THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

THE EFFECTS OF BROADBAND NOISE ON CERTAIN PARAMETERS OF THE CARDIOVASCULAR SYSTEM IN NORMAL RESTING ADULTS

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PHILOSOPHY

BY

LYLE B. CARTWRIGHT

Oklahoma City, Oklahoma

1973

THE EFFECTS OF BROADBAND NOISE ON CERTAIN PARAMETERS OF THE CARDIOVASCULAR SYSTEM IN NORMAL RESTING ADULTS

APPROVED BY

DISSERTATION COMMITTEE

ACKNOWLEDGMENTS

First I would like to thank the chairman of my committee, Dr. Robert N. Thompson, without whose support and guidance this work would never have been undertaken. Also, I am deeply indebted to the other members of my committee, Dr. Carl A. Nau, Dr. C. H. Lawrence, Dr. Grover C. Harrison, and Dr. Jerry Tobias, for their constructive criticism and direction.

My gratitude is also extended to Dr. Earl Folk and his assistants who were of invaluable help in the statistical analysis of the data. To Dr. Charles Brake, Mr. Jim Langwell, and the many others at the Federal Aviation Administration, Aeronautical Center, Civil Aeromedical Institute, Oklahoma City, Oklahoma, who gave freely of their time and special skills, my thanks is extended.

All of the experimental studies were conducted at FAA, CAMI, in Oklahoma City in the laboratory of Dr. Jerry Tobias.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF ILLUSTRATIONS

LIST OF ABBREVIATIONS

α, alpha - level of statistical significance

ASHA - American Speech and Hearing Association

β, beta - chance of making a Type II statistical error

BP - blood pressure

CAMI - Civil Aeromedical Institute

cm - centimeter

cm^2 - cubic centimeter

dB - decibel (re .0002 dyne/cm^2, unless otherwise specified)

Δ, delta - change of

dBA - sound level measured with the "A" weighted network

dBC - sound level measured with the "C" weighted network

EKG - electrocardiogram

FAA - Federal Aviation Administration

H_A - alternate hypothesis

H_0 - null hypothesis

Hg - mercury

Hz - Hertz

IBM - International Business Machine

I.M. - intramuscular

I.V. - intravascular

kg - kilogram

kHz - kiloHertz

mike - microphone

min - minute

mm - millimeter

msec - millisecond

N. - nerve

NASA - National Aeronautic and Space Administration

NS - not significant

p - statistical probability level

PTS - permanent threshold shift

re - reference

S.D. - standard deviation

S.E. - standard error

Sec - second

SPL - sound pressure level (re .0002 dyne/cm^2)

t - standard statistical t test

TTS - temporary threshold shift

USAF - United States Air Force

^oC - degrees Centigrade

USASI - United States of America Standards Institute

(trans. med.) - (translation by the National Medical Translation Service, Washington, D. C.)

THE EFFECTS OF BROADBAND NOISE ON CERTAIN PARAMETERS OF THE CARDIOVASCULAR SYSTEM IN NORMAL RESTING ADULTS

CHAPTER I

INTRODUCTION

Sounds perceived by the ear can have two somewhat overlapping effects on human beings. The first of these effects is neuropsychological and the second is neurophysiological (1, 2). A third effect, observed only under laboratory conditions, is the direct damage or destruction of both animal and human tissues by noise of extremely high intensity. The mechanism in operation in the third case is the conversion of acoustic energy into thermal energy with subsequent thermal damage to the exposed tissues. The ear and its associated neural mechanisms do not play a role in this phenomenon (3, 4, 5).

The neuropsychological effects of sound on human beings have been extensively studied and are not within the scope of this dissertation. They will be mentioned only as they interact with the neurophysiological effects of noise under study here.

In 1949, Davis et al. (6) published a short paper on the possibility of extra-auditory hazards from exposure to intense sound and

ultrasound. Rosenblith (7), in 1955, reviewed the literature on extra-auditory effects of noise on man and concluded that much more study was needed before it could be determined if such a hazard did exist.

By 1970 several studies on the non-auditory effects of noise on man (Russian and German literature) and on laboratory animals (American literature) had been reported. The year 1970 also saw the publication of two books in the United States on this subject. Brief quotes from each illustrate the still widely divergent opinions on the existence of non-auditory physiological damage to humans exposed to noise.

The first book, Physiological Effects of Noise (8), was 345 pages long. The publisher's advertising flyer stated:

> Without doubt, the ever increasing noise of modern society represents a threat, every bit as grave as air and water pollution, to the continued life of man on this planet. Few, however, are aware of the very real and pressing physiological effects of noise pollution on the human organism. Not only does noise pollution impair man's audio mechanism, but his heart, his endocrine functions, and his reproductive systems (emphasis added) are also attacked by the ceaseless barrage of sound that has become part and parcel of our technical world.
>
> Physiological Effects of Noise presents for the first time - all (emphasis added) existing information on the disruption of our psychological and physiological equilibrium by all kinds of noise, from sonic booms to rock and roll music . . . . We have already unknowingly committed acts of senseless violence against ourselves through our lack of understanding of noise pollution (9).

The other book published in the United States in the same year was The Effects of Noise on Man (10). The author, Kryter (11), in his summary chapter concluded " . . . other than as a damaging agent to the ear and as a maskor of auditory information, noise will not (emphasis added) directly harm people or interfere with psycho-motor performance."

The review of the literature which follows will help one to

understand how such widely divergent views can be held by internationally recognized researchers in the field of bioacoustics.

CHAPTER II

LITERATURE REVIEW

Non-auditory Effects of Noise on Man

In 1972 an article by Kryter (12) on the non-auditory effects of environmental noise presented a good overview on the current state of scientific knowledge about general non-auditory effects of noise on man. Kryter stated that much more controlled experimental work is needed in this area and that such experiments should look specifically at the effects of noise over prolonged periods of time.

In 1971 Glorig (13) published an article entitled "Noise Exposure - Facts and Myths". After reviewing the literature, he concluded that there was no firm evidence to date that would indicate an extra-auditory health hazard from noises ordinarily encountered in industry. He also pointed out the widely divergent conclusions reached by Lehmann as compared to those of Finkle and Poppen (14). In a retrospective study, Lehmann found that steel workers had a statistically significant higher incidence of vascular disorders and arrhythmias. In contrast, Finkle and Poppen reported no change in the cardiovascular state of subjects exposed to noise of intensities up to 120 dB.

Rosen (15), in 1971, related the high incidence of cardiovascular disease in industrial countries to noise exposure and stress.

He cited the animal studies of Friedman et al. (16) who had determined that rabbits exposed to 102 dB of white noise for ten weeks developed greater aortic atherosclerosis than did the control rabbits.

These and other studies led Rosen to conclude:

> Such investigations as I have described appear to point to noise as an environmental pollutant as well as a health hazard . . . . Life in this world is so full of stress and strain that every possible effort must be made to eliminate those which are amenable to elimination. The reduction of noise levels in everyday life is not an impossible dream (17).

In his 1970 text Kryter listed six principles necessary for the successful functioning of biological organism (18):

a. A sensory receptor, such as the ear, will be much more sensitive to the form of energy that normally activates it than will be any other part of the organism.

b. A sensory receptor will not transmit the energy it received to other parts of the organism, but will transmit only signals (usually nerve impulses) that the receptor system generates, i.e., the physical energy of the stimulus, as such, is not transmitted.

c. A sensory receptor will be damaged by excessive amounts of the physical energy to which it is especially sensitive before any other component of the organism adversely affected by that energy.

d. A sensory receptor will not generate signals (either electrical impulses or chemicals) that can harm any part or component of the normal organism.

e. The integrity of parts and mechanisms of the organism will tend to be maintained or strengthened as the result of responding to normal stimulation.

f. Responses of parts and mechanisms of the organism that serve no useful purpose will tend to be inhibited by the organism (i.e., adaptation will take place).

In 1970 Anticaglia and Cohen (19) reviewed the literature and concluded that noise could: (a) cause permanent hearing impairment, (b) hinder job performance on certain tasks, and (c) act as a source of annoy-

ance. They noted that, in general, American authors did not feel that noise constituted an extra-auditory health hazard, whereas most European authors believed that a very grave extra-auditory health hazard might exist. The authors concluded by calling for more study into the possible extra-auditory pathological effects of noise.

Writing on the effects of noise on physiological state in 1969, Jansen (20) noted the studies of Grandjean and many other investigators, all of which have demonstrated that unexpected noise can produce physiological changes in man. He warned that until it is proved that these physiological changes are negligible, noise must be considered as a possible detrimental influence on human health.

Jansen (20) reported the results of his laboratory study of healthy young students exposed almost daily to bursts of 90 dB white noise of 300 msec to 90 min duration. He found the expected changes of increased diastolic blood pressure and noted that this increase sometimes disappeared after several months of testing. Unfortunately, no numerical data on blood pressure or pulse rate was given and no mention of testing for statistical significance was made.

In 1964 Rosen et al. (21) noted the relationship between hearing loss and cardiovascular disease. While their studies were not and can not be statistically corrected for dietary, racial, cultural, and socioeconomic factors, the difference in the incidence rates for atherosclerosis rates between the Mabaans and people of "civilized" countries was amazing. The fact that the Mabaans did not develop presbycusis in their noise free environment was also most fascinating.

Grandjean (22) summarized the proceedings of the recent American

Speech and Hearing Association meeting on the non-auditory physiological effects of noise on man:

> We know well the existence and the mechanism of the habituation process, but unfortunately know very little about this protecting mechanism in every-day life, and especially against the increasing noise of our modern environment. More research in this field would be of great practical importance.

Shatalov and Murov (23) reviewed the Russian literature and noted marked inconsistency in the data collected by different researchers studying the response of the cardiovascular system to noise. They noted that Andryukin and others believe that intense noise increased arterial pressure while Arkhad'yevskiy and Vopilkina noted that intense noise caused hypotension.

Shatalov and Murov (23) performed a retrospective study on 3,930 workers (52 per cent male) of various occupations and ages. Their subjects were divided into four groups, classified as follows:

I. Laboratory technicians (controls) who were exposed to low intensity noise and were assumed to have low "neuropsychic tension".

II. Scientists who were exposed to low intensity noise and were assumed to have high "neuropsychic tension".

III. Automobile test drivers who were exposed to high intensity noise and were assumed to have high "neuropsychic tension".

IV. Carpenters and repairmen who were exposed to high intensity noise and were assumed to have low "neuropsychic tension".

Their results are summarized below:

a. The blood pressure in all groups was found to increase with age ($p < .05$).

b. The blood pressure of older workers in noisy occupations did not differ from that of the controls.

c. Younger male workers in noisy occupations had statistically elevated blood pressure compared to that of the controls.

d. The scientists had elevated blood pressure compared to that of the controls.

e. The automobile test drivers had much higher blood pressure than did the controls.

From these observations the authors concluded that "a combination of the two industrial factors, noise and neuropsychic tension, has a greater effect on the systolic and diastolic arterial pressure than each of them taken separately.

Mohr et al. (24) reported a statistically significant rise in the pulse rate of five test subjects exposed to wide-band noise generated by a NASA thermal structures tunnel during "blow down operation". The pulse rate increased from 10 to 40 per cent during the exposure which lasted about one minute at approximately 150 dBA.

Other Related Literature

In 1952 Karrasch (25) published a paper entitled, "The Effect of Noise on the Human Organism". It was his opinion that in the classification of work assignments, noise should be included in the list of "work impeding factors" along with heat, cold, humidity, and noxious gases. He discussed the great divergence of views on the possible extra-auditory effects of noise on industrial workers; in particular, he noted the views of Kryter, Muller, and Kellner:

a. Kryter rejects a general effect of the factory noise completely and maintains that the average worker accustoms himself to the noise of his working place completely and, apart from injury to hearing, experiences no ill effects (26).

b. Muller maintains that noise promotes accidents and causes general damage: irritability, vertigo, trembling of spread fingers, indigestion and heart trouble, distrubances of sleep, depression (26).

c. In studying textile workers exposed to 85-90 phons at head level, Kellner found the core of N. Vagus, the center of the so significant innervations for the regulation of the vegetative functions of the inner organs is affected by machinery noise to the utmost (26).

Karrasch attempted to reconcile these opinions with these statements:

> The effect of noise is a psychophysical problem! Perhaps the opposing nature of the opinions, at least partly, is to be traced back to the different way of thinking of the German and the American workers. The American has a different inner attitude towards technology and also to its acoustical mani festations; furthermore, many colored people, who are indeed physically more robust, work in American production (26).

Karrasch described his experiment relating to the effect of noise of 105 phon in the frequency range 1200 - 2400 Hz. Using three human subjects, bodily efficiency was measured by the "blood pressure - amplitude - pulse rate product of 10,000" and an ergometer. The faster the amplitude frequency-product was reached, the lower was the efficiency (after the testing arrangement of G. Lehmann). The results were not analyzed statistically in the article but Karrasch concluded:

> Figure 1 shows the results. One sees that the efficiency with noise of all three subjects is clearly reduced. The measurement itself reveals that the effect of noise had influenced the blood pressure of two subjects and the pulse rate of the third (27).

Unfortunately no numerical values of pulse rates and blood pressures were given. No mention was made of what type of controls (if any) were used or how many trials were undertaken.

In 1962 Bode (28) published an article entitled "General Illness as a Result of Noise". He listed authors who allegedly had shown the following noise induced illnesses and/or conditions:

a. Lehmann: reaction to rotation in which acclimatization does not occur.

b. Symanski: sleep disturbance.

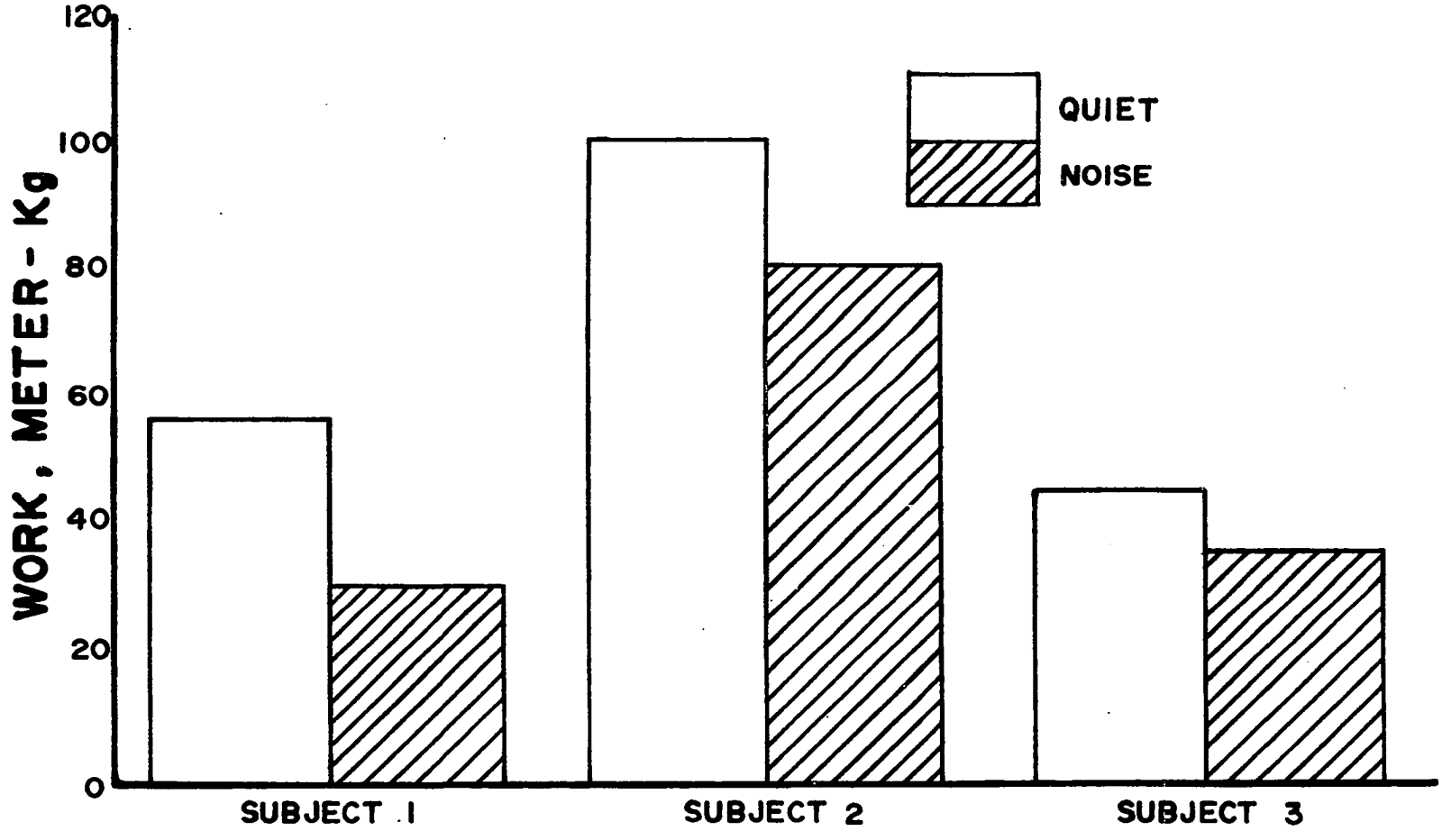

Figure 1.--Graphical representation of reduced efficiency under the influence of noise (from Karrasch).

c. Hennigsen: vasomotor instability.

d. "Forefathers": decreased blood flow to organs.

e. Smith and Laird: alteration in production of gastric juices and peristalsis.

f. Orlow: stomach and duodenal ulcers.

g. Holstein and Lehmann: nervousness, fatigue, and poor concentration.

h. Slawin: increase in strength of conditioned reflexes.

