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Does Urban Noise Represent a Hazard to Health?

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DOES URBAN NOISE REPRESENT A HAZARD TO
HEALTH?

by
FRANCES J. STORLIE

A dissertation in partial fulfillment of the
requirements for the degree of

DOCTOR OF PHILOSOPHY
in
URBAN STUDIES

Portland State University
1976

TO THE OFFICE OF GRADUATE STUDIES AND RESEARCH:

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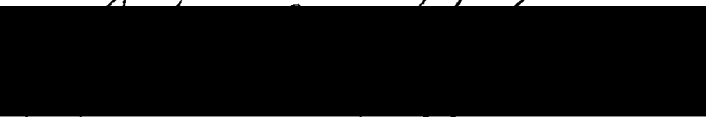
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AN ABSTRACT OF THE DISSERTATION OF Frances J. Storlie for a Doctor of Philosophy in Urban Studies presented November 12, 1976.

Title: Does Urban Noise Represent A Hazard To Health?

APPROVED BY MEMBERS OF THE DISSERTATION COMMITTEE:


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Charles Campbell, M.D.

The problem of noise as a potential health hazard to urban man has been raised. The literature was used to establish two premises: that cities are noisy environments, and that noise-free societies have less coronary artery disease (CAD) than do industrialized sections of the world. These differences also hold for rural and urban areas of the United States.

Geographical questions concerning rate differentials for CAD have been addressed by numerous disciplines. Subsequently, social, psychological, and physical explanations have been put forth. Throughout this paper the emphasis has

been placed on the physical aspects of noise exposure. The conceptual framework utilizes noise-load, overload, stress and deformation. Noise was described as a force capable of eliciting a predictable physiological response from the human organism. Noise was further conceptualized as a by-product of technology which exerts a stressor effect upon the cardiovascular system of man.

The investigation, from which the data were generated, was a micro-view of physiological effects, in that the only measurement taken was heart rate change in hospitalized patients in response to noise.

The heart rate was calculated under low noise conditions, and comparisons subsequently made to heart rate during noise. In addition, the noise climate for each of two coronary care units (CCU) was tabulated over a 24 hour period. Generally noise levels in the CCUs were higher than might be found in a man's own home. Only between the hours of 3:00 and four in the morning, was ambient noise equal to or below the suggested levels (45 dbA) for any sustained period of time.

Conditions of noise elicited heart rate change in 30 of 37 subjects ($P = .001$). This finding relates to the presence of a change and does not speak to the extent or meaningfulness of that change. Patients with heart attacks responded to noise conditions ($n=18$, $P = .01$) in that 17 of the eighteen patients experienced a change in heart rate when noise was introduced. No differences could be noted

for categories by site of infarctions.

It was further hypothesized that the extent of heart rate response (HRR) would be a function of the gap between low noise and high noise conditions. A regression analysis showed the response to be significantly correlated with noise gap for the total population (N=37, P= .05), however the correlation was minimal ($r=.4528$) with slightly less than 21 per cent of the variation in HRR explained by the variation in noise gap. Those subjects more than 60 years of age (n=20), also showed a significant correlation ($r=.5173$) with 26 per cent of the variation in HRR explained by the variation in noise gap. The highest correlation ($r=.7373$) was obtained for ten persons with a past history of heart disease ($r^2=.5436$, P=.05).

The implications for site planning and structure are many, particularly for hospitals, nursing, and convalescent homes where older persons with heart disease are housed. Site planning should give attention to noise environment; and structural planning, to sound-proofing. Interviews with architects and hospital builders showed this goal to be attainable mechanically, if somewhat costly.

It was agreed by those interviewed that such costs as evolve from noise-reduction or noise-proofing in hospitals would most certainly be passed on to the consumer and be reflected in his health care costs.

Additional research is needed which focuses on the effects of noise on the cardiovascular system over time,

using standardized criteria for cardiovascular health and cardiovascular disease. Other research might focus on larger samples of patients hospitalized with CAD, in an effort to identify an index of physiological and psychological responses to noise.

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CHAPTER I

INTRODUCTION

Urban noise creates stress. That is, it has the capacity to elicit a variety of physiological effects from the human body which may, in turn, be detrimental to the well-being of man. In this dissertation noise will be conceptualized as a noxious stimulus to persons exposed to it for more than very short periods of time.

Hospital patients, particularly those with coronary artery disease (CAD) may incur added risk should locational site decisions and/or room assignments within the institution, fail to take noise variables into account. Or, technological devices, if sufficiently noisy, may actually potentiate discomfort in the patient. Treatment facilities ranging from ambulatory clinics to hospital intensive care units, to structures erected for the elderly, may now have to consider location in terms of noise level, as well as the traditional economic variables such as access and transportation time.

A wide range of approaches should ultimately be employed in the study of any possible noise-effects on health, however, this dissertation will be limited to an examination of noise effect on the cardiovascular system, especially upon the heart rate, of a vulnerable population--that is, upon cardiovascular patients admitted to the coronary care unit (CCU).

To begin with, rate differentials for CAD will be examined. And evidence from such epidemiological and socio-cultural studies as are relevant will be presented to show that coronary artery disease is a more common occurrence in noise-saturated than noise-free societies. A review of the pertinent physiological data will detail the adverse effects of noise upon the cardiovascular system, particularly upon the heart.

The Problem to be Examined

A number of previous studies have identified noise sources around and inside the urban hospital; however, only a limited number of investigators have attempted to correlate noise levels with changes in vital functions of the body. In fact, the largest of these studies carried out by the U.S. Public Health Service in 1963, simply asked the respondents to identify noise that bothered them.

The framework for the present study draws heavily upon physical model of stress and proposes the presence of multiple stressors in the urban environment, one of which is noise. A field study will be designed with the purpose of measuring the effects of noise on patients with coronary artery disease. The stated hypothesis represents the problem to be addressed: Does noise affect heart rate? If so, are there relevant implications for health?

The data will identify noise sources common to an intensive coronary care unit (For example, suction apparatus,

breathing machines, bed rails) and catalogue their intensities. The Falk and Woods' study measured a small number of possible noise sources in an intensive care unit but did not correlate noise with changes in patient status. This dissertation will go on from the work of Falk and Woods, Marshall, and Shapiro to identify noise sources, measure their intensities, and finally, to correlate noise level with changes in heart rate. Twenty-four hour noise profiles for each of two units are expected to indicate hours that are typically noise-saturated and other times that are relatively noise free. Because the characteristics of patients with CAD are not homogenous (For example in age, sex, past medical history, and site of heart damage) differential responses of patient subgroups will be obtained.

Finally, it is estimated that approximately 28 million persons in the United States today suffer from coronary artery disease. One in every four adult males now living will experience a myocardial infarction* at some point in his lifetime. Many more men and women have, or are in the process of developing atherosclerotic coronary artery disease, without actual heart damage. Moreover, men and women over the age of 70 years are almost universally affected. Therefore the implications drawn from noise-health data have wide application for the urban setting, particularly for locational decisions regarding hospitals and rest homes, as well as for the placement of coronary care units within those structures,

*The general consensus is that the terms myocardial infarction, heart attack, and coronary occlusion are synonymous.

and the assignment of patients to hospital space.

NOISE IN THE COMMUNITY: AN OVERVIEW

Noise is a by product of industrialized society to which urban man is more or less continuously exposed. Transportation networks literally crisscross the once quiet plains and valleys delivering the roar of high speed motors and diesel trucks to populations hitherto unexposed. And within the boundaries of major cities the roar of aircraft flyover is a given. Freedom from noise is a luxury most residents cannot afford.

