found to have hearing losses (50%) at the described places of rolling mills. The initial degree of the decline among them was 23%, to a slighter degree 19%, moderate and great 9%. In addition to the impaired hearing, many workers were found to have changes in the neurological state. A rather large number complained of headaches, dizziness, irritation, not sleeping well, increased fatigue, pain in the region of the heart, heart palpitations, extended pain in the muscle extremities, cramps, a feeling of numbness, etc. The complex of such complaints was shown to be 25% of those studied after 3-5 years of work under noisy conditions. In many cases, the complaints were combined with objective symptoms. We noted a disruption of arterial pressure (often an increase of it), lability of the pulse, a decline of vibrational and pain sensitivity, up to complete anesthesia, thermoasymmetry, tremors of the hand and eyelids, instability of the Romberg posture.

Many of the listed changes, 17-45% of the cases were recorded on those surveyed already in the first year of work. In the same period, with 20% of the workers we noted a drop of more than 30 decibels in the perception of high tones with normal audibility in the speech range frequency. The early neurological symptom is the result of functional disorders of the central nervous system. Thus for the purpose of a well-timed exposure and taking dispensary accounting of such people, we required a doctor's examination and audiometric examination of the workers under noise conditions during the first year of work.

There was a certain interest in studying the workers' memory and attention functions. An analysis of different stages of work under noise conditions (Table 1) showed that visual memory and attention concentration got worse with an increase of the production stage and age and that there is a direct and close correlative connection with hearing disruption. We did not find a dependence of the condition of hearing memory on the character of the labor (Table 2). At the same time, the presence in groups of workers with a sufficient stage of close, direct correlation between a drop in hearing memory and the degree of hearing drop is evidence of the possible influence of functional noise on this condition. The absence of a similar connection in all the groups with a service life up to 10 years (see Table), as well as a percent which is similarly high independent of the persons with poor memory, allow us to assume that the drop in the hearing memory sets in with workers under noisy conditions (during the first year of work), sooner than the hearing decline (after 3-5 years). The visual memory and the attention concentration at the stage up to the 10th year was seldom lower in the case of workers with nerve tension labor (in 8-11% of the cases), but in 20-53% of the cases, the drop of these functions was observed in people with physical labor. Evidently, the determined influence here showed an elemental professional selection of the workers. The very character of the labor of the workers of the 2nd group required good memory and attention, which is important even with physical labor. A decline of such functions of high nerve

activity such as memory and attention concentration may be related to the number of adequate reactions of the organism to the effect of intense noise.

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**ШУМ ПРОКАТНОГО ПРОИЗВОДСТВА И ЕГО ВЛИЯНИЕ
 НА ОРГАНИЗМ РАБОЧИХ**

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По наблюдениям ряда авторов, влияние на организм человека прерывистого шума отличается от действия стабильного шума. Интенсивности, спектральный состав и временные параметры первого из них на участках пореза, правки, укладки и транспортировки различных видов проката определяли с помощью предварительно откалиброванного шлейфного осциллографа И-117 и измерители шума и вибрации ШИВ-1. Перед началом смены у 340 рабочих, подвергавшихся воздействию прерывистого шума, измерили пороговую слышимости, артериальное давление и пульс, определяли состояние болевой, вибрационной и температурной чувствительности, активно выявляли их жалобы. Изучали также функции памяти и внимания.

В трубо- и листопрокатных цехах на участках пилгримных станков, агрегатов поперечной резки шум типично ударный, с почти мгновенным временем нарастания переднего фронта импульса (40 мс), короткими длительностями звучаний (0,2 с) и высокими максимальными уровнями (122 дБ). По интенсивности и характеру прерывистости шум, генерируемый штепельными прессами и прессами типа «Пеле» и рельсоблочном цехе и цехе рельсовых скреплений, близок к описанному, но отличается меньшей частотой импульсов (20 имп/мин), несколько большим, приближающимся к 100 мс, временем нарастания переднего фронта и большей длительностью звучаний и пауз (в пределах 1—2 с). В сортопрокатных цехах шум, возникающий при работе под холодной и горячей резки металла, может быть отнесен к шуму трения. Он характеризуется относительно большим временем нарастания переднего фронта (более 100 мс), сравнительно длительными звучаниями и паузами (несколько секунд) и, следовательно, их малой частотой (2 имп/мин). При трении прокатных профилей о стелюги холодильников во время перемещения их шлеперами также возникает шум трения, но с более высокой средней интенсивностью в импульсе (115 дБ). На Коммунарском металлургическом заводе конструкция шлепера изменена с таким расчетом, чтобы металл при перемещении не касался стелюг холодильников; это исключило трение скольжения, благодаря чему интенсивность шума снижена на 12 дБ в импульсе и на 20 дБ по средней мощности.

Шум, генерируемый в толстолистовых прокатных цехах дисковыми и гильотинными ножницами, а также движением листов металла по рольгангам, представляет собой сочетание ударного шума с шумом трения и характеризуется непериодичностью, непостоянством параметров. В некоторых выборках отдельные реализации очень существенно отличались одна от другой (в 100—150 раз). Мгновенные величины колебались от весьма неблагоприятных (интенсивность в импульсе до 142 дБ, время нарастания 20 мс, длительность паузы 0,1 с) до относительно благополучных.

Хотя существует мнение о большей агрессивности непериодических шумов (Г. А. Суворов и Л. А. Маракучкин), мы этого не отмечали. Очевидно, причина здесь в том, что на производстве в отличие от камеральных условий при эксперименте чередование импульсов различной силы и пауз обусловлено ходом технологического процесса и появление очередного сверхсильного раздражителя (импульса) не является неожиданностью для работающих. В то же время количество высоких по интенсивности импульсов значительно меньше, чем, например, при ударном шуме соответствующей мощности. Это, по-видимому, и определило меньшую активность непериодического шума. Так, среди 37 работающих в условиях непериодического шума на участках гильотинных и дисковых ножниц выявлено снижение слуха начальной и легкой степени выраженности, согласно классификации В. Е. Остапкоич и Н. И. Пономаревой, у 43%. У 25 рабочих пилгримных станков и агрегатов поперечной резки под влиянием ударного шума изменения слуха были более глубокими: обнаружилось не только начальная и легкая, но умеренная тугоухость и тугоухость значительной степени (20%). Общее число тугоухих в этой группе (84%) было также существенно большим, чем в предыдущей ($P < 0,01$). Стабильный шум интенсивностью 105 дБ, эквивалентный по средней мощности непериодическому и ударному, вызвал соответствующие изменения у 68% обследованных (25 человек). По возрасту и стажу работы все сравнимые группы аналогичны: для обследования взяты рабочие 30—40 лет со стажем 5—15 лет.

Со снижением слуха на описанных участках прокатных цехов выявлено 168 человек (50%). Начальную степень снижения среди них имело 23%, легкую — 19%, умеренную и значительную — 9%. Помимо тугоухости, у многих рабочих найдены изменения в неврологическом статусе. Довольно многочисленными были жалобы на головную боль, головокружение, раздражительность, плохой сон, повышенную утомляемость, боли в области сердца, сердцебиение, тянущие боли в мышцах конечностей, судороги, чувство онемения и др. Комплекс таких жалоб предвдвляло 25% обследованных после 3—5 лет работы в шумных условиях. Жалобы во многих случаях сочетались с объективной симптоматикой. Отмечались нарушения артериального давления (чаще повышение его), лабильность

**Влияние производственного стажа и с
 (в % к числу)**

Состояние функций памяти и внимания	Стаж	
	до 1	1—
Снижение памяти: слуховой зрительной	41,7	61,1
	8,4	11,1
Снижение концентрации внимания	16,7	22,2

Влияние характера труда на функции па

Состояние функций памяти и внимания	физичес	
Снижение памяти: слуховой зрительной	50,0	20,0
	53,0	

Примечание: знак + при снижении от снижения слуха (коэф-т)

пульса, снижение вибрационной и болевой термодиссиметрии, тремор ручки век, неустойчивость зрения.

Многие из перечисленных изменений доанных уже в течение 1-го года работы чено снижение более чем на 30 дБ восприимчивости частот речевого диапазона. Ранняя неврофункциональных расстройств центрального вываления и взятия на диспансерный и аудиометрическое обследование работников работы.

Определенный интерес представляло исследование зависимости того и другого от стажа, что зрительная память и концентрация производственного стажа и возраста, имеют изменения слуха. Не выявлено зависимости от стажа (табл. 2). В то же время наличие в группе корреляционной связи между снижением слуха свидетельствует о возможном влиянии подобной связи во всех группах со стажем, как и одинаково высокий независимо от стажа допустить, что снижение слуховой памяти раньше (в течение 1-го года работы), чем концентрация внимания и слуха у рабочих перво-напряженного труда (в течение этих функций наблюдалось у лиц 2-й группы влияние здесь оказал стихийный професорский труд физическом труде это не является хорошей деятельностью, как память и концентрация адекватных реакций организма на возде

ЛИТЕРАТУРА. Остапкоич В. Е., Рашиер М. В., Колганов А. В. «Мед.», 1970, № 3, с. 79—83. — Суворов Г. А. и др. «Мед.», 1970, № 7, с. 105—106.

Таблица 1

Влияние производственного стажа и состояния слуха на функции памяти и внимания (в % к числу обследованных)

Состояние функций памяти и внимания	Стаж работы (в годах)					Коэффициент ранговой корреляции со снижением слуха при стаже	
	до 1	1—2	3—5	6—9	10 и больше	до 10 лет	более 10 лет
Снижение памяти: слуховой	41,7	61,1	40,7	50,0	65,0	0	0,9±0,01
зрительной	8,4	11,1	14,8	10,7	42,7	0	0,9±0,01
Снижение концентрации внимания	16,7	22,2	33,3	32,1	60,9	0,6±0,03	0,9±0,01

Таблица 2

Влияние характера труда на функции памяти и внимания (в % к числу обследованных)

Состояние функций памяти и внимания	Преобладающий компонент труда					
	физический	нервно-напряженный	не выражен			
Снижение памяти: слуховой	50,0	54,8	46,1	57,8+	60,0	79,4+
зрительной	20,0	38,7	7,7	27,7+	15,0	53,8+
Снижение концентрации внимания	53,0	54,8	11,5	63,8+	30,0	76,9+

Примечание: знак + при цифре — наличие прямой тесной корреляционной зависимости от снижения слуха (коэффициент корреляции $r > 0,6$).

пульса, снижение вибрационной и болевой чувствительности, вплоть до полной анестезии, термоасимметрия, тремор рук и век, неустойчивость в позе Ромберга.

Многие из перечисленных изменений в 17—45% случаев регистрировались у обследованных уже в течение 1-го года работы. В тот же период у 20% работающих отмечено снижение более чем на 30 дБ восприятия высоких тонов при нормальной слышимости частот речевого диапазона. Ранняя неврологическая симптоматика является результатом функциональных расстройств центральной нервной системы. Поэтому с целью своевременного выявления и взятия на диспансерный учет таких лиц необходим врачебный осмотр и аудиометрическое обследование работающих в шумных условиях в течение 1-го года работы.

Определенный интерес представляло изучение у рабочих функций памяти и внимания. Анализ зависимости того и другого от стажа работы в шумных условиях (табл. 1) показал, что зрительная память и концентрация внимания ухудшаются с увеличением производственного стажа и возраста, имеют прямую тесную корреляционную связь с нарушением слуха. Не выявлено зависимости состояния слуховой памяти и от характера труда (табл. 2). В то же время наличие в группах рабочих с достаточным стажем тесной прямой корреляционной связи между снижением слуховой памяти и степенью снижения слуха свидетельствует о возможном влиянии на состояние этой функции шума. Отсутствие подобной связи во всех группах со стажем работы до 10 лет (см. табл. 1), так же, как и одинаково высокий независимо от стажа процент лиц с плохой памятью, позволяет допустить, что снижение слуховой памяти наступало у работающих в условиях шума раньше (в течение 1-го года работы), чем снижался слух (после 3—5 лет). Зрительная память и концентрация внимания при стаже до 10 лет редко были понижены у рабочих нервно-напряженного труда (в 8—11% случаев), но в 20—53% случаев снижение этих функций наблюдалось у лиц физического труда. По-видимому, определенное влияние здесь оказал стихийный профессиональный отбор рабочих. Сам характер труда рабочих 2-й группы требует от них хорошего состояния памяти и внимания, тогда как при физическом труде это не является столь важным. Снижение таких функций высшей нервной деятельности, как память и концентрация внимания, может быть отнесено к числу адекватных реакций организма на воздействие интенсивного шума.

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Поступила 14/III 1975 г.

SECTION 22

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Y. Ando (Dritte Physikalisches Institut, Universitaet Goettingen, Federal Republic of Germany); H. Matorri		<u>Institution and address where research was performed</u> Kobe University Hospital Ikuta, Kobe Japan 650
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Ministry of Education Japan	
<u>Citation</u> Ando, Y. and H. Matorri. Effects of noise on human placental lactogen (HPL) levels in maternal plasma. Brit. J. Obstet. Gynaecol. 84(2):115-118, Feb. 1977		
<u># of Ref.'s</u> 7	<u># of Fig.'s</u> 5	<u>Language</u> English
<u>Type & duration of experiment</u> short-term epidemiological survey using blood samples taken at same times from both test and control groups	<u>Purpose for study</u> to test for significant differences in maternal HPL levels in women living in noisy or quiet areas	
<u>Description of test groups (subjects, #, age, etc.)</u> 2 groups: (1) tests-- in the Itami district near Osaka International Airport (noise area)--343 randomly selected pregnant women 22-41 weeks gestation (2) controls-- Kobe district (little aircraft noise)--112 randomly selected pregnant women, 22-41 weeks gestation		
<u>Control of other stressors</u> environmental conditions similar for test & control areas (air pollution, pop. density, weather, traffic, standard of living)	<u>Statistical Methods</u> chi-square two-sided test	
<u>Noise Stimulus</u> source: aircraft noise (people tested who lived near a large airport) spectral characteristics: not given noise level: WECPNL (weighted equiv. continu- ous perceived noise level)=75-95 length of exposure: variable <u># of trials</u> : 1 sampling per subject	<u>CVS Response Measured</u> none <u>Nonauditory effects</u> Human placental lactogen (HPL) levels measured in maternal plasma in both noisy and quiet groups; HPL levels lower after 30th week of pregnancy in noise-exposed groups; significant difference after 36th week.	
<u>Author's conclusions</u> Human placental lactogen (HPL) levels of subjects in the high aircraft noise area were lower than those in the control area after the 30th week of gestation. The percentage of HPL levels in the fetal danger range (4 micrograms/ml or less) was higher in the noise area. Lower birth weights of babies were associated with lower HPL levels in the noise area.		
<u>Evaluation & comments</u> (1) interesting implications for potential hazards of noise on human reproduction (2) It was not clear from the article whether HPL levels were tested in serum or plasma, since the terms were used interchangeably.		

EFFECTS OF NOISE ON HUMAN PLACENTAL LACTOGEN (HPL) LEVELS IN MATERNAL PLASMA

BY

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Summary

The levels of human placental lactogen (HPL) in the serum of expectant mothers both subjected to and not subjected to aircraft noise were measured. The HPL levels in the quiet reference area and in the noise area were similar before the 29th week of pregnancy. However, the HPL levels of subjects in the noise area tended to be lower than those in the reference area after the 30th week of pregnancy and the difference became significant after the 36th week of pregnancy. The lower HPL levels were associated with lower birth weight for infants of mothers who lived in the noise area.

The manner in which babies react to aircraft noise during their sleep as a function of the length of stay in a noisy area has been investigated by a statistical survey (Ando and Hattori, 1970, 1973 and 1974). It was found that babies born to mothers who moved to the noise area before or during the first five months of pregnancy showed little or no reaction to the noise. After the introduction of regular jet plane services, it was observed that the average birth weight of babies in the noise area (which surrounded an international airport) was clearly less than that from other neighbouring quiet areas. The incidence of low birth weight babies increased as the noise level increased. It has been suggested that such noise could be a possible cause of toxæmia of pregnancy (Ando and Hattori, 1974).

We now report HPL levels in maternal serum in a noise area and a quiet reference area.

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METHODS

In the Itami district around the Osaka International Airport (hereafter referred to as the noise area), blood samples were taken from 343 randomly selected expectant mothers, between 22 and 41 weeks gestation, who were attending the Itami City Hospital. In the Kobe district, a district with relatively little aircraft noise (hereafter referred to as the reference area), blood samples were obtained from 112 randomly selected expectant mothers of equivalent gestation who were attending either the Kobe University Hospital or the Kanebo Hospital. Other environmental conditions such as atmosphere pollution, density of population, standard of living, weather and traffic conditions were almost the same for the two areas. The noise area was residential and commercial while the reference area was only commercial and industrial.

One 5 ml sample of venous blood was taken from each subject and refrigerated overnight. On the following day about 2 ml of serum was

separated from each sample and then stored in a deepfreeze. The HPL levels were measured at Kobe University Hospital. The measurements were conducted a total of five times with 50 to 170 samples being processed in one batch.

The measurements were made using the Phadebas HCS Test Kit (Lot No. 1687, 6121 and 5546). As far as possible, samples from mothers in the noise area and mothers in the reference area were measured at the same time. At the time of blood sampling, the names of the mothers, the date of sampling, and the estimated date of delivery were recorded. In the Itami district, the time at which the mothers had moved into the noise area and their current addresses were also recorded. The birth weights of the babies were obtained from hospital records.

RESULTS

Figures 1 and 2 show the HPL levels in mothers in the reference area at varying stages of gestation. The graphs also show the mean ($\pm 2SD$) levels (Lindberg and Nilsson 1973a) and the fetal danger zone (FD Zone) is outlined. It is apparent that most of the HPL levels of mothers in the reference area remained within the normal range, while HPL levels of mothers in the noise area, where the noise levels were in the range of 75 to 95 WECPNL (see

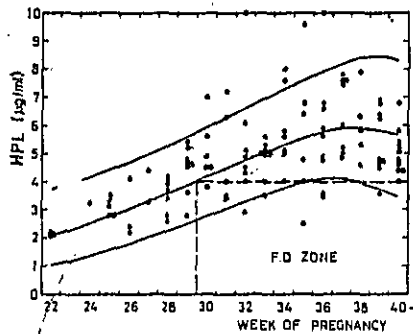


FIG. 1
HPL levels of 112 mothers in the reference area. The three lines give the mean $\pm 2SD$ values for normal HPL levels from Lindberg and Nilsson (1973a).

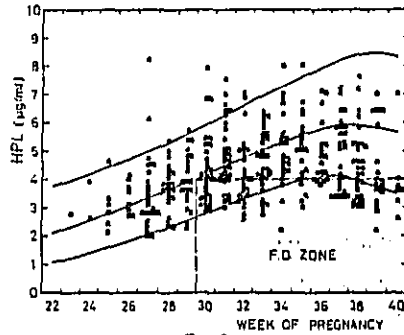


FIG. 2
HPL levels of 343 mothers in the noise area. The three lines show the mean $\pm 2SD$ values for normal HPL levels from Lindberg and Nilsson (1973a).

Appendix), were lower, particularly after the 30th week of pregnancy.

The total number of subjects and the percentage of subjects whose HPL levels were more than 1SD below the mean, from both the noise area and the reference area, are shown in Table I.

The incidence of HPL levels more than 1SD below the mean at various stages of pregnancy is shown in Figure 3. Table I and Figure 3 show that after the 32nd week of pregnancy, the HPL levels of more than 40 per cent of the subjects in the noise area had fallen to more than 1SD below the mean and that this percentage was higher for subjects at later stages of pregnancy. Significant differences were obtained after the 36th week of pregnancy (Chi square two-sided test; $P < 0.025$).

The correlation between the HPL level measured during the 30th to 40th weeks of pregnancy and the birth weight of the babies in the noise area was also examined. The mean ($\pm 1SD$) birth weight of babies born to mothers whose HPL levels were 6 $\mu\text{g/ml}$ or above was 3307 ± 359 g (39 subjects). On the other hand, the mean ($\pm 1SD$) birth weight of babies born to mothers with HPL levels of 4 $\mu\text{g/ml}$ or less was 2945 ± 263 g (11 subjects). In these two groups, the incidence of birth weights below 3.0 kg was 73 per cent (HPL levels 6 $\mu\text{g/ml}$ or above and 73 per cent (HPL levels 4 $\mu\text{g/ml}$ or

TABLE I
Percentage of subjects whose HPL levels were more than 1SD below the means

Gestation (weeks)	No. of subjects in reference area			No. of subjects in noise area			P value for difference
	Total	With HPL levels more than 1SD below mean	Rate (per cent)	Total	With HPL levels more than 1SD below mean	Rate (per cent)	
22-23	3	0	0	1	0	0	NS
24-25	6	0	0	8	1	13	NS
26-27	5	2	40	30	5	17	NS
28-29	15	3	20	43	8	18	NS
30-31	11	2	18	62	15	24	NS
32-33	16	4	25	56	27	48	<0.050
34-35	13	3	23	43	17	40	NS
36-37	17	4	25	46	24	52	<0.005
38-39	14	4	29	47	32	68	<0.010
40-41	12	3	25	7	6	86	<0.025
Total	112	25	22	343	135	39	

TABLE II
Percentage of subjects categorised according to HPL level

Category	Reference area		Noise-area	
	Number of Subjects	Rate (per cent)	Number of Subjects	Rate (per cent)
(a) 30th to 33th week of pregnancy				
4 µg/ml or less	40	28	160	36
Below the lower range of normal HPL level (~2SD)	40	8	160	13
6 µg/ml or more	40	25	160	17
(b) 36th to 41st week of pregnancy				
4 µg/ml or less	43	9	100	45
Below the lower range of normal HPL level (~2SD)	43	9	100	45
6 µg/ml or more	43	35	100	18

All results obtained at 36 to 41 weeks were significantly different ($p < 0.01$)

less) respectively; the difference was significant ($P < 0.005$).

DISCUSSION

This study shows that the HPL levels of subjects in the noise area tended to be lower than those in the reference area. The difference was particularly distinctive for the HPL levels measured after the 36th week of pregnancy. Furthermore, the percentage of mothers with

HPL levels of 4 µg/ml or less, a measure of fetal danger, tended to be higher in the noise area than in the reference area.

We also confirmed that the birth weight of babies born to mothers with low HPL levels tended to be low (Letchworth *et al.*, 1971; Spellacy *et al.*, 1971; Lindberg and Nilsson, 1973b). The decrease of the HPL level was associated with a lower birth weight for babies whose mothers lived in the noise area and this

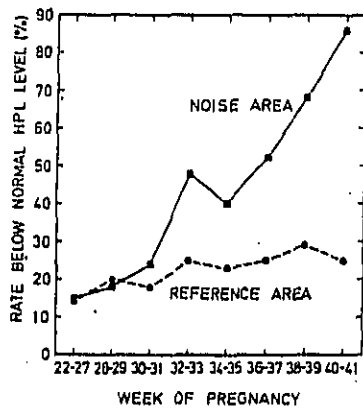


FIG. 3

The percentage of subjects with HPL levels more than 1SD below the mean by stage of pregnancy (see Table I).

confirmed previous results (Ando and Hattori, 1973 and 1974).

ACKNOWLEDGEMENTS

The authors are grateful to Dr Hirai of the Kanebo Hospital in Kobe for taking blood samples and measuring HPL levels and to Dr Kashima of the Itami City Hospital for taking blood samples. They thank Dr Mochizuki and Dr Morikawa, Faculty of Medicine, Kobe University for helpful discussions; the patients for their co-operation and M. Shobntake for help with translation. This research was supported by the Ministry of Education, Japan.

APPENDIX

The weighted equivalent continuous perceived noise level (WECPNL) is calculated as follows.

$$\text{WECPNL} = \overline{\text{dB}(A)} + 10 \log N - 27,$$

where $\overline{\text{dB}(A)}$ is the average value of peak noise level in dB(A) and N is the number of flights in a day, i.e. $N = N_1 + 3N_2 + 10N_3$, N_1 , N_2 , and N_3 are the number of flights between 0700 and 1859 hours, 1900 and 2159 hours and 2200 and 0659 hours respectively. WECPNL was nearly equal to 2+ECPNL; for the distribution of noise level in ECPNL (see Ando and Hattori, 1973).

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SECTION 23

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Edwin Boyle, M.D. (The Medical Univ. of S. Carolina, Dept. of Medicine, 80 Barre St., Charleston, S.C. 29041) Phillip A. Villanueva		<u>Institution and address where research was performed</u> Miami Heart Institute Research Division 4701 N. Meridian Ave. Miami Beach, Fla. 33140	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Miami Heart Institute		
<u>Citation</u> Boyle, E. and P.A. Villanueva. Hyperbaric oxygen seizures in rats: effects of handling and chamber noise. Laboratory Animal Science 26 (1): 100-101, Feb., 1976			
<u># of Ref.'s</u> 8	<u># of Fig.'s</u> 0	<u>Language</u> English	
<u>Type & duration of experiment</u> Incidence of seizures with various handling procedures and hyperbaric oxygen flow rates for laboratory rats were compared.		<u>Purpose for study</u> To test whether noise and handling of animals were significant stressors contributing to HBO-induced seizures.	
<u>Description of test groups (subjects, #, age, etc.)</u> <u>Test rats:</u> hyperbaric air of 5000 ml/sec which was reduced to 30 ml/sec and normal rough handling; 30 ml/sec air flow and gentle handling <u>Control rats:</u> normobaric air in similar conditions as tests			
<u>Control of other stressors</u> controlled laboratory conditions		<u>Statistical Methods</u> not given--mostly a qualitative study	
<u>Noise Stimulus</u> <u>source:</u> flow of oxygen in HBO chamber (loud hiss) <u>spectral characteristics:</u> not known <u>noise level:</u> varied with flow rate of oxygen <u>length of exposure:</u> 60 min. <u># of trials:</u> not given specifically, but at least 35 treatments of noise & handling methods		<u>CVS Response Measured</u> NONE <u>Nonauditory effects:</u> Controls--no seizures. Seizures decreased to 1/3 the original number in tests after the oxygen flow was reduced; after gentler treatment of the rats was used, the seizures ceased altogether.	
<u>Author's conclusions</u> A combination of rough handling and loud gas flow noise in a hyperbaric oxygen chamber is a significant contributor to hyperbaric oxygen (HBO)-induced seizures in rats. The seizures may be mediated by an adrenocortical mechanism.			

Evaluation & comments
(1) This study may be mostly of use to laboratory animal caretakers; (2) shows another aspect of noise as a stressor (3) effects of noise and handling were not studied separately.