Bode described his study of some 2,000 shipyard workers. Out of 280 workers classified as "noise workers", 105 came to the industrial clinic five or more times in a 1-year period. The most noteworthy finding, according to Bode, was that ulcers increased after eight years of noise work. He added, however, that this could be explained on the basis of age. Amazingly, only three of the 280 "noise workers" had hearing defects. Time loss for cardiovascular, central nervous system, and gastrointestinal disorders was greater for the noise workers. Time loss accident rates were greater for the noise workers than for the non-noise workers (1.12 days per year compared to 0.18 days per year).

Bode made no reference to any statistical analysis of the data presented but concluded, "It is at once seen that sympathetic variable persons, persons with stomach and intestinal disease, and in many cases, people with heart and related illness, would be advised not to take noise work if possible" (28).

In 1966 Kangelari et al. (29) published an article entitled "On the Influence of Vibration and Noise on General Disease." The authors collected data from 1960 to 1962 on two groups of workers: (a) 135 "testers" at motor stations exposed to noise of 116-120 dB (age not

given), and (b) 152 "trimmers" in a casting plant exposed to noise of 88-90 dB and aged 20-39 years. The results of time loss in days per 100 workers for the three year periods is presented in Table 1. The authors related the apparent increased incidence of "general disease" to "impaired autonomic functions of the nervous system under the influence of noise".

Odescalchi (30) reviewed the "more recent" literature and concluded:

> . . . the principal general effects of noise, especially of the industrial noise, are variable, but industrial noise is a harmful agent. (A harmful agent in a working environment being considered any factor which alone or together with other factors can induce statistically significant changes of the biological integrity of the subjects exposed for a standard working time.)

Farr (31) in 1967 speculated on the possibility of noise in the home environment being a harmful agent. He noted that home appliances, especially in the kitchen, often produce noise levels in excess of those permitted for industry under federal regulations. He further stated:

> The effects of noise in exacerbating disease may be seen in a specific infectious disease, such as tetanus. In other disease states such as anxieties, duodenal ulcer, and other kindred so-called tension ills, the additive deleterious effect of noise is real and immediate (32).

Semczuk and Gornz (33) in 1970 studied the "cardiorespiratory efficiency" in 200 healthy men subjected to noise from 90 to 115 dB. They reported that in 73 per cent of the subjects, there was a significant decrease in "cardiorespiratory efficiency" as a result of their exposure to the "harmful noise".

Culliton (34) published "Noise Menace Threatens Man" in 1966. The article was summarized by the author: "The constant whirl of jet planes and the blare of sirens, as great a menace as air pollution,

TABLE 1

DAY LOSS PER HUNDRED WORKERS

Type of Illness	116-120 dB Predominantly Noise 2-5 kHz	88-90 dB Controls 120-800 Hz
All sickness, including URI	103.1	77.0
Upper respiratory infection	55.9	39.1
Angina	8.9	7.8
Diseases of naso-pharynx	3.2	1.5
Diseases of the lungs	3.2	1.5
Diseases of peripheral nerves	2.3	1.5
Gastritis	1.6	0.4
Ulcers	2.9	1.6
Diseases of bone, joints, muscles	1.1	1.5
including myositis	0.8	1.1

threatens civilized man's hearing and sanity."

Welch in a 1969 article entitled "The Physiological Effects of Audible Sound" stated, ". . . through its activating effect upon subcortical neuronal systems of the brain, sound, either continuous or intermittent, modifies the pacing by the brain of cardiovascular, encocrine, metabolic, reproductive, and neurological function" (35). Welch cited no references or experimental data which led him to this conclusion.

Experimental Animal Studies and the Thermal Effects of Noise on Man

Most scientists are finally becoming aware of the many pitfalls involved in applying to human beings conclusions drawn from studies on experimental animals. A brief review of the physiological (non-auditory) effects of noise on various laboratory animals will help emphasize this point. The behavioral effects of noise on animals have been intentionally omitted as they are not pertinent to this investigation.

In May, 1948, the USAF Headquarters Air Material Command Engineering Division was directed to perform a study on the physiological effects of intense sound (36). In this series of tests, experimental animals and human volunteers were exposed to jet engine noise and intense siren noise at frequencies varying from 1,000 to 12,000 Hz. It was determined that in the case of furred animals, such as rats and guinea pigs, sound can be a lethal stimulus. The animal experiment results as a function of frequency are given in Table 2.

It is interesting to note from the data in Table 2 that 3,000 Hz appears to be particularly lethal to these animals. More important,

TABLE 2

DATA ON ANIMALS EXPOSED TO INTENSE SOUND FIELDS AS RELATED TO THE FREQUENCY OF THE SOUND FIELD

Animal	Frequency (Hz)	Sound Level (dB)	Air Temp (°C)	Fur Temp (°C)	Rectal Temp (°C)	Time to Death (min.)
Guinea Pig	1,000	153	31.0	50.5	42.0	29
Rat	1,100	156	32.0	42.0	41.0	45
Rat	3,000	157	30.5	-	43.0	9
Guinea Pig	3,000	155	34.0	52.0	42.0	8
Guinea Pig	3,000	156	36.0	46.0	44.0	9
Guinea Pig	5,000	156	33.0	49.0	51.0	21
Rat	5,000	157	33.0	43.0	63.0	11
Guinea Pig	7,000	157	-	46.0	44.0	16
Guinea Pig	10,000	154	34.0	42.0	46.0	60 (living)
Guinea Pig	12,000	154	35.0	42.5	44.5	29

perhaps, is the observation that the mechanism of death appeared to be thermal in nature as was indicated by post mortem histological measurements.

This mechanism of death (the conversion of acoustic energy to thermal energy) is extremely important when one theorizes that such exposure might cause similar effects on human beings.

Human volunteers were exposed to sounds of varying frequency (650 - 4,100 Hz) and intensity (137 - 163 dB) for periods up to 33 minutes. The auditory temporary threshold shift (TTS) was recorded and was found to be consistent with the results obtained by other authors in other experiments. It is interesting to note that there was no mention of any other physiologic change or injury other than TTS with the exception of the heating of the skin, to be discussed below.

A series of "pain withdrawal" experiments was performed in which a human volunteer would place his hand in the intense sound field of the siren and leave it there until he would withdraw it because of pain due to heating. The length of time to withdrawal was very much frequency dependent as shown in Figure 2.

It was unfortunate that the exact intensity level was not specified in this particular part of the experiment but from the equipment descriptions given it was presumed to range from 120 to 160 dB. Again, it must be emphasized that this element of heating of the organism (especially with furred animals) must be considered when attempting to extrapolate from the following animal data to the possible extra-auditory physiological effects of noise on human beings.

Anthony et al. (37), in 1959, conducted a series of experiments

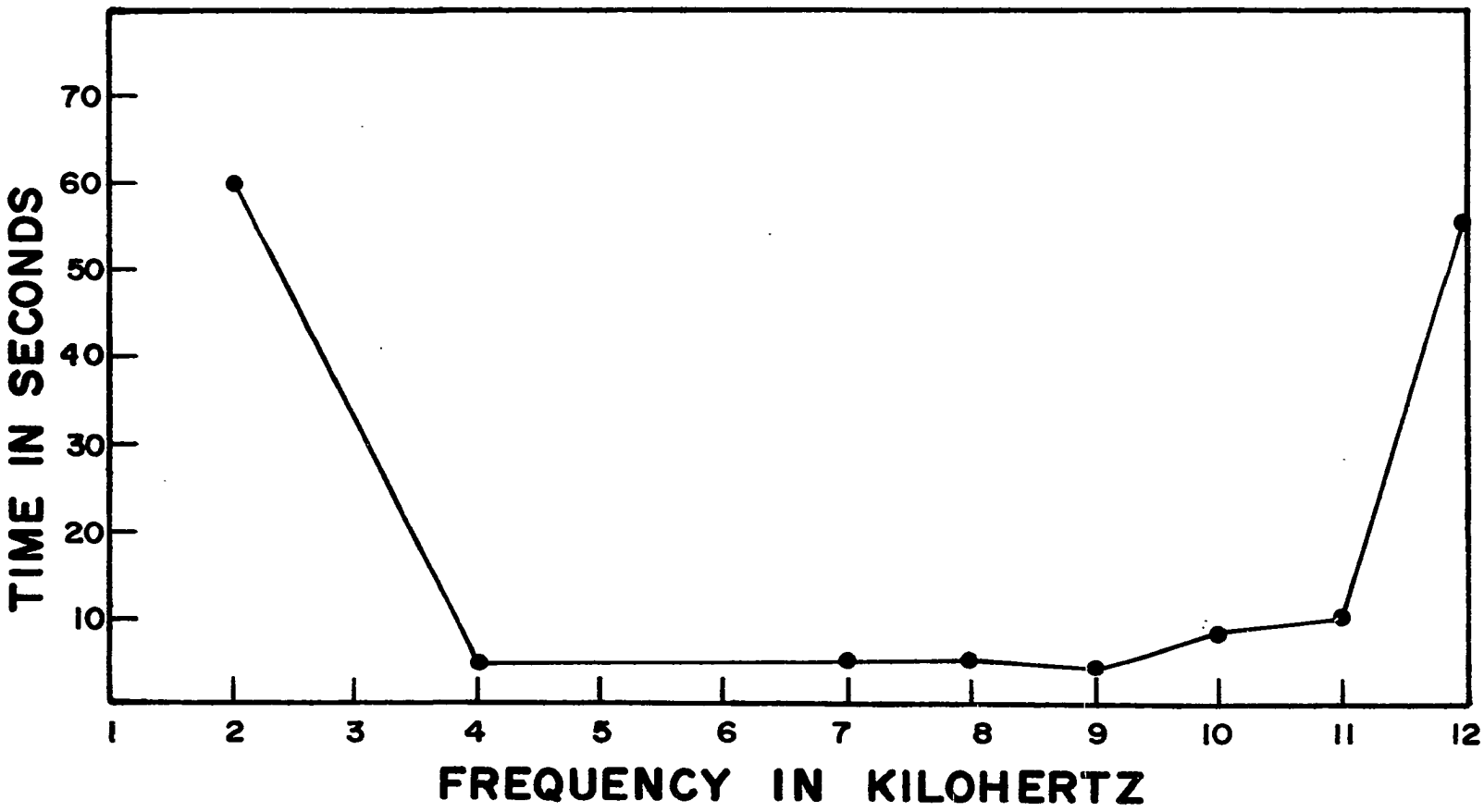

Figure 2.--Time to pain withdrawal as a function of frequency (from USAF study).

on mice, rats, and guinea pigs in an attempt to establish if noise could produce endocrine changes which might be harmful to such experimental animals. White noise at levels of 139 - 143 dB (re .0002 dyne per cm^2) for fifteen minutes per day over a period of four weeks was used. No significant visceral organ weight change was detected in the rats or guinea pigs but the female mice did develop significant adrenal hypertrophy compared to non-noise exposed controls. The authors concluded that the noise stress led to increased adrenal activity. No mention was made of fur, rectal, or core temperature in the test animals. Biochemical testing (serum electrolytes, serum ascorbic acid, and blood glucose) revealed no difference in test versus control groups with the possible exception of altered ascorbic acid metabolism in noise exposed guinea pigs. (This point could well be the topic of much further research in that guinea pigs are the only known sub-primate animals requiring exogenous ascorbic acid for life.) These authors alluded to one of their previous studies in which laboratory rodents were exposed to 135 dB of 2 - 40 kHz noise for 2 - 40 hours per week for from 2 - 9 weeks. At these exposures there was a "breakdown" in the normal endocrine defense mechanism resulting in microscopic pathological changes in the adrenals and other organs. The authors further stated that with "low frequencies" and similar exposure time no such pathology was demonstrable. They concluded the article with the statements:

> Physiological stress stimuli, such as noise, impose strains on certain bodily functions which are compensated for by changes within the endocrine system. As with purely mechanical or electronic feedback loops, it is possible to exceed the limits over which the system can operate; in the case of noise this appears to involve intense high frequency sounds (38).

Anthony and Ackerman (39) stated:

> . . . it is generally conceded that the entire process of homeostasis is geared in large part to the pituitary-adrenocortical system, especially during the exposure of an animal to a stressful situation. As one might expect, the most sensitive measure of an animal's physiological response to a stress such as noise involves the determination of changes in the activity of the adrenal gland.

Accepting this point of view, one would logically attempt to measure changes in the activity of the adrenal gland or its target organs in response to stressful stimuli.

In a series of experiments Anthony and Ackerman employed three types of noise situations:

a. single short noise bursts of 139 dB (re 0.0002 dyne per cm^2) at 150 - 4800 kHz and 2 - 20 kHz;

b. chronic exposure to intense low frequency noise for three to six weeks with an exposure time of 30 - 40 hours per week;

c. chronic exposure to intense high frequency noise for two to three weeks with an 18 hour per week exposure.

Following the exposures, the animals were sacrificed and examined for gross, histological and histochemical analysis of the adrenal gland and adrenal target organs. Experiments on the level of circulating blood eosinophils were also performed. The examinations indicated that:

a. both brief and prolonged exposure to low frequency noise causes increased adrenocortical activity in mice and guinea pigs, but the tolerance limits of the adrenal system were not exceeded and, hence, it was felt that "low frequency noise did not constitute a harmful physiologic stress."

b. chronic exposure of the test animals to high frequency noise caused an increase in adrenal activity manifested by adrenal hemorrhage and vacuolization of adrenal cells.

Geber and Anderson (40) studied the effect of chronic audio-

genic stress upon rats and rabbits and determined that three weeks exposure to six minute bursts hourly of 20 - 25,000 kHz noise with an average level of 83 dB (range 73 - 93 dB) produced cardiac hypertrophy (ventricles). Weights of the entire animal bodies and of most other visceral organs were decreased with the exception of some adrenal and renal hypertrophy in some of the older rats.

A Russian investigator, Klemparskaya (41), studied the influence of noise on the formation of circulating antibody titre. His noise stimulus was stated to be 58 - 62 dB (frequency characteristics not given) 24 hours per day for three or four weeks. This level of noise is quite low but the author did not specify his dB reference. It should be kept in mind that some foreign experimenters use 0.0001 dyne/cm^2 instead of 0.0002 dyne/cm^2 as their sound pressure reference. He did not state quiet dB values. He concluded:

a. This noise stimulus activated the formation of antibodies after a single injection (I.M.) of 500 million microbe bodies of paratyphoid vaccine of bacilli breslaviensis (sic).

b. Revaccination (I.M.) of the animals in the absence of noise was considerably less effective than in control rabbits which were vaccinated initially in the absence of noise.

c. Noise exerts an inhibitory effect upon antibody formation using I.V. immunization techniques compared to I.V. immunization of non-noise exposed control rabbits.