However, the recent industrialization of much of the United States has forced questions concerning the effects of technology upon the environment, and more importantly, upon man. Historically, these questions focused almost entirely upon air and water pollution. Only recently has noise been considered an environmental hazard to be measured and controlled. The question being posed with increasing frequency is whether noise affects the health and well-being of urban man.

Within the urban structure, the hospital is deemed the most sensitive of all institutions.¹ In no other does the question have equal significance. Planning decisions regarding the location of new health facilities have typically been

¹Baron, R., "Noise and Urban Man," AJPH 52:11:2060, November, 1968.

based on models of user-access and economics.² Furthermore, within the urban hospital, decisions about the placement of special care units and the channeling of traffic flows have been made without serious regard for the effects of noise upon the patients housed there.

Only in recent years has noise been conceptualized as a physical force. While noise may extensively explain the epidemic of hearing loss there is less consensus about the exact nature of the nonauditory health hazards attributable to noise.^{3,4,5,6} As far back as 1938 McChord stated that "the multiple and insidious effects of noise constitute an inadequately recognized baneful influence on the lives of millions of persons throughout the country, especially those who live in urban areas." He went on to say that, "noise deafness constitutes the most serious and tangible of the ill noise effects...but there is, in addition, a host of

²Clipson, C., and Wehrer, J., Planning for Cardiac Care: A Guide to planning and design. Ann Arbor, Michigan, The Health Administration Press. School of Public Health, 1973.

³Atherly, C.R., "Monday morning auditory threshold in weavers," British J. Indust. Med. No. 21:21, 1964.

⁴Davis, H., Hoople, G., and Parrack, H.O., "Hearing level, hearing loss and threshold shift," J. Acoust. Soc. Am. No. 30:478, 1958.

⁵Glorig, A., Ward, W., and Nixon, J., "Damage risk criteria and noise induced hearing loss," Arch. Otolaryng. No. 74:413, 1961.

⁶Kryter, K., "Exposure to steady-state noise and the impairment of hearing," J. Acoust. Soc. Am. No. 35:1515, 1936.

scarcely measurable injuries made evident by the neuroses, loss of sleep, excessive fatigue, emotional disturbances and the like, that jeopardize the complete well being of most persons.⁷

McChord's statements were prophetic, for many of his assumptions have been substantiated by later research. It has been shown, for example, that certain kinds of noises cause alterations in respirations, heart rate, circulation and muscle tension. Apparently noise demands a heavy burden in physiological adaptation. The adverse effects may occur when noise acts as a stressor upon the normal psychologic and physiologic mechanisms of man.

In addition, there is evidence to indicate that noise may, in fact, contribute to the accumulation of cholesterols in the blood.⁸ Particularly, noise has been implicated as an intermediary factor in the genesis of the so-called stress related disorders such as gastric ulcers, hypertension, and coronary artery disease. Before introducing the concept of noise as a health problem to man it might be useful to examine some of the commonly encountered sources of noise within the urban environment and to discuss their characteristics.

⁷McChord, C., Teal, E., and Witheridge, W., "Noise and its Effect on Human Beings," JAMA 110:1553, May 7, 1938.

⁸Breysse, P., "Sound Pollution--Another Urban Problem," in Hafen, B. Ed. Man and His Environment, Minneapolis, Minnesota, The Burgess Publishing Company, 1972. pp 103-107.

The Nature of Noise in the City

Noise in the city may be categorized in a number of ways. Perceived noise level (PNdB)--a term which indicates that the assessment of loudness is made by the listener--is a subjective measure, inasmuch as there may be wide variation between the actual quantitative measurement and the reported "loudness." And the same sound may elicit varying reports of intensity by different subjects. Absolute sound pressure (SPL) is considered to be the most sensitive noise measurement because it is a quantity independent of human perception. More often, however, measurements of noise are made using a special scale of decibels (dB_A) which has been correlated to simulate the range of sensitivities of the human ear.

Kryter's classic definition of noise includes other qualities such as unexpectedness, inappropriateness, intermittency, interference, and reverberation.⁹ Intermittency and reverberation are characteristics that lend themselves to quantitative measurement. Unexpectedness, inappropriateness and interference require qualitative rating scales.

Interference caused by the noise of construction or aircraft flyover has been well documented. Even so, noise from these sources cannot be said to be uniformly inappropriate. For workers involved with these activities such

⁹Kryter, K., "The Effect of Noise on Man," J. Speech and Hearing Disorders Monograph 1:17, Sept. 1950.

noise is both appropriate and expected, but for the surrounding home owners it may be completely unexpected. The interference "potential" of noise is being increasingly called out to argue against such projects as airport expansion and the location of new freeways. It should be pointed out, however, that such scales are peculiar to the adult population. Younger persons such as the modern teenager maintain their own view of what constitutes "noise."

Noise may arise from a number of sources in the urban community; from traffic and transportation, construction and industry, all manner of aircraft, as well as from a whole spectrum of residential acouterments such as power mowers and household appliances. More commonly, emissions from several sources combine to form what is referred to as a "standing wave" so that only the expert in acoustics could identify the various sources making up the wave.¹⁰ Most urban noise is of this variety.

Baron makes the argument that ambient noise is of little physiological import because its nuisance potential is not as great as that of intermittent or "intrusive" noise--the latter being that noise which stands apart from the ambient and is generally of short duration.¹¹

¹⁰Taylor, R., Noisc, Baltimore, Maryland, Penguin Books, 1970 page 81.

¹¹Baron, R., The Tyranny of Noise, New York. St. Martin's Press, an affiliate of the McMillan Company, 1970, page 130.

The entire notion of intrusive noise may have evolved from the early British studies in which a sampling of London residents identified traffic, aircraft, and neighbors' children as sources of noise which permeated their homes. Central to this identification was the belief that there are times and habitats where noise is not expected and therefore need not be tolerated.¹² The home, the school, library, and most certainly, the hospital are some examples of sensitive environments.

Persons living near airports have argued successfully that aircraft at takeoff and landing represent very intrusive noises which disturb their normal conversations and interrupt their sleep.¹³ Evidence of the public annoyance with airport noise is provided by the Inglewood experience in which residents have named the nearby Los Angeles Airport in litigation totalling 5 billion dollars.¹⁴

The most commonly encountered sources of noise in heavy industry and construction are, electric saws and motors, diesel-powered motors, transformers, fans, various types of presses, sorters, stackers, belts, pneumatic drills, milling

¹²Burns, W., Noise and Man, London, John Murray, 1968, page 215.

¹³Cohen, A., and Ayer, H., "Some Observations of Noise at Airports and in the Surrounding Community," Am. Ind. Hygiene Assoc. 25:139, March-April 1964.

¹⁴Still, H., In Quest of Quiet, Harrisburg, Pa. Stackpole Books, 1970, page 106.

and rotating machines, and grinders and lathes.¹⁵ Experts disagree on what constitutes the safe limits of industrial noise for plant workers, not to mention the safe levels for the surrounding community.

Although the period of time that nearby homeowners would be exposed to industrial noise would vary, it is safe to say that most manufacturing plants operate at least two shifts each day and many run three. The worker, himself, may commute to a quiet neighborhood and thus experience only the workday exposure while the surrounding homes may get little respite from noise day or night.