(11). The W1/Ten rat should be considered for studies on sarcomatous growth and tumor transplantation. Inbreeding of tumor-bearing rats might lead to a line of animals with increased incidence of spontaneous bone sarcomas.

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³ The authors thank Ms Cynthia Smith for her technical assistance.

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⁵ Reprint requests to Dr Machado at the University of Tennessee Memorial Research Center, Alcoa Highway, Knoxville, TN 37920.

⁶ Wayne Lab Biosci., Allied Mills, Inc., Chicago, Ill.

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HYPERBARIC OXYGEN SEIZURES IN RATS: EFFECTS OF HANDLING AND CHAMBER NOISE^{1,2}

EDWIN BOYLE AND PHILIP A. VILLANUEVA

The convulsive effects of hyperbaric oxygen (HBO) were first reported nearly a century ago (1) and have been reported often enough to be considered a true complication of exposure to oxygen under high pressure. Susceptibility of animals, and of rats in particular, to HBO-induced seizures and to oxygen toxicity in general may depend upon such factors as age (2), strain of rat (3), and temperature (4).

Due to these seizures, other effects of HBO have become difficult to analyze. Part of our group's study of the effect of HBO upon senile cognitive function in humans has been an examination of the effects of HBO upon neurotransmitters in rat brain. However, previous studies of the effects of HBO upon neurotransmitters in rats and mice have reported oxygen-induced seizures (5,6).

In one of our early studies, using male rats (Carr: COBS (W1) BR) from the Charles River Labs ranging in age from 24-26 mo of age, we noted frequent seizures when the animals were exposed to HBO at 30 psig. The animals were exposed for a total of 60 min (15 min for both compression and decompression, allowing a sustained 30 psig exposure of 30 min). Control rats exposed to normobaric air in similar chambers and at the same schedules showed no such convulsions. After 5 exposures we noted that the flow of oxygen made a loud hissing noise in the HBO chamber. The flow rate could not be precisely calculated with the available equipment but was approximately 10 cu ft/min, or approximately 5000 ml/sec. The flow of oxygen was then reduced to 30 ml/sec. The occurrence of seizures persisted in the test animals but declined to approximately one-third their previous

number. At the end of 20 treatments animal handlers were changed. The original handlers had treated the animals in what would be considered a normal manner, ie, lifting them by the tails to transfer them to and from the chambers and cages. The new technicians, in contrast, were more gentle in their treatment of the rats and especially avoided dangling the animals by their tails. Convulsions then declined steadily and by the thirty-fifth treatment no rats convulsed.

Noting the extinction of seizures, we adopted the following protocol for the exposure of test and control rats:

- 1) All animals, test and control, received human contact for at least 1 wk prior to exposure. This contact consisted of gentle lifting, petting, and sham transfer to and from chambers and cages.
- 2) For HBO test animals, 15 min were allowed for both compression and decompression, and 30 min for exposure at 30 psig.
- 3) In both oxygen and air chambers, gas flow was adjusted to 30 ml/sec.

Using this revised schedule of handling in over 500 subsequent exposures, we have not had a single convulsion among our test or control populations, which include over 150 rats, ranging in age from 6-26 mo. This would indicate that both chamber noise and rough handling may contribute significantly to HBO-induced seizures in rats. It has been shown that hyperbaric oxygen may induce susceptibility to autogenic seizures, and that rough handling—specifically lifting by their tails—has also been noted to induce convulsions in mice treated with certain CNS drugs (7). The pathophysiology of these seizures is unknown, but they may be mediated by an adrenocortical mechanism which could be triggered by the stress of rough handling or by the loud rushing sound of the gas flow. Such an adrenal mechanism has been pos-

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olated as a possible mediator of HBO-induced seizures, as adrenalectomy results in a marked attenuation of this activity (8).

Pre-exposure handling and gentle handling during treatment may eliminate one of the possible causes of seizure activity, and a reduced noise level in the chamber may reduce the risk of the other. We believe that the absence of convulsive activity since the adoption of our handling/exposure protocol bears out the efficacy of these measures and supports the possibility of HBO chamber noise and rough handling potentiating each others' epileptogenic activities.

In the light of these findings, the present literature dealing with the central nervous system toxicity of HBO may require re-evaluation.

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treatments animal handlers had at would be considered by them by the falls to the chambers and cages. (that, were more gentle and especially avoid their falls. Convulsions by the thirty-fifth treat-

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le of handling in over we have not had a test or control population of 150 rats, ranging in age would indicate that handling may contribute to seizures in rats. If hyperbaric oxygen may induce tonic seizures, and that lifting by their tails or convulsions in mice (page 7). The pathogenesis is unknown, but the theoretical mechanism of the stress of rough handling sound of the gas variation has been proposed.

SECTION 24

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Robert W. Cantrell Captain, Medical Corps, U.S. Navy Chairman, Dept. of Otolaryngology Naval Regional Med. Ctr.; San Diego, CA 92134	<u>Institution and address where research was performed</u> NA
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u>
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Citation
 Cantrell, R. W. Physiological effects of noise. In AGARD (Advisory Group Aerospace Res. Dev.) Conf. Proc. 171: C11-1--C11-11, 1975

<u># of Ref.'s</u> 31	<u># of Fig.'s</u> 7	<u>Language</u> English
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<u>Type & duration of experiment</u> NA	<u>Purpose for study</u> Review article on the physiological effects of noise (non-auditory)
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Description of test groups (subjects, #, age, etc.)
 NA

<u>Control of other stressors</u> NA	<u>Statistical Methods</u> NA
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<u>Noise Stimulus</u> NA source:	<u>CVS Response Measured</u> NA
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<u>spectral characteristics:</u> <u>noise level:</u> <u>length of exposure:</u> <u># of trials:</u>	<u>Non-auditory effects</u> peripheral vasoconstriction and other circulatory changes changes in gastrointestinal motility change in Galvanic Skin Response increased corticosterone levels increased cholesterol levels increased fatal resportions and birth defects increased catecholamine excretion
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Author's conclusions
 The physiological effects of noise are more dangerous than is generally believed. More study is needed.

Evaluation & comments | A very informative review of some of the major areas of the non-auditory physiological effects of noise; includes a table listing the functions of the autonomic nervous system (sympathetic and parasympathetic nerves) which is considered the mediator of physiological responses to noise. Theoretical mechanisms involved in the stress reaction to noise are discussed.

PHYSIOLOGICAL EFFECTS OF NOISE

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N76-17797

SUMMARY

It is well known that noise adversely affects mankind. Many studies have been performed which show the effects of noise on hearing, speech reception, sleep, mental health and work performance. Until recently, relatively little had been written on the physiological effects of noise. Studies which have been reported are generally retrospective on a group of people working in a noisy environment where precise controls over the intensity of the noise and the duration of exposure were lacking. Recent studies show that the effect of noise on the biochemistry of the body, the cardiovascular system, and the organ systems controlled by the autonomic nervous system are more serious than previously suspected.

Noise serves as a stressful stimulus which provokes the General Adaptation Syndrome as described by Selye. Noise is one of the several stressful stimuli which activate this syndrome via the hypothalamus to the pituitary which produces ACTH resulting in increased adrenocortical activity. There is considerable evidence to support this concept, and this theory along with recently carefully controlled studies, are reviewed.

INTRODUCTION

Noise has been recognized as harmful to man for at least 2500 years. The Sybarites of Greece banned metalwork involving hammering within the city limits as early as 600 B.C.¹ As industrialization has increased along with population density, noise levels have risen to a point where they have become a serious public health problem.

The effects of noise on hearing, annoyance, sleep and speech interference, and work performance have been studied extensively. Until recently relatively little has been written on the physiological effects of noise. Most of the work has been done as retrospective analysis on groups of people working in a noisy environment where precise measures of the intensity of the noise and the duration of exposure were lacking. In the studies, it was not always possible to separate the effects of heat, light, dust, noxious fumes or other environmental pollutants from the effects of noise alone.

Recent studies show that the effect of noise on the biochemistry of the body, the cardiovascular system, and the organ systems controlled by the autonomic nervous system are more serious than previously suspected.

In Germany, Jansen², Lehmann³, and Meyer-Delius⁴; in Russia, Andriukin⁵, Andrukovitch⁶ and Shatalov⁷ among others; and in the U.S. Davis⁸, Rosen⁹ and more recently Cantrell¹⁰, have studied the physiological effects of noise exposure. A symposium held in Boston in 1969 was devoted to this subject, and resulted in a book¹¹ which detailed much of the knowledge available at that time.

PATHWAYS

A pathway from the sound source to the target organ must be established in order to show a cause-and-effect relationship. For the purposes of this discussion, infra-sound (below 20 Hz) and ultra-sound (above 20,000 Hz) will not be considered. Nor will the possible physiological effects of vibratory energy on the body in general be considered. There is evidence that vibratory energy can affect the body; that the vibrations transmitted through structures is more significant than airborne transmissions; and that vibrations (sound) from 20 to 20,000 Hz may exert a physiological effect in ways other than through the auditory mechanism, i.e. through the skin. These studies are scarce and since the most damaging airborne vibratory (noise) effects are transmitted through the ear with its central auditory connections, it is those physiological effects which will be considered here.

Once sound enters the auditory canal, it causes the tympanic membrane to vibrate. This in turn moves the three ossicles which at the oval window create a wave in the inner ear fluids by the piston-like action of the footplate of the stapes. The relatively large area of the tympanic membrane compared with the small area of the stapedial footplate (17:1) plus a small lever advantage from the ossicles, transforms the small pressure of sound energy impinging on the tympanic membrane into a 22-fold greater force acting on the inner ear fluid. The fluid wave thus created distorts the basilar membrane and the hair cells of the organ of Corti are stimulated. Nervous impulses generated in the organ of Corti travel along the auditory neurons to the central auditory nuclei.

Sound is also transmitted through the bones of the skull directly to the inner ear, and we measure these two pathways (air conducted and bone conducted sound) to help diagnose hearing disorders. One who cannot hear air conducted sound but can hear by

bone conduction has a conductive hearing loss which in most cases can be remedied by appropriate medical or surgical treatment. If one has difficulty hearing both air and bone conducted sound, this is known as nerve deafness which is not correctable, and sound must be amplified in order for the patient to hear.

After stimulating the auditory nerve, the sound waves, which are now nervous impulses, travel to the central auditory nuclei in the medulla, where some fibers ascend through the midbrain via the lateral lemnisci on the same side, but more cross before ascending on the opposite side through the midbrain to reach the inferior colliculus, then the medial geniculate body and finally the auditory area of the temporal lobe where the sound is interpreted.

It is probable that after reaching the central auditory nuclei, impulses travel through the reticular formation to reach the hypothalamic nuclei. From the hypothalamic nuclei which are situated just superior to the pituitary, the products of stimulation travel to the pituitary which then produces endocrine effects and completes the auditory-hypothalamic-pituitary-endocrine pathway.

The hypothalamus is not the only part of the brain directing autonomic activity. The forebrain, the thalamus and the cerebral cortex are all integrated with the hypothalamus to utilize behavioral and autonomic adjustments which serve to adapt the individual to changes in both the internal and external environment.

AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system (ANS), also known as the vegetative nervous system, is a system of motor neurons whose cell bodies are collected into ganglionated chains in the thoracic region near the vertebral column and in isolated ganglions elsewhere in the body. Anatomically the A.N.S. is divided into the thoracolumbar (sympathetic) and cranio-sacral (parasympathetic) division. This system is generally not under voluntary control.

Table I lists the functions of the autonomic nervous system, which acts to maintain the constancy (homeostasis) of the fluid environment (internal milieu) of the body. The autonomic nervous system combats forces which tend to cause variations in this environment. It regulates the composition of body fluids, their temperature, quantity and distribution by effecting changes in circulatory, respiratory, excretory and glandular organs.

TABLE I
AUTONOMIC NERVOUS SYSTEM FUNCTION

SYSTEM	PARASYMPATHETIC (CHOLINERGIC)	SYMPATHETIC (ADRENERGIC)
<u>Eye (Pupil)</u>	Constricted	Dilated
<u>Heart Rate</u>	Decreased	Increased
<u>Blood Vessels</u>		
Coronary	Dilated	Dilated
Skin & Mucosa	Dilated	Constricted; Dilated
Skeletal Muscle	Dilated	Dilated; Constricted
Cerebral	Dilated	Constricted (?)
Pulmonary	Dilated	Constricted
Abdominal Viscera	Dilated	Constricted
<u>Bronchi</u>	Constricted	Dilated
<u>Glands</u>		
Sweat	Stimulated	Stimulated
Salivary	Stimulated (Thick)	Stimulated (Watery)
Gastric	Stimulated	Inhibited
Adrenal		Stimulated
<u>Smooth Muscle</u>		
Skin (Pilomotor)		Contracted
Stomach	Increased Tone	Decreased Tone
Small & Large Intestine	Increased Tone	Decreased Tone
Bladder	Contraction	Inhibition
General	Restorative	Energizing
<u>Mediator</u>	Acetylcholine	Sympathin Epinephrine Norepinephrine

The two divisions are antagonistic: one slows, the other speeds the cardiac rate; one constricts, the other dilates the pupil or the bronchi. Generally, the sympathetic strengthens the defense against such challenges as enemy attacks, temperature variations, and water deprivation. Animals who have had sympathectomies are incapable of working (no sugar is mobilized); cannot withstand exposure to temperature extremes (no sweating when hot; no vasoconstriction when cold) and they are less able than normals to withstand oxygen deprivation or hemorrhage. They can survive in a controlled environment.

The parasympathetic system is concerned with restoration of the body processes. It inhibits the heart rate, contracts the pupils and stimulates the digestive tract through which the energy stores of the body are replenished. It is primarily in control while one is sleeping.

DISCUSSION

Selye¹² described the General Adaptation Syndrome. According to this concept, an individual exposed to stress - cold, heat, drug reaction, fractures, infections, operations, burns or other trauma (NOISE) - responds by:

1. Stimulation of the hypothalamus which
2. Stimulates the anterior pituitary to release ACTH which
3. Stimulates the adrenal cortex to release cortisol which
4. Stimulates the body to protect against systemic anabolism of tissue.

This theory is well accepted even if there is not complete agreement as to the actual mechanism of action. Stress is known to be a factor in the development of such diseases as peptic ulcers, cardiovascular disease, including hypertension and coronary artery disease, and it is implicated in the aging process.

Noise, especially aversive, intrusive noise, is thought to be merely one of many agents which serves as a stress-provoking stimulus. Noise stimulates the sympathetic portion of the A.N.S. As such it should be minimized just as noxious fumes, excessive heat or cold or, indeed, even marked population density, should be diminished and controlled where possible in the environment.

Assuming this to be true, what evidence do we have that noise has any effect on these functions?

Mason¹³ reported the electrical stimulation of the hypothalamus of conscious Rhesus monkeys was associated with an increase in pituitary-adreno-cortical activity, as judged by the marked elevation of plasma 17-hydroxycorticosteroid.

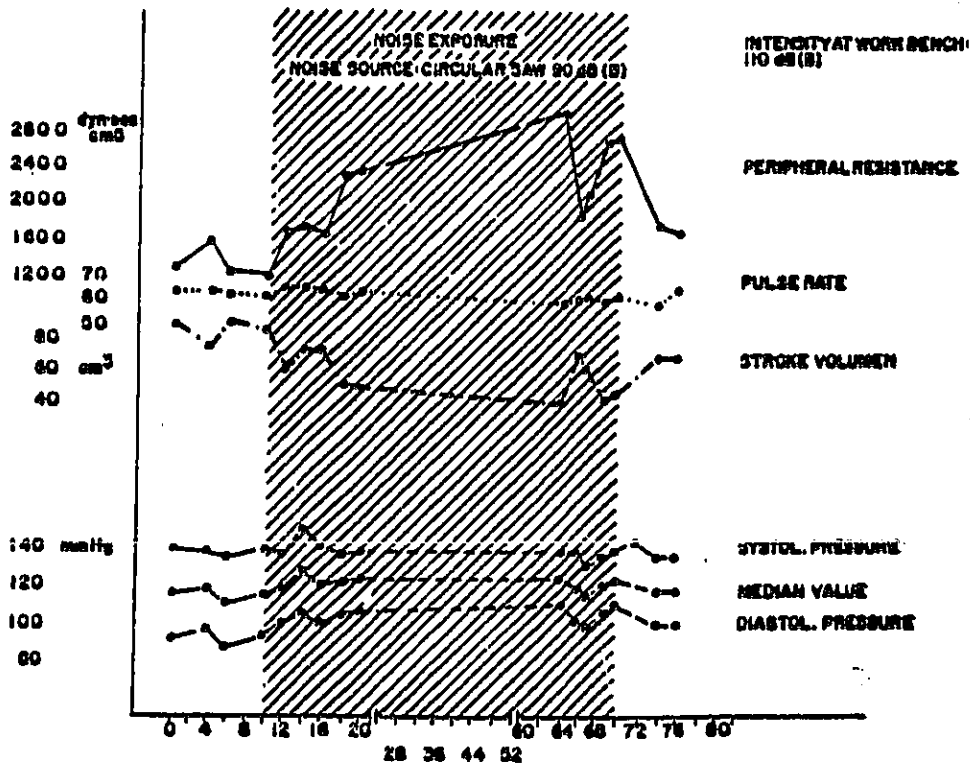


FIG. 1. Circulatory reaction during noise exposure in one subject employed in a noisy factory. (From Lehmann and Tamm. Forsch.-Ber. Wirtsch.-u. Verkehrsmin. NRW 517 [Kohn-Oplader, 1958]. Cited by G. Jansen, Transl. Del. Inst. Hear. Res. No. 26, 1972. With permission).

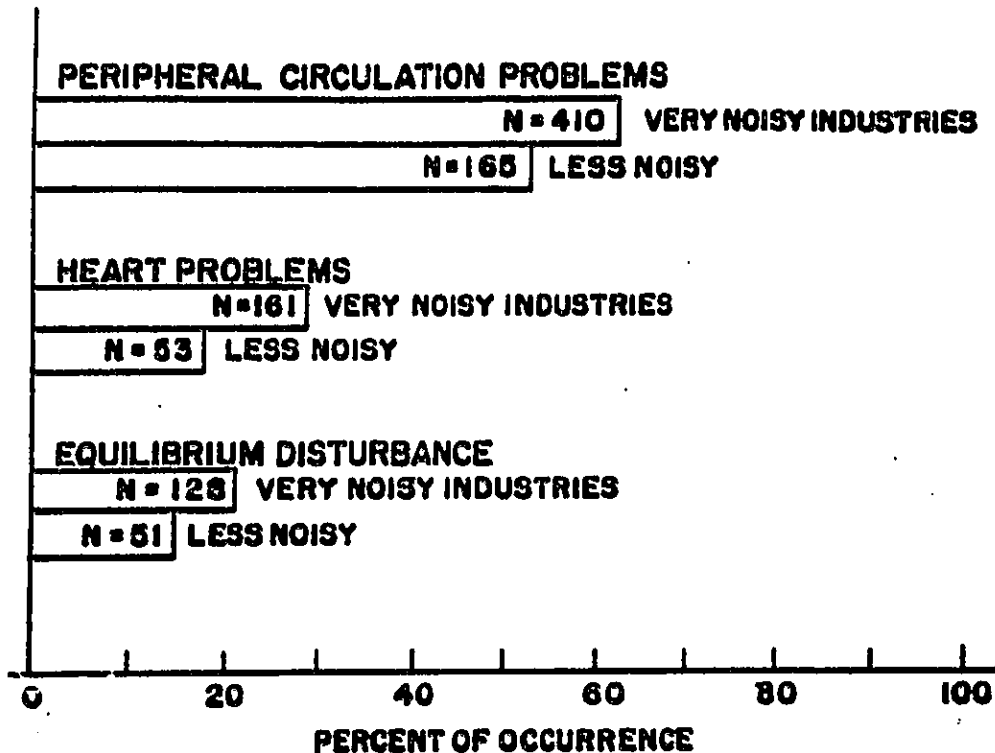


FIG. 2. Differences in percentages of occurrence of physiological problems in 1,005 German industrial workers. The differences in peripheral circulation and heart problems in the two classes of industry were statistically significant. (From G. Jansen, Stahl, Eisen, 81, 1961, pp. 217-220. With permission of the author and Stahl, Eisen.)

Henkin and Knigge¹⁴ exposed rats to 130 dB at 220 Hz for 48 hours and measured adrenal secretion of corticosterone. It was found that the output of adrenal corticosterone doubled in 30 minutes and tripled in 60 minutes. This tripled excretion rate was maintained for approximately 12 hours at which time it fell to normal or subnormal levels only to rise again to the former high rate where it was maintained for 25 to 48 hours.

Friedman, et al¹⁵, exposed rabbits to 102 dB of white noise for 10 weeks and showed a much higher level of blood cholesterol than non-exposed animals despite being on identical diets. The animals exposed to noise developed more aortic atherosclerosis and more cholesterol deposits in the iris than the control animals.

Geber¹⁶ exposed gravid female rats to noise intensities of 74-94 db from 20 to 2,500 Hz for 6 minutes of each hour followed by 54 minutes of quiet (ambient noise level was 64 db) and to a flashing light for the same period of time, throughout each day of pregnancy or to some other desired day (i.e. 16-20 days).

He found:

1. Total litter resorption occurred in 40-50 percent of the pregnancies.
2. Increased congenital anomalies, including meningoceles, spinal bifida, cranial hematomas, abdominal hernias, and defects of the eye, tail, hind- and forefoot.

Geber and Anders¹⁷ studied the effects of chronic intermittent noise stress on the body weight and the weight of the ventricles, adrenal glands, kidneys, and ovaries of young and old rats and rabbits. Significantly hypertrophied ventricles of both species were found at the end of three weeks' stress. Body and other organ weights were slightly decreased, with the exception of the adrenals and kidneys of the older rats which were increased.

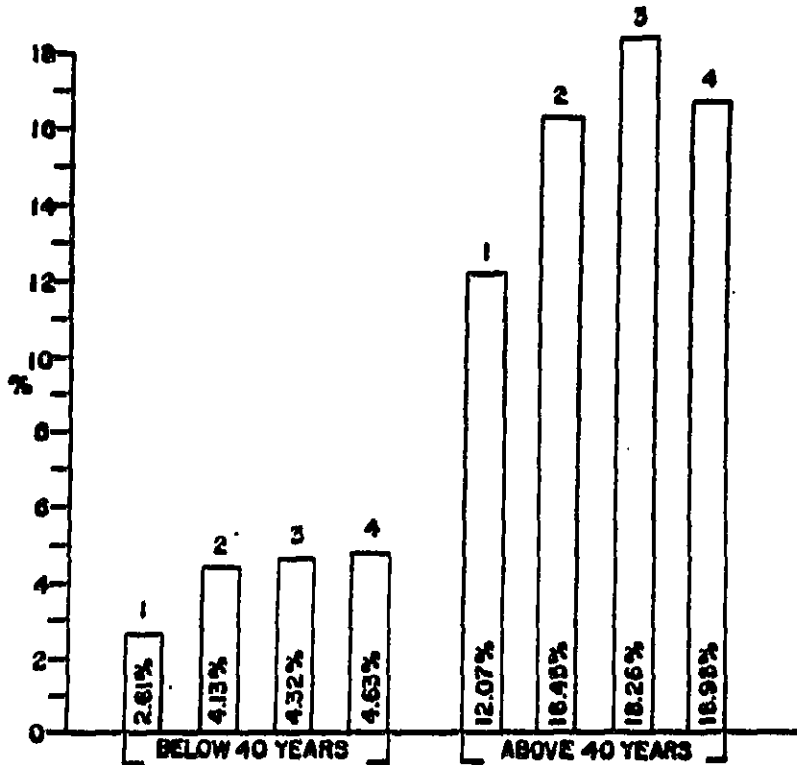


FIG. 3. Incidence of hypertension in male and female workers (in age groups under and above 40 years) in noisy workshops: 1. tool making workshop; 2. sorting workshop; 3. workshop with automatic lathes; and 4. workshop producing ball bearings. (From A.A.Andriukin, Cor.Vassa., 1961, pp.289-293. With permission).

Similar evidence is available in humans. Davis, et al¹⁸ labeled the following set of responses to noise the N-responses:

1. A vascular response characterized by peripheral vasoconstriction, minor changes in heart rate, and increased cerebral blood flow since cerebral vessels show no vasoconstriction to such stimuli.
2. Slow, deep breathing.
3. A change in the resistance of the skin to electricity (Galvanic skin response [G.S.R.]).
4. A brief change in skeletal muscle tension.

To this set of responses can be added:

1. Changes in gastrointestinal motility.
2. Chemical changes in blood and urine from endocrine glandular stimulation.

A tone of approximately 70 dB SPL at 1,000 Hz will elicit the N-response. This same tone continued for a long enough time may induce TTS or NIPTS and is near the level at which broadband noise may become significantly adverse to people¹⁹.

Davis and Berry²⁰ and Stern²¹ found that humans who could avoid an 80 dB, 10-second 800 Hz tone by pushing a switch at the correct time, exhibited greater gastro-intestinal motility during the tone (i.e. when they failed to press the switch) than did subjects who had no means of avoiding the tones. Kryter¹⁹ labeled this a response-contingent effect of noise. The noise thus became an aversive stimulus, primarily because it

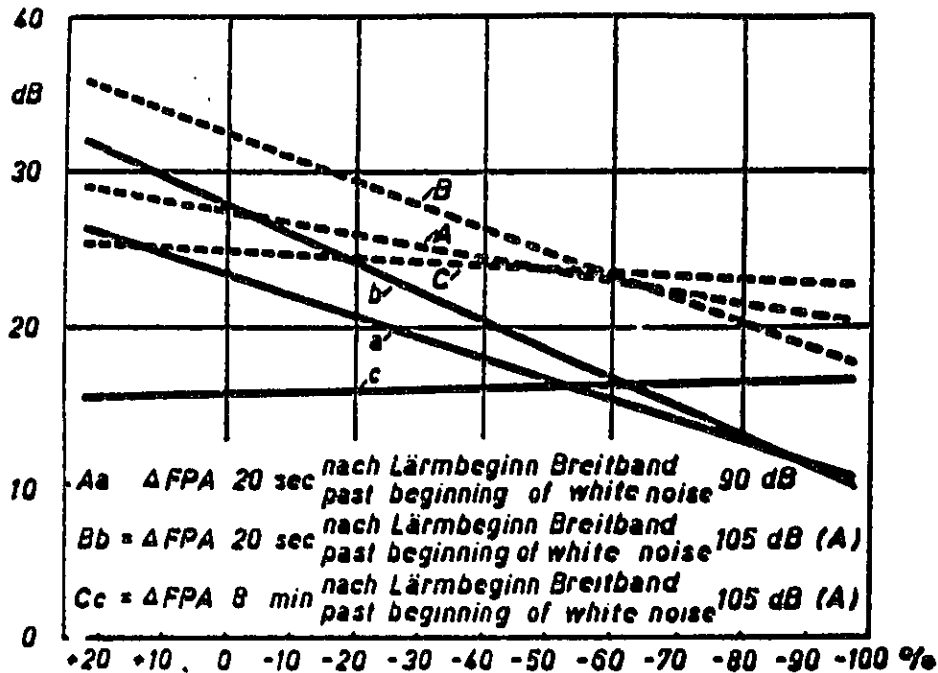


FIG. 4. Finger - pulse - amplitude and TTS. (From Jansen, G. Relation Between Temporary Threshold Shift and Peripheral Circulatory Effects of Sound. Physiol. Effects of Noise, ed. by Bruce L. and Annemarie S. Welch, Friends of Psychiatric Research, Inc., Maryland Psychiatric Research Center and The Johns Hopkins Univ. Sch. Med., Baltimore, Md., Physiol. Effects of Noise, Plenum Press - New York - London, 1970, pp. 67-72. With permission of the authors and Plenum Press).

indicated incorrect responses on the part of the subject; its aversive effect otherwise was small.