Lawrence et al. (42) studied the effects of noise on the vascular supply to the Organ of Corti in guinea pigs. Using various stimulus levels in the range of 120 dB SPL at 4 kHz, temporary threshold shifts were produced in the test animals. The animals were immediately sacrificed and microscopically examined. Important observations included:

a. In certain areas corresponding roughly to that area activated by the particular frequency, the vessels of both the stria

vascularis and the basilar membrane were found to be devoid of red cells. No change in the hair cells was observed.

b. Relatively low sound intensity levels can produce this effect.

c. It was not possible to stimulate one ear and use the other as a "control" because the "control" ear would invariably show "cross over" effects of sound (i.e., relative anemia); hence, non-noise exposed litter mates were used as controls.

These studies, which demonstrated such dramatic pathological effects of noise on certain experimental animals, can be put into proper perspective by the studies done on the heating of haired and hairless mice by intense sounds by Danner et al. (43). Anthony (3) also studied the effects of noise exposure on the adrenal glands in hairless mice. The hairless mice suffered much less damage because their hairless bodies reflected most of the sound energy which, in contrast, was absorbed by mice with hair. This absorption of acoustic energy resulted in increased core temperature and visceral organ damage.

Experimental Human Studies

Other than for very short duration noise where the "startle effect" predominates, there are very little experimental data available on the effects (if any) of noise on the human cardiovascular system performed under laboratory conditions. This is especially true concerning the effects of prolonged noise on the cardiovascular parameters of blood pressure and pulse rate. In fact, if the literature review of Finkle and Poppen (44) was complete, there was none available in English until 1948.

In 1947 Finkle and Poppen (44) studied some of the clinical effects of intense jet engine noise on nine normal male volunteers. The noise was broadband in nature and about 120 dB in intensity in those

frequencies up to 7,500 Hz. The fixed mounted engine also produced sound energy at higher frequencies but the authors stated that there was no equipment available to them at that time to analyze accurately noise above 7,500 Hz.

The actual amount of energy delivered to each subject's tympanic membrane was not measured and it would be difficult to estimate since all subjects were wearing some kind of ear defender. During the first two days of the experiment some of the subjects stationed around the engine exhibited a TTS persisting several hours after the cessation of the noise. None suffered a PTS and none had a TTS after the first two days. The authors stated that after two days of exposure all subjects were fitted with both earplugs and leather aviator helmets.

The subjects were usually seated around the test chamber mounted jet engine at various locations and were permitted to read magazines. Ten 1-hour and five 2-hour exposures to the noise were experienced by each of the nine subjects. Blood pressure and pulse rate were determined in the conventional clinical manner on each subject before and after each noise exposure. A medical officer also measured the pulse rate and blood pressure on some of the subjects during the noise exposure at random intervals.

During the noise test blood pressure was taken by palpating the radial artery while observing an aneroid sphygmomanometer. The authors noted the inherent inaccuracy of this method. Electrocardiograms were obtained at random on some of the subjects during each of the 15 noise runs. All subjects had normal pretest EKG's. Some other physiologic parameters were also studied. Unfortunately no control runs were made.

The authors concluded:

> Since negative findings predominate in the clinical observations, mention is made first of these. There was no change noted in the general physical condition of these men upon daily examination during the course of the experiment. The simple neurologic tests that were made were normal, as were the values for pulse rate, respiratory rate, and blood pressure obtained before, during, and after daily exposure to the engine. It is pointless to itemize these normal figures (emphasis added), or those for visual acuity, electrocardiographic and electroencephalographic pattern (45).

It is unfortunate that the authors felt it pointless to report normal figures since normal figures can point to pathological conditions if a firm trend is established.

In 1955 Steinmann et al. (46) studied the effect of various noises upon the blood pressure of humans. They observed the well known startle reaction caused by an unexpected loud noise and noted that there was an increase in the blood pressure "in the moment of shock". They also discussed the effect of certain association linked noises, such as music or the crying of a baby, and noted that blood pressure can respond to meaningful noise stimuli by varying either upward or downward.

The numerical data presented were very limited. The authors stated that 333 subjects, aged 20 to 70 years, were studied. Most subjects were normotensive but some were hypertensive. The only reported numerical change in blood pressure in response to a sound stimulus was contained in this single quoted sentence:

> So the clang and beating tones and the motor noises (of racing cars) in almost all cases produces a temporary blood pressure change; as a rule, it produced a blood pressure rise of 5 - 20 mm Hg, diastolic often somewhat more than systolic, so that the pulse pressure became somewhat smaller (47).

Unfortunately, again there was no reference to any control runs, to statistical analysis, or to the intensity of the sound stimulus

used to evoke the responses reported.

Lehmann and Tamm (48), in 1956, published what is perhaps the most widely quoted article about man's cardiovascular response to prolonged noise. The bulk of the article described the various cardiovascular responses of 18 healthy test subjects to various noises. Many cardiovascular parameters were automatically recorded including blood pressure and pulse rate. Ballisto-cardiograms were also made on each subject. During the one hour test run the subjects were exposed to various broad-band noises of loudness levels up to 90 phon.

The authors reported all of their results in tabular form and these tables indicated only if a particular parameter increased or decreased for each subject after exposure to the noise. In the text, it was stated:

> It is noteworthy that there was little change in pulse rate. There was occasionally a slight tendency to bradycardia. The pulse pressure amplitude showed a tendency to narrow, which was attributable primarily to an elevation of the diastolic values (49).

The above observations were not substantiated by any numerical data. Perhaps this is just as well since the method of statistical analysis used by the authors cannot be allowed to pass without some very critical comment. The reader is alerted to this fact by the authors when they stated:

> The statistical evaluation was based on the determination of significance, not in the usual manner, in terms of the mean values, but rather in terms of the deviations and fluctuations of the noise values as opposed to the values of a test without noise, since physiological fluctuations were always present at rest, in addition to those caused by measurement technology (50).

The authors then enumerated several equations allegedly used to determine if a change in a parameter were statistically significant.

These equations, in addition to containing several undefined terms, also contained several errors that appear to be typographical in nature. It was the opinion of three professional consultant biostatisticians (51) that if the equations described (even allowing for typographical errors) were used to determine the statistical significance of a change in a parameter, any conclusion based on such a test could not be accepted without violating very basic biostatistical theory and practice. Thus, this widely quoted work must be considered only with serious reservations.

Ponomarenko (52) studied the influence of several noises of various intensities and spectral characteristics upon the cardiovascular functions in normal adolescent boys, ages 15 and 16. Only the tests of broadband noise at 75 dB will be discussed here because lesser intensities produced smaller responses. (It should be noted that the dB reference for the noise stimulus was not stated by the author and some European authors do use 0.0001 dyne per cm^2 rather than 0.0002 dyne per cm^2 as a reference.)

Physiologic testing was performed before and after the 1-hour noise stimulus. The author stated that identical tests using the same boys served as his controls. He further noted "no notable effect of experimental conditions upon these functions could be determined" (53).

Ponomarenko concluded from his experiments:

a. 75 dB "wideband" noise (see Figure 7) lowers the pulse pressure by 15mm Hg on the average with a maximum of 25mm Hg in one subject.

b. The systolic blood pressure was decreased by 8.8mm Hg on the average.

c. The diastolic blood pressure increased 6.0mm Hg on the average.

d. The pulse rate decreased 10.9 beats per minute on the average.

e. The pre-test value of every parameter was re-established always in less than 20 minutes after cessation of the noise. The "controls" responded under the same conditions with the following changes:

1) pulse pressure change of 0.0mm Hg
2) systolic blood pressure change of 0.0mm Hg
3) diastolic blood pressure change of 0.0 mm Hg
4) pulse rate decreased 2.0 beats per minute.

Terent'yev et al. (54) in 1969 published an article on the reaction of the human nervous and cardiovascular systems to aviation noise. They described the cardiovascular response of 90 subjects who worked in noise areas from 100 to 136 dB daily from one to six hours per day. Technicians working in about 130 dB complained of general fatigue, decreased working capacity, headache, unpleasant sensations in the region of the heart, tinnitus, whole body pruritus, myalgia of the abdominal muscles, nausea, and vomiting. A few also reported poor sleeping pattern and decreased appetite. EKG changes were found in some subjects, chiefly a decrease or inversion of the T wave.

The authors conducted an experiment under laboratory conditions in which the subjects were exposed to one hour of noise of approximately 101 dB. The authors stated that as a result of the noise exposure, the heart rate decreased 10 - 20 beats per minute and the systolic and diastolic blood pressure decreased 10 - 20 mm of Hg. Unfortunately, neither the spectral characteristics of the sound nor the activity of the subjects was reported. There was no mention of "control" runs and there was no mention of statistical analysis.

CHAPTER III

PURPOSE AND SCOPE

The fact that noise can cause disease in human beings has been known since before 1831 when it was noted that boilermakers always became deaf when working for any length of time in the interior of a boiler under construction (55). Even today millions of American workers are exposed to noise levels of sufficient intensity to produce hearing loss. The World Health Organization estimates that noisy working environments in American industry result in a total annual loss of $4 billion per year (56).

Only in recent years has the question arisen as to the possibility that noise, over a prolonged period of time, damages organs other than the ear (19). The system which has received the most attention in this regard is the cardiovascular system. Several retrospective epidemiological studies, especially those by Rosen et al. (57), have shown an association between noise exposure and increased incidence of cardiovascular disease.

This study was designed to attempt to measure any statistically significant change (at the $\alpha = .05$ level) in the cardiovascular parameters of blood pressure, pulse rate, and pulse pressure that might be produced by noise similar to that frequently encountered in industry.

Assuming that if exposure to continuous noise under laboratory conditions does produce a change in one or more of these parameters, then one might conclude that similar exposure in industry over a period of many years might lead to or aggravate diseases of the cardiovascular system. On the other hand, if no changes are produced under laboratory conditions, at least a much needed base line of what constitutes normal cardiovascular function (as measured by these parameters) would be established and future "in the field" studies would be more meaningful.

Twenty healthy adult subjects were exposed to 91 dBA noise for one hour. Each subject served as his own control under identical conditions on a different day except that the ambient noise was approximately 38 dBA. During both test and control runs the cardiovascular parameters of blood pressure, pulse rate, and pulse pressure were automatically measured every two minutes.

The noise exposure level of 91 dBA was selected because it was a value just greater than that permitted for an 8-hour work day without ear protection in American industry at the time this experiment was conducted. This noise level was intense enough to produce temporary damage to the ear in most persons as detected by a TTS in their post stimulus audiogram. As mentioned earlier in the review of the literature under Kryter's six "principles" for successful organisms, his third principle was:

> A sensory receptor will be damaged by excessive amounts of the physical energy to which it is especially sensitive before any other component of the organism (is) adversely affected by that energy (58).

The 91 dBA stimulus then should produce some temporary damage to the ear and this excessive amount of energy might "spill over" into other systems causing a measurable change in one of the cardiovascular parameters being recorded.

CHAPTER IV

MATERIAL AND METHODS

Subjects

Twenty healthy adult volunteer subjects, eleven female and nine male, participated in the experiment. Their age, sex, occupation, educational level, and order of participation in the experimental and control test runs is given in Appendix A. None of the subjects had any history or physical findings suggestive of significant previous disease of the auditory or cardiovascular systems. All had normal audiograms and electrocardiograms.

Equipment

The electromechanical equipment used in making these experiments can be divided into two broad general categories. The first of these is the noise generation and measurement equipment and the second is the physiological measuring equipment.

Noise generation and measurement

The noise used in all of the tests was generated by a General Radio random noise generator set to the USASI mode driving a 100 watt Bodgen power amplifier. The output of the power amplifier was fed to a 6" x 9" loud speaker located inside of the audiometric test chamber.

The loudspeaker was positioned approximately 3 feet directly in front of and slightly above the subject's head. A condenser microphone, which was positioned about 3 inches from the subject's left ear, was connected to a Bruel and Kjaer sound level meter located outside the chamber. The output of the sound level meter was coupled to a matching strip chart recorder. With this arrangement the noise intensity could be adjusted to 91 dBA during test runs with the subject in place. All of this equipment is listed with identifying numbers in the List of Equipment and can be further identified schematically in Figure 3 and photographically in Figures 4, 5, and 6.

Physiological measurements

The blood pressure was automatically determined every two minutes. The internal timer in the cuff pump triggered the pump which inflated the cuff on the subject's left arm to 140mm Hg. A pressure line from the pump also led to the electrosphygmomanometer, where the line pressure was converted into an electrical signal by means of a linear core transducer. Korotkoff sounds detected by the microphone inside the cuff were amplified and mixed with the pressure signal. This combined output from the electrosphygmomanometer was fed to one channel of the strip chart recorder. The electrocardiogram lead I was connected via shielded cable to an EKG preamplifier and then to the polygraph. The radial pulse crystal was fed through an amplifier and filter into the polygraph. All of this equipment is identified by number in the List of Equipment, schematically in Figure 3, and photographically in Figures 4, 5, and 6.

Automatic audiometry was performed in the conventional manner

List of Equipment

I. Audiometric chamber: Industrial Acoustics Co., Model 1201-A

II. Sound level measurements and recording were all performed with Bruel and Kjaer equipment in accordance with their instruction book (59).

- A. Microphone: Model 4145
- B. Microphone amplifier: Model 2603
- C. Band pass filter set: Type 1612
- D. Pistonphone: Model 4220
- E. Recorder: Model 2305

III. Audiometry

- A. Grayson-Stadler recording audiometer: Model 1703 (pulsed tone mode)
- B. TDH 39 earphones with MX-41 cushions

IV. Noise source

- A. General Radio random noise generator: Model 1382
- B. Bodgen power amplifier: Model CHB100
- C. Loudspeaker, 6" x 9", manufacturer unknown

V. Blood pressure and pulse recording

- A. Narco Bio-Systems electrosphygmomanometer: Model ESG-300 with matching microphone and adult size blood pressure cuff
- B. Automatic cuff pump: Model 95-300-70
- C. Crystal pulse pickup: Part 705-0020

VI. Strip chart recorder: Grass Model 5 Polygraph simultaneously recording:

- A. Lead I electrocardiogram at 25 mm per sec paper speed
- B. Blood pressure every two minutes automatically
- C. Radial artery pulse

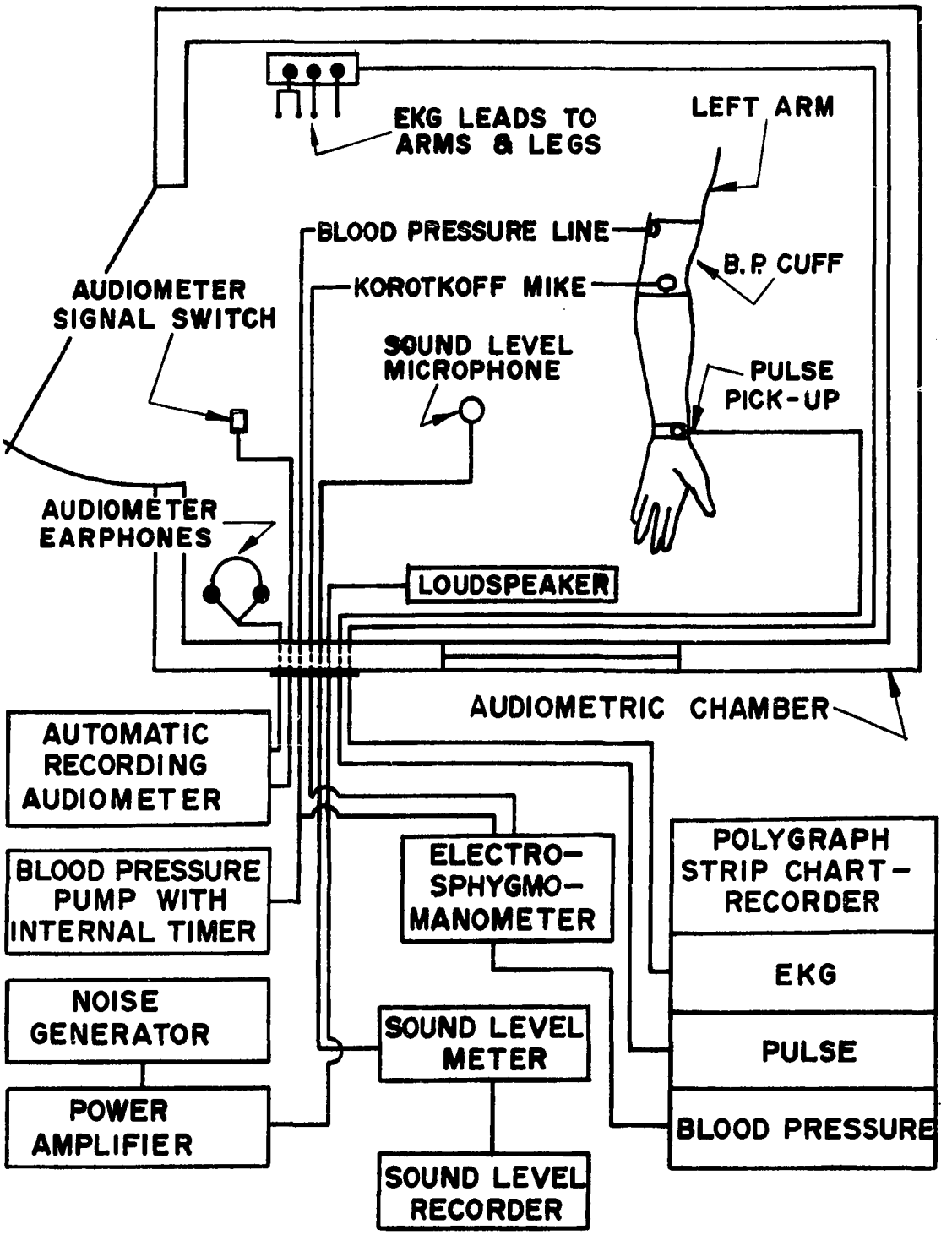

Figure 3.--Schematic diagram of equipment.