The home itself is the source of a variety of noises which could be classified as unsafe if exposure to them is prolonged. And within the household the kitchen has been called the "noise center" of the home. Farr measured some sounds common to households and found that the noise from a vacuum cleaner in a next door apartment was 72 dbA; the hi fi emitted 75 dbA, and the stove vent, dishwasher and garbage disposal all on together generated 84 decibels of noise as measured in the apartment next door.¹⁶

Not all urban homes have such an array of equipment by any means, furthermore the average housewife uses these

¹⁵Fuchs-Schmuck, A. "Studien uber das Pulsverhalten nach definiertem Industrielarm," Int. Z. angew Physiol. einschl. Arbeitphysiol. 22, 1-9, 1966.

¹⁶Farr, L., "Medical Consequences of Environmental Home Noise," JAMA, 202:3:171, October 16, 1967.

appliances for only short periods of time throughout the day. Nevertheless objections against the reverberation of household fans and disposal units have been widely reported and "unexpectedness" is undoubtedly a characteristic of all noise emissions from households other than one's own. Probably because such noise is of generally short duration, the impact upon health has not been investigated.

Finally, noise sources common to large metropolitan hospitals include heating and refrigeration systems, fans, paging apparatus, x-ray and cautery equipment, corridor and utility room traffic and many more. And noise generated from outside the structure which may penetrate patients' rooms includes all of the sources common to a busy community, particularly traffic and aircraft.

Eldridge argued that temporary threshold shifts* such as those expected after exposure to loud noise of short duration are benign--that only noise that leaves a residual hearing shift at the start of the next day's exposure is hazardous.¹⁷

Eldridge's argument seems valid when applied to hearing

*Threshold refers to the lowest point on a standardized scale at which sound can be detected by the human ear. A shift in threshold may be temporary or permanent reaction. This point--between hearing and not hearing--may vary between individuals and in the same individual over time.

¹⁷Eldridge, D. and Miller, J., "Acceptable Noise-Exposure--Damage Risk Criteria," in Proceedings of the Conference on Noise as a Public Health Hazard, Eds. Ward, D., and Fricke, J., ASHA Report #4, Washington D.C. February 1969, page 97.

loss but it may not hold for other physiological phenomena. For it is not positively known that the processes which result in hearing loss are the same as those which lead to cardiovascular disease.

The Concept of Noise as a Problem to Man

It has been generally accepted as fact, that noise is a problem to urban man. Harmful effects are said to occur when human beings are exposed to high levels of noise for more than very short periods of time.¹⁸

Numerous studies have attempted to identify linkages between noise in the urban environment and cardiovascular disease. Comparisons have been made between the morbidity and mortality of populations in "noisy" and relatively noise-free societies. Socio-cultural studies which selectively and systematically compared characteristics of populations and their environments have seemed well suited to the analysis of disease processes. Unfortunately, most of these have been done in areas other than the U.S. Thus generalization may be inappropriate because of the numerous qualifications that would apply. In one such study, Mann, et. al., compared the rates for cardiovascular disease in the Masae(Tanganyka)--a rural people who live in a quiet environment--to the rates for American males in the Framingham study.¹⁹ He and his

¹⁸Still, Op. Cit. Chapter I.

¹⁹Mann, G., Shaffer, R., Anderson, R., and Sandstead, H., "Cardiovascular Disease in the Masae," J. Atherosclerosis Research 4:289-312, July-August, 1964.

associates reported that for men aged 30 to 44 years, the incidence among the Masae was 2.8 per cent of the rate for men of the same ages residing in the U.S. For males in the age group 45 through 62 years, the rate for the Masae was 26 per cent of that for their American counterparts.

In other cross cultural research comparing the mortality rates among the Thai who had lived in Bangkok, and persons who lived in the metropolitan area of Los Angeles, it has been shown that less than three per cent of the Thai sample succumbed to myocardial infarction compared to 24 per cent of the Los Angeles group.²⁰

Rosen overcame some of the weaknesses* of Mann's methodology by employing multiple measurements (hearing tests, electrocardiograms, blood pressure levels and physical histories) as indices to CAD in his investigation among the Maaban people. It is noteworthy that not a single case of CAD or hypertension was diagnosed in this purely rural population, although the tests spanned a two month period with participants ranging in age from ten through 94 years.

The Maabans are a village people whose major activities

²⁰Hirst, A., Piyoratn, P., and Gore, I., "A Comparison of Atherosclerosis of the Aorta and Coronary Arteries in Bangkok and Los Angeles," Am. J. Clin. Path. 38:162, 1962.

*The lack of precise physiological measurements makes Mann's work difficult to evaluate. A diagnosis of CAD was assigned from examination of one electrocardiographic record. A definitive diagnosis requires ECGs both active and resting, serial blood enzyme tests and a history of angina pectoris or autopsy of the coronary arteries.

are gardening and hunting. Rosen found the noise levels to rarely exceed 40 decibels, by day or by night, even during their frequent ceremonial festivals. More significant perhaps, and contrasting with studies from Japan and Thailand, the Maaban people become prone to high blood pressure and CAD when they move to North Sudan where they are exposed to noise, a different diet, and the stress and strain of city life.²¹

Based on autopsy data, Florentin et. al. concluded that East African Ugandis, 40 years of age and older have far less coronary artery disease than do New Yorkers of similar age and sex. The morbidity of myocardial infarcts is extremely low in East and West Africans in contrast to the high rates for North Americans.²²

Other rural or farm peoples have been studied with similarly low rates of cardiovascular disease reported. CAD is almost unknown among the semi-nomadic Bedouin Jews of Southern Israel. This people, who subsist on an agricultural economy--are physically active and consume low quantities of animal fats. Simplicity of life style and a low level of

²¹Rosen, S., "Presbycusis Study of a Relatively Noise Free Population in the Sudan," Annals of Otology 71:3:727, September, 1962.

²²Florentin, R., Lee, K.T., Daoud, A., Davies, J., Hill, E., and Goodale, F., "Geographic Pathology of Arteriosclerosis: A Study of the Age of Onset of Significant Coronary Arteriosclerosis in Adult Africans and New Yorkers," Experimental and Molecular Pathology 2:103-113, 1963.

noise are characteristic of the life of the Bedouin.²³

And in Crete, the southernmost island of Greece, where a rural way of life prevails, it is uncommon to find coronary artery disease. Again, the way of life is simple; the population consists largely of farmers whose diet is low in animal fats.²⁴

As regards urban-rural differences in CAD within like cultures, the Guatamalan Indians of Latin America exhibit a low rate of coronary artery disease, except for those of the upper socioeconomic class and living in large urban centers. The investigators explain this finding by the difference in fat intake between the two groups. Mendez and his colleagues do not examine in any exhaustive way, other environmental factors which may also differ between the populations he studied.²⁵

Finally, Benson and others examined the causes of all deaths in the age group between twenty and 64 years, over a six months period in San Juan to support their conclusions. These investigators found that the mortality rates for CAD

²³Swartz, M., Rosenveig, B., Toor, M., and Lewitus, Z., "Lipid Metabolism and Arteriosclerotic Heart Disease in Israelis of Bedouin, Yeminite and European Origin," Am. J. Cardiol. 12:157, 1963.

²⁴Christakis, G., Severinghaus, E., Maldonado, Z., Kafatos, F., and Hashim, S., "Crete--A Study of the Metabolic Epidemiology of Coronary Heart Disease," Am. J. Cardiol. 15: 320, 1965.