Hormann, et al²² in a similar study verified the aversive effects of noise with three groups of subjects exposed to white noise at 95 dB. For Group A, the noise signaled they had made an error in a pseudo-tracking task; for Group B the same noise was the signal that they were on target in the pseudo-tracking test; and for Group C the noise was heard without any task.

Measures were: (a) TTS; (b) muscle tension measured by electromyography; and (c) subjective scaling of the amount of annoyance and disturbance induced by the noise and of the general sensitivity to noise, of the subjects.

The results showed subjects who invest the noise with positive emotional valence, feel themselves less annoyed, less disturbed and, in general, less susceptible to the noise than subjects who receive the noise with negative valence.

Muscle tension was highest for Group A, less for Group B, and least for Group C.

The amount of TTS was dependent upon the valence of the noise:

1. Negative valence (Group A) TTS=18.1 dB.
2. Positive valence (Group B) TTS=12.8 dB.
3. Neutral valence (Group C) TTS=11.0 dB.

The response-contingent effect apparently does not hold for all physiological reactions to noise. Jansen and Klensch²¹ found similar responses in the circulatory system (cardiac output, minute flow volume) in subjects exposed to random noise or music of equal intensity (about 90 Phon). Although the cardiac output and minute flow volume increased in some subjects and decreased in others, indicating an individual difference in

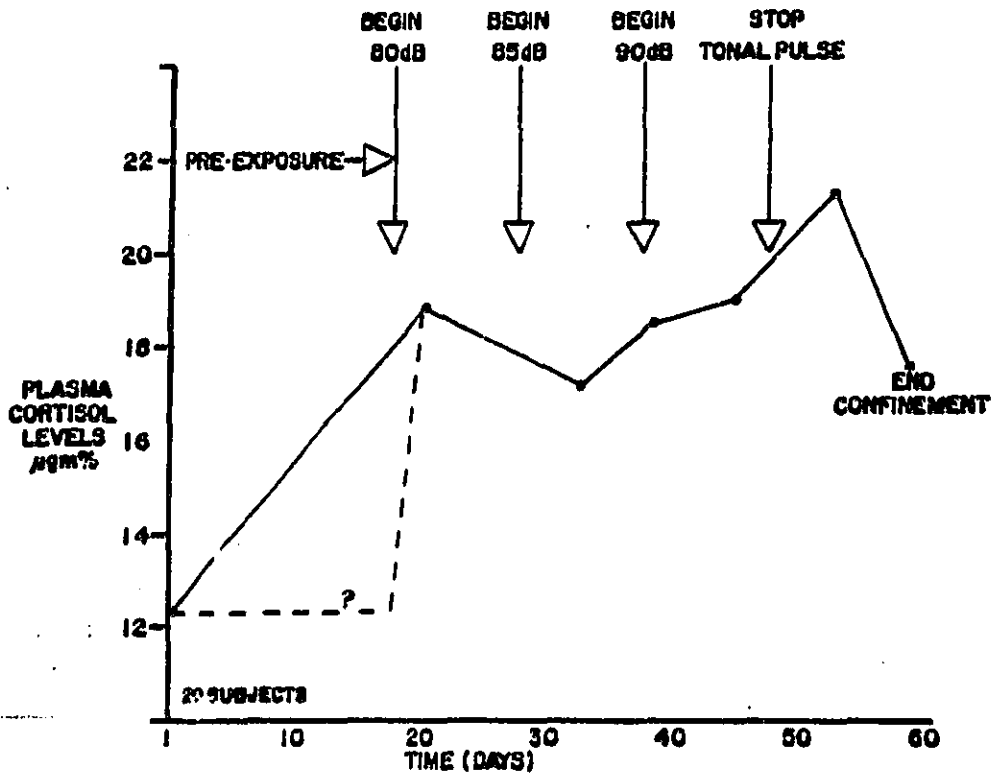


FIG. 5. Mean plasma cortisol levels before, during and after exposure to tonal pulses. A.M. sample. (From Cantrell, R.W.: Prolonged Exposure to Intermittent Noise: Audiometric, Biochemical, Motor, Psychological and Sleep Effects. The Laryngoscope, Suppl. 1, Vol. LXXXIV, No. 10, Part 2, Oct. 1974. With permission).

somatic responses to sound, it was the intensity of the sound and not its aversive (noise) or pleasurable (music) aspect which controlled somatic responses.

Levi²⁴ measured urinary catecholamines as a method of determining *H*-responses in human subjects. He found the following:

1. Pleasant stimuli (motion pictures evoking amusement) were nearly as potent as unpleasant stimuli (motion pictures evoking anger) in causing increased excretion of catecholamines.
2. Work in industrial noise and office work caused increased excretion of catecholamines.
3. Noise, light, or task have less influence on the catecholamine excretion levels than does the subject's attitude.
4. Under experimental stress, emotionally vulnerable people as a group do not excrete more catecholamines than normal people.

Lehmann and Tamm³ studied circulatory changes in human subjects exposed to noise. Peripheral arterial resistance was found to increase under the effect of noise, but pulse frequency and blood pressure remained unaffected. FIG. 1. summarizes the circulatory reactions observed by Lehmann and Tamm.

Meyer-Delius⁴ related the circulatory effects to the duration of noise exposure. An exposure of 90 dB(B) for 20 seconds increased peripheral arterial resistance for 80 seconds, i.e. the vasoconstriction mediated through the autonomic nervous system in response to noise exposure persists after the exposure.

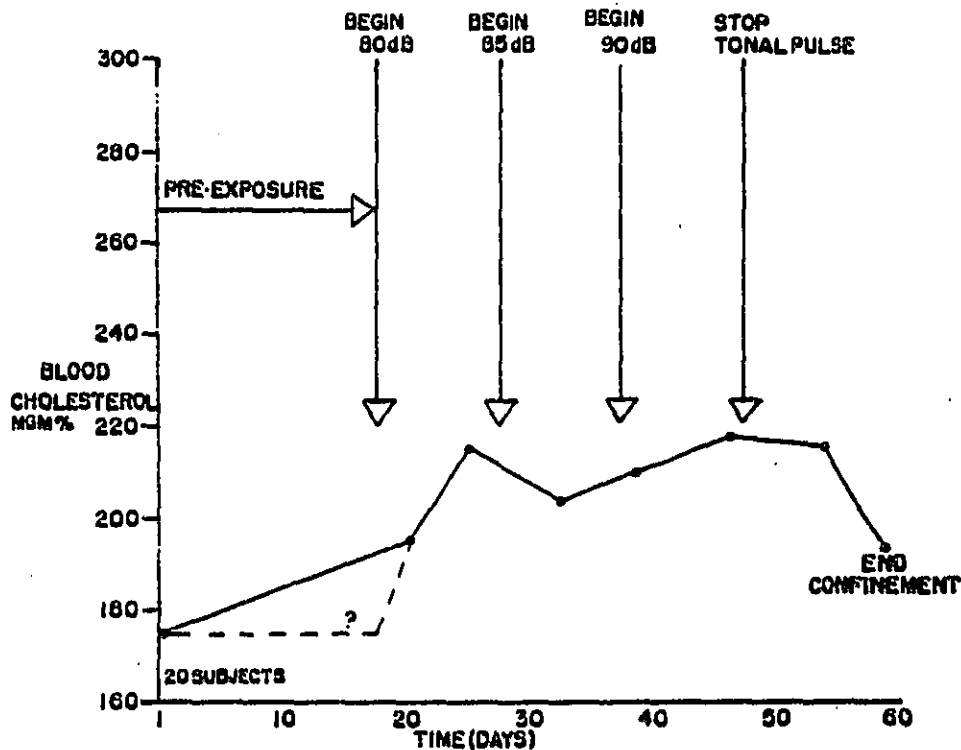


FIG. 6. Mean blood cholesterol levels. (From Cantrell, R.W.: Prolonged Exposure to Intermittent Noises: Audiometric, Biochemical, Motor, Physiological and Sleep effects. The Laryngoscope, Suppl. 1, Vol.LXXIV, No.10, Part 2, Oct. 1974. With permission).

There is a physiological adaptation to sound. Habituation might be a more correct term, but adaptation is used more frequently in the literature when referring to this phenomena. Bartoshuk²⁵ found that acceleration of the heart rate in unborn babies exposed to bursts of acoustic clicks at 85 dB adapted by the end of 40 trials. This adaptation is not complete and obviously does not cover all N-responses. Although man adapts to background noise, he will respond when the character or intensity of the noise is changed. Rossi, et al²⁶, found adaptation of vasoconstriction in subjects exposed to a background noise of 70 dB at 500 Hz did not reduce vasoconstriction to superimposed tones of 80 to 105 dB at 2000 Hz.

Janssen²⁷ plotted the increased numbers of peripheral circulatory problems, heart problems and equilibrium disturbances in German industrial workers in very noisy industries. The differences in peripheral circulation and cardiac problems in the two groups were statistically significant (FIG.2).

Andriukin⁵ showed a greater incidence of hypertension in men and women working in very noisy areas than their counterparts working in less noisy areas (FIG.3). There was also a relationship to age with older people appearing to exhibit more hypertension.

Additional data from Russia^{6,7} has shown that among workers in industries with high noise levels there is an unusually high incidence of circulatory, digestive, metabolic, neurologic and psychiatric problems.

Rosen and co-workers⁹ studied the Mabaans, a primitive tribe living in the Sudan. This tribe has no firearms or drums; their diet consists mainly of vegetables and some fish; and they live in very quiet surroundings (35-40 dB(C)) with relatively little stress. Hearing loss, hypertension and cardiovascular disease is virtually unknown even into old age.

Once the Mabeans move to noisy Kartoum, where they are exposed to noise, stress and a diet similar to city dwellers, they are reported to develop hypertension, coronary artery disease and hearing loss.

Critics of the concept that noise can cause pronounced physiological effects point out that a given noise exposure does not affect all persons similarly, nor does the same individual respond similarly to a given noise exposure occurring at a different time. These are valid observations which complicate the investigation of noise effects. A partially satisfactory answer is that no two humans are alike nor do they respond identically to any stress-stimuli. Normann's study²² of the valence effect of noise (i.e. a desired sound is less damaging than an unwanted sound) is an interesting concept. This may give some insight into why some people are content to work in a noxious stimulus and show little or no effects, whereas others are upset by the same noxious exposure and respond with the symptoms of stress.

A very interesting experiment is reported by Jansen²⁰. He measured the change in finger pulse amplitude 20 seconds after the onset of white noise at 90 dB(A); 20 seconds after the onset of white noise at 105 dB(A); and 8 minutes after the onset of white noise at 105 dB(A). Hearing was measured before exposure and the TTS at 2 minutes after exposure ceased. The TTS was measured at 4000 Hz. FIG. 4 is a graph relating TTS and change in finger pulse amplitude (peripheral vasoconstriction). Note that for short exposures wherein you would expect little or no TTS there was rather greatly decreased FPA. With longer exposures the TTS and change in FPA were similar. Jansen concluded that the vegetative response (VR), as manifested by changes in the finger pulse amplitude, and TTS can be influenced by noise acting through the vegetative system (A.N.S.). Furthermore, a man who will not have a hearing loss from high intensity noise is, nevertheless, highly endangered by the non-auditory physiological effects of high intensity noise.

In studies of our own¹⁰, we noted that even though 20 healthy young subjects showed little (3-6 dB) TTS after 30 days exposure to intermittent noise presented 0.66 seconds every 22 seconds 24 hours per day, they did have statistically significant shifts in plasma cortisol levels (FIG. 5) and blood cholesterol levels (FIG. 6). This noise was in the 3000-4000 Hz range and was presented at 80, 85 and 90 dB(A) each for 10 days. These are allowable levels by many damage risk criteria. This stimulus caused reduction in finger pulse amplitude during sleep and this, coupled with the relatively small TTS, supports Jansen's findings.

The shift in the blood cholesterol and plasma cortisol levels is interesting. Plasma cortisol is known to be influenced by ACTH and other studies^{29,30} have suggested that stress will elevate cholesterol and cortisol. Although controversy exists as to the normal values for serum cholesterol, the range is roughly 150-300 mgmt for all ages. Younger people should normally have lower levels. In our study, the mean age was 20.7 years and accordingly, the upper limit of normal should be 189 mgmt (Keys)³¹ to 240 mgmt (Fredrickson)³². If 189 mgmt is used as the upper limit of normal, all mean cholesterol levels during noise exposure are above normal. Even with the higher limits, all values are statistically significantly elevated from the mean, pre-exposure level, and they begin to decrease after the noise exposure ceases. In this case, the subjects acted as their own controls since all other factors including confinement, diet, and lack of exercise persisted for 10 days after the noise was stopped. Noise exposure was the only variable that changed.

These findings support the concept that the physiological effects of noise are more serious than previously supposed, and more studies of the effects of noise exposure are indicated.

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DISCUSSION

Q. (Johnson) Do you feel that noise that is so low in level that it will not damage the auditory system will cause any non-auditory effects?

A. (Centrell) Perhaps that is a loaded question because, as you know, there is a great deal of work going on to determine a safe level of noise that will not damage the auditory system. Some people think it is 90 dBA, others think it is as low as 70 dBA. If one accepts this later statement of 70 dBA being potentially hazardous, then I think that noise under 70 dBA will not cause any physiological effects. The level of noise at which physiological effects begin, and the seriousness of these effects, is not well-enough measured yet to answer your question. My personal opinion is that somewhere between 75 and 80 dBA, both for hearing and for physiological effects, will turn out to be the critical level. I would like to have Dr. Jensen comment on this question also.

A. (Jansen) One observes vegetative reactions at very low levels of noise. It is only a question what method one uses. For example, electrodermal response, or other sensitive physiological or psychophysiological methods, will show that there are quantitative influences at low noise levels. The question is what is the physiological relevance of these changes? I think the question cannot be answered until now as to where the point is that the normal physiological response is accumulated into a pathological one. This is the question one needs to answer. At the present one has no exact point to state where this begins. It is an increasing continuous augmentation of these reactions.

SECTION 25

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Ronald K. Chesser, Ronald S. Caldwell, and Michael J. Harvey		<u>Institution and address where research was performed</u> Ecological Research Center, Dept. of Biology Memphis State University Memphis, Tenn.	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> same as above		
<u>Citation</u> Chesser, R. E. et al. Effects of noise on feral populations of <i>Mus musculus</i> . <i>Physiol Zool</i> : 48(4):323-325, 1975.			
# of Ref.'s 12	# of Fig.'s 1	Language English	
<u>Type & duration of experiment</u> Part I -- Field experiment Part II -- Laboratory experiment		<u>Purpose for study</u> to see if adrenal size differences exist between noise-exposed and non-noise-exposed populations of wild mice; to test if these differences are due to noise.	
<u>Description of test groups (subjects, #, age, etc.)</u> feral <i>Mus musculus</i> --(wild house mouse). Part I--experimental group--field near aircraft--10 adult females, 32 adult males; control group--from rural field--13 adult males, 13 adult females-- no low-flying aircraft. Part II--from rural field--experimental group--10 adult males, 4 adult females; control group-- 9 adult males, 5 adult females.			
<u>Control of other stressors</u> stress of snap-trapping (which may not kill instantly) was not considered!		<u>Statistical Methods</u> student's t-test	
<u>Noise Stimulus</u> source: I. aircraft; II. recorded aircraft spectral characteristics: not given noise level: I--110 dB avg. aircraft noise II--105 dB length of exposure: 1 min. every 6 min (II) # of trials: Part II--lab. simulation 105 dB--1 minute every 6 minutes for 2 weeks		<u>CVS Response Measured</u> NONE <u>Nonauditory effects</u> adrenal weights--greater in noise-exposed animals than in controls in both field and laboratory conditions	
<u>Author's conclusions</u> Noise was the dominant stressor that contributed to the greater adrenal size in populations of wild mice near Memphis International Airport than in rural mouse populations.			
<u>Evaluation & comments</u> Summary form only			

Physiol. Zool. 48(4): 323-325, 1975.

EFFECTS OF NOISE ON FERAL POPULATIONS OF *MUS MUSCULUS*

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House mice, *Mus musculus*, were snap-trapped from two similar fields near Memphis International Airport, Shelby County, Tennessee, the only apparent difference being presence or absence of low-flying aircraft. Airport field noise levels ranged from 80 to 120 dB while rural field levels ranged from 80 to 85 dB. Mice from the airport field had significantly larger adrenal glands than those of the rural field. To determine if noise was the causative stressful factor, mice collected from the rural field were subjected to recorded jet noises at 105 dB in the laboratory for 2 wk. The experimental group had significantly larger adrenals than those of a control group.

INTRODUCTION

Investigations into the effects of noise on wild populations of animals are few, although laboratory studies are better documented. Fletcher and Harvey (1971) reviewed the literature concerning the effects of noise on wildlife and other animals. Greaves and Rowe (1969) subjected feral *Mus musculus* to noise levels between 85 and 120 dB in the laboratory. High noise levels deterred mice from entering rooms from which noise was emitted, especially when outside food availability was low. Marsh, Jackson, and Beck (1962) and Sprock, Howard, and Jacob (1967) found that noise had no dispersal effect on feral rodents.

Stress does not lend itself to quantification. However, one measurement of stress is adrenal hypertrophy which adapts the animal to stressful situations (Sawin 1969). Adrenal weights were utilized by many previous experimenters (Christian 1955; Davis and Christian 1957; King 1957; Bronson and Eleftherious 1963; Southwick 1964) as an indication of stress. Anthony and Ackerman (1955) subjected laboratory strains of *M. musculus* to 110 dB noise

levels and found slight but insignificant adrenal hypertrophy. However, no previous worker has studied adrenal weights under field situations involving stress. We wished to determine if adrenal weight differences existed in two field populations of *M. musculus*, one submitted to a possible stressful factor of noise. If adrenal differences did exist, we wished to determine if the differences were caused by noise.

MATERIAL AND METHODS

Thirty-two adult male and 10 adult female *Mus musculus* were collected from a field approximately 90 m from the end of a runway at Memphis International Airport, Shelby County, Tennessee. Thirteen adult males and 13 adult females were collected from a rural field 2.0 km from the airport field. These two fields were chosen due to similarity in habitat, close proximity, and dissimilarity in noise levels.

Differential noise levels of the fields were measured with a sound survey meter. Background noise levels of the fields were approximately equal, ranging from 80 to 85 dB. Noise levels of incoming and outgoing aircraft at the

airport field averaged 110 dB with the highest reading at 120 dB. Aircraft noises at the rural field were negligible over background. Mice for the field study were snap-trapped on November 26, 1973. Mice were sexed, weighed, and their adrenal glands removed and weighed in the laboratory. Total body weights were measured on a balance beam to the nearest 0.01 g. Adrenal weights were measured on a semiquantitative balance to the nearest 0.1 mg.

To determine if noise was causative, 28 additional mice were live-trapped January 10, 1974, from the rural field and taken to the laboratory. These mice were distributed into two cages measuring 146 × 98 × 60 cm. The experimental group contained 10 males and four females. The control group contained nine males and five females. After an acclimation period of 10 days, experimental mice were subjected to 1 min of 105 dB recorded jet aircraft noise every 6 min. Control mice were not subjected to jet noise. After 2 wk, adrenals were removed and weighed. Comparisons were made between adult male and female mice. Mice were considered adults if their body weights were 9.5 g or larger (Baker 1946).

Comparisons were made of gross adrenal weights, and differences were tested using Student's *t*-test. No comparison of adrenal weights to body weights was necessary due to insignificant differences in body weights of the different groups ($P < .05$).

RESULTS AND DISCUSSION

Adrenal weights of both male and female mice from the airport field were significantly greater than those from the rural field. In the laboratory study, experimental males and females had significantly greater adrenal weights than control mice (table 1). From these data we conclude that noise was the dominant stressful factor contributing to the adrenal differences between these two feral populations of *Mus musculus*.

In the field, other factors such as population density, air pollution by jet engines, or sight of aircraft may contribute to adrenal hypertrophy. Of the aforementioned, population density would probably effect greater stress. However, mark-recapture analysis indicated population density of the airport field was not greater than that of the rural field.

TABLE 1
MEAN ADRENAL WEIGHTS OF *MUS MUSCULUS* FROM FIELD AND LABORATORY

Sex	N	Adrenal weight (mg) ($\bar{X} \pm SE$)	N	Adrenal weight (mg) ($\bar{X} \pm SE$)
FIELD				
		Airport Field ($\bar{X} = 110$ dB)	Rural Field ($\bar{X} = 82$ dB)	
♂	12	2.5 ± 0.10	13	1.9 ± 0.17
♀	10	4.2 ± 0.17	13	2.6 ± 0.25
LAB CAGE				
		Experimental (with Noise)	Control (without Noise)	
♂	10	3.3 ± 0.11	9	2.4 ± 0.12
♀	4	4.3 ± 0.43	5	2.5 ± 0.18

NOTE.—Adrenals of all mice subjected to high noise levels were significantly different from those of the controls at least at the 95% confidence level.

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Control
(with Noise)

1.9 \pm 0.12
2.5 \pm 0.18

of the controls at

SECTION 26

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> R. D. Fell, C. J. Ellis, D. R. Griffith		<u>Institution and address where research was performed</u> Department of Zoology Iowa State University Ames, Iowa 50011	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u>		
<u>Citation</u> Fell, R. D. et al. Thyroid responses to acoustic stimulation. Environmental Research 12(2):208-213, 1976.			
<u># of Ref.'s</u> 21	<u># of Fig.'s</u> 2	<u>Language</u> English	
<u>Type & duration of experiment</u> Laboratory Experiment - 12 weeks Soundproof room		<u>Purpose for study</u> Since other stressors suppress thyroid activity through release of ACTH, the effects of noise-stress on thyroid responses (iodine uptake and body wt.) were studied for comparison purposes.	
<u>Description of test groups (subjects, age, etc.)</u> 100-150 day old male and female white rats (Sprague-Dawley strain) Test Group 1: exposed to noise for 12 weeks Test Group 2: exposed to noise for 2 weeks Control Group: no noise 12 weeks			
<u>Control of other stressors</u> Laboratory conditions; temperature 80°F; sound level - 68 dBA background		<u>Statistical Methods</u> Standard error	
<u>Noise Stimulus</u> source: single tone from audio-generator spectral characteristics: 1000 Hz, monotonic noise level: 95 dBA length of exposure: 15 minutes # of trials: every 15 min. daily for 8 hrs. each trial: 15 min. noise and 15 min. quiet for 8 hrs. daily		<u>CVS Response Measured</u> None <u>Nonauditory effects</u> Thyroid histology - no significant changes in follicle cell sizes. Iodine uptake - reduced due to noise Weight gain - noise induced suppression of wt. gain compared to the controls	
<u>Author's conclusions</u> Noise stress significantly suppressed cumulative weight gains in both test groups. Weight was suppressed earlier (in the first 2 weeks) in female rats than in male rats (in the 6th week). The weight suppression was correlated with suppression of iodine uptake by the thyroid during the same time periods.			
<u>Evaluation & comments</u> The use of rats in noise studies is considered suspect by many researchers. The number of animals in each group was not specified.			

Thyroid Responses to Acoustic Stimulation

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Received October 28, 1975

Male and female rats were subjected to monotonic, 1000 Hz, 95 dB(A), noise stress presented in 15 minute intervals 8 hours per day for 12 weeks. Body weights and thyroid I-131 uptake values were recorded. Relative body weight-gain rates were significantly reduced. Female rate of weight-gain decreased during the first 2 weeks, and remained lower than controls. Male rate of weight-gain decreased during Week 6 and remained low throughout the last 6 weeks. Thyroid I-131 uptake values were low for both sexes and a positive correlation between the time of decreased iodine uptake and suppressed weight-gain rates was noted.

INTRODUCTION

Undesirable sound is one of the more recently recognized forms of stress, particularly in the form of daily exposure to environmental noise (Cuadra, 1972; Jansen, 1969; Kerbec, 1972; U. S. Environmental Protection Agency, 1971). This stress is potentially of great detriment to biological systems (Welch and Welch, 1970).

The most common effect cited in past noise-stress studies is adrenocortical activation via the hypothalamic-pituitary axis (Anthony, 1973). Frequencies of 125, 1000, 5000, and 10,000 Hz at levels between 65 and 93 db caused substantial elevations (twice normal levels) in human free plasma 17 hydroxycorticoids (Arguelles *et al.*, 1962). Urinary 17-ketogenic steroid excretion was also markedly elevated. Apparently, adrenal cortical function in humans is remarkably sensitive to auditory stimulation probably through the effect of ACTH released by the stress of noise perception.

Another investigation (Henkin and Knigge, 1963) showed a triphasic adrenocortical response in female rats exposed to sound-stress (130 db, 220 Hz). Initial high adrenal corticosterone secretion was followed by a period of decreased secretion, which was followed by a return to high output levels. This study correlated with another (Geber *et al.*, 1966) which reported a biphasic adrenal ascorbic acid depletion due to noise-stress (73-93 db, 20-25 kHz). An initial decrease in ascorbic acid and adrenal weight followed by a rise in ascorbic acid and adrenal weight above control values was observed. These remained high for the experimental period (21 days).