Figure 4.--View of inside of test chamber.

Figure 5.--Test subject with physiological measuring equipment attached.

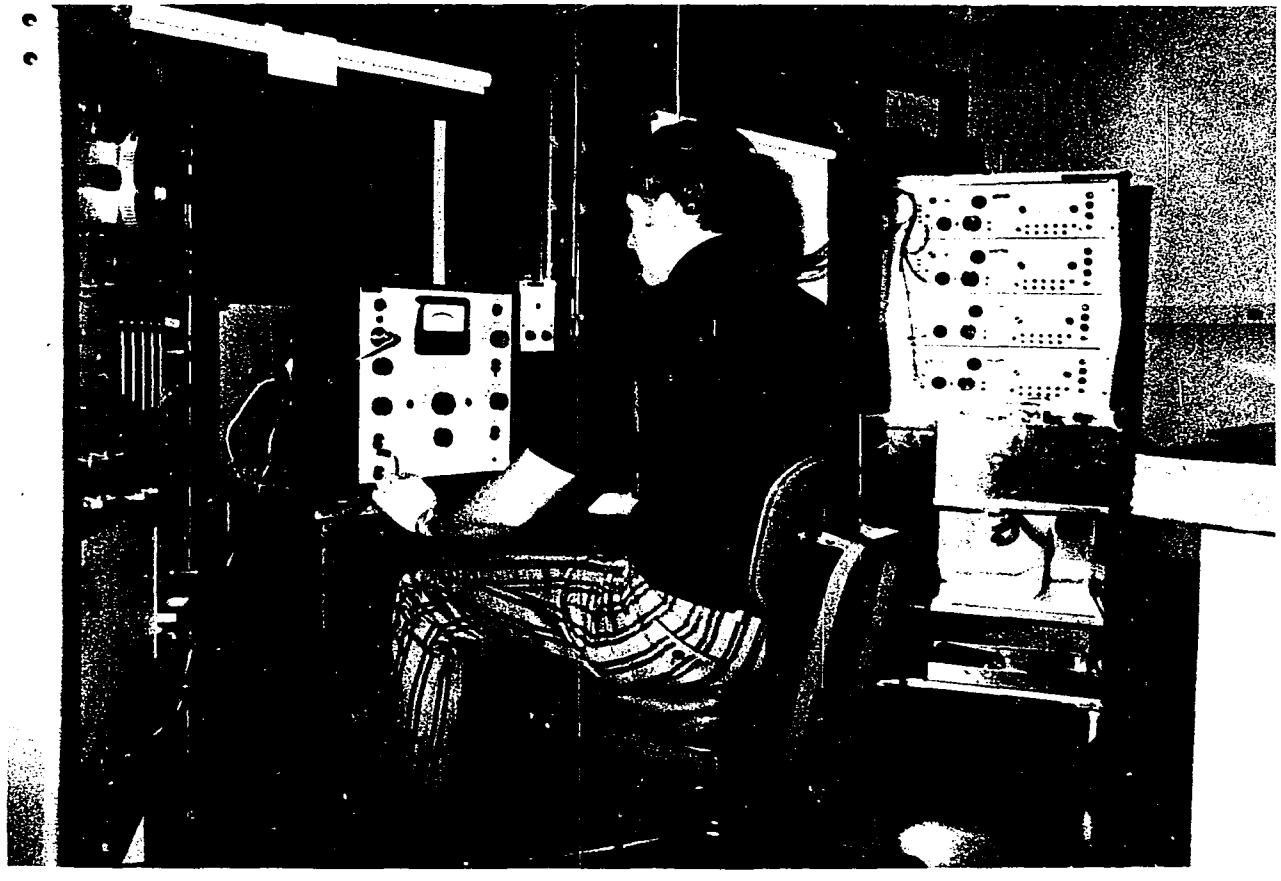

Figure 6.--External view of test chamber and operator monitoring test and recording equipment.

and the TTS was determined two minutes post stimulus at 4 kHz.

Measurements

Blood pressure

Clinically human blood pressure is ordinarily measured indirectly by the use of a device called a sphygmomanometer and a stethoscope. A blood pressure cuff is wrapped about the mid portion of the upper arm, while the bell or diaphragm of the stethoscope is placed over the brachial artery at a level slightly above the antecubital fossa. The pressure cuff is then inflated to a pressure sufficient to occlude the blood flow in the artery and then the pressure in the cuff is "bled off". As the pressure in the blood pressure cuff decreases, a point is reached where a small bolus of blood will pass through the relative occlusion now afforded by the blood pressure cuff. This bolus of blood creates a turbulence in the brachial artery which produces the so-called Korotkoff sounds heard by the examiner. The appearance of this Korotkoff sound is labeled systolic blood pressure phase I.

As the pressure in the occluding cuff continues to bleed down toward zero, four other phases of blood pressure may be detected. The diastolic pressures, phases IV and V, are of great medical significance, while phases II and III are of little or no medical significance. The determination of the diastolic blood pressure is not as easy as it might appear.

As the cuff pressure continues to decrease, the sound often becomes muffled (phase IV) and then quite suddenly ceases (phase V). According to Bordley et al. (60), phase V is the best indirect estimate of diastolic blood pressure. Quite often, however, the sounds undergo

no cessation and then the point of "muffling" (phase IV) is given as the diastolic blood pressure. In some persons the sounds undergo no distinct point of muffling and do not cease. In this case the diastolic blood pressure cannot be determined indirectly with accuracy.

The accuracy with which the diastolic blood pressure can be determined clinically by a physician varies greatly due to many factors. These factors include pulse rate, hearing acuity of the physician, ambient noise level, and anatomical characteristics of the patient. All these factors taken together led Bordley et al. (61) to conclude:

> . . . Furthermore, the distinctness or demarcation of the dulling phase varies in different subjects examined. Frankness must cause us to admit that all too frequently the reading of diastolic pressure by this criterion becomes merely a guess.

There are other indirect measurements of human blood pressure, such as the Riva-Rocci method (62) and the Broemser glass optical manometer. More recently indirect blood pressure measurements have been made utilizing the Doppler principle. Lategola and Harrison (63) describe an interesting electromechanical method requiring only about five seconds to determine indirectly human blood pressure.

All of the inherent variability in the indirect measurement of human blood pressure leads one to the question, why not measure the arterial blood pressure directly by catheterization of the radial or brachial artery. There are two overwhelming reasons for not using intravascular instrumentation. First, Stevens (64) did extensive work on cardiovascular instrumentation. He found that the anxiety, pain, discomfort, or whatever, of intra-arterial catheterization introduced such blood pressure changes as to make the procedure of little value in conscious human subjects. A second reason against the use of intra-arterial

catheterization is the possibility of vascular complications. Levin et al. (65), after studying the complications following intra-arterial catheterization of a group of volunteer subjects, concluded that repeated arterial catheterization leads to an increase in the incidence of thrombosis and peripheral embolization. There was also unexplained transient neutropenia in some of the subjects. Although platelet counts were normal in all, other complications included thrombophlebitis in two subjects whose veins were catheterized. Intra-arterial thrombosis as manifested by arterial embolization occurred four times, but this was observed only after multiple arterial catheterizations. So, the idea of using intra-arterial methods for the direct measurement of human blood pressure for this study was abandoned.

The method of blood pressure measurement selected for use in this study was a modified electromechanical system described by Geddes et al. (66). The cuff was of the adult size (14.2 cm width) placed on the subject's left upper arm at the level of the heart as recommended by Bordley et al. (60). The cuff was inflated to 140 mm of Hg every two minutes by an automatic pump. The bleed off rate was fixed at 6 mm of Hg per second. The Korotkoff microphone was located over the brachial artery under the cuff and was over the medial division of the brachial artery in those subjects having this anatomical variation. The pressure in the cuff and electronic transducer system was calibrated against a mercury column manufactured by the W. A. Baum Co., Inc. (67) at the beginning and end of each run. Stability for the entire system was ±1mm of Hg at center scale (80mm Hg) and ±2mm of Hg at the extremes (30 and 130mm of Hg).

The output of the Korotkoff microphone was amplified and filtered and then superimposed upon the output of the pressure transducer and fed into one channel of the Grass Polygraph (see photographs and schematic diagram).

The systolic pressure was determined by the appearance of the first Korotkoff sound as recorded on the polygraph. This is in keeping with the method of Geddes et al. (66) and also corresponded with the reappearance of the radial pulse wave recorded on a second channel of the polygraph. This method was first described by Davis et al. (68) and proved to be quite reliable when compared with the visual recording of the first Korotkoff sound or clinical measurements.

The diastolic blood pressure was taken to be the first recorded Korotkoff sound whose amplitude was less than one-third the height of the tallest recorded Korotkoff sound. This point was chosen somewhat arbitrarily but seemed to correlate well with clinical measurements taken in the conventional manner. Lategola (69) has used this as the diastolic pressure in his tilt table experiments and the use of complete disappearance of the Korotkoff sounds as suggested by Bordley et al. (60) did not correlate well with clinical measurements. Davis et al. (70) suggested using the maximum radial pulse following deflation of the cuff as a diastolic end point but again the clinical correlation was very poor so the "one-third" value was used as described above.

The radial pulse

The radial pulse was measured by placing a piezoelectric (pressure sensitive) crystal over the radial artery at the level of the left wrist where the artery is quite superficial. The output of the crystal

pickup was fed to a second channel of the Grass Polygraph (see Figure 3). The pulse pickup was useful especially when it was difficult to determine the exact occurrence of the first Korotkoff sound when movement artifact was present. At the time the experiments were performed no artifact suppression electronic equipment as described by Lagerwerff and Luce (71) was available to the investigator.

Electrocardiogram

A standard Lead I electrocardiogram was continually monitored by large silver electrodes and electrode paste on each arm and leg. The output of the electrodes was fed to a third channel of the polygraph through an appropriate preamplifier. The chart speed was 25mm per second throughout the entire two hours of each of the 40 runs. This high speed was necessary in order to measure the P-R, QRS, and Q-T intervals accurately. The electrocardiogram was not calibrated to 1 millivolt equals 1 cm because it was found that over the two-hour run the calibration would vary due to drying of the electrode paste and subject movement. The amplitude of the electrocardiogram was adjusted to make the intervals easy to read and their absolute voltage was not measured.

Pulse rate

The pulse rate was determined by counting either the QRS complexes or radial pulse waves for a 30 second interval every two minutes. This gave 60 measurements of the pulse rate over the two-hour recording period. The exact time that the pulse rate was measured was determined by using the two minute interval marks generated by the blood pressure pump timing generator.

Procedure

When each subject arrived at the laboratory, he was given a general explanation of the testing procedure and shown the equipment that would be used. He was instructed to enter the booth and sit down. After the examiner closed the door, he asked the subject to open the door from the inside and leave the chamber. Also the subject was instructed how to activate the intercom from the inside and how to push an alarm button if for any reason he did not want to complete a test run. The alarm was never pushed except in a demonstration preceding each run. The shades on the windows were never closed; the light inside the booth and the ventilation fan were left on continuously.

The subject then re-entered the test booth and the EKG electrodes, the blood pressure cuff, and the pulse pickup were attached and tested. The subject put on the earphones for the initial audiogram. The door to the test chamber was then closed and the recording equipment started. An automatic audiogram was completed during the first six minutes of each control run and a second audiogram was completed approximately ninety minutes into the run. The subject removed the earphones after each audiogram.

The subject had free use of his right arm and could read from The World Almanac (72) if he desired. Although movement of the left arm was somewhat restricted by the equipment attached to it, the subject was allowed to move it except during blood pressure measurements. He was instructed to hold his left arm as still as possible for 20 seconds following cuff inflation, which occurred every two minutes. Sixty systolic and 60 diastolic automatic blood pressure readings were obtained

on each of the 20 control and 20 test runs. The EKG and the radial pulse were continuously recorded. During a quiet control run, the ambient noise inside the booth was recorded continuously with the B & K equipment previously described.

During the noise test run the procedure was the same. The same instructions and practice were given. An automatic audiogram was recorded six minutes into the run. The first 30 minutes of each noise test were quiet. The ambient noise level averaged about 38 dBA measured 3 inches from the subject's left ear. After 30 minutes of quiet the noise was turned on. The noise was broadband in type and was adjusted to 91 dBA $\pm$ 1 dB as measured 3 inches from the subject's left ear. The spectrum analysis is given for both the quiet and noise conditions in Figure 7. Continuous recording of the noise level was done.

At the end of one hour of noise the noise was turned off and the subject was instructed to put on the earphones for his second audiogram. This audiogram was begun immediately to determine the presence of temporary threshold shift. After completion of the audiogram the earphones were removed and the subject remained in the now quiet chamber until the two-hour run was completed.

Each of the 20 subjects served as his own control in a separate run on a different day. Six subjects experienced the quiet control run first and fourteen subjects experienced the noise test run first.