²⁵Mendez, J., Tejada, C., and Flores, M., "Serum Lipid Levels Among Rural Guatamalan Indians," Am. J. Clin. Nutr. 10:403, 1962.

among the Puerto Ricans of San Juan were low compared to those for the United States as expressed in actuarial tables of the latter. In a comparative study of an urban and rural community it was found that urban Puerto Rican males had higher blood pressures, higher mean serum cholesterol levels, were more obese and smoked more cigarettes than did rural men of comparable age.²⁶

Studies based on autopsy data are subject to all of the errors and differences in death reporting procedures across unlike cultures and, in addition, the possibility exists that "incidence" based on autopsy data is grossly inaccurate. In particular, the number of deaths due to heart attacks, the variable most investigators measured, is closely related to the type of care patients receive. Lacking modern coronary care facilities more of the Thai, for example, may succumb to their infarctions than do persons in Los Angeles where the latest in specialized service is available. Taking this fact into account, would however, simply widen the already existing difference between the mortality rates for the two cultures.

Bruhn in evaluating almost a hundred of such cross cultural studies criticized their non use of standard laboratory criteria for CAD. He reported the greatest weakness to be

²⁶Benson, H., Costas, R., Garcia-Palmieri, M., Feliberti, M., Aixala, R., Blanton, J. and Colon, A., "Coronary Heart Disease Risk Factors: A Comparison of Two Puerto Rican Populations," Am. J. Pub. Health 56:1057, 1966.

the "absence of information...regarding many of the risk factors commonly thought to be important in the development of CAD other than dietary and serum lipids." He suggests that research be pursued in populations where environmental variations are more uniform and characteristics of individuals more homogenous.²⁷

In spite of certain methodological weaknesses, the data comparing rate differentials for cardiovascular disease in various sections of the United States tend to support the premise that noise-saturated areas experience a higher incidence of CAD than do less "noisy" locations. For example, Learner and Anderson reported that mortality rates due to coronary artery disease were found to be highest in the heavily industrialized and urban States of New York, New Jersey, and Pennsylvania. In contrast, the lowest rates appeared for the more rural states of Minnesota, Iowa, North and South Dakota, Nebraska and Kansas. Even after the data were adjusted to age, the highly urbanized states showed the greatest mortality in the middle and upper ages from coronary artery disease.²⁸

Sauer's data--again comparing coronary artery disease between the East Coast and the Western Plains States--support

²⁷Bruhn, J., and Wolf, S., "Studies Reporting Low Rates of Ischemic Heart Disease: A Critical Review," Am. J. Pub. Health 60:8:1477-1495 August, 1970, page 1491.

²⁸Learner, M., and Anderson, O., Health Progress in the United States 1900-1960, Chicago, University of Illinois Press, 1963, pp 122-170.

the earlier conclusions of Learner and Anderson. It is of interest that the two areas studied by Sauer differed in the extent of their industrialization, moreover, the state having the highest rate of CAD, had a rate more than twice as great as the state having the lowest.²⁹

It must be conceded that environmental qualities other than noise differ from one geographical area to the next and differ markedly across cultural settings. As Bruhn has indicated, diet, climate, humidity, elevation, and a host of other factors may influence a population's potential to develop coronary artery disease. In addition, sociologists have argued that geographic and occupational instability, as well as the loss of primary support systems, represent stresses that are more common to urban man than to his rural counterpart.

It is obvious that noise, of itself, cannot "cause" coronary artery disease, much less myocardial infarction. However, it will be argued here that noise acts as a stressor upon the cardiovascular system of man. Such a force has valence for the urban population and must be taken into consideration in any discussion of the stress related disorders. Indeed, the sonic environment should be given greater weight in planning decisions regarding the location of urban hospitals and the placement of specialized units within the institution. For noise has a measurable effect upon the heart

²⁹Sauer, H.J. "Epidemiology of Cardiovascular Mortality--Geographic and Ethnic," AJPH 52:1:164, January, 1962.

and vascular system of man.

The Effect of Noise on the Cardiovascular System

A review of the literature shows that noise exerts at least a fourfold effect on the human organism: the startle response,³⁰ an autonomic nervous system response, the endocrine response, and an effect on sleep.

Statements about a relationship between stress and cardiovascular disease are based upon what is presently known concerning the response of the heart and blood vessels to sympathetic nervous system stimulation. When stimulated by noise, hormone-like substances are released into the circulating blood. These compounds, known as catecholamines, or more specifically, adrenalin and nor-adrenalin, serve to selectively narrow the lumen of certain blood vessels in the body. Heart rate is accelerated.

Measurement of catecholamines in the urine is considered an accurate index of physiological stress since most of the circulating adrenalin and nor-adrenalin is excreted via the kidneys within a very short period of time.

Other measures that have been employed as indicators of the physiological response to stress are: a change in heart rate, a decrease in amount of blood flowing to the skin, dilatation of the pupils, and pale, cool skin. Rosen implicated

³⁰Landis, C., and Hunt, W., The Startle Pattern, New York, Farrar and Rinehart, 1931, Chapter 2.

noise as a stressor and stated: "Noise can cause constriction of the blood vessels, pale skin, dilated pupils, and voluntary and involuntary muscle tensing, all of which are related to a response by the autonomic nervous system."³¹

The research concerning noise and the cardiovascular system can be divided into three categories for ease of discussion; laboratory studies on human subjects, laboratory studies using animals, and finally field studies. Each of these will be dealt with in turn.

In a series of experiments, Lehman found that the autonomic nervous system, especially the sympathetic division, reacts to loud noise, and further, that the metabolism of adrenalin is somehow involved in this reaction.³² And Buckley and Smookler were able to demonstrate significant enlargement of the left ventricle of the heart, as well as of the adrenal gland, in mice, as outcomes of exposure to noise.³³ When Friedman and his associates exposed rabbits to noise over a ten week period, although the diet was identical for both

³¹Rosen, S., "Vegetative Reactions to Auditory Stimuli," Transactions of the American Academy of Ophthalmology and Otolaryngology. May-June, 1964.

³²Lehmann, A., "Psychopharmacology of the Response to Noise with Special Reference to Audiogenic Seizure in Mice," reported in Welch, B., and Welch, A., Eds. Physiological Effects of Noise, New York, Plenum Press, 1970. page 251.

³³Buckley, J., and Smookler, H., "Cardiovascular and Biochemical Effects of Chronic and Intermittent Neurogenic Stimulation," Dept. of Pharmacology, University of Pittsburgh School of Pharmacy, as reported in Welch, B. and Welch, A., Op. Cit. page 83.

groups, a higher level of blood cholesterol was found in the exposed animals compared to the nonexposed. Noise-exposed animals also developed a greater degree of atherosclerosis of the aorta than did their controls.³⁴ While not of specific interest to this study it should be noted that a crucial link between noise and cardiovascular disease may lie in identification of the processes leading to elevated blood cholesterol. For a cholesterol level of more than 250 mg. per cent is a primary risk factor for coronary artery disease.³⁵

In another type of investigation, Black presented rats with 40 decibels of noise for varying periods of time. Eighty-eight per cent of the animals decelerated their heart rates in response to the initial stimulus, but by the fifth trial all subjects had an increased heart rate which continued to accelerate throughout the remainder of the presentations. Although the latency of the heart rate response lengthened with increased duration of exposure, the absolute heart rate continued to accelerate.³⁶ The failure of the heart rate to show terminal habituation was supportive of earlier findings.

³⁴Friedman, M., Byers, O., and Baron, A., "Plasma lipid responses of rats and rabbits to an auditory stimulus," Amer. J. Physiology 212:1174-1178, 1967.