Activation of adrenocortical secretions because of noise-stress correlated with other forms of stress which cause similar results. It has long been known various stressors (leg fracture, formalin injection, shock, surgery, ether, etc.) cause increased adrenocortical hormone secretion concomitant with increased release of ACTH.

Increased secretions of adrenocortical hormones and ACTH have been related to decreased thyroid function due to suppressed TSH release from the

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adenohypophysis (Brown-Grant *et al.*, 1954; Harris, 1955; Knigge, 1960; Nicoloff *et al.*, 1970; Wilber and Utiger, 1969). Although the mechanism of this action is obscure, evidence has accumulated favoring a common precursor for ACTH and TSH (Ducommun *et al.*, 1966; Guillemin, 1968; Retiene *et al.*, 1968).

Due to stress release of ACTH and ACH, and their suppressing effects on thyroid activity, examination of the thyroid response to moderate noise-stress seemed appropriate.

METHODS

Throughout this investigation, 100-150 day old albino rats raised in our laboratory (Sprague-Dawley origin) were used. They were maintained on Wayne lab blox and water *ad libitum*. Male and female animals were divided into one control and two experimental groups.

Control rats were kept in a moderately soundproof room where ambient conditions of temperature (80°F), light cycle (12 hours), and sound level, 68 ± 2 db(A), existed. Experimental rats were subjected to sound-stress in an environmental chamber (7 x 12 x 10 ft) where ambient conditions identical to the control room existed.

A single tone (1000 Hz), recorded from a Heathkit IG72 audio-generator, was played to experimental animals in 15 minute periods followed by 15 minutes of ambient sound conditions. This routine was continued during the light phase 8 hours per day for 12 weeks. Group I experimental animals remained in the chamber 12 weeks. Group II experimental animals were placed in the chamber at the start of the 10th week, therefore only receiving sound-stress for 2 weeks.

During the experimental period all animals were observed for signs of otitis media, and at time of sacrifice, 25% of the animals were randomly inspected for visual indications of middle ear infections. Based on these inspections, animals were found to be free of gross hearing abnormalities.

Throughout this experiment body weights were measured weekly. At termination of the 12th week of sound-stress all rats were lightly anesthetized with ether and injected with five μ Ci of I-131 per animal. All animals remained in the radioisotope control room 24 hours. At the end of this period they were again lightly anesthetized and external thyroid counting, utilizing a Nuclear Chicago 1620A countrate meter, was carried out. A percentage of iodine uptake was calculated and significance testing carried out by the Iowa State University Statistical Laboratory. Animals were sacrificed after external thyroid counting. Thyroid glands were excised and fixed in 10% buffered formalin and embedded in paraffin for later histological examination.

RESULTS

Mean cumulative weight gains for all groups appear in Table 1. Control animals exhibited increases in weight throughout the experiment. Control males averaged 242 g at Day 1, and gained weight to an average of 427 g at sacrifice. Control females averaged 224 g at Day 1 and after 12 weeks averaged 275 g. The rate of gain for females slowed during the last several weeks.

Group I female rats averaged 259 g at the onset of sound-stress and after 12 weeks of it their average weight had increased only to 264 g. At no time during the

TABLE I
MEAN CUMULATIVE WEIGHT GAINS* (GRAMS) \pm STANDARD ERROR

Animal groups	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9	Week 10	Week 11	Week 12
CF ^a	11.7 ± 2.4	16.0 ± 2.7	27.5 ± 4.8	29.8 ± 4.4	33.2 ± 5.6	36.7 ± 5.7	38.8 ± 6.4	42.6 ± 7.8	44.5 ± 7.3	47.3 ± 7.8	52.3 ± 8.5	51.2 ± 9.3
CM ^a	30.7 ± 0.9	54.4 ± 1.8	76.1 ± 2.1	86.3 ± 2.6	98.1 ± 3.5	111.3 ± 3.7	123.3 ± 4.6	135.7 ± 3.5	151.6 ± 3.6	163.0 ± 3.9	175.0 ± 4.4	185.8 ± 4.9
EF12 ^b	-2.9 ± 0.9	-0.4 ± 1.9	2.9 ± 1.8	2.4 ± 2.0	1.5 ± 2.7	4.2 ± 2.0	3.5 ± 3.2	4.4 ± 2.7	3.1 ± 2.7	5.2 ± 2.8	6.8 ± 3.8	5.2 ± 4.3
EM12 ^b	21.0 ± 5.8	46.7 ± 6.9	57.7 ± 10.5	67.4 ± 10.9	77.0 ± 11.4	77.1 ± 12.2	87.6 ± 12.3	93.3 ± 12.8	102.6 ± 15.8	104.7 ± 13.3	110.4 ± 13.5	111.7 ± 13.4
EF2 ^c	1.6 ± 2.5	6.2 ± 2.7										
EM2 ^c	6.7 ± 3.9	18.8 ± 6.9										

* Relative to starting weight.

^a Control females (ambient sound).

^b Control males (ambient sound).

^c Group I—Experimental females (sound stressed 12 weeks).

^d Group I—Experimental males (sound stressed 12 weeks).

^e Group II—Experimental females (sound stressed 2 weeks).

^f Group II—Experimental males (sound stressed 2 weeks).

12 week period were significant differences found between any of the weekly weighings. However, when experimental females in group I were compared to control females, highly significant differences ($P < 0.001$) existed between all 12 week cumulative weight gain average values. The body weight gains of experimental females were significantly suppressed throughout the experiment, in fact, during Week 1 they lost weight.

Group I male rats were placed in the experimental chamber having an average weight of 316 g and at termination of 12 weeks of sound-stress these animals averaged 428 g. Although these animals gained weight, as did their control counterparts, significant differences ($P < 0.05$) showed at Week 6 and became increasingly significant until time of sacrifice. A suppressed growth rate was displayed for experimental males and experimental females but for the latter the growth suppression commenced immediately after being subjected to sound-stress.

Group II rats were exposed to sound-stress for only 2 weeks. Females began their stress period averaging 236 g and after 2 weeks in the chamber weighed 242 g. Males averaged 320 g when initially placed in the chamber and after 2 weeks of sound-stress weighed 339 g.

When compared to control animal cumulative weight gains, highly significant differences ($P < 0.001$) were found in both males and females.

Group II females did not lose weight, but group I females showed suppressed growth immediately after sound-stress began. Males in group II did not continue gaining as control animals. Instead their growth was suppressed at once but not to the extent of females in the same group.

Percent 1-131 uptake for 24 hours (Table 2) showed a significant sex difference among control rats ($P < 0.01$) and a similar sex difference among group II rats (P

TABLE 2
MEAN PERCENTAGE IODINE-131 UPTAKE VALUES \pm STANDARD ERROR*

Group	Mean percentage uptake	Standard error
Control females	3.53	0.28
Control males	2.48	0.22
Group I females	2.32	0.14
Group I males	1.67	0.11
Group II females	2.56	0.21
Group II males	2.49	0.28

* Group I animals were sound-stressed 12 weeks, group II rats exposed only 2 weeks.

< 0.05). No significant sex difference was shown among group I animals.

A significant decrease in iodine uptake resulted between control females and group I females ($P < 0.01$). The difference in this uptake between group II females and control females was also highly significant ($P < 0.001$). However, no significance was recorded between females in groups I and II.

Control males and group I males displayed no difference in I-131 uptake. Yet a highly significant difference existed between control males and group II males ($P < 0.001$). Only a significant difference was noted between group I males and group II males ($P < 0.02$).

Female iodine uptake values were reduced in this study sometime during the first 2 weeks of sound exposure. Their uptake stayed at that level throughout the remaining 10 weeks. On the other hand, male iodine uptake was equal and constant in level the first 2 weeks of the study but decreased sometime between the 2nd and 12th weeks (Table 2).

Thyroid glands were sectioned and analyzed for follicle and follicle cell size. No significant alterations in structure or size were noted.

DISCUSSION

This study shows growth rates are altered by sound-stress at the level reported herein and that these alterations are sexually differentiated both in amount and time of response. Suppression of cumulative growth rates in female rats occurred more rapidly than in males and appeared to be more severe.

Depression of body weight has been demonstrated using various types of stress and attributed by some (Sackler *et al.*, 1959; Sackler *et al.*, 1960) to decreased food intake during the stress, as well as other hormonally induced factors. Results presented in this paper reveal a close correlation between decreased relative weight gains and depression of I-131 uptake values for both male and female rats. Females lost weight during the first 2 weeks, and their iodine uptake values were suppressed during this same period. Males began to lose weight significantly during the sixth week of sound-stress. Their iodine uptake values were depressed during the last 10 weeks. This depressed I-131 uptake may have been initiated during the sixth week.

Suppressed body weight gains of stressed animals, correlated chronologically with decreased I-131 uptake values (groups I and II), suggest a physiological difference in the response to sound-stress between male and female rats.

Sound of various levels has been used by other investigators to observe stress reactions (Anthony, 1973; Arguelles, 1962; Geber *et al.*, 1966; Henkin and Knigge, 1963). A stress response to sound elicits higher levels of ACTH and the catabolic glucocorticoids. Adrenal cortical hormones have an inhibitory effect on the thyroid gland or anterior pituitary release of TSH (Brown-Grant *et al.*, 1954; Knigge, 1960; Nicoloff *et al.*, 1970; Wilber and Utiger, 1969). Another suggested possibility is that sound through extrahypothalamic or hypothalamic influences reduced TSH secretion by the anterior pituitary and thus indirectly altered thyroid secretion. This altered thyroid function may in turn decrease the secretion of growth hormone, antagonize the effects of growth hormone, or decreased thyroid function may alter the synergism between thyroid hormone and growth hormones (Daughaday *et al.*, 1975). These endocrine interactions, along with decreased food intake, are possible explanations for the loss of weight gain observed in these rats.

Parameters measured in this investigation (relative weight gain and iodine uptake values) exhibited sex differences. Gonadal hormones may play a role in these differences since gonadotrophin-secreting pituitary basophils increase in number under sound-stress (Sackler *et al.*, 1959; Sackler *et al.*, 1960). Ovaries increased in size and weight, uteri decreased in weight, whereas males exhibited no testicular change in normal spermatogenesis during such stress (Zondek and Tamari, 1964). Along with these anatomical effects, decreased fertility rates have been reported among similarly stressed rats (Zondek and Tamari, 1964). The mechanism of gonadal hormone involvement is still unknown and highly variable with the amount and level of sound-stress used.

Results of this investigation emphasize more research is needed to examine effects of sound-stress on both male and female physiological and endocrinological systems, specifically in regard to thyroid functions.

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SECTION 27

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u>	
<u>Citation</u> Guha, D. et al. Effects of sound stimulus on gastric secretion and plasma corticosterone level in rats. Research Communications in Chemical Pathology and Pharmacology. 13(2):273-281, Feb. 1976.		
<u># of Ref.'s</u> 21	<u># of Fig.'s</u> 3	<u>Language</u> English
<u>Type & duration of experiment</u> Laboratory experiment in sound-proof chamber; 4 hour sessions - 32 days total	<u>Purpose for study</u> To determine the effects of noise on gastric secretion, and correlate these effects with blood corticosterone levels	
<u>Description of test groups (subjects, #, age, etc.)</u> 250 - 300 g Wistar rats (including unspecified numbers of males and females) with implanted stomach cannulas. The test and control groups contained 6-8 rats each.		
<u>Control of other stressors</u> Implanted cannulas may have added additional stress; experiments began 2 weeks post-operative	<u>Statistical Methods</u> Mean, standard error, and student's t-test	
<u>Noise Stimulus</u> source: through a speaker spectral characteristics: 4000 cycles per second noise level: 80 dB length of exposure: continuous for 1 or 2 hr. # of trials: Phase A - quiet in tests and controls Phase B - noise for 1 or 2 hr. in tests Phase C - quiet in tests and controls	<u>CVS Response Measured</u> None <u>Nonauditory effects</u> Significant decrease in gastric secretion and increased corticosterone levels during periods of noise exposure, returning to normal in one hour.	
<u>Author's conclusions</u> Continuous exposure to noise stress produced a significant decrease in the volume of gastric secretion and an increase in the plasma corticosterone level. An increase in gastric secretion occurred in the first hour before the noise stimulus (in phase B), an anticipatory mechanism that may be adaptive.		
<u>Evaluation & comments</u> This article supports the idea that noise stress could be a cause of stomach ulcers by decreasing the volume of gastric secretion, which increases stomach acidity. Evidence that noise acted as a stressor is indicated by increased plasma corticosterone levels.		

EFFECTS OF SOUND STIMULUS ON GASTRIC SECRETION AND
PLASMA CORTICOSTERONE LEVEL IN RATS

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Abstract

The effects of sound stimulus were studied on the gastric secretion in rats with chronically implanted cannulas. Attempts were made to correlate the changes in the secretion with those of the plasma corticosterone level. Exposure of the animals to sound stimulus (1 hr or 2 hr) produced a marked decrease in gastric secretion and a concomitant increase in plasma corticosterone. It appears that in producing these effects sound stimulus acted as a stressor. Furthermore, a paradoxical increase in secretion was noted in the first hour collection prior to the sound stimulus. This initial increase in secretion may be due to an adaptive compensatory mechanism in anticipation of its marked inhibition.

Introduction

Continuous exposure to sound stimulus has been shown to produce certain disease states such as peptic ulcer, hypertension and fetal skeletal defects in man and in animals (Mahl, 1950; Russek, 1967; Geber, 1973). Stress-induced gastric ulcer has been reported to occur as a result of increased stomach acidity concomitant with a marked decrease in the volume of gastric secretion (Brodie *et al.*, 1962). Studies from this laboratory have also demonstrated

that different types of stress including sound stimulus inhibit the growth of transplanted 4M mammary carcinoma and 7,12-dimethylbenz[a]anthracene-induced tumor (Pradhan and Ray, 1974). Rosecrans et al. (1966) observed an increase in both plasma corticosterone and urinary catecholamines on exposure to combined auditory, visual and motion stress.

The present study was therefore undertaken to determine the effects of chronic exposure to auditory stimulus on the volume of gastric secretion in rats. Attempts were also made to correlate the stress-induced changes in gastric secretion with blood corticosterone level.

Methods and Materials

Rats of Wistar strain weighing 250-300g and of either sex were used. The animals were implanted with stainless steel cannula in their stomach according to the method of Pare (1972) for repeated collections of gastric secretion over prolonged periods (Guha et al., 1974). Experiments were started following a post-operative period of 2 weeks during which rats were also adapted to the experimental situations. The experimental and control groups each consisted of 6-8 rats.

During a daily session lasting for 3 or 4 hours animals were kept inside a sound-proof cabinet fitted with a speaker and gastric secretion was collected every hour. Sessions were carried out 6 days a week at the same hour of the day.

Sound stimulus (80 db, 4000 cps) was presented to the experimental group during the 2nd hour in two experiments (I and II) and during the 2nd and 3rd hours in one experiment (III); these sound exposure periods were preceded and followed by an hour of collection of control secretion.

Gastric secretion was experimental period with presence of sound stimulus period without any sound hours; and phase C, as detailed analysis of periods lasting for 3-5 minutes, the gastric secretion under same conditions as experimental.

Micro-determination to the method of Gilchrist experimental and control gastric secretion. Paired comparison between Student's t test.

A. Gastric secretion

(1) Effect of sound : experiments performed in the control group, 1.2-1.7 ml and in the experimental group there was a marked reduction in secretion during phase A. In phase B secretion returned to its control value. Phase C showed a progressive

Gastric secretion was measured during the sessions throughout the whole experimental period which was divided into three phases depending on the presence of sound stimulus in the experimental group: phase A, control period without any sound; phase B, exposure to sound stimulus for 1 or 2 hours; and phase C, post-stimulus control in absence of sound. For detailed analysis of data each of these phases was further subdivided into periods lasting for 3-6 days as shown in Tables 1 and 2. In each experiment, the gastric secretions of a control group was monitored under the same conditions as experimental rats but without exposure to sound.

Micro-determination of the plasma corticosterone was done according to the method of Ulisk *et al.* (1964) at the end of each phase in both experimental and control groups. The mean and standard error of the hourly gastric secretion in different phases were determined in both experiments. Paired comparison between the data from the relevant groups was done by Student's t test.

Results

A. Gastric secretion

(1) Effect of one-hour sound exposure. The results of one of the two experiments performed with 1-hour sound exposure, are presented in Table 1. In the control group, the hourly secretions in three phases ranged from 1.2-1.7 ml and did not differ significantly from each other. In the experimental group there was also no apparent difference in the hourly secretions during phase A. In phase B, following exposure to sound, the secretion was markedly reduced in the 1st hour. In the 2nd hour, the secretion returned to its control value as in phase A. The 3rd hour secretion in this phase showed a progressive increase over that in phase A. In phase C, the hourly

TABLE 1. Effect of 1-hour sound exposure on gastric secretion^a in rats.

Phases and Days	Experimental Group ^b			Control Group		
	1st hr.	2nd hr.	3rd hr.	1st hr.	2nd hr.	3rd hr.
A 1-5	1.7 ± 0.6	1.5 ± 0.1	1.6 ± 0.6	1.4 ± 0.1	1.5 ± 0.1	1.3 ± 0.2
6-10	1.6 ± 0.2	1.3 ± 0.1	1.4 ± 0.1	1.5 ± 0.1	1.2 ± 0.1	1.3 ± 0.1
B 1-5	2.1 ± 0.3	1.4 ± 0.1	1.4 ± 0.2	1.7 ± 0.2	1.6 ± 0.3	1.3 ± 0.2
6-10	2.5 ± 0.1	1.2 ± 0.1	1.8 ± 0.2	1.6 ± 0.1	1.4 ± 0.1	1.5 ± 0.2
11-15	2.8 ± 0.1 ^c	1.2 ± 0.1 ^d	1.7 ± 0.2	1.7 ± 0.1	1.7 ± 0.1	1.7 ± 0.2
C 1-4	2.4 ± 0.1	1.6 ± 0.3	1.5 ± 0.2	1.6 ± 0.3	1.7 ± 0.5	1.5 ± 0.2
5-7	1.6 ± 0.1	1.2 ± 0.1	1.4 ± 0.1	1.4 ± 0.2	1.4 ± 0.2	1.4 ± 0.2

^a ml/hour (mean ± S.E.).

^b Rats of this group were exposed to sound during the 2nd hour of phase B.

^c Significantly (P < .001) greater than the 1st hour secretion during day 6-10 of phase A.

^d Significantly (P < .001) less than the corresponding 1st hour secretion.

secretion returned to control level as in phase A, except for a slight increase during the 1st 4 days leading to a higher mean value compared to that of phase A.

(ii) Effect of two-hour sound exposure. The control animals showed only slight variations in the hourly secretions during the different phases of the experiment (Table 2). In the experimental group no marked difference was also observed in the hourly secretions during phase A. Exposure to sound for 2 hours markedly reduced the secretions in the 2nd and the 3rd hours of phase B. In the 4th hour (after withdrawal of sound) the volume of secretion returned very close to its own control value as in phase A. The 1st hour secretion in this phase showed a small increase in secretion compared to its corresponding

TABLE 2. Effect of 2-hour sound exposure on gastric secretion^a in rats.

Phases and Days	Experimental Group ^b			Control Group		
	1st hr.	2nd hr.	3rd hr.	1st hr.	2nd hr.	3rd hr.
A 1-5	1.7 ± 0.6	1.5 ± 0.1	1.6 ± 0.6	1.4 ± 0.1	1.5 ± 0.1	1.3 ± 0.2
6-10	1.6 ± 0.2	1.3 ± 0.1	1.4 ± 0.1	1.5 ± 0.1	1.2 ± 0.1	1.3 ± 0.1
B 1-5	2.1 ± 0.3	1.4 ± 0.1	1.4 ± 0.2	1.7 ± 0.2	1.6 ± 0.3	1.3 ± 0.2
6-10	2.5 ± 0.1	1.2 ± 0.1	1.8 ± 0.2	1.6 ± 0.1	1.4 ± 0.1	1.5 ± 0.2
11-15	2.8 ± 0.1 ^c	1.2 ± 0.1 ^d	1.7 ± 0.2	1.7 ± 0.1	1.7 ± 0.1	1.7 ± 0.2
C 1-4	2.4 ± 0.1	1.6 ± 0.3	1.5 ± 0.2	1.6 ± 0.3	1.7 ± 0.5	1.5 ± 0.2
5-7	1.6 ± 0.1	1.2 ± 0.1	1.4 ± 0.1	1.4 ± 0.2	1.4 ± 0.2	1.4 ± 0.2

TABLE 2. Effect of 2-hour sound exposure on gastric secretion^a in rats.

Phases and Days	Experimental group ^b				Control group			
	1st hr.	2nd hr.	3rd hr.	4th hr.	1st hr.	2nd hr.	3rd hr.	4th hr.
A 1-5	1.4 ± 0.3	1.0 ± 0.2	1.1 ± 0.1	1.1 ± 0.1	1.3 ± 0.2	1.3 ± 0.1	1.3 ± 0.2	1.3 ± 0.2
6-10	1.2 ± 0.1	1.0 ± 0.1	1.2 ± 0.2	1.2 ± 0.2	1.2 ± 0.1	1.1 ± 0.2	1.2 ± 0.3	1.2 ± 0.3
B 1-5	1.6 ± 0.2	1.3 ± 0.2 ^c	0.5 ± 0.1 ^c	1.3 ± 0.3	1.4 ± 0.2	1.2 ± 0.2	1.3 ± 0.3	1.4 ± 0.2
6-10	1.7 ± 0.2	0.6 ± 0.2	0.7 ± 0.1	1.4 ± 0.2	1.4 ± 0.1	1.4 ± 0.4	1.4 ± 0.4	1.6 ± 0.3
11-15	1.6 ± 0.2	0.9 ± 0.2	0.8 ± 0.1	1.4 ± 0.1	1.4 ± 0.2	1.1 ± 0.4	1.3 ± 0.2	1.5 ± 0.2
C 1-6	1.5 ± 0.2	1.5 ± 0.2	1.5 ± 0.2	1.5 ± 0.1	1.5 ± 0.1	1.3 ± 0.1	1.3 ± 0.2	1.6 ± 0.1

^a ml/hour (mean ± S.E.).^b Rats of this group were exposed to sound during the 2nd and 3rd hours of phase B.^c Significantly ($P < .001$) less than the corresponding 1st hour secretion.

controls in phase A. In phase C, all hourly secretions returned very close to their control values as in phase A.

B. Plasma corticosterone level.

In the experimental groups of rats plasma corticosterone levels increased significantly after sound exposure for 1 hour (in experiment I) or 2 hours, (in experiment III) in phase B, while there was no significant change in its levels in the control groups (Table 3). During the later phases due to technical difficulties the plasma corticosterone levels could be correctly measured only from a limited number of rats. These data are scanty and variable and not presented here.

TABLE 3. Plasma corticosterone levels^a in the control and experimental^b rats.

Experiment No. and phases	Experimental group	Control group
Experiment I		
Phase A	8.5 ± 0.5 (6)	8.8 ± 0.3 (4)
Phase B	14.6 ± 0.5 (7) ^c	9.6 ± 0.4 (4)
Experiment III		
Phase A	5.7 ± 0.5 (7)	5.8 ± 0.2 (4)
Phase B	7.1 ± 0.4 (5) ^d	5.2 ± 0.5 (4)

^ang/20 µl; mean ± S.E.

^bRats were exposed to sound for 1 or 2 hours during phase B of Experiments I and III respectively.

^cP < .001; ^dP < .05.

Repeated exposure o decrease in the volume of in the plasma corticoster with the results of other of any kind not only decr serum cortisone level (Br Ogle, 1973; Smookler and Pare, 1964). If the stre effect on gastric secreti induced inhibition of sec tolerance to sound or exh as reported by Card

Many hypotheses hav stress-induced decrease i Okabe, 1970). This decre ischemic change in the ge increased gastric motilit

Sound-induced chang closely agree with those increase in blood steroid hyperfunction in all 3 ac during the sound exposure been stimulated resultin; leading to elevation of

Discussion

Repeated exposure of rats to auditory stress produced significant decrease in the volume of gastric secretion concomitant with a marked increase in the plasma corticosterone level. These findings are in close agreement with the results of other investigators who showed that environmental 'stress' of any kind not only decreased the gastric secretion, but also increased the serum cortisone level (Brodie et al., 1962; Brodie and Hooke, 1971; Dai and Ogle, 1973; Snookler and Buckley, 1970; Mikhail, 1971; Friedman and Adar, 1965; Jare, 1964). If the stress is continued for a prolonged period, its inhibitory effect on gastric secretion gradually becomes less. This decrease of sound-induced inhibition of secretion may have resulted from the development of tolerance to sound or exhaustion of available elements of gastric secretion, as reported by Card (1967) in relation to drug-induced changes.

Many hypotheses have been put forward to explain the mechanism for the stress-induced decrease in gastric secretion (Dai and Ogle, 1974; Takagi and Okabe, 1970). This decrease induced by sound stress may be due to transient ischemic change in the gastric mucosa (Goldman and Rosoff, 1968) and to increased gastric motility produced during stress (Watanabe, 1966).

Sound-induced changes in blood steroid levels observed in this study closely agree with those of Snookler and Buckley (1970). They reported an increase in blood steroid following auditory stress in rats concomitant with hyperfunction in all 3 zones of the adrenal cortex. They also suggested that during the sound exposure central noradrenergic neuronal centers could have been stimulated resulting in liberation of ACTH from the anterior pituitary leading to elevation of adrenal steroid secretion.

During the first hour of phase B of these experiments the gastric secretion showed a progressive increase. This paradoxical increase was similar to that observed in relation to inhibition of gastric secretion by Guha *et al.*, (1974) following epinephrine treatment and by Mahl (1952) during chronic fear. This increase may be attributed to an adaptive compensatory mechanism in anticipation of its marked inhibition induced by the stress (Guha *et al.*, 1974).

Acknowledgement

The authors are thankful to Dr. Rajendra Kumar for his assistance in estimating the plasma corticosterone levels in the later part of this work.

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SECTION 28

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> National Science Foundation NSF Grant GY-11519
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Citation
Hanson, J. D. et al. The effects of control over high intensity noise on plasma cortisol levels in Rhesus monkeys. Behavioral Biology 16:333-340, 1976.