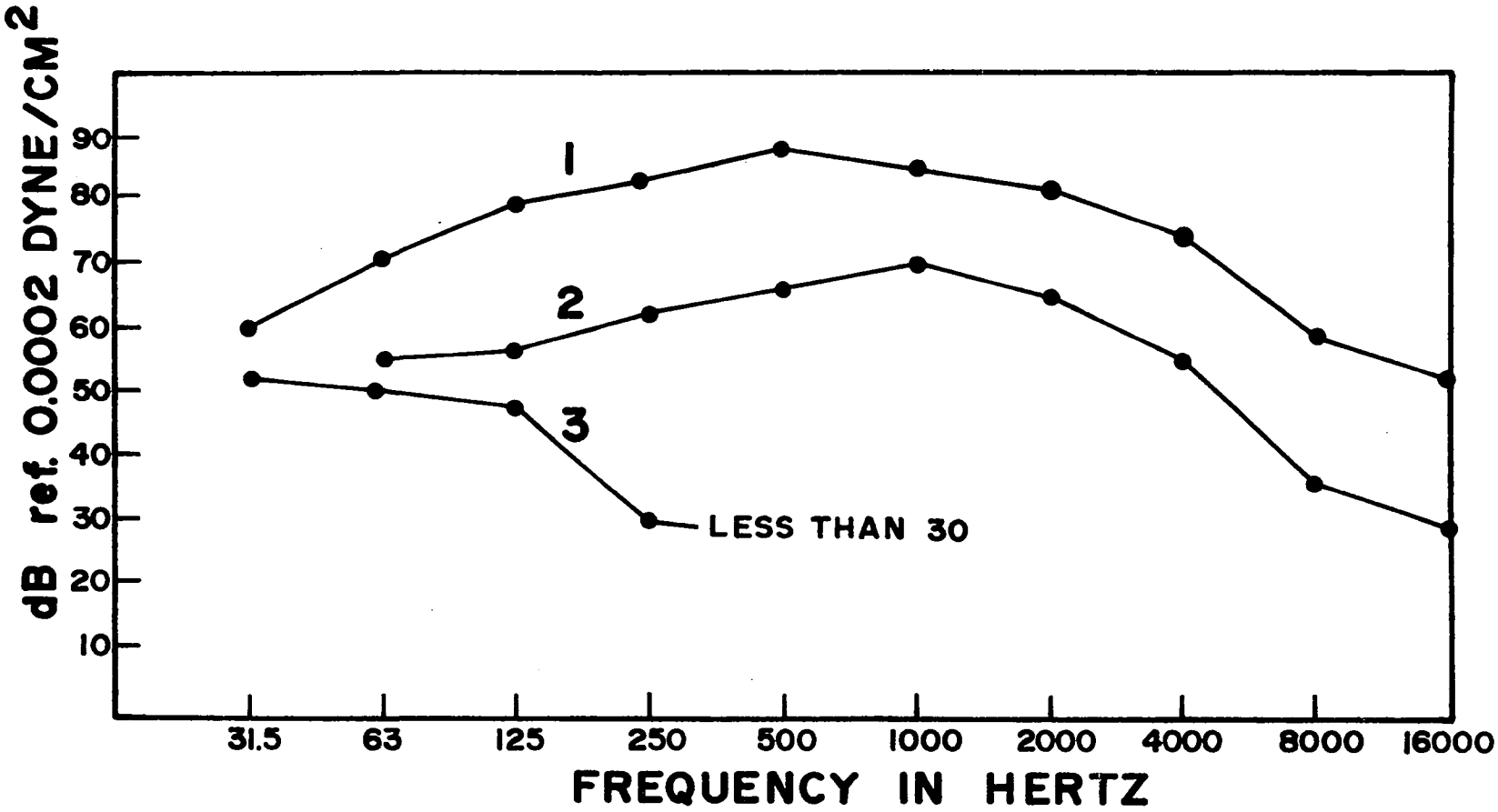

Figure 7.--Spectral analysis of: 1) noise used in this study, 2) noise used by Ponomarenko, and 3) "quiet" conditions of this study.

CHAPTER V

RESULTS AND DISCUSSION

The cardiovascular parameters of blood pressure, pulse and pulse pressure are continuously changing to meet body demands and stresses. For this reason, during the two-hour course of each experimental run, both control and test, each of the four cardiovascular parameters under study was determined every two minutes. This, of course, then gave 60 numerical values of raw data for each parameter for all forty test runs or a total of 9,600 raw data points.

In order to reduce this amount of raw data to manageable quantities, it was transferred to IBM computer cards and the computer determined "means of size three", thus reducing the original data to 3,200 mean data points. These mean data points are time dependent in the following manner. For each of the four parameters there were originally 60 numerical values. These 60 values were converted into 20 "means of size three" so that data point one represented the mean of measurements made at two, four, and six minutes into the run; and data point two was the mean of measurements made at eight, ten, and twelve minutes into the run. This conversion was continued up to data point 20 which consisted of measurements made at 116, 118, and 120 minutes into the run (the last six minutes).

The raw data for the parameters of blood pressure and pulse rate were averaged for each point in time for all subjects for both control and test conditions and are given numerically in Table 3 and presented graphically in Figures 8 and 9.

It is readily apparent from the graphical representation of the raw data that the cardiovascular parameters under study do undergo change during the course of the two hours of the run. The important question here is whether or not the changes observed in the several parameters are the same under both noise and quiet conditions. From the graphs it can be seen that there are differences in the two curves. Statistically paired t tests of several of these differences were performed to determine if the differences were due to any factor other than chance alone. The standard paired t test as described by Dixon and Massey (73) was employed. In all, 20 separate t tests on these raw data were performed. The t tests compared the amount of change between the test and control runs at five different points in time at the $\alpha = .05$ level. The "change values" represent the amount and direction of change for each parameter from the average value of that parameter during the first 30 minutes of the run.

The five time periods during which tests were performed are identified below by the letters A through E:

a. Point A: the first six minutes of the noise.

b. Point B: the average of the central twelve minutes of the noise.

c. Point C: the last six minutes of the noise.

d. Point D: the first six minutes after the cessation of the noise, i.e., the start of the quiet period after the one-hour exposure to the noise.

TABLE 3

AVERAGE VALUES OF RAW DATA

Time into Run (in min.)	Mean for All Subjects Corresponding Data Point	Systolic blood pressure in mm of Hg		Systolic blood pressure in mm of Hg		Pulse rate in beats per minute	
		Control	Test	Control	Test	Control	Test
6	1	110.6	108.5	65.4	66.5	80.5	79.9
12	2	108.3	105.1	63.9	63.8	80.5	79.3
18	3	104.7	104.2	62.8	63.8	79.6	78.0
24	4	104.3	103.1	62.2	62.2	78.9	76.9
30	5	103.7	101.9	62.7	62.9	78.8	75.6
36	6	104.1	103.3	62.3	64.5	77.8	76.0
42	7	103.5	102.2	62.8	64.6	76.5	76.0
48	8	102.6	101.8	62.8	64.8	77.6	75.6
54	9	104.1	102.2	63.7	65.2	76.5	76.7
60	10	102.8	102.1	64.0	65.2	76.3	76.2
66	11	102.5	102.7	62.6	65.2	75.6	75.7
72	12	102.2	102.1	62.9	65.8	75.4	76.6
78	13	102.4	102.0	63.8	64.9	75.5	75.4
84	14	103.8	101.6	64.1	65.0	76.5	74.6
90	15	103.5	102.3	64.9	66.1	75.9	75.2
96	16	104.6	105.2	65.5	66.7	74.3	73.4
102	17	105.0	105.1	66.7	67.4	74.8	73.9
108	18	103.7	102.7	65.3	66.3	74.5	73.5
114	19	103.9	103.4	65.5	67.9	74.8	72.7
120	20	103.5	103.6	66.9	67.8	74.2	73.2

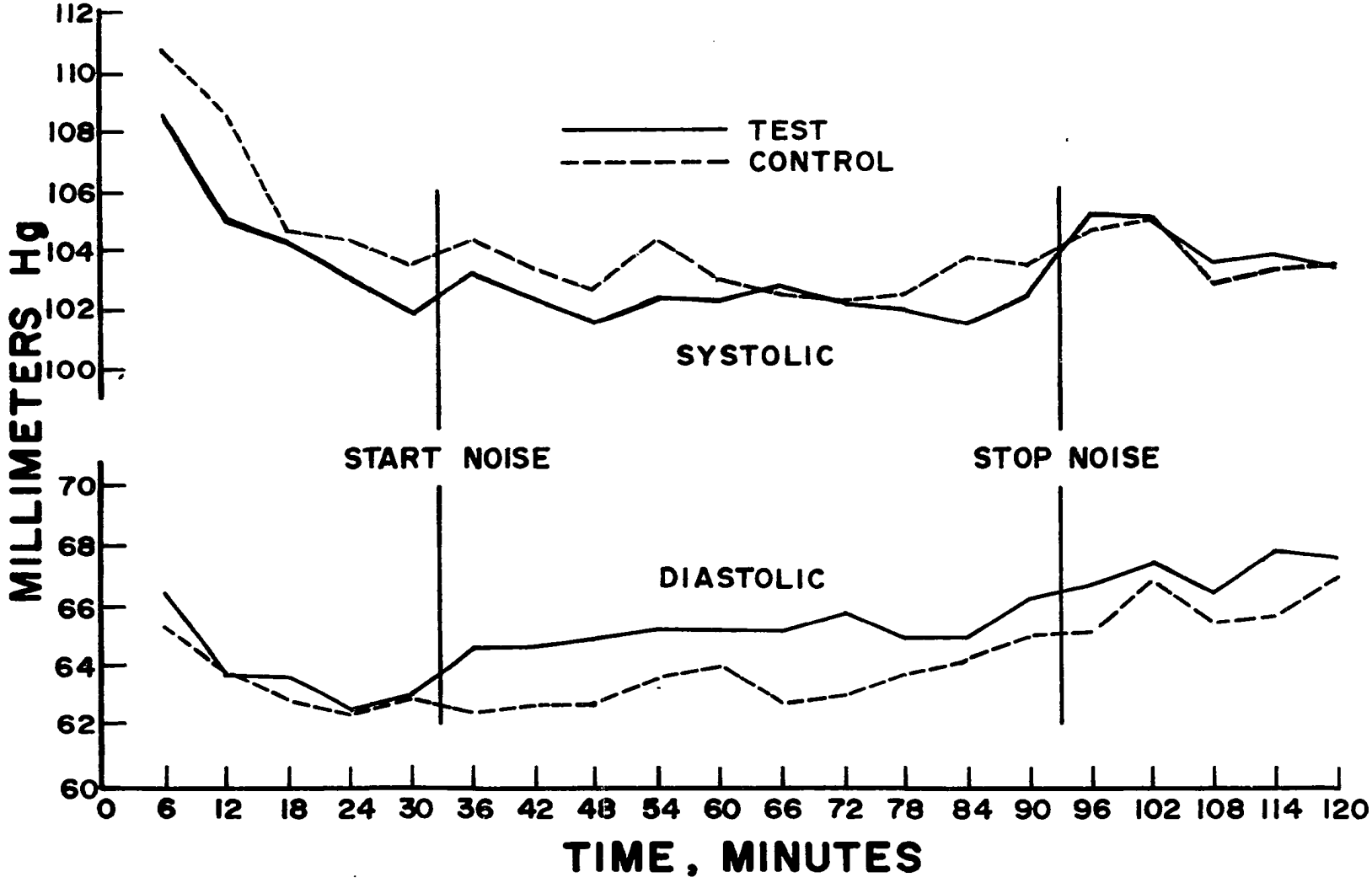

Figure 8.--Mean systolic and corresponding mean diastolic blood pressures based on raw data for all subjects during all experimental runs (20 control, 20 test).

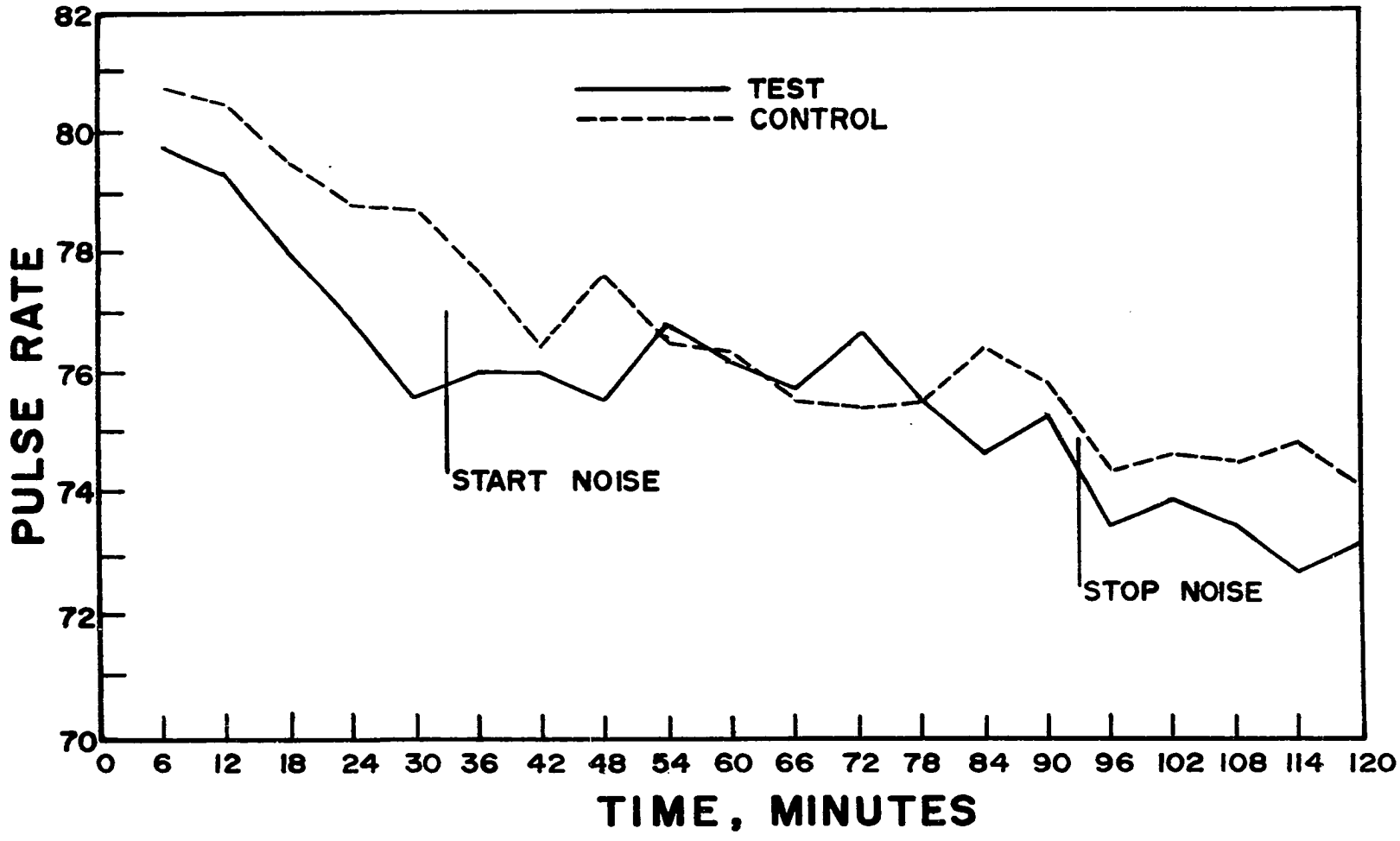

Figure 9.--Mean raw data pulse rate for all subjects during all experimental runs (20 control, 20 test).

e. Point E: the last six minutes of the two-hour run.

The results of the paired t test are given in Table 4. It can be seen that only two of the 20 t tests performed were significant with α at the .05 level and both of these occurred in the systolic blood pressure test.

The more important tests are the comparisons of the change in any given parameter at point C (the last six minutes of the noise) because at this point the subject would have been exposed to the test noise for the longest period of time. None of the parameters showed a statistically significant change at this point in time. It should be noted also that out of any series of t tests at the $\alpha = .05$ level (even if the data were completely random) one "significant" result would appear on the average in every 20 tests.

The original null hypothesis (H_0: noise causes no statistically significant change in any of the cardiovascular parameters under study here) was not rejected because the t value for all parameters at point C was too small for statistical significance at the $\alpha = .05$ level. When the null hypothesis is not rejected, the 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 β power of the t test at the $\alpha = .05$ level was determined in the standard manner as described by Dixon and Massey (74). Using the alternate hypothesis (H_A: noise will produce a $\pm$ 6 mm or greater change in the pressure tests or a six beat per minute or greater change

TABLE 4

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

in the pulse rate), the β power of each test is given in Table 5.