³⁵Primary Prevention of the Atherosclerotic Diseases. Report of the Inter-Society Commission for Heart Disease Resources, Circulation XLII December, 1970.

³⁶Black, H., "Heart rate response to auditory stimuli of varying duration," Psychonomic Sci. 1:171, 1964.

Early, classical studies by Hildebrandt correlated changes in the respiratory and heart rate of human subjects with degrees of stress. His hypothesis that both heart rate and breathing rate would increase as a function of noise intensity was substantiated.³⁷ And other investigators have correlated positively increases in heart rate with an increase in threshold for hearing. Jansen's conclusions--that noise will elicit a predictable and measurable response from the sympathetic nervous system--were similar to earlier findings.³⁸

From a later investigation, De Leon concluded that an increase in heart rate is a predictable response to noise exposure, however he was unable to consistently condition the response in eight human subjects.³⁹ Finally, using a human neonatal population, Bartoshuk measured the heart rate response to noise over 40 exposures. Cardiac acceleration persisted from the initial trial through the fortieth, although a decrement in the degree of the increase was noted. The faster rates were more pronounced in the older infants,

³⁷Hildebrandt, G., "Die Koordination von puls und Atemfrequenz bei der Arbeit," Internationale Zeitschrift fur Angewandte Physiologie, einschliesslich Arbeit Physiologie, (Berlin) 21:27.

³⁸Jansen, G., Institute for Hygiene und Arbeit Medizen, reported in Welch, B., and Welch, A., Op. Cit. page 67.

³⁹De Leon, G., "Conditioning the human heart rate with noise as the unconditioned stimulus," J. Exp. Psychol. 68: 518-520, 1964, page 518.

however, the rate increase existed independent of the sex of the neonates.⁴⁰

Laboratory investigations have provided ample evidence that vasoconstriction of the peripheral blood vessels is one factor in noise trauma reactions in both human and animal subjects. Other outcomes such as changes in heart rate, and skin temperature are thought to be secondary to a sympathetic nervous system response. Replication of such physiological effects in the laboratory settings has produced only minor variations in the conclusions of the investigators. However, isolating noise as a cause of similar effects in the field setting has been fraught with difficulty.⁴¹

Work situations have proved what has been, perhaps the most fruitful of the "natural habitat" studies. Typically, heart rate, blood pressure, electrocardiographic changes (ST segment), or incidence of MI among workers are used as outcome measures and subjected to a variety of tests.

⁴⁰Bartoshuk, A., "Response decrement with repeated elicitation of human neonatal cardiac acceleration to sound," J. Comp. Physiol. Psychol. 55:9:13, 1962.

⁴¹German Physiologists, Hawel and Starlinger measured the stressor effects of noise in two groups of students who performed problem-solving tasks during quiet and noise conditions. The controls received neither treatment. A positive correlation was found between acceleration in heart rate and secretion of adrenalin in the urine, but the stressor effect of noise was not differentiated from the stress experience when taking a problem-solving test. Hawel, W., and Starlinger, H., "Einfluss von wiederholtem viewstundigem intermittierendem sogenannten rosa Raufchen auf Catecholamin-ausscheidung und Pulsfrequenz," Int. Z. Angew. Physiol. Arbeitsphysiol. 24: 351, 1967.

In one such investigation, workers who had been exposed to industrial noise of from medium to high frequency (85-120 dbA) in the course of their work day, experienced deceleration of the heart rate and an increase in vascular tone. Electrocardiographic changes were more common in workers who were exposed to high levels of noise.⁴²

In a similar study, a group of normally hearing adolescents were subjected to noise at frequencies ranging from 100 to 200 Hz at 85 decibels during the course of their ordinary day. Among the cardiovascular effects reported was a decrease in pulse rate which was transient in nature and disappeared after the subjects were removed from the noise conditions.⁴³ Moreover, Ward and Suedfeld were unable to document any changes in heart rate for subjects in their five day investigation on a college campus. Increased ambient noise provided by highway traffic had adverse effects on interaction in group problem-solving and no reliable data were generated on individual cognitive performance.⁴⁴

The results of the Ponomarenko, and of the Ward and

⁴²Shotolov, N., Saitonov, A., and Glotov, K., "On the state of the cardiovascular system under conditions involving exposure to the action of permanent noise" Labor Hygiene and Occupational Health 6:10:14.

⁴³Ponomarenko, I., "The effect of high-frequency industrial noise on certain physiological functions in adolescents," Hygiene and Sanitation 31:188-193.

⁴⁴Ward, L., and Suedfeld, P., "Human responses to highway noise," Environ. Research 6:3:306, September, 1973.

Suedfeld studies differ from those obtained from experimental situations with animals, as well as from the data reported previously from Fuchs-Schmuck, and Hawel and Starlinger. The latter investigators found noise to systematically induce an increase in heart rate in their subjects. And Hobbs found that people just walking up and down the hallways outside their experimental room, caused the heart rates of their subjects to accelerate.⁴⁵

If Deave and Zearman's premise has validity, whether heart rate tends toward bradycardia (slowing) or tachycardia (acceleration) may depend more upon the degree of anxiety present, than on the actual level of noise. They reported that strong anxiety leads to an increase in heart rate whereas weak anxiety may actually decelerate the heart.⁴⁶

In addition to the specific effects upon the heart and vascular system that have been discussed, a classic study by Mohr reported some less well established effects. Mohr and his associates, all researchers in noise phenomena, exposed themselves to two minute periods of high intensity, broad band, pure tone, low frequency noise. They reported experiences of giddiness, chest discomfort, and flushing and tingling of the skin. Specifically, at 60 Hz (cycles per second)

⁴⁵Hobbs, E., Department of Veteran Affairs, London, Ontario, Canada, Personal Communication, January 30, 1975.

⁴⁶Deave, G., and Zearman, D., "Human Heart Rate During Anxiety," Percept. Motor Skills 8:103-106, 1958.

and 153 decibels they experienced severe chest pain, choking respirations and salivation and pain upon swallowing.⁴⁷ It must be noted that exposure of a population to similar intensities would be rare indeed. In addition, these subjects were not randomly selected--they were, in fact, researchers entering the experimental situation with the hypothesis that noise would elicit adverse responses from the human body. The addition of a control group would have strengthened the credibility of the findings.

Mohr's research is important, however, for it makes clear that feelings of annoyance are not prerequisite for the cardiovascular physiological response. At least at the outset, the Mohr responses can be categorized as purely physiological in nature. Sound pressure created by noise, of more than 70 decibels apparently can bring about a cardiovascular response independent from feeling states. This point is emphasized because of the widely held notion that the response (R) to a noise stimulus (S) occurs solely as a function of perception and socialization (P).

The conclusions of Lehmann and Tamm support those of Mohr and his associates. They found that both short and long term exposure to noise of greater than 70 dbA caused narrowing of the small arteries of the body--a vasoconstriction which occurred independently of annoyance or any other emotion

⁴⁷Mohr, G., Cole, J., Guild, E., and von Gierke, H., "Effects of low frequency and infrasonic noise on man," Aero-space Medicine 36:9:817, September 1965.

in the participant. Furthermore, similar effects were obtained after each noise stimulus and at all intervals. An interesting conclusion from the Lehmann and Tamm report was that the same degree of narrowing took place in persons long accustomed to noise as it did in persons unaccustomed to it.⁴⁸

Conditioning by exposure seems not to be consistently operative in the effect of the noise stimulus. The effects, whether vascular narrowing or heart rate responses remain unmitigated over multiple trials.