<u># of Ref.'s</u> 15	<u># of Fig.'s</u> 2	<u>Language</u> English
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<u>Type & duration of experiment</u> Type: Laboratory experiment in a soundproof chamber Duration: 11 days - monkeys trained to control noise. 28 days - experiment	<u>Purpose for study</u> To study the effects of control over a stressful stimulus (noise) on plasma cortisol levels
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Description of test groups (subjects, #, age, etc.)
24 Rhesus monkeys: 12 one-year olds (6 males, 6 females)
12 3-year olds (6 males, 6 females)
Three groups: 1. no noise 2. control over noise 3. loss of control over noise (same animals as group 2)

<u>Control of other stressors</u> Laboratory conditions; some stress may have been associated with venipuncture	<u>Statistical Methods</u> 2x3x3 factorial design; Fisher LSD test; analyses of variance
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<u>Noise Stimulus</u> source: (power tools, heavy machinery, drills) Ampex Model AG 600 tape recorder spectral characteristics: reported in previous paper noise level: 100 dB length of exposure: 4 13.-min. noise periods with 2 min quiet periods inbetween # of trials: 2	<u>CVS Response Measured</u> none <u>Nonauditory effects</u> 1) increased plasma cortisol levels in animals with no control over the noise 2) levels of plasma cortisol in animals with control over noise were similar to that of controls with no noise. 3) aggressive behavior increased in animals with loss of control over noise. 4) anti-social behavior (decreased social contact) was greater in animals with no control over noise.
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Author's conclusions
Monkeys who had control over high intensity noise had blood cortisol levels similar to that of the controls (no noise). Loss of control over noise increased blood cortisol levels to that of the monkeys who never had control over noise. Cortisol levels may be a reliable measurement of the short term effects of stress in humans.

Evaluation & comments
This research is mainly concerned with the adverse effects of noise stress on primate behavior and is a fairly well controlled study.

The Effects of Control over High Intensity Noise on
Plasma Cortisol Levels in Rhesus Monkeys¹

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The effects of control over exposure to high intensity noise on plasma cortisol levels and social behaviors were examined in rhesus monkeys. There were four conditions: control over noise, loss of control over noise, no control over noise, and no noise. Plasma cortisol data indicated that animals with control over high intensity noise stimulation did not differ from animals exposed to no noise at all. Plasma cortisol levels were significantly elevated in animals with no control over high intensity noise and in animals experiencing a loss of control over noise. Animals which experienced loss of control over noise showed increased aggressive behavior while animals with no control over noise showed significantly less social contact than other animals.

In recent years considerable attention has been given to research involving the effects of control over aversive stimuli both with human and nonhuman subjects. Research with humans has used electric shock or high intensity noise as the aversive stimulus and GSR, self-report, and performance tasks as measures of stress. While the results with GSR measures have been contradictory, task performance seems generally to have been less impaired when the subjects perceived that they had control over the stressful stimulus than when they had no control over the stressful stimulus (Bandler, Madras, and Bem, 1968; Corah and Boffa, 1970; Champion, 1950; Geer, Davison, and Gatchel, 1970; Glass, Reim, and Singer, 1971; Staub, Tursky, and Schwartz,

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1971). Lefcourt (1973) reviewing the results of several studies has argued that the perception of control over an aversive stimulus is a salient feature in the reduction of stress reactions to the aversive stimulus.

Research conducted using non-human subjects concerning the effects of control over stress-causing stimuli has been limited and contradictory. Brady (1958) using rhesus monkeys and electric shocks, reported development of gastro-intestinal ulcers among animals with control over shock presentation whereas no such ulceration developed among animals with no control over shocks. Weiss (1968) subjected rats to severe electric shocks and reported finding ulcers in the rats with no control over the administration of shocks and no ulcers in rats with control, a direct contradiction of Brady.

The present study used plasma cortisol levels as a physiological measure rather than ulceration or GSR skin conductance measures. While gastro-intestinal ulceration is recognized as a long term stress index our concern was with the short term effects of exposure to stress. GSR measures have produced contradictory results whereas plasma cortisol levels provide a high degree of reliability and specificity (Bowman and DeLuna, 1969). This study used continuous high intensity noise as a stressor since it has been shown previously to produce short term increases in cortisol levels in rats (Henkin and Knigge, 1963), in monkeys (Nealis and Bowman, 1972) and in humans (Arguelles, Ibeas, Ottone, and Chekherdemian, 1962). Social behaviors were measured immediately following noise exposure. The major concern of the present study is the effect of control over the aversive noise stimulus and the effect of no such control on cortisol levels and on measures of social behaviors.

METHOD

Subjects. Twenty-four rhesus monkeys (*Macaca mulatta*), 12 one-year olds (six males and six females) and 12 three year olds (six males and six females) were used in this experiment. All monkeys were laboratory born animals raised in small social groups. The monkeys had not previously been exposed to high intensity noise. Feeding, handling, and maintenance schedules were held constant throughout the study.

Apparatus and Materials. The noise stress was modeled after that used by Nealis and Bowman (1972). One hour of continuous noise (power tools, heavy machinery, pneumatic drills, snowmobiles, etc.) at 100 db (re 0.0002 dynes/cm²) was administered in a soundproof chamber (10 X 10 X 8 ft l.d.). Nealis and Bowman (1972) reported that this continuous noise was the most effective form of high intensity noise and that it produced its maximum effect on cortisol levels after one hour of stimulation. An Ampex Model AG 600 tape recorder was used to record and playback the

auditory stimuli. The noise was amplified by an Altec Model 1594A amplifier and an Electrovoice Sentry IV horn loaded speaker system (Frequency response 50-20,000 Hz). Behavioral observations were made in a playroom described by Harlow, Rowland, and Griffin (1964).

Design. The experiment employed a $2 \times 2 \times 3$ factorial design. There were two age groups, one and three year olds, and two experimental parts. Each of the experimental parts had three groups. In the first part these groups included control over noise, no control over noise, and a no noise group. In the second part, the monkeys from the control over noise condition lost their control over noise stimulation. The no control over noise animals, and the no noise animals were treated as in the first part. Each monkey was tested twice in each part of the experiment.

Procedure. Monkeys in the control over noise group were trained to terminate the noise by depressing a retractable lever. Each animal in the no control over noise group was paired with one in the control over noise group and was exposed to equal durations of noise with the lever made inoperable. At the end of 11 days of training all monkeys in the control over noise group performed perfectly, i.e. upon presentation of the lever, during noise, the subjects depressed the lever within 1-3 sec. No noise monkeys were familiarized with the sound room environment but were never exposed to noise. During this training period, all animals were familiarized with the Playroom.

The experimental conditions were administered to all groups over a period of twenty-eight days. Treatments of specific individual subjects were separated by a minimum of seven days to insure alleviation of any temporary noise-induced stress. The experimental condition consisted of four 13-min periods of noise separated by 2 min intervals of silence. In the control over noise group, the lever was presented at the end of the 13 min noise interval. These trained monkeys always pressed the lever within a few seconds and the noise stopped.

In the no control over noise group, noise was again presented in the four 13 min periods followed by 2 min of silence, however, the retractable lever was not presented. In the no-noise group, animals were exposed to neither the noise nor the retractable lever. In the second part of the experiment the monkeys previously in the control over noise group were now presented with the retractable lever at intermittent times, but pressing the lever did not terminate the noise stimuli. Each animal in this group received two such extinction trials.

Immediately following the experimental treatment, venipuncture of the saphenous vein was performed and 1 ml of whole blood was obtained. Centrifugation followed and the resulting plasma was analyzed by the protein-binding radio-immunassay for cortisol defined by Bowman and DeLuna (1969). At least six replicate cortisol determinations were made on each sample. The cortisol value for each sample was the mean of the first

three determinations that were within $5\mu\text{g}/100\text{ml}$ of each other. Basal non-stress cortisol levels were established on blood drawn 24 hr prior to each noise stress test.

The behavioral activities were scored 1/2 hr after blood drawing in the playroom for 30 min using a modified frequency scoring technique. A modification of the Hansen-Seay Behavior Definitions (Suomi, Harlow, and Kimball, 1971) with 24 categories was used. Behaviors showing extremely low frequency were dropped from analysis leaving a total of 13 behavior categories. Two observers scored the test animal simultaneously. Three animals of comparable age, weight, and dominance were placed in the playroom with the test animal. The same four animals were always tested in the playroom together.

Prior to the start of the experiment, pre-tests for social behavior measures were conducted in order to familiarize the monkeys with the playroom. Inter-observer reliability was maintained throughout the experiment above the 0.95 level (Pearson product-moment correlation).

Data Analysis. Analyses of variance were employed for evaluating the data of the present study. The Fisher LSD test was used to determine significant differences between means.

RESULTS

Biochemical Data. An analysis of variance of the basal cortisol levels over groups and parts of the experiment showed no significant effects. Since basal levels were equivalent, all subsequent cortisol analyses were based on difference scores. Plasma cortisol difference scores (cortisol levels following noise exposure minus the cortisol levels found 24 hr earlier) for all animals were subjected to an analysis of variance. The Treatment X Parts interaction was significant ($F(2,18) = 7.86, P < .025$) (Fig. 1). The plasma cortisol difference scores of monkeys which had control over high intensity noise did not differ from those of animals which received no noise stimulation at all ($P > .10$). However, the monkeys which received identical amounts of high intensity noise but which had no control over the noise showed significant elevations in plasma cortisol relative to both the no-noise group ($P < .05$) and the control over noise group ($P < .05$). The animals in the loss of control over noise condition showed an elevation of cortisol to levels comparable to animals which had no control over noise ($P > .05$). These levels were significantly greater than the levels found in the same animals when they had control over the noise stimulus ($P < .05$) and significantly greater than animals in the no noise condition ($P < .05$).

Social Behavior Data. Analyses of variance of the measures of social behavior yielded only two significant effects involving control over noise. The

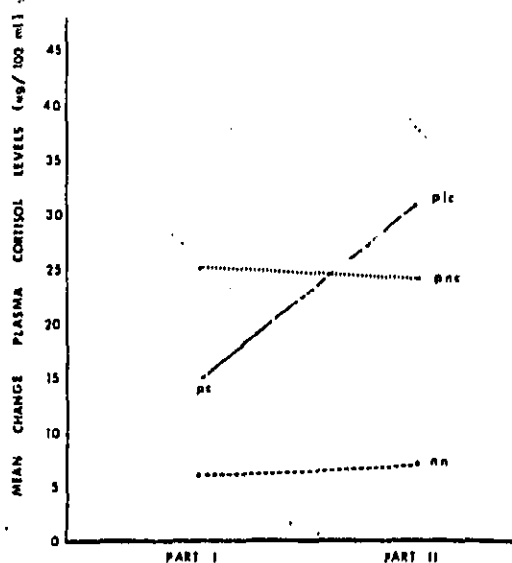


Fig. 1. Changes in cortisol levels following exposure to high intensity noise. (nn = no noise; pc = control over noise; plc = loss of control over noise; pnc = no control over noise.)

TABLE 1
Frequency of External Aggression Per Session in Three Year Old Monkeys

Treatment	Mean frequency
No noise	1.19
Control over noise	.88
No control over noise	.63
Loss of control over noise	2.50 ^a

^aSignificantly different from other conditions, P 's < .05.

measure of External Aggression had a significant Treatment X Age X Parts interaction ($F(2,18) = 7.50, P < .01$). Three year old monkeys in the loss of control over noise condition exhibited higher levels of aggression than animals in all other conditions. Means are presented in Table 1. There was a significant Treatment main effect for Social Contact ($F(1,18) = 9.51, P < .01$). The animals with no control over noise showed significantly less social contact than animals in the other groups. Means are presented in Table 2. This is

TABLE 2
Mean Frequency of Social Contact per Session

Treatment	Mean frequency
No noise	33.2
Control over noise/loss of control over noise	30.0
No control over noise	16.5 ^a

^aSignificantly different from other conditions P 's < .05.

similar to the findings reported by Nealis and Bowman (1972) whose noise stressed animals (without control over noise) showed a reduction in social interactions.

DISCUSSION

The control over aversive high intensity noise produced significant reduction of the physiological response to stress as measured by plasma cortisol levels. Changes in cortisol levels relative to non-stress periods were similar in animals with control over the high intensity noise and those who were never exposed to noise at all, while elevated cortisol levels were found in animals exposed to identical amounts of high intensity noise, but who had no control over the presentation of noise. When the animals which had control over the noise stimulus had that control removed, there was an increase in their cortisol difference scores. The levels were significantly higher than they had been when the same animals had control over the noise and were similar to those of noise exposed animals which had never perceived control over the noise. Thus, control over an aversive stimulus markedly reduced the cortisol response that normally accompanies the aversive stimulus.

This increased cortisol response from control over noise to loss of control over noise could not have been due to the passage of time or increased exposure to the noise stimulus, since both the noise animals and the animals with no control over noise exposure showed no cortisol changes over the same period. Variance due to diurnal changes in cortisol levels was controlled in this study since cortisol difference scores were used with the sample taken directly following noise exposure being compared with one taken exactly 24 hr previously when no noise exposure had occurred. Seasonal variations in cortisol levels are likely to have been of little consequence since the entire study was carried out in 28 days during the summer. The cortisol difference levels obtained in the present study from animals in the no noise and the no control over noise condition are similar to those found for

comparable treatments of monkeys by Nealis and Bowman (1972), while the difference level of the loss of control over noise animals is close to the maximal cortisol response to ACTH injections reported by Meyer and Bowman (1972). In light of this close correspondence of data from noise stressed animals with other data, the finding of a low cortisol response in animals with control over noise is the more striking. Clearly, control over an aversive noise stimulus can greatly attenuate the cortisol response to the aversive stimulus.

Some of the behavioral measures further substantiate these findings. Among three year old animals the loss of control over noise manipulation produced levels of aggression that were significantly greater than the levels found in any other condition. The animals in the no control over noise group showed a significantly lower level of social contact than did animals in other treatment groups. This finding is similar to the finding of Nealis and Bowman (1972) that animals following a one hour noise stress session displayed a reduction in social behaviors and an increase in self-oriented behaviors.

However, many results of social behavior measures did not correspond to the pattern of results found with cortisol levels. Social behavior measures are highly dependent upon the context and behavior of the other animals. In this study social groups were carefully constructed several weeks prior to the onset of testing, and only one noise stressed animal was in a group at any time. Nealis and Bowman (1972) found a greater number of significant changes in playroom behavior following noise stress, but all of the animals in their playroom groups had been noise stressed at the same time, producing a quite different behavioral context from the present study.

The effects of control or lack of control over an aversive stimulus on cortisol levels reported here parallel the results of Weiss (1968) finding gastrointestinal ulceration in stressed rats and the various performance measures used in several human studies (Champion, 1950; Geer *et al.* 1970; Glass *et al.* 1971; Staub *et al.* 1971). Plasma cortisol difference measures would seem to be of use in evaluating the short-term effects of stress and of control or lack of control over stress. Cortisol measures might be a more reliable index of stress in humans than the GSR measurements typically used.

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SECTION 29

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> C. S. Harris, H. C. Sommer, and D. L. Johnson	<u>Institution and address where research was performed</u> Biological Acoustics Branch 6570th Aerospace Medical Research Lab Wright-Patterson AFB, OH 45433
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> U.S. Air Force
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Citation
 Harris, C. S. et al. Review of the effects of infrasound on man. Aviation, Space, and Environmental Medicine. 47(4):430-4, Apr. 1976.

# of Ref.'s 37	# of Fig.'s 0	Language English
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<u>Type & duration of experiment</u> Not applicable	<u>Purpose for study</u> Review article on effects of infrasound on reaction time, equilibrium, nystagmus, and human performance.
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Description of test groups (subjects, age, etc.)
 Not applicable

<u>Control of other stressors</u> Not applicable	<u>Statistical Methods</u> Not applicable
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<u>Noise Stimulus</u> source: sound having alternating, low frequency pressure changes spectral characteristics: infrasound - below 16 Hz noise level: 105-120 dB length of exposure: not applicable # of trials: not applicable	<u>CVS Response Measured</u> None <u>Nonauditory effects studied</u> equilibrium vestibular response nystagmus (involuntary eyeball movements) reaction time performance
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Author's conclusions
 The effects of infrasound at low intensity levels have been exaggerated or do not exist at all. The level at which infrasound becomes dangerous is still unknown.

Evaluation & comments
 Authors try to refute the results of many infrasound studies done on human performance, nystagmus, and subjective response.

Review of the Effects of Infrasound on Man

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HARRIS, C. S., H. C. SOMMER, and D. L. JOHNSON. Review of the effects of infrasound on man. *Aviat. Space Environ. Med.* 47(4):430-434, 1976.

Claims that infrasound adversely affects human performance, makes people "drunk," and directly elicits nystagmus, have not been clearly demonstrated in any experimental study. The effects obtained at low intensity levels of 105 to 120 dB, if they can be substantiated at all, have been exaggerated. Recent well-designed studies conducted at higher intensity levels have found no adverse effects of infrasound on reaction time or human equilibrium. The levels at which infrasound becomes a hazard to man are still unknown. However, the hazardous levels are certain to be much higher than have been suggested in some of the literature. The preliminary exposure limits which were proposed several years ago for use in the U.S.A. are still considered safe and adequate based on present knowledge. Caution is necessary in future research because artifacts produced by faulty experimental procedures can suggest genuine psychological or physiological effects.

WE ARE SURROUNDED by infrasound, those acoustic frequencies below approximately 16 Hz according to the definition of the U.S. Environmental Protection Agency (32). Any alternating, low-frequency pressure change may be defined as infrasound. Any pulsating or throbbing piece of equipment, the wind blowing around corners and between buildings in our city streets, or wind whipping through the windows of our car, driving in the mountains, diving, and swimming in water, etc., is a potential source of infrasound. It does not strain the imagination to think of many other sources. However, until recently it was thought that most of these sources were relatively unimportant and that only very intense levels of infrasound— >140 dB, such as produced by rockets and extremely large jet engines—clearly much above the threshold of perception, were any cause for concern. And, of course, only a relatively small percentage of people are, or will be, exposed to such intense infrasound.

This paper was prepared by personnel of the Aerospace Medical Research Laboratory, Aerospace Medical Division, Air Force Systems Command, Wright-Patterson Air Force Base, Oh. The opinions are those of the authors and do not necessarily reflect the views of the U.S. Air Force. This paper has been identified by Aerospace Medical Research Laboratory as AMRL-TR-74-70.

In recent years, infrasound has been suggested as having effects at much lower intensity levels; indeed, at levels below the threshold of perception. This suggestion is of considerable concern to the Air Force since many maintenance men work around jet aircraft that routinely produce levels up to 130 dB (7).

Some studies on the effects of infrasound on man claim so many dire effects that most investigators will question their credibility. For example, Gavreau (13) states: "—weak infrasounds affect the semicircular canals—the balancing mechanism in the ear—and produce fatigue, dizziness, irritation and nausea. They are certainly one of the many causes of allergies, nervous breakdowns and other unpleasant 'phenomena of modern life' which are found in industrial cities . . ." Subsequent to Gavreau's publication (13), the study of infrasound appears slightly more "scientific;" however, many ominous claims have resulted. Authors have suggested that infrasound can: a) make people feel subjectively drunk (4,29), b) adversely affect human performance (21), and c) serve as a useful clinical tool for assessing vestibular function because of its potential for eliciting nystagmus (9).

There are good reasons for questioning the conclusions contained in most studies on the effects of infrasound on man. Most studies are weak in experimental methodology and in scientific reporting. Experimental procedures, statistical methods, and even the number of subjects are often either omitted entirely or so cursorily presented that the reader cannot be sure what was done.

PERFORMANCE MEASURES

Infrasound at levels from 105 dB* to 120 dB has been reported to produce a state akin to alcoholic intoxication in people and to increase their reaction time (10,21). Evans and Tempest (10) state: "It would appear that these psychological effects of infrasound, in the context of transportation, are perhaps more important than the balance disturbance effects. The subjective

*Throughout this paper, dB is the R.M.S. sound pressure level referenced to $20 \mu\text{Pa} = 20 \mu\text{N/m}^2$.

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descriptions of the subjects as feeling intoxicated are very accurate, since the changes in reaction time are similar to those experienced by a man of average weight who would not pass a breathalyzer test . . ." Aside from the questionable reasoning, the authors (10) are suggesting that infrasound at higher levels, above 120 dB, adversely affects "balance" by directly stimulating the vestibular system and, at lower levels of 105 dB to 120 dB, producing a state similar to alcoholic intoxication through some unknown mechanism. Why adopt two different mechanisms for explaining the effects of infrasound since one of the most sensitive indicators of alcohol ingestion is a disturbance of balance? Fregley *et al.* (11,12) demonstrated that standing with eyes open on a narrow rail was the most sensitive measure of their ataxia task battery to the percent level of alcohol in the blood stream. Therefore, the more interesting question concerning the infrasound studies is why the subjects' reaction times increased, and why they reported feelings of drunkenness at lower intensity levels than were required to adversely affect their balance. Hood, Leventhall and Kyriakide (21) found a balance test, standing with eyes open on a 1-in. wide rail, was unaffected by levels of infrasound that they report produced an increase in reaction time.

More important than the discussion of the interpretation of the results is an examination of the results themselves. In three different articles where it is suggested that infrasound at levels of 105 to 120 dB may produce a state similar to alcoholic intoxication in the subjects, either no data are presented or it is not obvious that the data support the claims. Moreover, the article by Evans and Tempest (10) warns the transportation industry about possible adverse psychological effects of infrasound without presenting details of procedure, experimental design, statistical analysis or, indeed, the number of subjects used to come up with the conclusion. Yet they state: "No level or frequency of infrasound in the 1-20 Hz region caused any visual disturbance but relatively low levels (115-120 dB) cause a 30-40% increase in reaction time. (It is intended that a more detailed study of this effect will be published in due course.)" The publication of these claims is, at best, premature but, unfortunately, it is also deceiving to present reaction time results in terms of percentage change without presenting the means and standard deviations of the original reaction time measures. Even then the practice is questionable since a very small change in reaction time can result in a considerable percent change.

In another article (21), the investigators purport to show an effect of infrasound on a pointer-following task and a reaction time task. There is only a brief description of the reaction time task: ". . . a simple single button reaction-time experiment was performed 50 times (21)." One would want to know what stimulus the subjects were reacting to and what the conditions were under which the subjects reacted 50 times. No reference is made to the fact that procedures for conducting simple reaction time experiments are pretty well standardized (37), and details of training, counterbalancing, and manner of stimulus presentation are quite important in

conducting a valid experiment. Nevertheless, the authors state (21): "Pointer following and reaction time experiments showed a deterioration in performance in the noisy environment with a significance better than 0.1%." Graphs are presented for individual subjects on the basis of percent increase in error and percent increase in reaction time. This is very poor form since the analyses of variance should not have been conducted using such scores. Nevertheless, these graphs make it seem unlikely that such a high significance level could have been obtained.

It is possible that they could have meant the 10% level? The acceptance of the 10% level is unsatisfactory in the view of the present authors because of the unusual nature of the findings and the small number of seven subjects. The effect reported for reaction time and pointer following was unusual because the percent increase in error and percent increase in reaction time was larger at 110 dB than it was at 120 dB. Although the results for the two sets of scores tend to agree, there is essentially no correlation between the two sets of scores, as one can readily determine by comparing each subject's relative position in both tasks. Therefore, one could not predict from the reaction time scores the score a subject would receive on the pointer-following task. This is unusual, since both scores supposedly change because of a state analogous to alcoholic intoxication created in the subject by infrasound.

In discussing the results obtained with a rail balancing task, the same authors (21) state: ". . . In the balance experiment only two of the subjects were significantly affected by infrasound . . ." This conclusion was reached even though an analysis of variance showed no statistically significant effects. At least no effects may have been shown; the authors point out that an analysis of variance was used with the balance test but do not report the results except the above statement that two subjects were "significantly affected." This type of reasoning in interpreting experimental results is misleading and time consuming since it can lead an investigator to discover effects that do not exist. A behavioral measure is not a fixed quantity but is subject to change, not only as a function of the independent variable but also a function of procedural, task, and subject variables as well. The purpose of statistical analysis is to determine whether uncontrolled factors can explain the observed differences.

The statement is made in another article (4) that: "Experiments have shown that a band of noise 2 Hz-15 Hz wide at a level of 105 dB, or a 7 Hz tone at the same level, can produce an increase in visual reaction time of 10% in half of the subjects in a test group . . ." Such statements are not informative and no substitute for the use of statistical methods.

A recent, well-designed study by Borredon (2) casts further doubt on the reliability of the above results. He found, using 42 young men as subjects, that a 7.5 Hz infrasound stimulus presented for a 50-min duration at an intensity level of 130 dB had a negligible effect on simple reaction time.

Until evidence is presented in a clearly documented fashion, there is no reason to assume that low levels of

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infrasound, 120 dB and below, increase reaction time or that infrasound has an effect at low levels that is on a different dimension from that obtained at higher levels.

NYSTAGMUS MEASURES

Another suggestion made by several authors (8-10) is that infrasound stimulates the vestibular receptors and thereby elicits nystagmus. Nystagmus should be a less-variable measure than reaction time; however, when the response is very weak this may not be the case. Nystagmus is subject to many artifacts of measurement, and there are numerous types of nystagmus that are not vestibular in origin. The results in these articles are presented in a case history fashion or there is a simple designation of the number or percentage of subjects showing clear nystagmus, or a slight nystagmus partially masked by random eye movements. No attempt was made to quantify the nystagmus results in terms of eye movement velocity or frequency of beats, and the criterion used to determine the presence of nystagmus was not defined. This could be quite important since, in two figures purporting to show a nystagmus response to infrasound, the nystagmus is quite small compared to that elicited by caloric stimulation and rotational tests (1,5). Furthermore, the authors (10) seem to assume that nystagmus should be obtained since they state: "... the majority of observers show some vertical nystagmus under intense infrasonic stimulation, and in fact, examination of eye movement traces indicated that those who did not show a clear nystagmus were, in almost all cases, producing random eye movements sufficient to mask a small nystagmus..." It is also possible, in recording random eye movements, to find sections of the graph paper where the eye movements look like nystagmus. In studies conducted in our laboratory (17) artifacts have often been produced that looked like nystagmus. Control procedures demonstrated that the "nystagmus" was due to eye blinks (and partial eye blinks and squinting), reflexive muscle contractions about the eyes, turning the head, extreme deviation of the eyes (gaze nystagmus), amplifier drift, and, indeed, a combination of many of the above.