TABLE 5

BETA POWER TABLE

Test	Time Period	H_A	Power
Systolic B.P.	15	$\pm$ 6mm Hg	$p > .995$
Diastolic B.P.	15	$\pm$ 6mm Hg	$p > .975$
Pulse Rate	15	$\pm$ 6 beats per min.	$p > .995$
Pulse Pressure	15	$\pm$ 6mm Hg	$p > .975$

Several factors can influence the raw data values of the cardiovascular parameters under study here. Among these are the emotional "set" of a subject on a given day (75) and the season of the year (76). For these reasons it was decided to convert the raw data for each subject into percentage change values which would help to eliminate these additional sources of error. The results of the conversion of the raw data into percentage change values are given in Tables 6 through 13 and are shown graphically in Figures 10 through 13.

It is not statistically permissible to use the same type of paired t test on the data after it has been manipulated in this fashion because, on the average, the t values will be zero due to the manipulation.

The graphs do present an accurate visual representation of the behavior and trends of the various parameters. It should be noted that under conditions of both quiet and noise the following occurred:

TABLE 6

PERCENTAGE CHANGE FROM CONTROL SYSTOLIC BLOOD PRESSURE MEAN
DURING CONTROL RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	6.2010	3.7339	0.8349	13.55	0.06	13.49
2	4.4980	4.3599	0.9749	12.45	-4.72	17.17
3	0.4790	2.3865	0.5336	5.85	-3.88	9.73
4	0.0480	2.6054	0.5826	5.51	-4.35	9.86
5	-0.4515	2.4383	0.5452	3.35	-6.14	9.49
6	-0.0950	2.0602	0.4607	4.60	-3.35	7.95
7	-0.6820	1.9920	0.4454	2.26	-4.90	7.16
8	-1.5935	1.4752	0.3299	1.85	-4.48	6.33
9	-0.1170	2.2014	0.4923	4.23	-4.29	8.52
10	-1.3710	3.0722	0.6870	4.91	-9.46	14.37
11	-1.7385	2.5726	0.5753	3.78	-6.54	10.32
12	-1.9300	2.1186	0.4737	2.87	-7.31	10.18
13	-1.8070	1.9117	0.4275	1.48	-5.81	7.29
14	-0.4090	2.2686	0.5073	5.36	-4.15	9.51
15	-0.8015	2.4823	0.5551	3.15	-4.79	7.94
16	0.4000	2.4763	0.5537	4.20	-4.88	9.08
17	0.8015	2.9298	0.6551	8.64	-4.47	13.11
18	-0.4780	2.6907	0.6017	3.99	-4.90	8.89
19	-0.3315	1.6954	0.3791	3.13	-3.45	6.58
20	-0.6205	2.7932	0.6246	4.31	-6.17	10.48

TABLE 7

PERCENTAGE CHANGE FROM THE MEAN OF THE SYSTOLIC BLOOD PRESSURE
DURING TEST RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	5.2160	4.8019	1.0737	18.44	-1.40	19.84
2	1.7765	3.0060	0.6722	7.96	-5.61	13.57
3	0.9095	2.5867	0.5784	6.19	-3.65	9.84
4	-0.1120	2.1196	0.4739	3.72	-4.25	7.97
5	-1.3150	2.4978	0.5585	3.82	-7.40	11.22
6	-0.0425	2.5952	0.5803	4.35	-4.49	8.84
7	-1.0175	2.2141	0.4951	3.26	-4.22	7.48
8	-1.4385	2.1104	0.4719	2.46	-5.97	8.43
9	-1.1175	2.2219	0.4968	2.04	-6.75	8.79
10	-1.1810	2.8261	0.6319	3.98	-7.14	11.12
11	-0.5835	1.2793	0.2861	1.79	-3.30	5.09
12	-1.1815	1.9322	0.4321	2.16	-5.11	7.27
13	-1.2465	2.2190	0.4962	3.17	-6.56	9.73
14	-1.5955	2.2055	0.4932	3.75	-4.69	8.44
15	-0.8535	2.3572	0.5271	4.06	-6.12	10.18
16	1.8875	2.8542	0.6382	9.04	-3.26	12.30
17	1.8175	2.3738	0.5308	6.87	-1.51	8.38
18	-0.4955	2.2810	0.5100	4.59	-4.38	8.97
19	0.2185	2.5172	0.5629	6.39	-3.16	9.55
20	0.3520	3.0155	0.6743	6.71	-5.72	12.43

TABLE 8

PERCENTAGE CHANGE FROM THE MEAN OF THE DIASTOLIC BLOOD PRESSURE
DURING CONTROL RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	1.7990	8.3498	1.8671	18.64	-12.73	31.37
2	-0.5515	5.6396	1.2611	11.43	- 9.06	20.49
3	-2.0730	3.3434	0.7476	2.72	- 7.68	10.40
4	-2.9360	4.8009	1.0735	5.17	-12.73	17.90
5	-2.1475	4.6309	1.0355	8.89	-11.09	19.98
6	-2.7145	2.5819	0.5773	3.68	- 6.46	10.14
7	-2.1260	3.3414	0.7472	3.26	- 9.12	12.38
8	-2.0275	3.3650	0.7524	4.14	- 6.98	11.12
9	-0.5725	4.7054	1.0521	13.75	- 8.54	22.29
10	-0.0225	4.3716	0.9775	6.18	-10.40	16.58
11	-2.1525	3.6867	0.8244	5.44	-10.31	15.75
12	-0.2945	5.5152	1.2332	9.29	-10.63	19.92
13	-0.3210	5.4390	1.2162	6.89	-16.69	23.58
14	0.1845	5.1549	1.1527	10.79	- 9.07	19.86
15	1.3250	5.3443	1.1950	10.32	- 8.54	18.86
16	2.0550	3.8300	0.8564	8.25	- 5.97	14.22
17	4.0185	4.2226	0.9442	12.46	- 4.79	17.25
18	1.7955	4.5014	1.0065	13.40	- 4.98	18.38
19	2.1395	4.1726	0.9330	10.24	- 6.83	17.07
20	4.6215	5.6756	1.2691	13.40	-10.63	24.03

TABLE 9

PERCENTAGE CHANGE FROM THE MEAN OF THE DIASTOLIC BLOOD PRESSURE DURING TEST RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	1.8375	6.6921	1.4964	16.15	-11.90	28.05
2	-2.3665	5.6180	1.2562	13.37	-12.73	26.10
3	-2.3035	4.5255	1.0119	4.26	-12.66	16.92
4	-4.8115	4.0223	0.8994	2.86	-11.61	14.47
5	-3.6245	4.5534	1.0182	6.55	-11.86	18.41
6	-1.3005	4.3925	0.9822	7.14	- 9.67	16.81
7	-1.1545	4.1980	0.9387	4.97	-10.11	15.08
8	-0.8605	3.6913	0.8254	6.17	-10.95	17.12
9	-0.3960	4.1105	0.9191	7.04	- 9.67	16.71
10	-0.3220	3.7008	0.8275	5.65	- 7.74	13.39
11	-0.3420	3.6161	0.8086	7.64	- 7.08	14.72
12	0.6335	3.8366	0.8579	9.22	- 6.38	15.60
13	-0.5845	4.5855	1.0254	7.70	-14.25	21.95
14	-0.4845	4.1490	0.9277	8.75	- 7.43	16.18
15	1.1480	6.1959	1.3854	11.51	-10.32	21.83
16	2.0325	5.0995	1.1403	11.86	-11.56	23.42
17	3.3160	4.6109	1.0310	11.45	- 6.28	17.73
18	1.5825	5.0680	1.1332	13.12	- 9.29	22.41
19	4.1270	5.2238	1.1681	16.57	- 3.16	19.73
20	3.8700	3.9927	0.8928	10.71	- 2.75	13.46

TABLE 10

PERCENTAGE CHANGE FROM THE MEAN OF THE PULSE RATE DURING CONTROL RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	5.2100	5.8286	1.3033	15.62	- 6.25	21.87
2	4.9395	4.4819	1.0022	13.26	- 3.12	16.38
3	3.7455	4.9254	1.1013	11.86	- 3.12	14.98
4	2.8010	3.5399	0.7915	9.05	- 4.63	13.68
5	2.7550	3.3722	0.7540	9.43	- 3.12	12.55
6	1.2660	3.8356	0.8577	6.28	- 7.24	13.52
7	-0.3965	4.1277	0.9230	5.84	-10.08	15.92
8	1.2190	2.4568	0.5493	5.26	- 6.21	11.47
9	-0.3660	3.4052	0.7614	3.76	- 8.28	12.04
10	-0.6975	3.1917	0.7137	5.060	- 8.28	13.34
11	-1.4470	3.3438	0.7477	7.90	- 6.16	14.06
12	-1.7995	3.6267	0.8110	4.55	- 8.26	12.81
13	-1.7055	4.6434	1.0383	12.50	-10.75	23.25
14	-0.5350	3.6800	0.8229	8.59	- 7.58	16.17
15	-0.9285	3.5654	0.7972	5.15	- 8.41	13.56
16	-3.1910	3.8191	0.8540	3.25	- 9.20	12.45
17	-2.5490	4.1789	0.9344	7.98	- 9.20	17.18
18	-2.7990	4.3947	0.9827	7.98	-10.66	18.64
19	-2.4010	4.2702	0.9548	5.62	-10.82	16.44
20	-3.1130	5.0612	1.1317	4.03	-14.47	18.50

TABLE 11

PERCENTAGE CHANGE FROM THE MEAN OF THE PULSE RATE DURING TEST RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	5.4715	6.2970	1.4081	21.36	- 3.94	25.30
2	4.6980	6.0948	1.3628	16.34	- 5.03	21.37
3	2.6870	6.5839	1.4722	20.29	- 7.84	28.13
4	1.4365	4.1729	0.9331	10.53	- 8.79	19.32
5	-0.3265	4.2853	0.9582	9.45	- 7.78	17.23
6	0.3850	3.1985	0.7152	7.26	- 5.02	12.28
7	0.3855	3.1992	0.7154	6.63	- 5.92	12.55
8	-0.0675	3.4643	0.7747	6.45	- 7.36	13.81
9	1.3505	2.2347	0.4997	5.32	- 4.01	9.33
10	0.6750	2.8377	0.6345	7.01	- 4.67	11.68
11	0.0360	2.0287	0.4536	4.41	- 3.37	7.78
12	1.1695	5.2517	1.1743	8.50	-14.95	23.45
13	-0.4930	4.3935	0.9824	6.52	-15.52	22.04
14	-1.6370	4.2936	0.9601	4.44	-14.95	19.39
15	-0.5835	3.6998	0.8273	6.52	-10.25	16.77
16	-2.9885	5.5908	1.2501	7.58	-11.71	19.29
17	-2.4695	3.2486	0.7264	2.80	- 8.53	11.33
18	-2.7720	5.2086	1.1647	11.43	-10.95	22.38
19	-3.9000	4.8531	1.0852	4.44	-12.44	16.88
20	-3.0575	5.6373	1.2605	10.47	-14.63	25.10

TABLE 12

PERCENTAGE CHANGE FROM THE MEAN VALUE FOR THE PULSE PRESSURE
DURING CONTROL RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	12.6300	10.5710	2.3637	29.50	-13.43	42.93
2	11.9180	11.2825	2.5229	31.88	-19.90	51.78
3	4.2945	9.2332	2.0646	20.96	-15.86	36.82
4	4.8800	7.9175	1.7704	17.46	- 8.51	25.97
5	2.3590	9.0028	2.0131	26.84	-11.33	38.17
6	4.5965	7.7066	1.7233	23.74	- 9.63	33.37
7	1.4465	6.2289	1.3928	12.83	-11.11	23.94
8	-0.7845	6.8039	1.5214	14.31	- 9.54	23.85
9	0.8470	8.4384	1.8869	15.96	-21.29	37.25
10	-3.6830	7.2473	1.6206	10.84	-19.02	29.86
11	-1.3995	8.6458	1.9333	13.27	-17.14	30.41
12	-4.2800	9.6847	2.1656	14.08	-19.01	33.09
13	-3.8970	8.6829	1.9416	17.03	-15.16	32.19
14	-0.7915	9.1617	2.0486	16.22	-17.27	33.49
15	-4.5435	11.5571	2.5843	14.89	-21.04	35.93
16	-2.5450	8.0800	1.8067	9.23	-19.62	28.85
17	-4.2670	9.1766	2.0520	12.77	-17.90	30.67
18	-3.8115	10.1907	2.2787	11.85	-17.68	29.53
19	-4.5245	7.7431	1.7314	8.34	-17.26	25.60
20	-8.4410	8.6286	1.9294	6.80	-22.57	29.37

TABLE 13

PERCENTAGE CHANGE FROM THE MEAN VALUE FOR THE PULSE PRESSURE DURING TEST RUNS (WITH OTHER STATISTICAL DATA)

Data Point	Percentage Change from Mean	S.D.	S.E. of Mean	Maximum	Minimum	Range
1	11.4785	11.5917	2.5920	35.15	- 4.14	39.29
2	8.5150	9.9255	2.2194	20.39	-13.52	33.91
3	6.0700	9.8652	2.2059	25.05	-13.39	38.44
4	7.5640	9.0296	2.0191	22.93	-10.98	33.91
5	2.5765	10.8463	2.4253	20.03	-23.46	43.49
6	2.2395	10.7360	2.4006	23.80	-14.40	38.20
7	-0.1810	8.4040	1.8792	22.69	-11.92	34.61
8	-2.2065	7.8481	1.7549	11.95	-24.95	36.90
9	-2.5795	7.8388	1.7528	7.70	-16.41	24.11
10	-3.1760	9.0243	2.0179	13.51	-21.04	34.55
11	-1.0725	4.9927	1.1164	8.37	-10.31	18.68
12	-4.6660	8.2470	1.8441	10.10	-17.16	27.26
13	-2.3375	10.6602	2.3837	27.90	-26.28	54.18
14	-3.3795	10.4152	2.3289	21.70	-23.73	45.43
15	-4.2840	11.9438	2.6707	21.95	-28.02	49.97
16	0.9385	9.3886	2.0993	22.09	-17.78	39.87
17	-0.7830	9.1268	2.0408	21.59	-18.26	39.85
18	-3.7635	9.7894	2.1890	15.61	-19.39	35.00
19	-5.9530	9.9304	2.2205	12.79	-19.43	32.22
20	-4.9940	10.2756	2.2977	14.80	-20.97	35.77

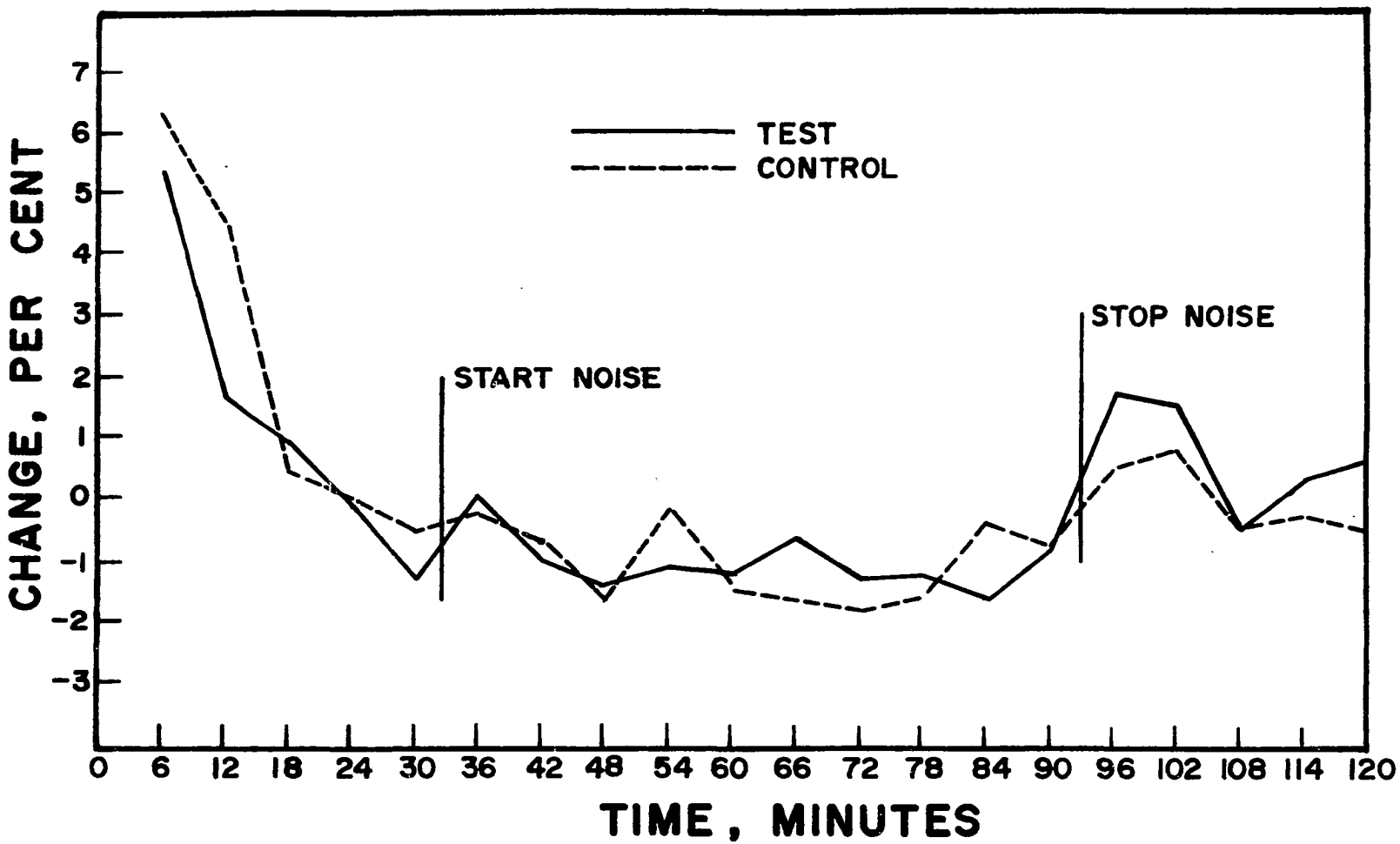

Figure 10.--Percentage change in systolic blood pressure from mean.