It must be pointed out that there is some question regarding the impact of momentary changes in cardiovascular dynamics, upon the health of man. That is, linkages between minute changes and frank pathology are by no means proved. Alterations in blood pressure and subsequent changes in heart rate are compensatory mechanisms that are mediated by the autonomic nervous system. Regarding the studies reviewed here, the variables selected for study, the methodology, as well as the subjects have differed greatly among investigations. Two commonalities emerge, however. That is that noise of sufficient intensity leads to vasoconstriction of peripheral arteries and a change in heart rate. And, as Jansen, Lehmann, and others have so clearly demonstrated, feelings of annoyance or socialization are not prerequisite for the physiological response.

⁴⁸Lehmann, A., and Tamm, J., "Über Veränderungen der Kreislaufdynamik des ruhenden Menschen unter Einwirkung von Gerauschen," Internationale Zeitschrift für Angewandte Physiologie, einschliesslich Arbeitphysiologie (Berlin) 16:217-18, 1970, Reported in Welch, B. and Welch, A., Op. Cit. page 57.

In this dissertation, the sum of these cardiovascular effects, whether explicitly defined as responses, or abstractly stated, shall be conceptualized as cardiovascular stress. However, noise will be suggested as but one of a host of stressors inherent in an urban environment which must be taken into account in discussing a stress phenomena for coronary artery disease.

THEORETICAL FRAMEWORK FOR THE STUDY

The analysis of micro phenomenon such as heart rate response to noise gains credibility when it is based on a theory of environmental stressors and multicausality. Moreover, through a broad framework of stressors and stress the psycho-sociological approaches come into harmony with the purely physical in their attempts to explain the disproportionate rate of coronary artery disease in U.S. cities.

The social, psychological, and physical components of stress form a matrix of complex interrelationships. Acceptance of this statement is essential to a fruitful discussion of cardiovascular disease causality, even though the exact nature of the relationships remains obscure. The major emphasis of this dissertation is on the physical model of stress. At the same time, the synergistic effect of all types of stressors is acknowledged.

The physical model of stress is used herein to describe the effort (or change) that occurs when a stressor is

applied to an object. Strain may be present over time (for example, weight upon a foundation) before the load of stress is sufficient to produce deformation. Deformation is generally thought to be irreversible. As increasing loads are applied to an object, or as a stable load is applied to an object that has weakened, a linear increase in strain will become evident in that object. Elasticity, recoil, tension and contractility are qualities that are always implied. In addition, discreet boundaries between stress and deformation are defined. The physical models of stress have great potential for establishing possible linkages between noise and cardiovascular disease, for the above properties are also characteristics of heart muscle. For example, the stress that results from fever, fright, or noise, brings about a change in heart rate that to a point (which can be defined in terms of magnitude over time) is purely physiological. In fact, these mechanisms are compensatory and allow man to vary his exercise without suffering ill effects. Apparently the harmful effects occur at the interface of normal, and overburden on the cardiovascular compensatory mechanisms.

The physical model of stress does pose a danger for arriving at simplistic explanations, however. One cannot dismiss coronary artery disease with simple deductions i.e. stress leads to strain when the limits of accommodation have been surpassed. While this statement is generally true in the case of heart failure, it cannot be applied to the

genesis of coronary artery disease. And, the heart rate response to noise may or may not be a function of accommodation in every instance.

Levine and Scotch have created a model for social stress which includes three types of situations. The first two are primarily concerned with social phenomena; the third of Levine's postulates--the existence of a physical component--includes situations in which heavy or implement loads are added to the organism.⁴⁹ While Levine's interest was socially generated stresses, "situations" can be readily expanded to include the environmental. For example, "urban situation" brings to mind crowds, traffic, smoke, diverse odors, and plentiful noise. Bright lights, banks of monitors, and terse conversations are very much a part of the intensive care "situation." The sociological view holds that such stresses are detrimental only if perception and previous socialization predetermine such a reaction. The point will be made again, that beyond any psychological effect, noise represents physical force that is measurable in units of sound pressure. It is this latter characteristic--the physical--which probably accounts for the cardiovascular response that is exclusive of perception.

The environmental concept of stress implies that pathological agents, one of which is noise, are present in

⁴⁹Levine, S., and Scotch, N., "Toward the Development of Theoretical Models, II." reprinted in Milbank Memorial Fund Quarterly XLV No. 2 Part 2, April 1967. page 163.

an urban environment. The saturation point, that is the concentration, has been defined as pollution. These agents exert incremental loads of stress upon the urban dweller, and, at some point in concentration over time, culminate in pathological adaptation. Hearing loss is one example. Deformation will not be found uniformly in a population because the evolution of disease depends, in part, upon the susceptibility of the host. Within this framework noise can logically be defined as a stressor.

Stressors, as used in this dissertation, are noise conditions which may persist over time, which can be described as stimulatory, although the potency of the stimulation may vary from exposure to exposure. Stressors bring about change, while at the same time, weaken the organisms' capacity to withstand change, or to recoil from change. Finally, such a feedback loop requires adaptation--probably at the cost of health. The outcomes are measurable in descriptive, and often in quantitative terms.

The question arises whether stressors can be differentiated from stimuli that do not stress. Can the same noise condition at one time and intensity represent a stimulus, and at another, a stress? In the field setting answers can only be arrived retrospectively by examination of the outcomes. As a result of continuous adaptation to various stressors the total organism, or a subsystem within it, undergoes strain. That is, the energy cost per unit of work

rises markedly and efficiency is diminished.^{50,51} Deformation occurs with the passage of time. As regards the cardiovascular response to stressors, alterations in blood pressure, heart rate, size of the myocardium, cardiac output,* and utilization of oxygen by the heart muscle have been used singly and in various combinations as measures of myocardial strain. The beginning of pathology may lie at the point when these parameters of myocardial health fail to revert to their pre-exposure levels of function.

In summary, this study will argue that noise is the waste of urbanized society. Noise is further conceptualized as a by product of technology which exerts a stressor effect on the cardiovascular system of man. As such, it represents a negative influence on health. This argument is based on the fact that measurable responses can be induced in the sleeping or awake state, by selective exposure to a critical level of noise. Intensity alone, satisfies the necessary criteria for this dissertation, that is it provides a means of accurate measurement and elicits a predictable response.

⁵⁰Bing, R., "Myocardial Metabolism," Circulation 12: 1955 pages 635-647.

⁵¹Sonnenblick, E., and Skelton, C., "Oxygen Consumption of the Heart, Physiological Principles and Clinical Implications," Mod. Concepts Cardio. Dis. Vol. XL No. 3 March 1971. pp 9-10.

*The amount of blood, expressed in milliliters, pumped out of the heart in one minute of time. (CO=SV times HR)

It is further suggested that the sonic environment of urban hospitals may exceed the acceptable safe levels of 40 to 45 decibels.⁵² Within the hospital technological devices that monitor vital functions for the critically ill, may at the same time, represent a noise hazard to the patient.

For the purposes of this paper noise will denote unwanted or untoward sound as defined through its physical, social and psychological properties. The denotation "unwanted" is retained to get at a link between noise and its frustration potential, and from a physical perspective an abstract quality of "untowardness" is introduced. Noise may be unwanted or untoward, or both, but it cannot be neither and still be classified as noise.