The initial assumption that led these investigators to study the efficiency of infrasound in eliciting nystagmus is not based on sound logic. The authors (9), in writing about the effects of low-frequency acoustic signals in the range of 2-20 Hz state: "... This is within the normal frequency range of the semi-circular canals and the upper frequency range of the otolith." This statement is partly true since there is some overlap between the two frequency ranges (26). However, why should there be a relationship between frequency of infrasound, and the frequency sensitivity of the vestibular system to accelerative forces? Certainly, there is no necessary relation. In addition, 7 Hz was reported to be the most sensitive frequency for eliciting nystagmus, and 7 Hz is certainly not within the most sensitive frequency range for either the semicircular canals or the otolith receptors. If there is a relationship between the frequency of infrasound and the frequency sensitivity of the vestibular receptors, one

would expect greater sensitivity, more nystagmus, at the lower frequencies below 2 to 4 Hz.

In one article (9) a tracing of "vertical nystagmus induced by a 7 Hz stimulus at 142 dB" is presented as a Fig. 3. The authors do not say whether the eye movement recording was made with eyes open or closed. They state: "Throughout the tests, each experiment was repeated with eyes opened and closed." In another article (10) they recorded with eyes open to reduce the possibility of obtaining spontaneous nystagmus. Therefore, one would assume the tracing was obtained with eyes open. However, it is an unusual tracing since there do not seem to be any blinks and the recording is longer than 40 s. Regardless of whether the recording was made with the eyes open or closed, the question remains whether this eye movement record demonstrates nystagmus. The authors state (9): "Fig. 3 shows a typical nystagmus response in the form of two periods of nystagmus with a few seconds interval between them." If this figure illustrates a typical nystagmus response to infrasound, it is indeed a weak response. In this over 40 s long tracing, only approximately the first 15-s segment shows movements that resemble nystagmus beats. If one scores the six beats occurring just during this period for eye movement velocity in the slow phase, based on the author's calibrations for 20° of eye displacement and for time, the slow phase velocity is approximately 1.5°/s. It would be difficult to classify this response as nystagmus under any system for evaluating nystagmus response (1,20). Such questionable nystagmus might be accepted by some investigators as threshold levels for vestibular stimulation with caloric or rotational stimuli; however, with these stimuli a clearcut nystagmus that is easily scoreable, can be demonstrated at a slightly higher intensity level. Whether this can be demonstrated with infrasound seems questionable. And the fact that these authors, on the basis of their data, propose the use of infrasound as a clinical tool for assessment of vestibular function is truly astonishing. To further complicate this interpretation the authors state (9): "... In the course of the objective (nystagmographic) measurements, no horizontal eye movements were recorded but some 2/3 of the observers showed some evidence of vertical nystagmus, which rather surprisingly, occurred at the end of the stimulus rather than during stimulation." This finding is surprising and so is the statement that no horizontal eye movements were recorded (they probably meant horizontal nystagmus) since Fig. 2 in the same article is titled: "Horizontal eye movements induced by a 10 Hz stimulus at 140 dB (observer 2)." Finally, the authors conclude (9): "... The fact that the eye movements produced are vertical rather than horizontal suggests that the low frequency effect operated mainly on the otolith system rather than the vestibular system." The otolith system is generally considered to be part of the vestibular system; however, if by "vestibular system" they are referring to the semicircular canals, then the conclusion is still unwarranted. There are four vertical canals and two horizontal canals, and one may legitimately question whether vertical nystagmus indicates primarily a stimulation of

the otolith system.

Fig. 2 in a subsequent article (10) is an eye movement tracing of "Vertical nystagmus induced by a 7 Hz stimulus at 130 dB." This figure is "... a typical vertical nystagmus trace recorded with binaural stimulation. (— binaural stimulation presented in antiphase at the two ears.)" In this tracing, the nystagmus looks larger and more consistent than in the Fig. 3 referred to in the previous article (9), where subjects were presented with a monaural stimulus; however, part of this is due to the difference in the calibration scale and to the fact that the figure is presented upside down relative to Fig. 3 (in Fig. 2, up is down and vice versa). Turn Fig. 2 over and at least three of the larger nystagmus beats look exaggerated because of an accompanying eye blink or muscle tension. If one eliminates the first beat in the record, because there is no indication of where the baseline began, and also eliminates the three beats that seem combined with blinks, and corrects for the time in which these eye movements occur, a nystagmus with a slow phase velocity of $1.5^\circ/s$ or less is again obtained. The authors do not state whether these eye movements were obtained during infrasonic stimulation or after infrasonic stimulation, as they were in the preceding study (9). Nevertheless, the eye movement tracing presented as typical of nystagmus produced by a binaural stimulation with 7 Hz infrasound is unimpressive and open to question as to whether it is a vestibular nystagmus, particularly since the nystagmus response was reported to occur at approximately 10 dB above threshold as indicated by their own threshold curve presented in Fig. 3 (10). Matters of procedure must be clarified before such "low level nystagmus" can be accepted as accurate.

Other forms of "inadequate" stimuli, such as alcoholic (1,16) and caloric (5) stimulation, can produce a vigorous nystagmus, whereas the two eye movement tracings of vertical nystagmus in the studies on infrasound (9,10) show the eye velocity to be in the same range as a weak, spontaneous nystagmus. If, according to the threshold curve (10), nystagmus was elicited at 120 dB after 60-70 s, why not show the response at 150 dB after the same period of time. Shouldn't a vigorous nystagmus be produced? Their discussion and threshold curves would lead one to expect at least a quasi-linear relationship (9,10). In a preliminary study conducted in our laboratory (19), no nystagmus was obtained at the 155 dB level, with a 5 min stimulus.

SUBJECTIVE MEASURES

The discussion of the "subjective" effects of infrasound is just as confusing as the discussion of the performance and nystagmus measures. The authors do not tell how the judgments were obtained or how they varied with the duration and intensity of the stimulus. The possibility of suggestion affecting the content of subjective judgment is very real, and particularly in this series of articles on infrasound because of the type of subjective response that was elicited. Evans *et al.* (9) point out: "... The subjective sensation most commonly reported is one of 'swaying' the apparent movement being away from the

ear under stimulation." Howard and Templeton (22) point out that the sway response is very susceptible to suggestion, and increase in sway as a function of suggestion has been proposed as a measure of hypnotizability. These authors (22) were concerned with the sway response and not with subjective sensation of sway; nevertheless, assessment of a person's subjective feelings must be handled meticulously because of the possibility of suggestion affecting the results. Investigators in the area of drug research typically use double-blind procedures and placebo groups because of the difficulty in obtaining valid reports of subjective experiences (28). These methods should prove useful in the study of the effects of infrasound since the stimulus is unique and ambiguous—ambiguous because the subject knows he is participating in an experiment on sound (if he is not told, he can still see the speakers or headphones)—and yet, as has been pointed out (6), the infrasonic stimulus below approximately 14 Hz loses its tonal quality and is experienced as "pulsating" or "throbbing." Furthermore, in discussing subjects' responses to monaural stimulation, Evans, *et al.* (9) point out: "... although the sound pressure levels required to stimulate a normal person are relatively high, the region of 140 dB, the sensations experienced are not particularly unpleasant." Therefore, how would a cooperative subject respond to a throbbing stimulus that is not unpleasant if he is pressed for a judgment? He might report that he felt swaying. People generally tend to try to make "sense" even out of ambiguous stimuli (see Schultz (33) p. 177 for an interesting summary of the role of set or suggestion in sensory restriction research, and see Geldard (14) p. 416 for a discussion of illusionary movements.

DISCUSSION

Regardless of whether performance, nystagmus, or subjective measures are considered, it seems certain that the adverse effects of infrasound reported at low-intensity levels either do not exist or have been exaggerated. In a recent popular article (3), infrasound has been discussed in a more conservative manner than in most of the articles reviewed in the present paper. However, the author emphasizes that there may be "sensitive subjects" who are affected at much lower intensity levels of infrasound than the general population. It would also have been desirable to point out that the burden of proof is on those investigators who claim to have demonstrated effects at very low levels, particularly on those who claim to have demonstrated effects at levels below the threshold of perception.

In another recent review, the author makes several suggestions from his review of the literature that the present authors believe are questionable. For example, the author (36) states: "... even fairly low amplitude (in the range of 70 dB) 'may' have physiological effects." And in another part of the paper, he suggests that "vestibular and cochlear dyscrasias" stem from infrasound in the 110-130 dB range. These suggestions would probably not have been made if the author had also reviewed the results of the "Colloquium on Infrasound" held in Paris, France, September 1973 (24).

REVIEW OF INFRASOUND—HARRIS ET AL.

The question remains concerning the intensity levels of infrasound that can be considered "safe." The United States Environmental Protection Agency, in the document "Public Health and Welfare Criteria for Noise, July 27, 1973," considers levels of infrasound below 130 dB as not constituting a public hazard (32). Studies conducted in our own laboratory (19,23,24,27,30,31,34,35) as well as the study by Borredon (2) suggest that considerably higher levels than 130 dB are also safe. We have been unable, using human subjects, to elicit nystagmus at intensity levels to 155 dB—our data also suggest that the level must be greater than 170 dB in animal subjects—and unable to demonstrate adverse effects on equilibrium at levels to 140 dB (19,24).

Certainly, more research is needed on the effects of infrasound on man. Duration of exposure and interaction of infrasound with sound in the audible range must be studied systematically. However, a statement by Lipscomb (25) concerning the effects of noise in general is worthy of consideration by anyone undertaking research in the infrasound area: "Be cautious. Many people are willing—yea, eager—to hop onto the 'cry wolf' bandwagon and rant about the grave problems noise and other contaminants are causing. Careless overstatements can result in unfortunate dilution of factual evidence. Alarmists create a credibility question which fosters contemptuous disregard for new knowledge."

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SECTION 30

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Public Health Service Grant #5A07 AH 00221-02	
<u>Citation</u> Haskell, B. S. Association of aircraft noise stress to periodontal disease in aircrew members. Aviation, Space and Environmental Medicine 46(8):1041-1043, Aug. 1975.		
<u># of Ref.'s</u> 19	<u># of Fig.'s</u> 5	<u>Language</u> English
<u>Type & duration of experiment</u> Short-term field study using x-rays and hearing threshold tests to evaluate groups of aircrew members for periodontal disease	<u>Purpose for study</u> The relationship between aircraft noise exposure and periodontal disease in aircrew members as measured by alveolar bone (the bony tooth sockets) loss.	
<u>Description of test groups (subjects, #, age, etc.)</u> 3 groups of 25 men from the Pennsylvania Air National Guard: Group 1 - jet pilots; Group 2 - pilots and crew of propeller aircraft; Group 3 (controls) - enlisted men not exposed to aircraft noise. (Average age of groups 1 and 2 similar, 8 years younger for controls)		
<u>Control of other stressors</u> Not controlled	<u>Statistical Methods</u> 2 x 2 factorial F test analysis of variance; correlation coefficients	
<u>Noise stimulus</u> source: aircraft noise spectral characteristics: not specified noise level: 94-118 dBA (various stages of flight) length of exposure: 1000 - 4000 flight hrs. # of trials: not applicable	<u>CVS Response Measured</u> None measured, but alteration of blood flow in coronary disease could be related to periodontal disease. <u>Nonauditory effects</u> Alveolar bone loss using full-mouth x-rays - the more flight hours, the more alveolar bone loss in propeller crew; propeller crew suffered greater bone loss than jet crew.	
<u>Author's conclusions</u> Alveolar bone loss was much greater in propeller crew members than in jet crew members, which was only slightly higher than in the controls with no aircraft noise exposure. Flight hours were correlated to hearing loss in both jet and propeller crew members. The difference may be due to the greater vibration associated with propeller aircraft.		
<u>Evaluation & comments</u> 1) More controlled laboratory studies are needed to understand this phenomenon. 2) The ages of the subjects were not specified.		

Association of Aircraft Noise Stress to Periodontal Disease in Aircrew Members

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MASKELL, B. S. Association of aircraft noise stress to periodontal disease in aircrew members. *Aviat. Space Environ. Med.* 6(8):1041-1043, 1975.

A review of the literature reveals a multitude of effects that noise may contribute to periodontal disease, including cardiovascular disease, angiospasm of peripheral vessels, hypertension, and an increase in inflammatory cells with concurrent inhibition of healing. Three groups of 25 men were selected from the Pennsylvania Air National Guard for study. Group 1 consisted of F-102 jet fighter pilots; Group 2, pilots and crew of a four-engine, propeller-driven C-121 aircraft; and Group 3, enlisted men not exposed to aircraft noise, as a control. The degree of alveolar, intraseptal bone loss for each subject was measured from full-mouth radiographs of all groups. The greatest amount of bone loss occurred in crew members of propeller-driven aircraft. Jet pilots had considerably less bone loss while the average number of millimeters of bone lost per tooth revealed a difference between the three groups to the 0.01 significance level ($F=24.7$). The data suggests there is a degree of alveolar bone loss over a period of years associated with exposure to propeller aircraft noise and vibration, and negligible loss for jet aircraft noise.

FOR MANY years it was believed that periodontal disease (pyorrhea) was caused largely by local irritating and mechanical factors, and was treated with some success in many of the cases. However, the disease occurred or persisted in many of the patients treated. It is now known that periodontal disease is the result of a complex of both local and systemic factors with no single etiology. There have been reports that psychosomatic factors defined as "anxiety" or "stress" often give rise to definite pathological processes in the periodontal structures. In this paper, the subject of noise "stress" as a factor in periodontal disease will be considered.

The autonomic nervous system exerts control over the blood supply and, thereby, the nutrition for all parts of the body. It is known that blood vessel adventitia contracts in an exaggerated fashion and may remain contracted for extended periods of time from emotional tension. It is also possible that periodontal pathosis

could occur as a physical response to an inadequate supply of oxygen and other nutrients or a continued blood vessel constriction.

Manhold (12), summarizing the previous reports on tissue metabolism, reported that gingival tissue respire at a normal rate commensurate with its state of health or disease. Under normal circumstances, increased activity on the part of tissue requires an increased quantity of oxygen and other nutrients. This is provided by (1) abstraction of a larger amount of oxygen from any given blood volume by elevation of the oxygen utilization coefficient, and (2) an increase of total blood flow to the tissue. The total blood flow can be increased by dilation of the blood vessels and opening new capillaries to the area.

If incipient periodontal breakdown were precipitated, as in oral neglect, the reparative process might not be adequately initiated as a result of stress. In this case, the total blood flow increase to the affected area might not occur because of interference with formation of new vessels, with vessels already dilated, or with any repair process previously underway.

Noise might contribute a multitude of effects to periodontal disease. Evidence for this may be summarized as follows:

- 1) Periodontal pathology and the weight of the supra-adrenals in experimental animals increased with auditory and other stress (9-11,13).
- 2) People professionally exposed to noise are subjected to cardiovascular stress severe enough to cause pathology (14).
- 3) Adolescents have a tendency to develop angiospasm of the peripheral vessels due to noise and vibration (16).
- 4) There is more arterial hypertension among workers subjected to noise than among office workers or manual laborers (15).
- 5) The reaction of bodily defense to inflammation in animals is partly inhibited with auditory stress (17,18).
- 6) There is an increase of inflammatory cells in the blood with noise stress (19).
- 7) Anxious and mentally disturbed people are more sensitive to noise, and this group as a whole is more liable to have periodontal disease (1-8).

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TABLE V. TABLE OF MEANS FOR ALVEOLAR BONE LOSS.

Flight Hours	Type of Aircraft		Total of Means
	Prop A)	Jet C)	
Less than 2400	0.57 mm	0.30 mm	\bar{X} A & C 0.44 mm
	B)	D)	\bar{X} B & D 1.23 mm
Greater than 2400	1.95 mm	0.51 mm	\bar{X} A & B \bar{X} C & D 1.22 mm
Total of means	1.22 mm	0.39 mm	

that the more hours aloft, the more alveolar bone loss was present, regardless of type of aircraft flown.

In Table V, the means for alveolar bone loss demonstrate that officers in the propeller group had the greatest bone loss $\bar{X}=1.22$, compared with the jet groups $\bar{X}=0.39$. Officers in the propeller group with over 2400 h of flight time had the greatest bone loss, $\bar{X}=1.95$. A T test conducted between the propeller group and the control group for bone loss also proved significant to the <0.05 level of confidence.

DISCUSSION

It is not surprising that there are similarly high correlations between flight hours and hearing thresholds of $r=0.67$ and $r=0.70$ for both Groups 1 and 2, respectively. However, it seems unusual that the correlation between flight hours and mm/T is negligible for Group 1, and so significant for Group 2. The analysis of variance also clearly indicates a significantly high amount of bone loss in propeller aircraft pilots with a high number of flight hours.

The noise level that jet pilots experience is negligible during flight, yet severe on the flight line, while the propeller-driven aircraft pilots and crew are exposed to intense noise in both situations. An explanation for the comparatively large amount of alveolar bone loss for the propeller pilots, while the correlations between flight hours and hearing threshold for both Groups 1 and 2 are nearly identical, may lie in the extreme difference in the type of noise and vibration for the two types of aircraft. An analogy might be drawn to Tsysar's work, in which subjects worked with jackhammers and other noisy and vibratory low-frequency construction instruments (16). In this group, a tendency for the development of angiospasm of the peripheral vessels was experienced. Another factor may be the difference of in-flight atmospheric pressure between jet and propeller aircraft.

Although aging plays a large role in the amount of alveolar bone loss, the average ages for Groups 1 and 2 are only 1 year apart. Group 3, whose average age is approximately 8 years younger than the other groups, has a significantly lower mm/T score.

The importance of the role of psychological stress has already been reported. It is not known if there is inherently more stress in flying a large multieinged aircraft with a crew and dozens of passengers, than in flying the single-seat jet aircraft, but this too may be a factor.

It is assumed that there is no difference in the type of person who becomes a jet or propeller pilot, since the choice is made by the Air National Guard strictly on the basis of need. A pilot may be asked to switch from one type of aircraft to another when accepting a position with the Guard.

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AIRCRAFT NOISE & PYORRHEA—HASKELL

MATERIALS AND METHODS

In order to test the possibility that noise stress contributes to periodontal bone loss in humans, an experiment was devised to ascertain the degree of alveolar bone loss in persons subjected to aviation noise stress. Three groups of 25 men with complete medical and dental records were selected from the Pennsylvania Air National Guard. Group 1 consisted of F-102A jet fighter pilots; Group 2, of pilots and flight crew of the four-engine, propeller-driven C-121 aircraft; and Group 3, of enlisted office workers not exposed to aircraft noise, as a control. In order to determine the total amount of noise exposure for Groups 1 and 2, the number of hours of flight time was noted for each man.

In Group 1, the jet pilots wear custom-fitted helmets, specially equipped with ear protectors, thereby making cockpit noise in flight negligible. While on the ground at the flight line, these pilots may be exposed to extreme noise, usually without the benefit of the protective flight helmets. It was only 6 months prior to this study that a new directive went into effect requiring ear plugs or a helmet to be worn at the flight line.

In Group 2, the flight crew is without protective headgear and is continually exposed to noise on the ground and while flying.

Current full-mouth radiographs, of all three groups, taken with the prescribed USAF Dental Corps Technique, were examined on a standard radiograph view-box. The degree of existing bone loss was charted and measured with a boley gauge caliper to the nearest 0.1 mm. The total number of millimeters of bone loss was then divided by the total number of existing teeth, thus giving each individual a score of the average number of millimeters of alveolar bone lost per tooth (mm/T). These scores represent an underestimate in all films because it is impossible to measure bone height on the buccal and lingual surface of each tooth in the radiographs.

Hearing threshold tests, performed on the Maico audiometer at the ISO setting, were also recorded for all groups. (ISO: International Standard Organization (1964) F=1000 Hz 0-6.5 dB re 0.0002 dyn/cm²). Since prolonged noise is known to be damaging to hearing, it seemed reasonable to compare the easily identifiable parameter of hearing threshold to the amount of flight hours, thereby indicating a measurable degree of hearing loss for Groups 1 and 2. Hearing loss was then compared for each individual to the number of flight hours and with the increase of alveolar bone loss to determine possible associations. An average was calculated for the hearing threshold of right and left ears, at a frequency of 4,000-6,000 Hz.

RESULTS

The levels of sound in decibels measured with a sound level meter for Groups 1 and 2 are given in Table I. The present Federal Standard is 90 dBA for an 8-h exposure. The averages of flight hours, mm/T, hearing threshold, and age for all three groups are given in Table II.

An F test analysis of variance revealed a difference between the three groups to the 0.01 level of significance for both average mm/T ($F=24.7$) and average hearing loss in decibels ($F=7.54$).

TABLE I. AIRCRAFT NOISE LEVELS IN DECIBELS.

Flight Line*	Group	
	Group 1 F-102 A Jet Aircraft	Group 2 C-121 Propeller Aircraft
Aircraft at idle	105	94
Maintenance generators and other support equipment	101	102
In-Flight**		
Takeoff	Negligible	118
Climb	"	116
Level Flight	"	113

*Flight line conditions to which all pilots have been exposed without mandatory ear protection.

**In-flight ear protection maintained only by jet pilots.

TABLE II. GROUP AVERAGES FOR FLIGHT HOURS, BONE LOSS, HEARING THRESHOLDS, AND AGES FOR STUDY POPULATION.

	Group Averages \pm S. D.*		
	1 Jet	2 Propeller	3 Control
Flight hours	2,301 \pm 1,222	2,813 \pm 2,440	none
mm/T	0.39 \pm 0.28	1.22 \pm 0.90	.17 \pm .18
Hearing threshold in decibels	24.6 \pm 12.7	20.8 \pm 13.4	14.8 \pm 6.7
Age	34 \pm 4.9	33 \pm 8.7	26 \pm 3.0

* = Standard Deviation

TABLE III. GROUP CORRELATION COEFFICIENTS FOR FLIGHT HOURS WITH ALVEOLAR BONE LOSS AND HEARING THRESHOLD.

	Flight Hours/ (mm/T)	Flight Hours/ Hearing Threshold*
1. Jet	$r = 0.29$	$r = 0.67$
2. Propeller	$r = 0.75$	$r = 0.70$
3. Control	no flight hours	no flight hours

*Pearson's r correlation coefficient = significant correlation.

TABLE IV. THE ANALYSIS OF VARIANCE RESULTS FOR JET VS. PROPELLER SCORES.

Source of Variation	df	Sum of Squares	Mean Squares	F Ratio
Hours in Flight Time	1	7.59	7.59	36.14*
Jet-Prop	1	8.83	8.83	42.04*
Interaction	1	4.06	4.06	19.33*
Error	44	9.49	0.21	
TOTAL	47	29.97		

* = significant at < 0.01

Correlations between flight hours and mm/T, and between flight hours and hearing thresholds for Groups 1 and 2 are given in Table III. This analysis indicated there was no significant correlations in Group 1 between flight hours and mm/T, while all the other correlations indicated are significant to the 0.01 level of confidence.

In Table IV, the results of a 2×2 factorial analysis of variance indicate that there are significant interaction effects between the number of flight hours and the type of plane. An examination of the flight times also indicated

SECTION 31

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> P. M. Nealis R. E. Bowman, Ph.D.	<u>Institution and address where research was performed</u> University of Wisconsin, Primate Laboratory 22 N. Charter Street Madison, Wisconsin 53706
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> National Science Foundation U.S. Public Health Service
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Citation
 Nealis, P. M. and Bowman, R. E. Behavioral and corticosteroid responses of rhesus monkeys to noise-induced stress... (unpublished paper).

<u># of Ref.'s</u> 22	<u># of Fig.'s</u> 3	<u>Language</u> English
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<u>Type & duration of experiment</u> Laboratory experiment; over 36 days.	<u>Purpose for study</u> The effect of different types of noises and varying exposures on corticosteroid response and behavior of rhesus monkeys.
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Description of test groups (subjects, #, age, etc.)
 16 wild adolescent rhesus monkeys (8 males, 8 females).
 3 test groups - 2 males, 2 females each - 3 noise types presented randomly for each
 1 control group - 2 males, 2 females - no noise

<u>Control of other stressors</u> laboratory conditions - in sound-proof chamber; visual & auditory contact with 3 other subjects in each group, but no tactile contact.	<u>Statistical Methods</u> 3 x 3 factorial design; f-tests, analysis of variance
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<u>Noise Stimulus</u> source: continuous, variable, or impulse tape recorded noise spectral characteristics: frequency varied with variable noise (music) noise level: average level = 100 dBA length of exposure: 1, 2, and 5 hrs. (2 30-min. recordings of each noise type) # of trials: unspecified	<u>CVS Response Measured</u> None <u>Nonauditory effects</u> plasma cortisol level - initial rise during first hour of noise exposure; normal after 3 and 5 hrs. noise behavior - greater fatigue due to noise after 3 hrs. continuous or variable noise or after 5 hrs. impulse noise
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Author's conclusions
 Corticosteroid levels were elevated significantly after one hour of noise exposure but not after 3 or 5 hrs. This indicates an adaptation after 3 and 5 hours of noise. Type of noise did not affect the plasma cortisol responses, but did affect behavioral response.

Evaluation & comments
 Plasma cortisol responses to noise seem to be similar in monkeys and humans. Behavioral responses would be more difficult to compare.

Behavioral and Corticosteroid Responses of
Rhesus Monkeys to Noise-Induced Stress¹

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Adrenal and Behavioral Responses to Noise

Abstract

Adolescent rhesus monkeys were exposed in randomized block order to 1, 3, and 5 hr sessions of continuous, variable, and impulse noise. As an index of physiological trauma, plasma cortisol concentration was measured immediately at the termination of each auditory exposure. Immediately thereafter behavioral and social activities of the monkeys were charted by a modified frequency counting technique. At the termination of all 1 hr noise exposures, plasma cortisol levels were elevated to 80 per cent of maximum rise, but had returned to control levels at the termination of all 3 and 5 hr noise exposures. After 1 and 5 hr exposures to continuous and variable noises, the monkeys generally exhibited control levels of behavior, but after 3 hr exposures they showed reduced behavioral activity with corresponding increases in nonsocial behavior, especially sleep. In contrast, this behavioral alteration was not induced by impulse noise after 1 and 3 hr exposures, but was seen after 5 hr exposures. These findings suggest extra-auditory effects of noise in reducing arousal or in producing a general fatigue that is manifest in a "free response" social situation.

Corticosteroids Noise Rhesus Monkey Social Behavior Stress

Behavioral and Corticosteroid Responses of
Rhesus Monkeys to Noise-Induced Stress

The prevalence of noise in residential areas, places of public use, and work situations has generated considerable concern about the possible effects on man of this kind of sensory stimulation. A well-documented effect of exposure to intense noise is hearing loss manifested as either a transient or a permanent shift in auditory sensitivity thresholds. However, the extra-auditory effects of noise are not well understood (see Leake, 1970). Research has concentrated mainly on the effects of noise on task performance by human subjects, although the methods and findings of such experimentation remain somewhat controversial (Broadbent, 1957; Kryter, 1966). Moreover, noise-induced changes in task performance offer little generalization to other important aspects of human behavior. In particular, the possible effects of noise on social behavior have been largely unexplored. A major objective of the present study, therefore, was to delineate some of the consequences of prolonged noise exposure in regard to the behavioral responses of a primate species. The rhesus monkey was employed since it has often proved to be an appropriate species for modeling human behavior.