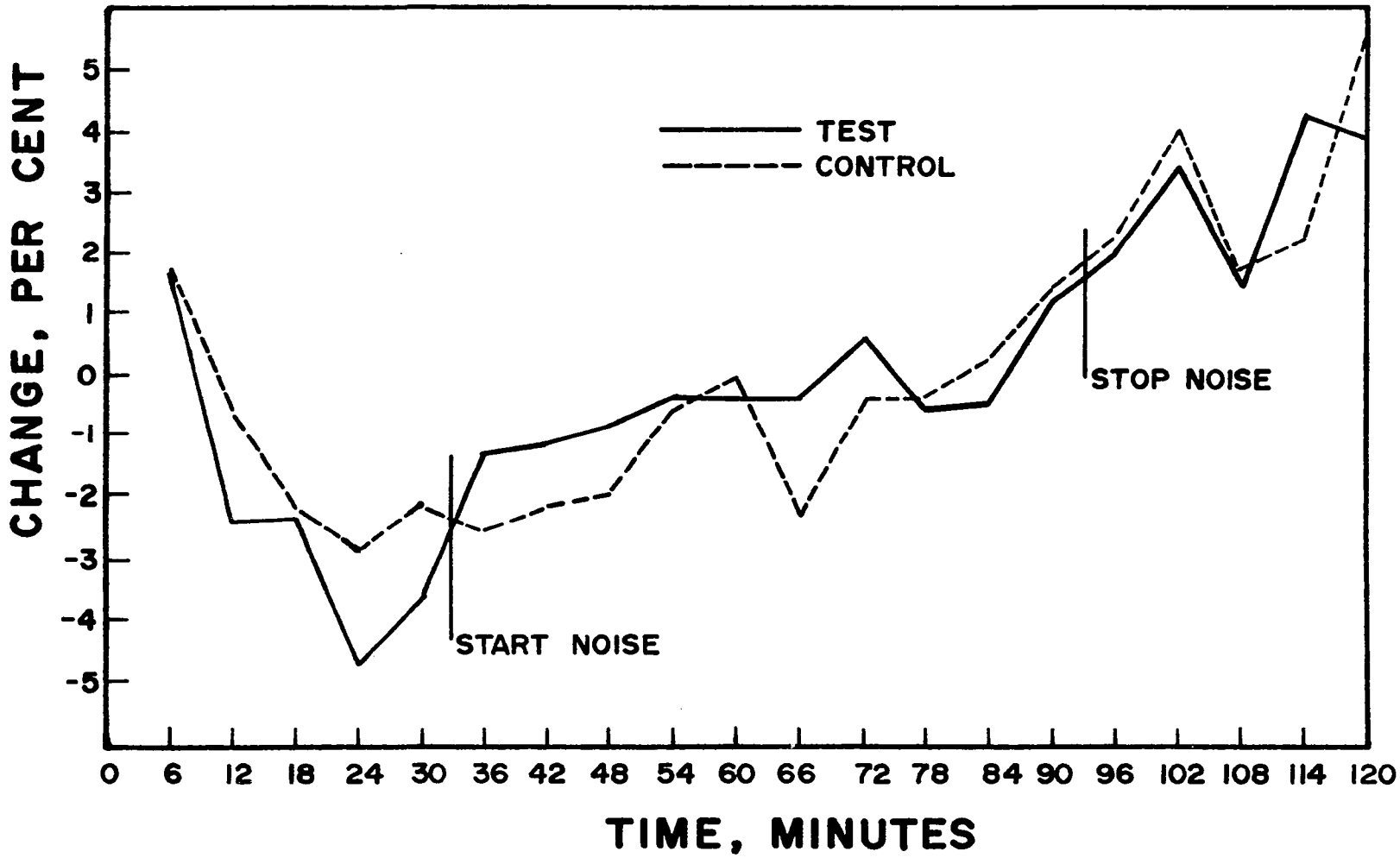

Figure 11.--Percentage change in diastolic blood pressure from mean.

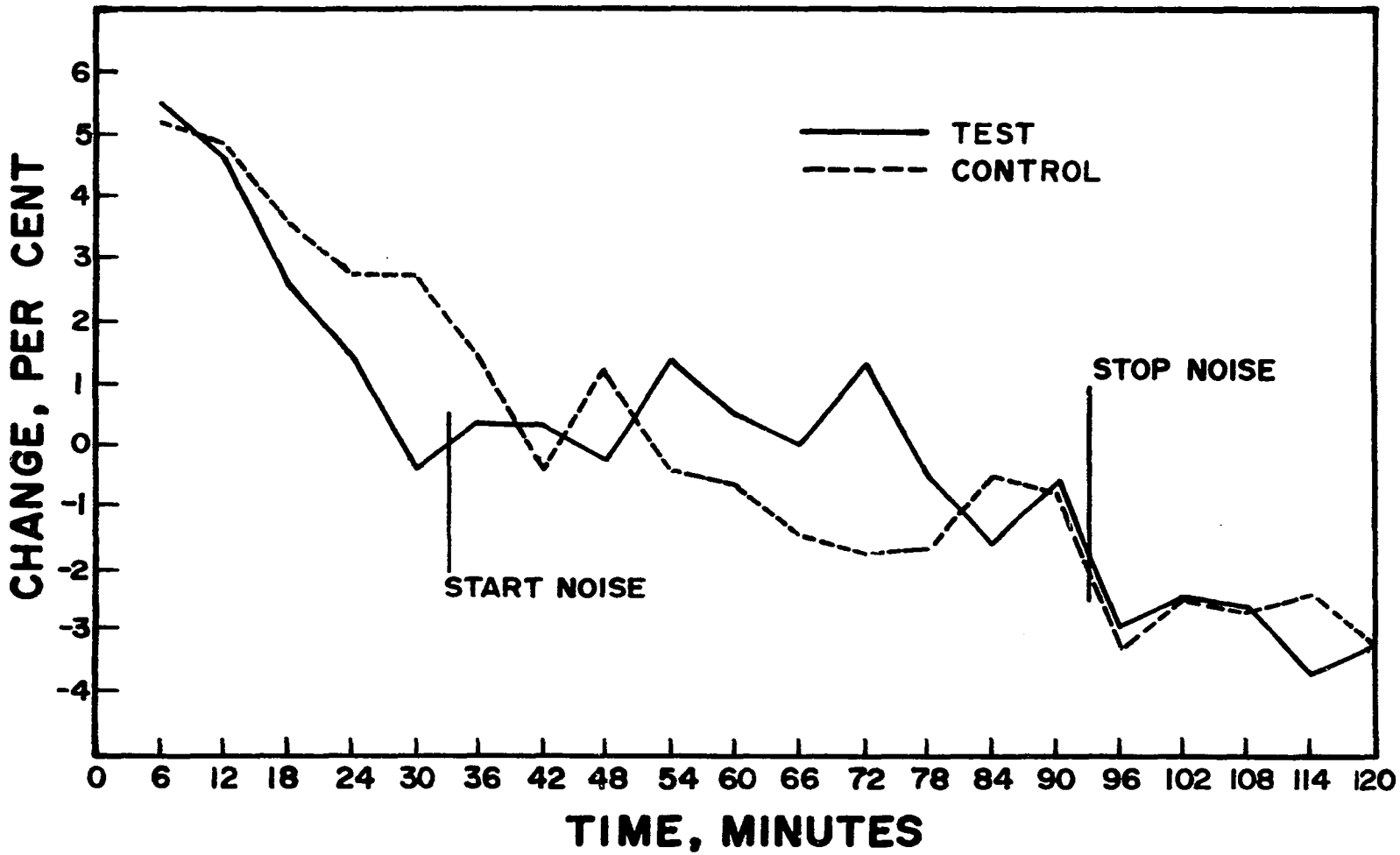

Figure 12.--Percentage change in pulse rate from mean.

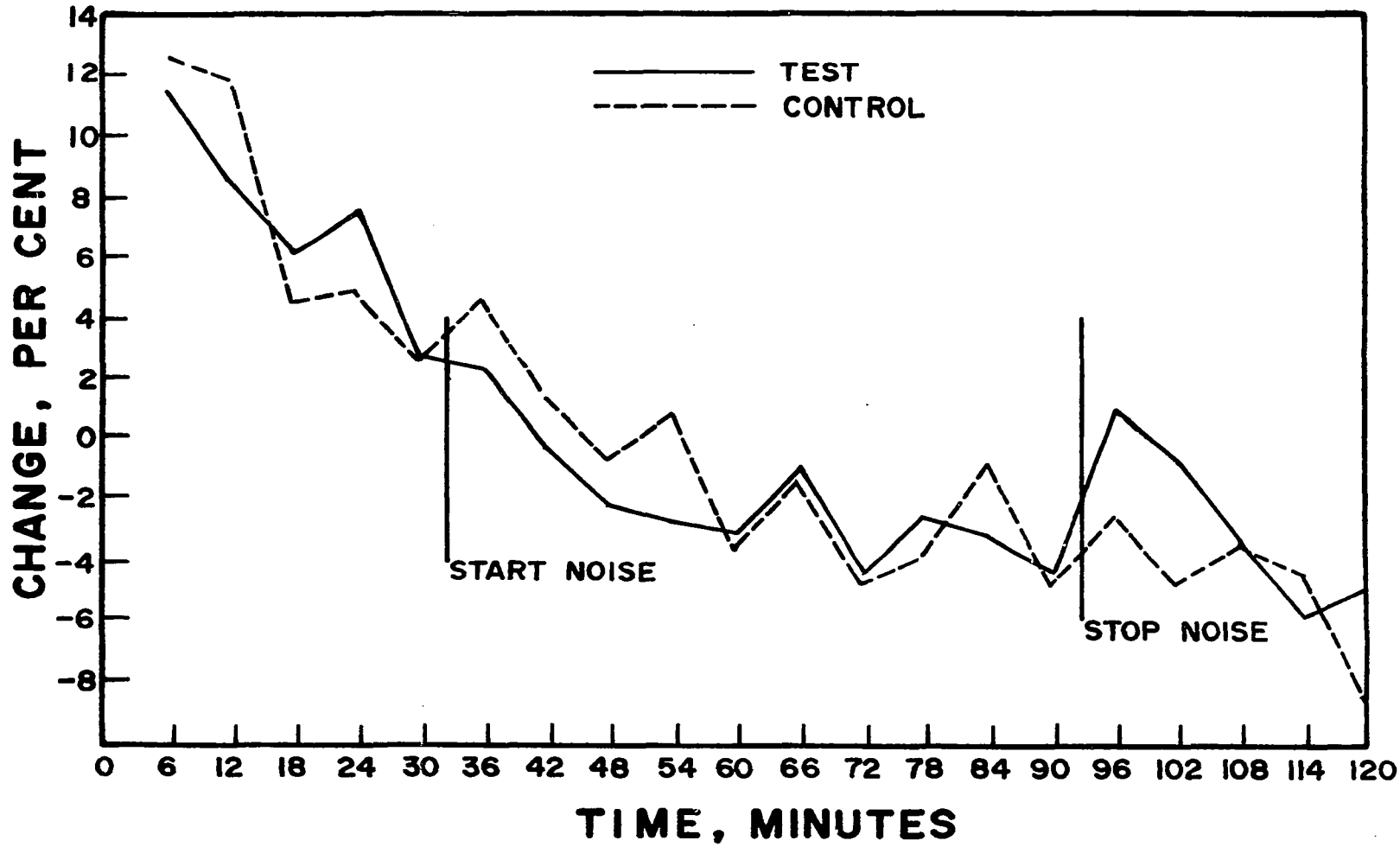

Figure 13.--Percentage change in pulse pressure from mean.

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

Eight paired t tests were performed on the percentage change data comparing each parameter's initial value (data point 1) to its value at the end of the experiment (data point 20). The results of the eight paired t tests are given in Table 14.

TABLE 14

RESULTS OF t TESTS PERFORMED ON
PERCENTAGE CHANGE DATA

Parameter	Condition	Comparison	Significance
Systolic BP	control	point 1 to point 20	$p < .001$
Systolic BP	test	point 1 to point 20	$p < .001$
Diastolic BP	control	point 1 to point 20	$p < .001$
Diastolic BP	test	point 1 to point 20	$p < .001$
Pulse Rate	control	point 1 to point 20	$p < .001$
Pulse Rate	test	point 1 to point 20	$p < .001$
Pulse Pressure	control	point 1 to point 20	$p < .001$
Pulse Pressure	test	point 1 to point 20	$p < .001$

CHAPTER VI

CONCLUSIONS

The influence of long term noise on the cardiovascular system of normal human beings was investigated in this study. The cardiovascular parameters of blood pressure, pulse rate, and electrocardiogram were continuously monitored during noise exposure and during quiet control conditions. Because these cardiovascular parameters are constantly changing, an attempt was made to detect a difference in these changes under noise stress as opposed to these changes under quiet control conditions. From the results of this study the following conclusions were drawn:

a. The exposure of resting normal adult volunteer subjects to broadband noise of 91 dBA for a period of one hour produces no statistically significant change in any of the parameters measured (at the $\alpha = .05$ level). The Beta power of the tests was always greater than 97.5 per cent using the alternate hypothesis that said noise would produce a change of 6 mm Hg in the blood pressure tests or 6 beats per minute in the pulse rate.

b. Two hours of chair rest in normal subjects produces the following cardiovascular changes which are all 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
   4) a marked decrease in the pulse pressure.

   These findings are true for experimental noise levels of both 30 dBA and 91 dBA.

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

CHAPTER VII

SUMMARY

This research was designed to attempt to determine whether or not random broadband noise of 91 dBA produces any statistically significant change in certain cardiovascular functions in normal adults.

Twenty healthy subjects (eleven female and nine male), ages 17 to 49 years, were exposed to the 91 dBA noise stimulus for a period of one hour while resting in a chair inside of an audiometric chamber. Each subject served as his own control by repeating the experiment under identical conditions except that the ambient noise was only about 38 dBA during the control runs.