This theoretical definition has the advantage that sound level in decibels may, but need not be specified. Perceived loudness is not a criterion. Socially unwanted noise may be of low, intermediate, or high intensity, and other specifications such as band width are relatively unimportant to this aspect of the definition. Untoward noise, on the other hand, refers to a specific decibel level sufficient to arouse a physiological response. Similar responses could be expected to recur within a specified range of probability, and independent of subjective overlay. A broad definition such as this makes possible measurement of changes in

⁵²Phelps, A., "The Need for Frequency Analysis in the Identification and Solution of Noise Problems," APCA Journal 23:1:17-22, January, 1973.

cardiovascular function, whether or not the noise is socially desirable. Changes in heart rate, blood pressure, in skin temperature or in the lumen of the blood vessels--all physiological parameters--may be linked to noise exposure and hypotheses made.

The Problem

There is ample evidence to show that noise represents a deleterious influence of unspecified magnitude, upon the cardiovascular system of man. The sonic environment has not been taken fully into account regarding the location of hospitals in metropolitan areas, and equally important, in the placement and administration of coronary care units within the hospital structure.

The noise environment of the patient who is housed in the coronary care unit has not been thoroughly researched nor has an accurate 24 hour profile of noise sources been attempted. Since the CCU hosts the most vulnerable of all cardiac patients, i.e. those with damaged hearts and thus prone to develop rate and rhythm disturbances, the major purposes of this study are to obtain:

- 1) a description of the sonic environment of the metropolitan hospital by examining the
 - a. external noise sources
 - b. internal noise sources
 - c. weight given to noise milieu by hospital planners.

2) a profile of common noise sources in the coronary care unit, as well as a record of their intensities.

3) discreet measurements of the heart rate response (HRR) to noise as demonstrated by patients admitted to the unit with a diagnosis of cardiovascular disease.

The first of the these objectives will be achieved through a comprehensive review of the literature, while the second and third topics form the basis for research for this study.

Chapter II presents a review of the classic and current literature concerning noise and hospitals with special reference to locational and planning decisions.

CHAPTER II

NOISE AROUND AND IN HOSPITALS

At the outset, it should be noted that the majority of metropolitan hospitals were built shortly after the turn of the century. Many are massive structures with facilities that have been remodeled not once, but several times. Most are located downtown.

Navarro's study of hospitals in Baltimore, Maryland showed a concentration of acute care beds (167/1000 pop.) in the downtown area. A lesser number of beds (4.1/1000 pop.) were located in outlying areas and even fewer (2.0/1000 pop.) were found in the suburban districts of Baltimore.¹

Similar dispersions would probably be found in other large cities of the United States. For example, Maricopa County's two largest institutions are situated in highly congested areas of Phoenix. The entire length of the 800-bed Good Samaritan Hospital borders a busy thoroughfare; and, St. Josephs Hospital--a slightly smaller institution--is located at the intersection of Central Avenue and Thomas Road, both heavily traveled by trucks and buses. Downtown locations were also found for facilities in Denver, Colorado; Seattle, Washington; and Portland, Oregon.

Hospitals located in such areas are particularly

¹Navarro, V., "The City and the Region, A Critical Relationship in the Distribution of Health Resources," Am. Behavioral Scientist 14:6:865-891, July/August, 1971.

vulnerable to external noise, due to the numerous intersections and stoplights which interrupt the smooth flow of traffic on the surrounding streets. The argument made for these choices is that downtown locations require minimal transportation time for patients and staff. Accessibility is optimal. And historically, accessibility to the community it serves has been considered an asset to any hospital.

Nevertheless, as Michaelsen states: "...with accessibility comes traffic and the noise it produces. The general volume of air and highway, rail and boat traffic, that might be close to a hospital site should be examined carefully with particular reference to the peak periods of activity and consequent noise production. Vehicular traffic patterns should be studied.² Michaelsen went on to say that traffic lights dotting a thoroughfare give rise to some very annoying sounds, namely the deceleration, braking and acceleration of trucks and buses.

The quality and quantity of the external noise load at any given hospital are functions of vehicular and aircraft traffic patterns, as well as the spatial arrangement of the surrounding buildings and the contour of the land. Obviously, surrounding structures can act either as noise buffers, or actually reflect and increase the intensity of noise at a

²Michaelsen, G., "Noise Production and Control," in Environmental Health and Safety in Health Care Facilities, Eds. Bond, R., Michaelsen, G., and R. De Roos. New York, The MacMillan Company, 1973. page 139.

hospital.³ The contour of the ground site is an additional consideration, since there will be a difference in noise load among hospitals constructed in a hollow, on flat ground or high on a hill.

Traffic Noise: The Washington Study

In the most extensive study of external traffic noise done to date, measurements were taken outside of ten hospitals in Seattle, Washington. Nine of these facilities are immediately adjacent to major highways, and the tenth was located near the central business district.⁴

The average noise level in patients' rooms, as measured in perceived noise level, (PNdB)* varied to nearly 80 decibels, depending upon whether the windows were open or tightly closed. Even in the corridors external noise measured as high as 74 decibels. The investigators found that patients were not particularly disturbed by noise below 65 PNdB, but noise of more than 72 decibels disturbed them considerably. The authors reported further that noises generated

³---Airborne, Impact, and Structure-Borne Noise-Control in Multi-family Dwellings. National Bureau of Standards and U.S. Department of Housing and Urban Development. Washington, D.C., 1967.

⁴---Main Report, "Noise in Hospitals Located Near Freeways," U.S. Department of Commerce, Bureau of Public Roads and Washington State Highway Commission, Department of Highways, January 1964. pp 3,4.

*Perceived Noise Level is defined by Taylor as the sound pressure level of between one-third of an octave and one octave of random noise at 1000 Hz, which is considered by normal people to be equally noisy to the sound of interest. $PNdB = 40 + 10 \log_2 Noy$. (Taylor, R., Noise, Ibid. p 254).

within the hospital were at least as great or greater than external noise. Nevertheless, their conclusions were that hospital noise had little effect on recovery rate (as measured in days of hospital stay) or on the physician's choice as to where patients should be admitted.

The weaknesses of drawing any conclusions from such a study must be pointed out in the light of this dissertation. First, The Washington study used days of hospital stay as an indicator of effect. Nowhere in the entire physiological literature is so gross a criterion applied to the effects of noise exposure. Conclusions might have been more meaningful had the investigators measured urine catecholamines in noise-exposed patients, or correlated the use of sedative and tranquilizers with variations in noise level. In addition, the correlation of noise climate with hospital choice by physicians was equally weak as a criterion measure. For a physician may be as naive about noise effects as the layman, and there is no reason to suppose he would use data with which he is unfamiliar to make decisions about where to admit patients. Even if this argument did not hold, a physician acquires staff privileges at one, or perhaps two hospitals in accordance with strict guidelines set by the hospital in question. Often, the decision of where to apply for staff privileges is influenced more by where his colleagues admit patients, or by where he is likely to be accepted than by environmental considerations. Moreover, the physician is never free to choose from among ten hospitals as

to where he will admit his patients.* Thus, the information concerning physician choice becomes almost nonsensical when applied to the Washington data.