Emotional states in humans following noise exposure have been extensively investigated by the self-report method (e.g., Kryter, 1966; Laird, 1929; Laird and Cove, 1933). For research on the monkey, there seems to be no objective index of emotionality that embodies specificity as to the quality or intensity of the sensation produced. However, considerable evidence exists to suggest that the activity of the pituitary-adrenal cortical system provides

a reliable measure of physiological and psychological trauma induced by environmental stressors (Bohus, 1968; Mason, 1968; Meyer and Bowman, 1972). A few investigators have, in fact, used corticosterone levels as indices of noise-induced stress in both humans (Arguelles, Ibeas, Ottone, and Chekherdeman, 1962) and rats (Henkin and Knigge, 1963). The general characteristics of the pituitary-adrenal system of the monkey are well documented as well as the response of this system to psychologically stressful conditions (Mason, 1968; Meyer and Bowman, 1972). Consequently, it was thought that total plasma cortisol concentration could be an effective measure of physiological trauma in rhesus monkeys following noise stimulation.

The physical characteristics of noise are apparently related to certain deleterious effects on the organism (Kryter, 1950; Laird, 1929; Lindquist, Neff, and Schuknecht, 1954; Reese and Kryter, 1944). Apart from loudness, the method of presentation appears to be a critical factor since steady-state noise is less disturbing to humans than intermittent sounds (Plutchik, 1959; Smith, 1951). These findings come from noise studies that employed unusual stimuli such as clicks and white noise presented at extremely high amplitude, e.g., 120-160 dB. In the present experiment, however, noises commonly found in human situations, e.g., in the operation of power tools, were presented at an intensity level that would be common to such situations. It was hypothesized that monkeys would require more time to adapt to intermittent noise than to constant noise, and that increases in noise exposure duration would produce increases in the severity of any deleterious effects observed.

Method

Subjects

Sixteen feral adolescent rhesus monkeys (Macaca mulatta), 8 males and 8 females, served as subjects. To facilitate the scheduling of treatments and observations, the monkeys were assigned to 4 groups of 4 subjects. Pairs of subjects within each group were housed together in wire cages throughout the study with feeding and maintenance schedules closely controlled.

Apparatus

An Ampex model AG600 monaural tape recorder was used to record and play back auditory stimuli. A Neumann omnidirectional condenser microphone provided acoustic pickup while reproduction of the sound was accomplished with an Altec model 1594A amplifier and Electrovoice Sentry IV speaker system (frequency response of 50-20,000 Hz). Noise treatments were administered in a sound-proofed chamber (1.8 X 1.8 X 7 m) that served to attenuate external incidental noise by 30 dBA. A previously described playroom (Harlow, Rowland, and Griffin, 1964) was utilized during pre- and post-treatment behavioral tests.

Auditory Stimuli

Three types of noise stimulation were employed: continuous (C), variable (V), and impulse (I). Two 30 min. recordings of each noise type were produced on magnetic tape at 7.5 ips. In Condition C the noise consisted of continuous sounds of power tools (21 min) and land vehicles (9 min) with the total time for each distributed randomly over the 30 min recordings. The noise for Condition V consisted of "rock" music of the type currently popular; both instrumental and instrumental-vocal arrangements (15 min each) were randomly distributed throughout a recording. The amplitude and frequency of V noise, unlike the steady-state characteristics of C noise, tended

to vary within any given segment because of the nature of music. The I condition was a random collection of shotgun blasts, pistol shots, and bursts of machine gun fire. The occurrence of any one of these sounds was at random. The total noise time in Condition I was approximately 2 min per 30 min recording in contrast to conditions C and V in which noise was present for the entire 30 min period.

Design and Procedure

A 3 X 3 factorial design with repeated measures was employed that consisted of 1, 3, and 5 hr exposures to C, V, and I noise presented at an average amplitude of 100 dBA. Three groups of monkeys were assigned to the noise conditions with the order of treatments randomized differently for each group. The remaining group of four monkeys served as a no-noise control. This latter group followed the same experimental procedure as the other three groups except that 3 no-noise sessions were given at each of the 3 durations. The experimental conditions were administered to all groups over a period of 36 days with treatments separated by a minimum of 90 hr. The monkeys were treated and tested in their respective groups throughout the study and also during 36 days of pre-experimental sessions that were designed to facilitate acclimation to test procedures. The subjects were caged individually during confinement to the sound-proofed chamber and had visual and auditory, but no tactual, contact with other members of their group. Noise and no-noise sessions commenced at different times, depending on the duration of each, but always ended at 15:15 hr.

Cortisol Assay. Plasma cortisol concentrations were monitored throughout the study. A blood sample of 0.4 ml of saphenous or femoral blood was taken with a dried, heparinized syringe at 15:15 hr on the day prior to each

noise or no-noise treatment. Blood samples were also collected immediately after noise or no-noise treatments, i.e. at 15:15 hr. All blood samples were centrifuged immediately, four 10 μ l plasma aliquots taken, each diluted with 190 μ l of distilled, deionized water, and frozen at -15° C until assayed blind in duplicate by protein binding radioassay for cortisol content (Bowman and De Luna, 1969). An assessment was also made of each monkey's maximal cortisol response to an intramuscular injection of 16 units/kg of ACTH (Armour Acthar), a method that results in maximal stimulation of the adrenal cortex in rhesus monkeys (Bowman and Wolf, 1969). Blood samples were collected at 0, 1, 3, and 5 hr post injection and assayed in the manner just described.

Behavioral Test. Noise-induced changes in the behavioral activity and social interactions of the monkeys were charted by a modified frequency counting technique (Suomi, Harlow, and Kimball, 1971) that consisted of 45 consecutive 20-sec intervals per subject/session. The behavioral taxonomy reported in this study is presented in Table 1. Interobserver reliability reached or exceeded the 0.95 level by the product moment correlation method for each behavioral category. A pre-treatment observation period commenced at 9:15 hr and a post-treatment observation period began at 15:45 hr on each day of noise and no-noise treatments. The monkeys were given 15 min to acclimate to the playroom before testing began.

Data Analysis. All data were converted to differences between pre- and post-treatment measures in order to assess changes due to treatments. These pre-post differences are termed "change scores" in this report. The change scores exhibited by the no-noise control monkeys under the various exposure durations were assumed to reflect the effects of no-noise experimental procedures such as confinement to the treatment chamber, handling, and venipuncture. Therefore the change scores of noise-treated subjects for each

exposure condition were corrected by subtraction of the mean change score exhibited by no-noise control subjects at the corresponding exposure duration. In this regard, it should be noted that analysis of variance revealed no significant effects of exposure duration on the change scores of no-noise controls for any of the dependent measures reported here. Under the null hypothesis of no effect of noise, the expected value, μ , of these corrected change scores would be zero. The corrected change scores for each dependent variable were then subjected to analysis of variance with planned contrasts.

Insert Table 1 about here

For significant F 's (linear) at $\alpha = .05$, subsequent orthogonal F tests were done on the differences of the mean corrected change scores from the null hypothesis value of zero.

Results

Cortisol Data. The monkeys' cortisol responses did not appear to be differentially affected either by the type of noise stimulation or by the interaction of noise type and exposure duration (F 's ≤ 1), although the main effect of exposure duration was statistically significant ($F = 24.4$, $df = 1/11$, $p < .0005$). An elevation in plasma cortisol concentration was observed at the end of 1 hr noise exposures, but no elevation was seen after 3 and 5 hr noise exposures. The mean changes in plasma cortisol concentration of noise-treated monkeys, corrected by the mean changes exhibited by control monkeys, are shown in Fig. 1 as a function of exposure duration.

Insert Figure 1 about here

When these changes in plasma cortisol levels were compared with maximal plasma cortisol responses to injection of ACTH, the noise stimuli did not appear to be maximal stressors. The activity of the pituitary-adrenocortical system was at 80 per cent of maximum following 1 hr noise exposures (45 μ g% vs 57 μ g%). After 3 and 5 hr exposures to noise, the monkeys exhibited plasma cortisol concentrations of 40 per cent (35 μ g% vs 86 μ g%) and 33 per cent (32 μ g% vs 96 μ g%) of maximal response, respectively, which approximates control levels of plasma cortisol under no-noise conditions. Only the plasma cortisol concentrations following 1 hr noise exposures reached significance at the 99 per cent confidence level. It can be noted that the control monkeys showed nonsignificant pre- to post-treatment increases in plasma cortisol concentrations of 4, 5, and 6.5 μ g% after 1, 3, and 5 hr no-noise exposures, respectively.

Behavioral Data. The following behavioral categories showed pre- and post-treatment frequencies that were too low to justify analysis: active disturbance, aggression, and submission. The occurrence of locomotion and exploration was of higher frequency, but yielded nonsignificant F ratios (all p 's $> .08$). However, corrected change scores for dependent measures of sleep ($F = 8.0$, $df = 1/11$, $p < .02$), passive disturbance ($F = 4.8$, $df = 1/11$, $p < .05$), social contact ($F = 7.9$, $df = 1/11$, $p < .02$), and social play ($F = 5.2$, $df = 1/11$, $p < .05$) were statistically significant in the analysis of the interaction of noise type and noise exposure duration.

Insert Figure 2 about here

After 1 hr exposures to C noise, the monkeys exhibited significant increases in passive disturbance and social contact, although levels of sleep and social play remained unchanged (see Fig. 2). However, after 3 hr exposures to C noise, significant increases in sleep were observed and were accompanied by significant decreases in both social play and social contact. Following 5 hr exposures to C noise, the monkeys exhibited significant increases in social play, but other behaviors remained at normal levels.

One-hour exposures to V noise resulted in significant decreases in sleep and significant increases in social play (see Fig. 2). This play behavior appeared to be contact-type play as reflected by significant increases in social contact for 1 hr exposures to V noise. After 3 hr exposures to V noise, both disturbance activity and sleep were significantly increased and social play was significantly decreased. After 5 hr exposures to V noise, the monkeys exhibited baseline levels in all behavioral categories except social play which remained at subnormal levels.

All behaviors remained unchanged following 1 hr and 3 hr exposures to I noise (see Fig. 2). Following 5 hr exposures to I noise, however, significant increases in sleep and passive disturbance were observed, accompanied by significant increases in social contact, and no change in social play.

Discussion

The present data indicate that the pituitary-adrenocortical response to continuous, variable, or impulse noise was similar to that of humans (Arguelles *et al.*, 1962) and lower mammals (Henkin and Knigge, 1963). There was an initial rise in plasma cortisol concentration during the first hour of noise exposure, but an adaptation to normal cortisol levels after 3 and 5 hr

noise exposures. This suggests an initial stress or fear followed quickly by an emotional adaptation.

By contrast, the social behavioral data indicate a more complex picture, relatively uncorrelated with the adrenocortical response. The generalization most apparent in these data was that noise produced a soporific or fatiguing effect on social behavior immediately following certain durations of exposure to the different noises. For example, continuous machine noise produced increased passive disturbance after 1 hr of exposure and increased sleep after 3 hr of exposure; the monkeys then appeared to adapt or recover following 5 hr exposures, showing normal sleep, passive disturbance, and social contact levels, but also showing elevated social play. By contrast, variable noise (music) produced increased social play after 1 hr of exposure, very heightened sleep and passive disturbance after 3 hr of exposure, and recovery to nearly normal behavioral levels after 5 hr of exposure. Finally, impulse noise (gunshots) produced no measured effects on subsequent social behavior after 1 hr and 3 hr exposures, but very heightened sleep, social contact, and passive disturbance after 5 hr of exposure.

Within the exposure durations tested, the monkeys exhibited both onset of and recovery from the soporific effect of both continuous and variable noise, and a more delayed onset of a soporific effect after the longest exposure to impulse noise. Unfortunately, these data in themselves offer little guide to prediction of the effects of longer durations of exposure to noise, or of longer sequences of repetition of exposures. However, these data do raise the possibility that one cost of noise exposures in the human environment may be a fatigue effect that will depress free responding such as that seen in social situations during periods immediately following the noise exposures.

The present data further clarify the monkey's pituitary-adrenocortical response to noise stressors. Rhesus monkeys, like humans (Arguelles *et al.*, 1962) and lower mammals (Henkin and Knigge, 1963), show initial high rates of cortisol secretion in response to noise exposure, but this response is attenuated by further exposures to the stressor. However, the corticosteroid response shows no differentiation among different types of noise. Contrariwise, the behavioral data suggest a parameter of noise effects that seems sensitive to the nature of the noise stressor. Whether or not the behavioral responses of a nonhuman primate species to noise stimulation generalize to the human situation is purely speculative at this time. Perhaps cognitive mediating factors play an important role in the human's struggle to adapt to unusual noise conditions as it appears might be the case in rhesus monkeys (Hanson, Larson, and Snowden, 1976). Little is known about this factor except that humans show reduced tolerance for frustration under noise conditions, but to a lesser degree when given the impression that they can exercise control over the noise stressor (Glass, Singer, and Friedman, 1969). Although the study of human behavioral reactions to noise is a difficult area of investigation, it may be of significance to determine how man socially behaves following exposure to noise, particularly in light of the present findings.

Footnotes

1. This research was supported by NSF Grant GY-9634 to the first author, and by USPHS Grants MH-11894 and RR-00167 to the University of Wisconsin Primate Laboratory and Regional Primate Research Center, respectively. The authors wish to thank R. De Luna, R. Goldschmidt, J. Klann, E. Hoffman, P. Lehninger, M. Levinson, P. O'Neill, P. Plubell, C. Polnazcek, and M. Walker for their efforts in carrying out the experiment.
2. Reprint requests should be addressed to Robert E. Bowman, University of Wisconsin Primate Laboratory, 22 N. Charter Street, Madison, Wisconsin 53706.

Figure Captions

1. Mean changes in total plasma cortisol concentrations as a function of noise exposure duration. No change with respect to control levels is represented by zero on the ordinate. The 99 per cent confidence interval is shown as the shaded area.
2. Mean changes in the frequency of four behavioral measures as a function of continuous noise (C) vs variable noise (V) vs impulse noise (I) and exposure duration. No change with respect to control levels is represented by zero on the ordinate. The 99 per cent confidence intervals are shown as shaded areas.

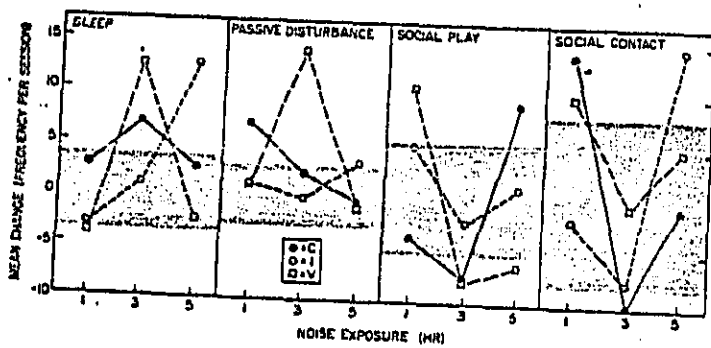
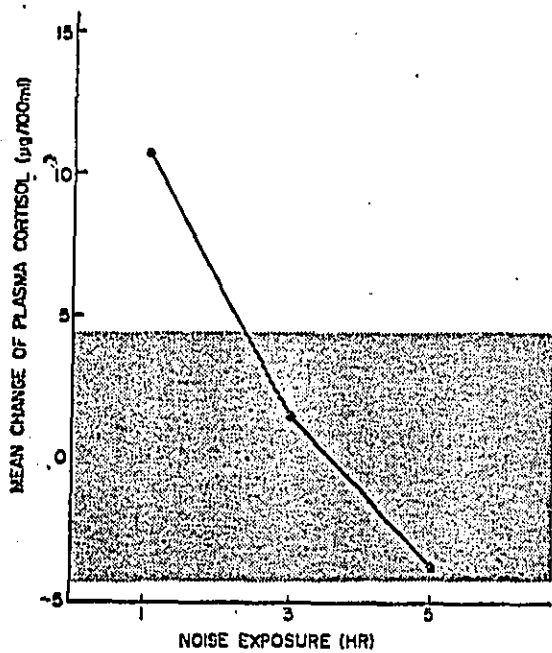


Table 1

Behavioral Taxonomy Employed in Playroom Testing

Social Behavior

Social Play: any socially directed play activity, including rough-and-tumble and noncontact play.

Social Contact: tactual contact with another subject exclusive of aggression, grooming, and play.

Aggression: hair pulling, biting, and/or facial threat (an expression consisting of ears pulled back with lower jaw pulled down and out).

Submission: fear grimace (facial expression resembling a smile, produced by pulling the lips back to expose teeth) and/or withdrawal from, or sexually presenting to, an aggressor.

Nonsocial Behavior

Locomotion and Exploration: ambulation of one or more full steps and/or visual, oral or tactual exploration/manipulation of environmental objects.

Sleep: resting in a sitting or prone position with head lowered and eyes closed.

Active Disturbance: self-mouth (oral contact, excluding discrete biting, with any part of the body), self-bite (specific, vigorous, self-directed bite), rock (repetitive, nonlocomotive forward and backward movement), or stereotypy (identical movements, rhythmic and repetitive, maintained for at least three cycles).

Passive Disturbance: self-clasp (manual or pedal clutching of any part of the body) and/or huddle (self-enclosed "fetal" position with head lowered beyond shoulder level, including any other patterns of self-clasp and self-embrace).

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SECTION 32

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> Lt. Col. Prof. G. Paolucci, IAF, MC	<u>Institution and address where research was performed</u> Centro di Studi e Ricerche di Medicina Aeronautica e Spaziale; Via P. Gobetti, 2/a 00185; Roma (Italy)
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u>
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Citation
 Paolucci, G. Influence of noise on catecholamine excretion. IN AGARD (Advis. Group Aerosp. Dev.) Conf. Proc. 171: C9-1-C9-2, 1975.

<u># of Ref.'s</u> 0	<u># of Fig.'s</u> 1	<u>Language</u> English
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<u>Type & duration of experiment</u> Field, short-term (1½ - 5 hr.)	<u>Purpose for study</u> To examine the effects of two different high intensity noises (continuous and intermittent) as stressors on urinary catecholamine excretion.
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Description of test groups (subjects, #, age, etc.)
 20 Air Force ground specialists usually employed in noisy tasks (wearing ear plugs) divided into two test groups of 10 each: Group A - continuous workday noise of 120 dB for 1½ hrs. Group B - intermittent impulse noise of 80 - 100 dB for 5 hrs. (Each subject served as his own control - catecholamines tested on nonworking days for control values)

<u>Control of other stressors</u> no control	<u>Statistical Methods</u> not given
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<u>Noise Stimulus</u> source: Group A - jet engine noise Group B - jet takeoff runway noise spectral characteristics: not specified noise level: Group A - 120 dB continuous Group B - 80 - 100 dB intermittent length of exposure: Group A - 1½ hrs. Group B - 5 hrs. intermittent impulse (a few sec.) noise every 20 min. # of trials: 1 workday and 1 non-workday per subject	<u>CVS Response Measured</u> none <u>Nonauditory effects</u> urinary catecholamine levels - no significant increases after a few hours noise exposure (no adrenal gland response); the levels were all within a normal range
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Author's conclusions
 Catecholamines did not increase significantly as a result of noise exposure for a few hours, indicating lack of adrenal gland response. Lack of response may be due to ear protection and habituation to noisy work environment.

Evaluation & comments
 The inclusion of a control group not normally exposed to noise would have been useful. The age and length of aviation service of the subjects was unspecified.

"INFLUENCE OF THE NOISE ON CATECHOLAMINE EXCRETION"

by
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SUMMARY

Aim of the work was to know whether a few hours exposure to hazardous noises could act as a stressing factor and so able to give increase on catecholamine excretion.

The study was carried out on aviation specialists, daily exposed to high noises, fully protected against hearing damage by ear plugs; the exposed people were divided in two groups of ten subjects, each one exposed to different noisy conditions:

- the one was exposed to continuous and steady noise of 120 dB for 1 hour and 1/2;
- the other to intermittent noise of 80-100 dB for 5 hours, with intervals between impulsive bursts of 20', lasting each one only a few seconds.

The subjective tolerance was good and no disturbance or fatigue reactions appeared at the end of the exposure. Urinary catecholamine excretion was assayed the day before the test (in noiseless place) and the next one at the end of the exposure.

As the test values, compared with the blank, showed no changes in catecholamine release, it can be argued that, upon trained people, with hearing fully protected, noise might not act as a conventional stressor, at least at the same conditions of the present research.

INTRODUCTION AND AIM OF THE WORK

The exposure to high levels of noise for a certain duration, apart the auditory effects, can lead to a loss of working efficiency.

The damages of noise upon eardrum and hearing organ are not be taken into consideration in this paper, in which only endocrinological aspects are to.

Since the long exposure to noise, in most people can produce headache, drop on attention, loss of resistance, we intended to establish whether the noise could act as a stressing factor, able to give rise to increased output of catecholamines from adrenal gland.

METHOD

On 20 IAF ground specialists, usually employed in noisy tasks (wearing ear plugs during the job), this research was carried out. The people was divided in 2 groups of 10 persons, unhomogeneous for age and body size, which were exposed for different durations to different high noise conditions.

In particular, the exposure was executed in the following way in two different places:

- "A"- "engine test area": the workers remained exposed for 1 hour and 1/2; the noise was continuous and steady at level of about 120 dB;
- "B"- "take-off runway": the workers' exposure lasted 5 hours, but the noise was intermittent and lower; it arised at every 5-10' take-off (one every 20') lasting only a few seconds and reaching about 80-100 dB.

In such people catecholamines were determined in a day off and after the noise exposure; the former was indicated "BLANK" and the latter "TEST". The analyses were executed according to BIO-RAD technique and the excretion values are reported in mcg/h.

As the "TEST" urinary specimen was collected at the end of exposure, and corresponded to the urine flown in bladder during all time of exposure, also urinary specimen of "BLANK" was collected for the same time, in the day before the test, in the same subjects resting in a noiseless place.

CMJ

RESULTS

The following table shows the data obtained:

	Area "A" (continuous noise)	Area "B" (intermittent noise)
NOISE LEVEL (dB)	120	80-100
EXPOSURE DURATION (h)	1 1/2	5
BLANK (mcg/h)	4.82 ± 2.91	4.07 ± 4.74
TEST (mcg/h)	9.36 ± 3.03	4.74 ± 3.00

(We recall that normal catecholamine excretion, in other previous research determined, is 3-5 mcg/h)

At the end of the exposure all the subjects didn't feel tired and no fatigue symptoms appeared

CONCLUSIONS

This research has shown that no change happens on catecholamine excretion after a noise exposure for a few hours; all this seems to mean that noise can be heard without any adrenal gland response (perhaps human tolerance can be due to ear protection and noise training).

The catecholamine release was similar in all the workers but one, high duty responsibilities charged, whose "TEST" value was 4 times higher than "BLANK", confirming what we have already achieved in previous experiences, that psychic loads can cause catecholamine increases.

DISCUSSION

Q. I believe your method was concerned with the total measurement of catecholamine excretion. Have you obtained no effects by measuring total catecholamine excretion although the results were very variable? Is it possible that if you had measured differential catecholamine excretion such as the separate components of it that you would have found some changes? Also, subcortical-steroids, at least as far as animal experiments are concerned, are very sensitive to noise. Have there been experiments done in man or is it more appropriate to use cortico-steroids in these types of estimations?

A. Catecholamine excretion is different from the cortico or the adreno cortico-steroid reaction because the catecholamine excretion is quickly responsive to stress while the cortico or 17-cortico-steroids produce a progressive reaction. Our experiments lasted for a short time and in this time no stress reaction was observed. We think that such stress might occur in humans to prolonged exposures at approximately 100 db.

SECTION 33

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> J. F. Pritchett, R. S. Caldwell, R. K. Chesser, J. L. Sartin	<u>Institution and address where research was performed</u> Dept. of Zoology-Entomology Agricultural Experiment Station Auburn University; Auburn, Alabama
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<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u>
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Citation
 Pritchett, J. F. et al. Effect of jet aircraft noise upon in vitro adrenocortical response to ACTH in feral Mus musculus. Life Sciences 18(4):391-6, Feb. 15, 1976.

<u># of Ref.'s</u> 16	<u># of Fig.'s</u> 2	<u>Language</u> English
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<u>Type & duration of experiment</u> Study of field exposure to noise; short term mice were collected over a 2-day period and adrenals assayed in 1-day.	<u>Purpose for study</u> To examine <u>in vitro</u> adrenocortical response of wild mice exposed to noise vs. a control group when given ACTH; to study difference in response to noise in domestic and wild mice.
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Description of test groups (subjects, #, age, etc.)
 2 groups of wild field mice: Group A - 14 adult male mice collected near the end of a runway at Memphis International Airport; Group B - 9 adult male mice from a control field 2 kilometers from the airport field..

<u>Control of other stressors</u> animals exposed in the field - no control background noise levels for test and controls about 85 dB	<u>Statistical Methods</u> Student's t-test; standard error; 2x2 factorial design for analysis of variance
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<u>Noise Stimulus</u> source: jet aircraft noise spectral characteristics: not specified noise level: average of 110 dB length of exposure: not known but through whole life of each animal # of trials: not applicable	<u>CVS Response Measured</u> None <u>Nonauditory effects</u> Adrenal glands assayed <u>in vitro</u> for corticosterone secretion rates with and without added ACTH. Basal secretion rates without ACTH were higher in noise-exposed mice than in controls; adrenal response to added ACTH was significantly greater in control than in noise exposed mice.
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Author's conclusions The jet aircraft noise to which the mice in this study were exposed interacted in the body such that the in vitro cortical responsiveness to added ACTH was decreased. Previous researchers have found that ACTH did increase the cortical response of adrenals in noise exposed animals. The present results may disagree due to the lack of a pre-incubation period for the adrenals.

Evaluation & comments
 More standard and more accurate adrenal assay methods need to be devised so that different studies can be compared more easily.