The cardiovascular parameters of blood pressure, pulse rate, and pulse pressure were determined every two minutes using automatic electromechanical sensors and recording equipment. The values obtained under noise test conditions were compared to the corresponding quiet control values using standard paired t testing procedures.

It was determined that at the statistical significance level of $\alpha = .05$, the 91 dBA noise stimulus had no effect on any of the cardiovascular parameters under study. The β power using the alternate hypothesis (H_A: 91 dBA noise will produce a $\pm$ 6 unit change in any given parameter) resulted in a β power greater than .975 in all cases.

LIST OF REFERENCES

1. Cohen, A. "Effects of Noise on Psychological State," ASHA Reports, 4:74-88 (1969).

2. Jansen, G. "Effects of Noise on Physiological State," ASHA Reports, 4:89-98 (1969).

3. Anthony, A. "Changes in Adrenals and Other Organs Following Exposure of Hairless Mice to Intense Sound," J. Acoust. Soc. Am., 28:270-274 (1956).

4. United States Air Force Headquarters Air Material Command Engineering Division Memorandum Report. Subject: Physiological Effects of Intense Sound, 1948.

5. Danner, P. A., Ackerman, E., and Frings, H. W. "Heating of Haired and Hairless Mice in High Intensity Sound Fields from 6 to 22 kc," J. Acoust. Soc. Amer., 26:731-739 (1954).

6. Davis, H., Parrack, H., and Eldredge, D. "Hazards of Intense Sound and Ultra Sound," Ann. Oto.-Rhino.-Laryng., 58:732-738 (1949).

7. Rosenblith, W. A. "Effects of Noise on Man: Problems for Study," Noise Control, 1:22-27 (1955).

8. Welch, B. L. and Welch, A. S. Physiological Effects of Noise. New York and London: Plenum Press, 1970.

9. Anonymous. Publisher's Flyer, Physiological Effects of Noise, B. L. Welch and A. S. Welch (Eds.). New York and London: Plenum Press, 1970.

10. Kryter, K. D. The Effects of Noise on Man. New York and London: Academic Press, 1970.

11. Kryter, K. D. The Effects of Noise on Man. New York and London: Academic Press, 1970, p. 587.

12. Kryter, K. D. "Non-Auditory Effects of Environmental Noise," Amer. J. Pub. Health, 62:389-398 (1972).

13. Glorig, A. "Noise Exposure - Facts and Myths," Trans. Amer. Acad. Opth. and Otol., 75:1254-1262 (1971).

14. Finkle, A. L. and Poppen, J. R. "Clinical Effects of Noise and Mechanical Vibrations of a Turbo-jet Engine on Man," J. Applied Physiology, 1:183-204 (1948).

15. Rosen, S. "Noise and Health," Mt. Sinai J. Med., 38:489-496 (1971).

16. Friedman, M., Byers, S. O., and Brown, A. E. "Plasma Lipid Responses of Rats and Rabbits to an Auditory Stimulus," Amer. J. Physiol., 212:1174-1178 (1967).

17. Rosen, S. "Noise and Health," Mt. Sinai J. Med., 38:495 (1971).

18. Kryter, K. D. The Effects of Noise on Man. New York and London: 1970, pp. 487-489.

19. Anticaglia, J. R. and Cohen, A. "Extra-Auditory Effects of Noise as a Health Hazard," Am. Ind. Hyg. Assoc. J., 31:277-281 (1970).

20. Jansen, G. "Effects of Noise on Physiological State," ASHA Reports, 4:89-98 (1969).

21. Rosen, S., Plester, D., El-Mofty, A., and Rosen, H. V. "Relation of Hearing Loss to Cardiovascular Disease," Tr. Am. Acad. Ophth. and Otol., 68:433-444 (1964).

22. Grandjean, E. "Summary," ASHA Reports, 4:99-102 (1969).

23. Shatalov, N. N. and Murov, M. A. "The Influence of Intensive Noise and Neuropsychic Tension on the Level of Arterial Pressure and the Incidence of Hypertensive Vascular Disease," Klin Med (Moskva), 48:70-73 (1970) (trans. med.).

24. Mohr, G. C., Cole, J. N., Guild, E., and von Gierke, H. E. "Effects of Low Frequency and Infrasonic Noise on Man," Aerospace Med., 36:817-824 (1965).

25. Karrasch, K. "Die Wirkung des Larms auf den menschlichen Organismus (The Effect of Noise on the Human Organism), Arbeitswiss, 6: 177-182 (1952) (trans. med.).

26. Karrasch, K. "Die Wirkung des Larms auf den menschlichen Organismus (The Effect of Noise on the Human Organism), Arbeitswiss, 6: 179 (1952) (trans. med.).

27. Karrasch, K. "Die Wirkung des Larms auf den menschlichen Organismus (The Effect of Noise on the Human Organism), Arbeitswiss, 6: 180-182 (1952) (trans. med.).

28. Bode, H. "General Illness as a Result of Noise," Z.arztl. Fortbild., 54:1282 (1962) (trans. med.).

29. Kangelari, S. S., Abramovich-Poliakov, D. K., and Rudenko, V. F. "K voprosu o vliianii vibratsii i shuma na obshchviv zabolevaemost" (On the Influence of Vibration and Noise on General Disease), Gig Tr Prof Zabol, 10:47-49 (1966) (trans. med.).

30. Odescalchi, C. P. "The General Effects of Noise: Noise and Working Efficiency, Industrial Music," Med. Lavoro, 61:629-644 (1970).

31. Farr, L. E. "Medical Consequences of Environmental Home Noises," JAMA, 202:171-174 (1967).

32. Farr, L. E. "Medical Consequences of Environmental Home Noises," JAMA, 202:173-174 (1967).

33. Semczuk, B. and Gornz, H. "Studies of the Effect of Noise in the Cardiorespiratory Efficiency," Polski Tygodnik Le Karski (Warszawa), 25:83-85 (1970) (trans. med.).

34. Culliton, B. J. "Noise Menace Threatens Man," Science News, 90: 297-299 (1966).

35. Welch, B. L. "Physiological Effects of Audible Sound," Science, 166:533 (1969).

36. United States Air Force Headquarters Air Material Command Engineering Division Memorandum Report. Subject: Physiological Effects of Intense Sound, 1948.

37. Anthony, A., Ackerman, E., and Lloyd, J. A. "Noise Stress in Laboratory Rodents. I. Behavioral and Endocrine Response of Mice, Rats, and Guinea Pigs," J. Acoust. Soc. Am., 31:1430-1437 (1959).

38. Anthony, A., Ackerman, E., and Lloyd, J. A. "Noise Stress in Laboratory Rodents. I. Behavioral and Endocrine Response of Mice, Rats, and Guinea Pigs," J. Acoust. Soc. Am., 31:1437 (1959).

39. Anthony, A. and Ackerman, E. "Stress Effects of Noise in Vertebrate Animals," WADC Technical Report 58-622, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, 1959, p. 1.

40. Geber, W. F. and Anderson, T. A. "Cardiac Hypertrophy due to Chronic Audiogenic Stress in the Rat, Rattus Norvegicus Albinus, and Rabbit, Lepus Cuniculus," Comp. Biochem. Physiol., 21:273-277 (1967).

41. Klemparskaya, N. N. "Influence of Noise on Formation of Antibodies," Med. Radiol. (Moskva), 9:54-60 (1964) (trans. med.).

42. Lawrence, M., Gonzalez, G., and Hawkins, J. E. "Some Physiological Factors in Noise-Induced Hearing Loss," Am. Ind. Hyg. Assoc. J., 28:425-430 (1967).

43. Danner, P. A., Ackerman, E., and Frings, H. W. "Heating of Haired and Hairless Mice in High Intensity Sound Fields from 6 to 22 kc," J. Acoust. Soc. Amer., 26:731-739 (1954).

44. Finkle, A. L. and Poppen, J. R. "Clinical Effects of Noise and Mechanical Vibrations of a Turbo-jet Engine on Man," J. Applied Physiology, 1:183-204 (1948).

45. Finkle, A. L. and Poppen, J. R. "Clinical Effects of Noise and Mechanical Vibrations of a Turbo-jet Engine on Man," J. Applied Physiology, 1:190-191 (1948).

46. Steinmann, B., Jaggi, U., and Widmer, J. "Über den Einfluss von Gerauschen and Lärm auf den Blutdrock des Menschen" (On the Influence of Noise and Uproar on the Blood Pressure of Humans), Cardiologic (Basel), 27:223-229 (1955) (trans. med.).

47. Steinmann, B., Jaggi, U., and Widmer, J. "Über den Einfluss von Geräuschen and Lärm auf den Blutdrock des Menschen" (On the Influence of Noise and Uproar on the Blood Pressure of Humans), Cardiologic (Basel), 27:224 (1955) (trans. med.).

48. Lehmann, G. and Tamm, J. "Über Veränderungen der Kreislaufdynamik des ruhenden Menschen unter Einwirkung von Geräuschen" (Concerning the Changes of the Circulatory Dynamics of Resting Men under the Influence of Noises), Internat. Z. angew Physiol. einschl. Arbeitsphysiol., 16:217-227 (1956) (trans. med.).

49. Lehmann, G. and Tamm, J. "Über Veränderungen der Kreislaufdynamik des ruhenden Menschen unter Einwirkung von Geräuschen" (Concerning the Changes of the Circulatory Dynamics of Resting Men under the Influence of Noises), Internat. Z. angew Physiol. einschl. Arbeitsphysiol., 16:219-220 (1956) (trans. med.).

50. Lehmann, G. and Tamm, J. "Über Veränderungen der Kreislaufdynamik des ruhenden Menschen unter Einwirkung von Geräuschen" (Concerning the Changes of the Circulatory Dynamics of Resting Men under the Influence of Noises), Internat. Z. angew Physiol. einschl. Arbeitsphysiol., 16:221-222 (1956) (trans. med.).

51. Folk, E., Deal R. B., Jr., and Parker, D. E. Chief of Biostatistics, FAA Acronautical Center, Professor, Department of Biostatistics and Epidemiology, University of Oklahoma Health Sciences Center, and Professor, Department of Biostatistics and Epidemiology, University of Oklahoma Health Sciences Center, respectively, Oklahoma City, Oklahoma, 1973, personal communication.

52. Ponomarenko, I. I. "The Influence of Noise of Different Intensity Levels and Spectral Compositions on Some Physiologic Functions of Adolescents," Labor Hyg. and Occup. Diseases, 10:32-37 (1966) (trans. med.).

53. Ponomarenko, I. I. "The Influence of Noise of Different Intensity Levels and Spectral Compositions on Some Physiologic Functions of Adolescents," Labor Hyg. and Occup. Diseases, 10:34 (1966).

54. Terent'yev, V. G., Sheludyakov, E. E., and Sviridova, E. S. "The Reaction of the Human Nervous and Cardiovascular Systems to the Influence of Aviation Noise," Voennomed Zh (Military Medical Journal), 6:55-58 (1969) (trans. med.)

55. Hunter, D. The Diseases of Occupations. Boston: Little, Brown, and Co., 1969, p. 872.

56. Hildebrand, J. L. "Noise Pollution: An Introduction to the Problem and an Outline for Future Legal Research," Columbia Law Review, 70:652-692 (1970).

57. Rosen, S., Plester, D., El-Mofty, A., and Rosen, H. V. "Relation of Hearing Loss to Cardiovascular Disease," Tr. Am. Acad. Ophth. and Otol., 68:433-444 (1964).

58. Kryter, K. D. The Effects of Noise on Man. New York and London: Academic Press, 1970, p. 489.

59. Broch, J. T. The Application of the Bruel and Kjaer Measuring Systems to Acoustic Noise Measurements. Copenhagen: K. Larsen and Son, 1971.

60. Bordley, J., Connor, C., Hamilton, W. F., Kerr, W. J., and Wiggers, C. J. "Recommendations for Human Blood Pressure Determinations by Sphygmomanometers," Circulation, 4:503-509 (1951).

61. Bordley, J., Connor, C., Hamilton, W. F., Kerr, W. J., and Wiggers, C. J. "Recommendations for Human Blood Pressure Determinations by Sphygmomanometers," Circulation, 4:506 (1951).

62. Bazett, H. C. and Laplace, L. B. "Studies on the Indirect Measurement of Blood Pressure: Sources of Error in Riva-Rocci Method," Am. J. Physiol., 103:48-67 (1933).

63. Lategola, M. T. and Harrison, H. F. "A Device and Method for Rapid Indirect Measurement of Human Systolic and Diastolic Blood Pressures," FAA-AM 70-21, 1970.

64. Stevens, P. M. "Cardiovascular Dynamics during Orthostasis and the Influence of Intravascular Instrumentation," Am. J. Cardiol., 17:211-218 (1966).

65. Levin, H. S., Messer, J. V., and Pines, J. "Repeated Venous and Arterial Catheterization in Man," Am. Heart J., 73:475-483 (1967).

66. Geddes, L. A., Spencer, W. A., and Hoff, H. E. "Graphic Recordings of the Korotkoff Sounds," Am. Heart J., 57:361-370 (1959).

67. Burch, G. E. and DePasquale, N. P. Primer of Clinical Measurement of Blood Pressure. St. Louis: C. V. Mosby Co., 1962.

68. Davis, R. C., Siddons, G. F., and Stout, G. L. "Apparatus for Recording Autonomic States and Changes," Amer. J. Psychol., 67: 343-351 (1954).

69. Lategola, M. T. Chief of Experimental Physiology, FAA Aeronautical Center, Oklahoma City, Oklahoma, 1973, personal communication.

70. Davis, R. C., Siddons, G. F., and Stout, G. L. "Apparatus for Recording Autonomic States and Changes," Amer. J. Psychol., 67: 343-352 (1954).

71. Lagerwerff, J. M. and Luce, R. S. "Artifact Suppression in Indirect Blood Pressure Measurements," Aerospace Med., 41:1157-1161 (1970).

72. The World Almanac, 1971. New York: Newspaper Enterprise Association, Inc., 1971.

73. Dixon, W. J. and Massey, F. J. Introduction to Statistical Analysis. New York: McGraw-Hill, 1969, pp. 119-122.

74. Dixon, W. J. and Massey, F. J. Introduction to Statistical Analysis. New York: McGraw-Hill, 1969, pp. 84-87.

75. Anonymous. "Responsibility Brings Jump in Pulse," JAMA, 201:23, 28 (1967).

76. Schneider, R. A. and Costiloe, J. P. "Seasonal Variations in Cardiovascular Functioning," Arch. Environ. Health, 24:10-16 (1972).

APPENDIX

SUBJECTS' PERSONAL DATA

Subject No.	Age	Sex	Occupation	Highest Education Completed	Date Quiet Control Run	Date Noise Test Run
1	31	M	Grad Student	M.P.H.	10- 7-72	7- 7-72
2	24	M	Grad Student	College	12-29-72	7-14-72
3	38	M	Physician	M.D.	12-19-72	7-17-72
4	30	M	Salesman	College	1- 9-73	7-18-72
5	32	F	Housewife	M.C.D.	11- 3-72	7-24-72
6	30	M	Grad Student	College	10-11-72	7-26-72
7	21	F	Secretary	High School	11-15-72	7-31-72
8	45	F	Scientist	M.S.	11-30-72	8- 2-72
9	28	M	Chemist	M.S.	9-27-72	8- 7-72
10	23	F	Secretary	High School	10-11-72	8-23-72
11	27	F	Secretary	College	11- 1-72	8-30-72
12	26	F	Secretary	College	10-13-72	9- 6-72
13	27	M	Anthropologist	Ph.D.	10-15-72	9-13-72
14	17	F	Student	High School	12-22-72	9-20-72
15	49	M	Indust. Hygien.	Ph.D.	10- 4-72	10-27-72
16	30	F	Physician	M.D.	10-13-72	10-25-72
17	30	F	Housewife	College	12- 1-72	12- 5-72
18	26	M	Nurses Asst.	High School	12-14-72	12-20-72
19	22	F	Nurses Asst.	High School	12-21-72	1- 2-73
20	28	F	Nurses Asst.	High School	12-27-72	12-29-72