EFFECT OF JET AIRCRAFT NOISE UPON IN VITRO ADRENOCORTICAL
RESPONSE TO ACTH IN FERAL MUS MUSCULUS

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(Received in final form December 29, 1975)

Paired adrenals from feral male Mus musculus trapped from fields adjacent to the Memphis (TN) International Airport and from suitable control areas were incubated in the presence or absence of ACTH. Incubation media were assayed fluorometrically for corticosterone. Control animals exhibited lower basal secretion rates of corticosterone when compared to their noise exposed counterparts. However, ACTH elicited a significantly greater increase in corticosterone secretion in controls as compared to the noise exposed group. The data suggest a noise related decline in adrenal cortical responsiveness to ACTH.

A review of the effects of high intensity noise upon domestic and feral animals suggests that little is known of specific noise-induced changes in adrenal cortical physiology in feral populations (1). Such changes in domestic and laboratory species are better documented.

Osintseva and co-workers reported a decline in adrenal weight in rats exposed to 80 db SPL (sound pressure level) for periods up to 126 days (2). Others have reported increases in adrenal weight in rats exposed to various types of noise ranging from 95 to 100 db SPL for various lengths of time (3, 4). We have observed significant increases in adrenal weight in feral Mus musculus exposed to jet aircraft noise both in the field and with laboratory simulation (unpublished data).

Decreases in numbers of circulating eosinophils have been reported in mice, rats, and guinea pigs subjected to 140-140 db SPL for intermittent periods up

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to 12 weeks (5-8). In addition, the depletion of adrenal ascorbic acid has been noted in rats incident to chronic exposure to intense noise (2). Jurtschuk and co-workers reported an increase in adrenal ascorbic acid with a shorter noise (120 db SPL) exposure time (11-15 days) (3).

Hiroshige, et al. have presented data from laboratory rats which indicate a noise related increase in hypothalamic release of corticotrophin releasing factor (CRF), a substance which in turn elicits the release of ACTH from the adenohypophysis (9). In related studies we have observed both an elevated secretion rate of corticosterone and lack of adrenocortical response to exogenous ACTH in feral *Sigmodon hispidus* subjected to intermittent jet aircraft noise (100 db SPL) for 4 weeks.

With few exceptions, most reports involving adrenal cortical-high intensity noise interactions have centered upon indirect indices of cortical activity, i.e., adrenal weight, vascular formed element changes, plasma corticoid levels, etc. In many physiological states, such parameters may not accurately reflect the true activity of the gland. Direct determination of secretory activity utilizing *in vitro* incubation methods may provide a productive alternative approach.

This investigation was designed to detect possible changes in both basal and ACTH stimulated *in vitro* corticosterone secretion rates of feral animals living in close proximity to a high intensity noise source, i.e., a metropolitan jetport. Since most previous studies have dealt with laboratory species, the present investigation is especially pertinent since Seabloom and Seabloom have recently reported significant differences in both basal and ACTH stimulated corticosterone secretion rates in feral *M. musculus* as compared to their laboratory counterparts (10). It has been suggested that such differences may be due to a genetically related decrease in adrenal cortical sensitivity in laboratory species, thus raising the possibility of a differential adrenocortical response to noise in comparisons of laboratory with feral species.

Methods

Experimental Animals and Collection Sites

Two groups of animals were utilized in this study. All animals were collected within a 2-day period in February, 1975 and, prior to sacrifice, were housed for 1 day at 24°C, were subjected to a 14-hr. photoperiod (0700-2100), and were given Purina lab chow and tap water *ad lib.* Fourteen adult male *M. musculus* were collected from a field approximately 90 meters from the end of a runway at Memphis International Airport, Shelby County, TN. Nine adult males of the same species were collected from a control field approximately 2 kilometers from the airport field.

Both fields were similar in habitat in that they were abandoned fields in early stages of secondary succession. Vegetation consisted mainly of grasses and herbs with a few scattered small trees. Additionally, mark-recapture analysis indicated similar population densities at both sites. The close proximity of the two fields insured similarity in genetic composition.

Differential sound levels of both sites were measured with a sound survey meter. Background levels were essentially equal with maximal recorded levels infrequently ranging to 85 db SPL. Noise levels due to incoming and outgoing aircraft at the airport field averaged 110 db SPL. Aircraft noises at the control field were negligible over background.

In Vitro Incubati.

All animals were weighed between 0900 and 1100 noise-exposed animal a dish of cold (2-4°C) trimmed to remove adrenal gland and incubated on analytical balance (quartered and incubated 200 millunits per ml carried out in a Dubuque CO₂ atmosphere). At removed to glass vial

Twenty-four hr. late determination of corticoid 110 fluorometer.

Weight Relationships

Control mice were similar to counterparts (Table 1). adrenal pair weights nutrient availability exposed animals since

	N
Control	9
Noise	14
1 Mg Adrenal/100 g Bod	
2 Mean ± Standard Error	

Adrenal Secretory Rat

Secretory data were presented and noise, no ACTH and noise and ACTH as well between appropriate mice analyzed included adrenal gland pair/90 min, percent of glandular weight summarized in Table 2.

Analysis of variance effects ($p < 0.001$), an ASR means revealed a 28% rate in the noise exposure in secretion rates over (26% and 59%, respect

In Vivo Incubation and Fluorometric Determination of Corticosterone

All animals were weighed to the nearest 0.1 g and then killed by decapitation between 0900 and 1100 hours on a single day. Sacrifice order of control and noise-exposed animals was randomized. Adrenal glands were rapidly removed to a dish of cold (2-4°C) Krebs Ringer bicarbonate (KRBC). The gland pairs were trimmed to remove adipose tissue, blotted on filter paper, and weighed on an analytical balance to the nearest 0.1 mg. Glands from each animal were then quartered and incubated in either 2 ml KRBC or a like volume of KRBC containing 200 milliunits per ml ACTH (Sigma Biochemicals). Incubations (90 min.) were carried out in a Dubnoff metabolic shaker (38°C, 60 oscillations/min., 95% O₂-5% CO₂ atmosphere). At the end of the incubation period, incubation media were removed to glass vials and frozen (-20°C).

Twenty-four hr. later, samples were thawed and extracted for the fluorometric determination of corticosterone (11). Fluorescence was determined with a Turner model 11G fluorometer utilizing #110-813 (primary) and #110-818 (secondary) filters.

Results and Discussion

Weight Relationships

Control mice were significantly heavier ($p < 0.05$) than their noise exposed counterparts (Table 1). No significant differences were noted in comparisons of adrenal pair weights or adrenal-body weight ratios. It is unlikely that lack of nutrient availability was related to the smaller body weights observed in noise exposed animals since the habitats were remarkably similar in that respect.

TABLE 1

	N	Body and Adrenal Weight Relationships		Adrenal Wt./ Body Wt. ¹
		Body Weight	Paired Adrenal Weight	
Control	9	15.34 ± .45 gm ²	8.8 ± .54 mg	57.10 ± 4.20
Noise	14	14.10 ± .36	8.4 ± .43	60.15 ± 3.38

¹µg Adrenal/100 g Body Wt.

²Mean ± Standard Error

Adrenal Secretory Rates

Secretory data were prepared utilizing a two by two factorial design (no noise and noise, no ACTH and ACTH) for the analysis of variance. The main effect of noise and ACTH as well as noise-ACTH interactions were analyzed. Differences between appropriate mean pairs were analyzed with Student's "t" test. Variables analyzed included absolute secretion rate (ASR or units of hormone produced/gland pair/90 min), relative secretion rate (RSR or units of hormone produced/unit of glandular weight/90 min) and RSR/unit of body weight. The data are summarized in Table 2.

Analysis of variance of ASR revealed significant noise effects ($p < 0.002$), ACTH effects ($p < 0.001$), and noise-ACTH interaction ($p < 0.001$). Comparison of the ASR means revealed a much greater (but insignificant, $p < 0.15$) basal secretion rate in the noise exposed group. ACTH induced a significant ($p < 0.001$) increase in secretion rates over basal levels in both control and noise exposed groups (260% and 59%, respectively). However, the response to ACTH in the noise ex-

TABLE 2

Noise, ACTH, and Corticosterone Secretion Rates				
	N	Absolute Secretion Rate ¹	Relative Secretion Rate ²	Relative Secretion Rate/g Body Wt. ³
Control				
Basal	5	0.72 ± .12 ug ⁴	7.73 ± 3.03 ug	0.51 ± .23 ug
ACTH	4	2.69 ± .14	35.47 ± 3.39	2.34 ± .25
Noise				
Basal	7	0.97 ± .09	11.79 ± 2.56	0.83 ± .15
ACTH	7	1.54 ± .11	18.71 ± 2.56	1.36 ± .19

¹Micrograms/Adrenal Pair/90 min.²Micrograms/100 mg Adrenal/90 min.³Micrograms/100 mg Adrenal/g Body Wt./90 min.⁴Mean ± Standard Error

posed animals was significantly less ($p < 0.001$) when compared to the control response, even though noise exposed basal levels were higher than control basal values.

Analysis of the variable RSR also revealed a significant noise effect ($p < 0.04$), ACTH effect ($p < 0.001$), and noise-ACTH interaction ($p < 0.002$). As before, the source of the interaction involved relative responsiveness of control and noise exposed glands to ACTH. Control animals exhibited a significant ($p < 0.001$) increase while noise-exposed groups showed a moderate yet insignificant elevation in RSR. Analysis of RSR per unit of body weight revealed a similar pattern: a significant ($p < 0.001$) increase in secretion with ACTH in the control group and a moderate but insignificant increase with ACTH in the noise exposed group. Noise-ACTH interaction also proved significant ($p < 0.01$).

The data presented strongly indicate that jet aircraft noise of the type and intensity studied interacts with the organism so as to diminish *in vitro* cortical responsiveness to exogenous ACTH. Although decreased responsiveness of mammalian adrenal cortices to ACTH may reflect lower endogenous ACTH titers (12), we do not believe the data indicate subcontrol ACTH levels in noise exposed animals. No apparent differences in adrenal weights or adrenal-body weight ratios were observed between control and noise exposed animals. Indeed the latter group showed elevated basal secretion rates. De Wied, *et al.*, have demonstrated that the *in vitro* production of corticosterone utilizing a similar incubation protocol (no preincubation period and in the absence of exogenous ACTH) is a reliable index of *in vivo* cortical activity (13). Since the amount of ACTH bound to the gland at the moment of the animals' death is responsible for rate of activity of the gland *in vitro*, the data suggest slightly elevated ACTH levels may have been present in noise exposed animals.

In vitro responsiveness to exogenous ACTH has been shown to reflect the recent history of exposure of the adrenal cortex to circulating ACTH. Glands from rats subjected to chronic ACTH treatment *in vivo* produced significantly greater amounts of corticosterone in response to *in vitro* ACTH stimulation than did glands from animals not subjected to prolonged ACTH administration (14). In addition Bakker and De Wied, utilizing essentially the same incubation methods as employed in our investigation, demonstrated that glands from stressed animals produced substantially more corticosterone *in vitro*, both in the basal state and when stimulated with given levels of ACTH, than did control glands (15). It was noted, however, that since the basal values of the stressed animals were greater, the percentage increase in steroid production due to ACTH

stimulation was not our observations on the latter authors, the from noise-exposed animals exposed to ACTH than did in the type and duration of ACTH responsiveness. since there is no interaction group, the data tend to group had a diminished response to ACTH stimulation.

At this point we must the assay of ACTH response longer with no ACTH) in a fortified medium. Considering such a protocol (responsiveness of corticosterone) may be due to the gland at the time of the substance present. ACTH activity (15). to be released from the possibility of non-cortical

In the present study, Since all glands were of responsiveness to as yet undetermined differences observed or may have been the substance. Further differences is not within the

It is therefore possible so as to decrease the intensity to markedly enhance. Should this be the case, stress response mechanism that intense noise effects and significant (7). Since the data preclude cortical activation by species, these reports significant differences in feral *M. musculus* are in question, it is suggested to question, it is suggested ability to respond to

1. J. L. FLETCHER and Pub. No. NTID 300. 74 pp. (1971).
2. V. P. OSINTSEVA, N. Hygiene and Sanita.
3. P. JURTSUK, A. S.

stimulation was not as great as in glands from non-stressed animals. Although our observations on basal rates are in agreement with those obtained by the latter authors, the data obtained from ACTH stimulated glands are not; glands from noise-exposed animals produced significantly less corticosterone when exposed to ACTH than did glands from control animals. This may be due to differences in the type and duration of stressful stimuli studied. Therefore, assuming that ACTH responsiveness of glands from our control animals is representative, and since there is no indication of greater circulating ACTH titers within the control group, the data tend to support the hypothesis that glands from the noise-stressed group had a diminished capacity to increase corticosterone production in response to ACTH stimulation.

At this point we must emphasize that most conventional *in vitro* techniques for the assay of ACTH responsiveness utilize a preincubation period (30 minutes or longer with no ACTH) after which the adrenal tissue is placed in fresh, ACTH fortified medium. Cortical responsiveness to ACTH is greatly enhanced following such a protocol (14). It has been suggested that the lack of maximal responsiveness of cortical tissue to ACTH stimulation in the absence of preincubation may be due to either (a) competition between endogenous ACTH bound within the gland at the time of death and exogenous ACTH or (b) the presence of a heat labile substance present within the tissue which has the ability to diminish ACTH activity (15). The latter substance was presumed by Bakker and De Wied to be released from damaged cells during quartering of the adrenals although the possibility of non-cortical origin was not excluded (15).

In the present study, ACTH responsiveness was determined without preincubation. Since all glands were handled in a like fashion, we must conclude that the lack of responsiveness to ACTH of glands from noise-exposed animals was due to some as yet undetermined factor(s) or state not present in the control group. The differences observed may have been related to changes within the glands proper or may have been the result of the continuing influence of some extra-adrenal substance. Further insight into the specific source or nature of these differences is not within the scope of the present investigation.

It is therefore possible that noise interacted at some as yet undetermined point so as to decrease the functional reserve capacity of the gland, i.e., the ability to markedly enhance secretory activity in response to physiological stimuli. Should this be the case, a significant segment of the noise-exposed animals' stress response mechanism may have been impaired. Several reports have indicated that intense noise superimposed upon other stresses could have damaging effects and significantly reduce life span, at least in laboratory species (5-7). Since the data presented by Seabloom and Seabloom (10) indicate the adrenal cortical activation system may be more sensitive to stressful stimuli in feral species, these reports assume added significance. In conclusion, there is a significant differential *in vitro* adrenal cortical response to exogenous ACTH in feral *M. musculus* exposed to jet aircraft noise as compared with controls. Although the specific site of noise interaction with the adrenal cortex is open to question, it is suggested that such interaction may restrict the animals' ability to respond to physiologically stressful stimuli.

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SECTION 34

SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<u>Principal Investigator(s)</u> A. J. Vander, L. L. Kay, M. E. Dugan, D. R. Mouw		<u>Institution and address where research was performed</u> Dept. of Physiology University of Michigan Ann Arbor, Michigan 48109	
<u>Investigator's Phone No.</u>	<u>Sponsoring Organization</u> Michigan Heart Association		
<u>Citation</u> Vander, A. J. et al. Effects of noise on plasma renin activity in rats. (unpublished paper).			
<u># of Ref.'s</u> 14	<u># of Fig.'s</u> 4	<u>Language</u> English	
<u>Type & duration of experiment</u> Laboratory experiment; 1-2 hours		<u>Purpose for study</u> The effects of noise on plasma renin activity in unanesthetized rats.	
<u>Description of test groups (subjects, #, age, etc.)</u> Male Sprague-Dawley albino rats 200-250 g. One test group was fed a standard Purina chow. The other test group was fed a sodium-free diet; 1 control group.			
<u>Control of other stressors</u> handling was minimized		<u>Statistical Methods</u> Multiple comparison procedure of Dunnett (variation of t-test)	
<u>Noise Stimulus</u> source: sound speakers spectral characteristics: broadband noise and 2000 cycles per second (CPS) noise level: 90 dB, 100 dB, 115 dB (each at the 2 frequencies) length of exposure: 30 minutes # of trials: 1 trial per animal		<u>CVS Response Measured</u> none <u>Nonauditory effects</u> Plasma renin activity increased due to 115 dB broadband noise in rats on the normal diet and due to 90-100 dB noise in rats on a sodium-free diet. The 2000 cps type of noise failed to increase plasma renin activity at even 115 dB.	
<u>Author's conclusions</u> Intensities of noise below the human pain threshold can increase plasma renin activity in rats. Plasma renin substrate levels were not altered by noise. The increase in plasma renin activity may be a factor in the development of hypertension.			
<u>Evaluation & comments</u> Plasma renin activity due to noise should be studied in humans. Increased renin activity stimulates salt retention leading to water retention, which can create a hypertensive state.			

Effects of Noise on Plasma Renin Activity in Rats¹

Category: Endocrinology

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Running Title: Noise and renin

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FOOTNOTES

1. Research supported by a grant from the Michigan Heart Association
2. Octive-band analysis of the broad-band noise at 106 dB overall was as follows (all value expressed as dB-SPL): 61dB at .065KHz; 87dB at .125KHz; 89dB at .5KHz; 96dB at 1KHz; 101dB at 2KHz, and 4KHz; 97dB at 8KHz; 82dB at 16KHz.

Noise has been shown to induce a wide variety of non-auditory physiological effects (1-3), including persistent hypertension in laboratory animals (4-8). The present study was designed to determine whether acute noise can stimulate renin secretion in the unanesthetized rat.

Methods

All animals used were male Sprague-Dawley albino rats, 200-250 g. They were fed either standard Purina chow (containing 1% NaCl) or a sodium-free diet (Nutritional Biochemicals Corporation) and were housed individually in metal cages; the housing room was on a 6 a.m. to 6 p.m. light-dark cycle. Experiments were performed in separate workrooms; the rats were conditioned to being moved in their cages from the housing room to the experimental sound-box and remaining in the box (with the sound turned off) for five minutes on each of three days preceding the day of the actual experiment; the cages were then returned to the housing room and the animals were removed from the cages and handled briefly. The same six cages were placed in the box together each day and in the same position on the sound-box floor. The sound-box was a sound-proofed wooden box with speakers built into its top.

On the experimental day, six rats were similarly placed in the box, the door closed, and the sound turned on at the desired frequency and intensity for thirty minutes; immediately thereafter, with the sound still on, the rat cages were removed one at a time, and carried to the adjacent room where the rats were decapitated, blood collection lasting thirty seconds. Control animals were subjected to exactly the same sequence of events except that the sound was not turned on. All experiments with animals on the standard diet were performed from 8:30 - 9:30 a.m.; controls and experimentals were run on consecutive days (the order was rotated from experiment to experiment). The low-sodium animals were studied from 8:30-10:30 a.m., the longer time being made possible by the fact

that a low-sodium diet eliminates most of the circadian rhythm for renin (personal observation).

Plasma renin activity (PRA) and plasma renin substrate (PRS) were determined by a modification (9) of the method of Haber et al. (10), which utilizes the generation of angiotension I during a standardized period of incubation and its measurement with the radioimmunoassay kits supplied by New England Nuclear.

All data are presented as means \pm one standard error (SEM). The analysis used to determine statistical significance between a set of noise levels and the control for that set was the multiple comparison procedure of Dunnett (11). This method calculates the Student t-statistic in the usual way but then uses the Dunnett tables for P rather than the usual Student t-tables. This is necessary whenever a single group of animals is used as the control for multiple experimental doses (in this case, the multiple noise levels).

Results

Figures 1-3 summarize the effects of broad-band² and 2000 cps sounds on PRA in rats on the normal-sodium diet. Broad-band noise caused a significant increase in PRA only when the intensity was increased to 115dB (Fig. 3); 2000 cps sounds failed to increase PRA at any intensity-level studied.

Figure 4 demonstrates that the animals on a low-sodium diet have a greater PRA-response to sound; a highly significant increase in response to broad-band noise occurred at 100dB ($P < .01$). The response to 90dB was not statistically significant ($P = 0.14$).

Plasma renin substrate (PRS) was measured in all experiments and no differences were observed between any groups.

Discussion

These data demonstrate that intensities of noise well

below the human pain-threshold can acutely increase PRA in unanesthetized rats maintained on either a standard or a sodium-free diet; plasma renin substrate did not change, indicating that the PRA increase reflects increased plasma renin concentration. That sodium deprivation reduced the threshold for this effect is consistent with the fact that sodium-deprivation enhances the renin-releasing effects of various stimuli (12).

Given the ability of noise to increase the activity of the sympathetic nervous system (13,14), it is logical to postulate that the increased renin secretion is mediated by increases in circulating catecholamines or enhanced activity of the renal sympathetic nerves. This pathway has previously been shown to be involved in the renin response to other types of stress in the rat (9).

The finding that noise increases PRA may have implications for the pathophysiology of hypertension. Experimental hypertension has been produced in laboratory animals using either pure auditory stimuli (4-6) or mixed auditory-visual-vibratory stimuli (7,8); the possible role of renin in these types of hypertension has not been investigated. Finally, it has been hypothesized that noise may be a risk-factor for hypertension in people (2,3,14).

Summary

The effects of noise on plasma renin activity (PRA) were studied in unanesthetized rats. An intensity of 115dB (broad-band) was required to increase PRA in animals eating a normal diet; the threshold was only 90-100dB for sodium-deprived rats. Stimuli at 2000 cps of up to 115dB were ineffective in elevating PRA.

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FIGURE LEGENDS

Figure 1.

Effect of broad-band noise on plasma renin activity in rats on a normal diet.

Figure 2.

Effects of 2000 cps noise on plasma renin activity in rats on a normal diet.

Figure 3.

Effects of 115dB noise on plasma renin activity in rats on a normal diet.

Figure 4

Effects of broad-band noise on plasma renin activity in rats on a low-sodium diet.

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Figure 1

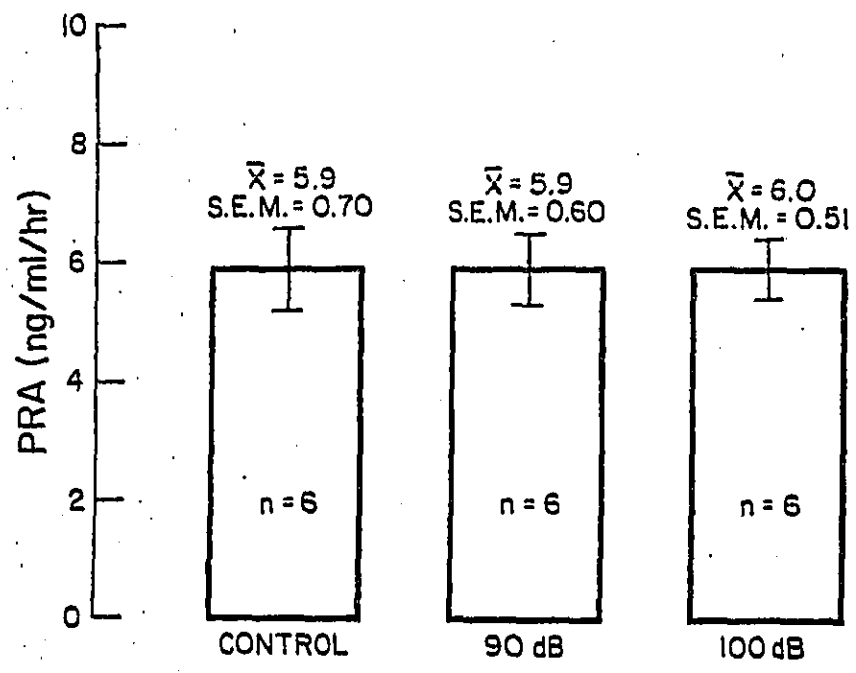


Figure 2

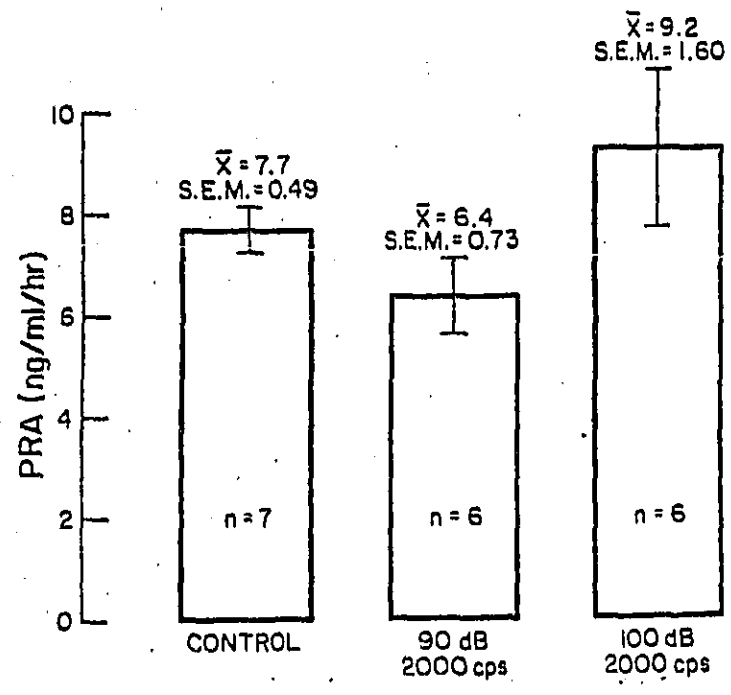


Figure 3

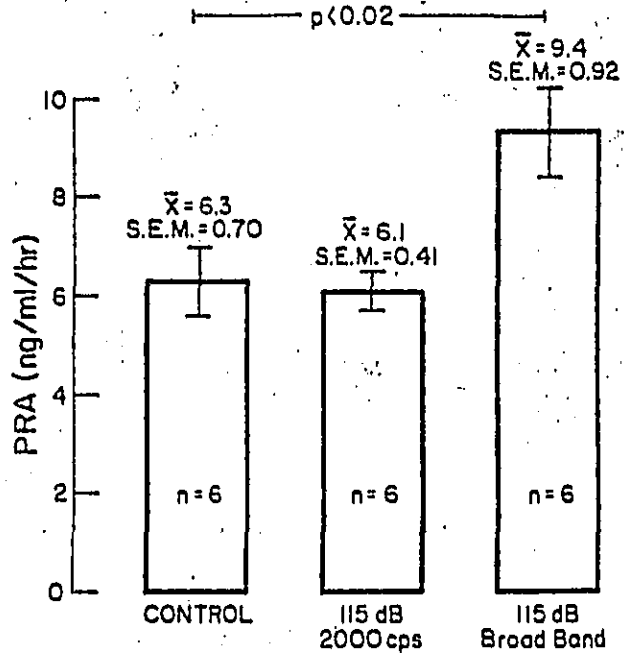


Figure 4