Patients' Views of Noise:

Interestingly, a comprehensive study by the U.S. Public Health Service indicated that 61 types of noise bothered patients. A subsequent analysis showed that only eleven of these noises emanated from outside the hospital, while 50 noises came from in-hospital sources.⁵

There is at least one major risk in generalizing from data that use patient self-reporting. Bothersome, or annoying qualities cannot be assumed to be necessary for the physiological noise effect. Based on the principles of physical pressure, or force (both of which apply to sound waves) noise has the capacity to elicit an effect without any feelings of annoyance on the part of the listener. On the other hand, frustration with noise may also produce cardiovascular effects, so that annoyance may be a sufficient, but not a necessary, cause in physiological effects of noise. As has been pointed out previously, noise represents sound

*Physicians in the Portland, Oregon Greater Metropolitan Area generally have staff privileges at one or two, and usually not more than three hospitals. This means that they do not admit patients to hospitals other than where they enjoy staff privileges, regardless of the number of considerations in the area.

⁵--Noise in Hospitals, Pamphlet, U.S. Public Health Services Publications 930-D 11, U.S. Government Printing Office, 1963.

pressure, and when that pressure is greater than 70 decibels (on the A scale) known physiological changes will result.

It has been postulated that the symphony, a rock and roll band, and the jackhammer have something in common. When the pressure of noise reaches more than 70 decibels, a quickened heart rate and peripheral vasoconstriction may result in those exposed. These assumptions remain to be proved in the laboratory, however.

The greater weight given to in-hospital noise, by patients, tends to deemphasize external noise as a problem. In addition, several arguments will be made here to support the lesser emphasis this dissertation places on external noise load.

The first has to do with the reduced number of hospital starts projected over the next few years. With the future availability of Hill-Burton funds in doubt, and the reorientation of those funds from hospital starts to hospital remodeling, the need for locational decisions has decreased. In fact, in 1972, the Hill-Burton Project announced a shift from providing funds for new, acute care facilities to the modernization of existing institutions.⁶ Emanuel Hospital in Portland, Oregon is one example of this policy applied. After spending a great deal of time and expense in planning an ultramodern 14-story facility, its application for a new physical plant was denied. Subsequently, Hill-Burton monies

⁶--Report from Washington, Trustee, 1973, page 48.

were granted to Emanuel for remodeling and rennovating the existing structure.

Secondly, the nature of sound-proofing has been perfected to the point that a structure can be rendered almost impermeable to external noise. Taylor argued that complete noise-proofing is economically unfeasible for most existing facilities, but the load of structurally borne noise can be sharply reduced with but a minimum of expense.⁷

Thirdly, as hospital planners make use of professional city planners, and the wealth of data the latter can supply, locational site planning will become a joint venture. With the increasing sophistication of such indicators as population forecasts, transportation projections, and pollution-measuring devices, hospital planners will have additional data available to them. It is assumed that locational site choices will then be rationally based.

The future emphasis may lie not so much with locational decision-making, as on what types of noise proofing are structurally necessary to guarantee comfort and safety to the hospitalized or convalescing patient. As technical appliances proliferate and scientific knowledge is applied to the care of patients in thousands of intensive care units, the control of in-hospital noise may become the major problem.

Noise in Hospitals: A Review of the Literature

Many investigators have addressed themselves to this

⁷Taylor, R., "Silencing and Soundproofing," in Noise, Middlesex, England, Penguin Books, Ltd. 1970.

topic. The largest of the studies, perhaps, was carried out in a London hospital.⁸ "Noise," as defined by these British investigators consisted of "sound that patients called annoying." The report, which was subsequently published in Modern Hospital, does not indicate whether the measurements were made in decibels A or linear scales. Precise noise level measurements were taken of such equipment as the sterilizing machine, and the floor polisher when in operation, on the maternity, long term and acute care wards. Noise ranged as high as 78 to 86 decibels for varying periods of time throughout the day.

A report by the U.S. Public Health Service cited previously, summarized a number of studies which were done in U.S. hospitals. Again, in most of these investigations, patients were asked to list the sounds that bothered them. Particularly prominent complaints were floor polishers, the clatter of dishes and pans, and "the conversation of night duty nurses during coffee break." In addition, to the noise from radios, television sets, paging systems and telephones, patients complained about talking in the corridors, children crying and the sounds of other patients in distress.

It would be of interest to know whether patients were bothered more by noise with which they were unfamiliar, however, the studies did not take this factor into account.

⁸Carlson, D., "Noise Control Program is Quiet Success," The Mod. Hosp. 105:6:82, December, 1965.

While any particular combination of these hospital noises might be present in some homes, they would be unfamiliar in others.

More recently, attention has turned to correlating noise levels with quantifiable vital signs; for example, heart or respiratory rates, blood pressure or other physiological functions such as the excretion of catecholamines by the kidney. Haslam⁹ measured noise on a surgical ward using a sound level meter calibrated to read such pressure in decibels. The meter was adjusted to a frequency weighting network (A) which closely approximates reception by the human ear. Haslam began with the premise that a critical level of noise would elicit a widespread response from the cardiovascular system, namely, changes in blood pressure, perspiration, respiratory, and heart rates. The monitoring equipment was placed in the center of the ward and insulated from floor waves. Whenever an event produced a major fluctuation--an entity the report fails to define--on the sound level meter, the patients' vital signs were measured. Changes were noted and assumed to be the result of the previous noise occurrence. In general, the level of noise generated by the staff was well below background noise and ranged from 35 to 73 decibels. Haslam concluded that noise on the surgical unit was well below the levels of human tolerance.

⁹Haslam, P., "Noise in Hospital, Its Effect on the Patient," Nurs. Clin. N. Amer. 5:4:715, December, 1970.

The nonspecificity of certain critical terms (for example, major event and human tolerance) make it difficult to generalize findings from the Haslam study. Other shortcomings have to do with the methodology used. Noise fluctuations were observed from the center of the ward and readings taken in decibels A. Noise reception would hardly have been the same at each patient's room, since the patients would be at various distances from the noise sources. The inverse square law states that sound level will decrease by six decibels for each doubling of the distance from the sound source to the receiver. Sounds heard by any one patient may not have registered as a "major fluctuation" on the meter, since it was positioned some twenty feet away. Or, noise causing a "major fluctuation" at the center of the room may have been perceived by patients in corner rooms or behind heavy curtains as a very slight noise. More specific data could have been generated had the microphone of the sound level meter been placed at the height of the patient and no more than two feet from his head. The present dissertation will go on from Haslam and correct for these weaknesses by placing the sound level meter as suggested, and taking nearly simultaneous records of the level of noise and vital signs.

Noise and its source, in a surgical suite of one of Chicago's hospitals, was investigated by Shapiro and Berland.¹⁰

¹⁰Shapiro, R., and Berland, T., "Noise in the Operating Room," New Eng. J. Med. 287:12:36, December 14, 1972.

The measurements were made during the course of an operation. Sounds generated by opening packages of gloves, wheeling equipment, hitting steel instruments together, and compressed air sounds registered higher than did the freeway traffic. The investigators concluded that the surgical suite is a very noisy place! In their report, the cardiovascular response to noise is reviewed, however, these investigators did not measure the physiological effects in either staff or the patient during their study. Nor do they indicate whether their interest was with the effect of noise on the surgeons and nurses, or upon the anesthetized patient.

The emphasis of Minckley's work was on patient discomfort in the recovery room and its possible linkage to noise levels.¹¹ Minckley defined noise as all sounds that registered on a noise level meter. Three categories of noise were used: low level (40 to 50 decibels); medium level (50 to 60 decibels); and high noise level (from 60 through 80 decibels). A correlation was postulated between noise level and the amount and number of sedatives that were administered. The latter was to be used as a measure of discomfort.

A portable, battery-operated sound level meter was used to measure sound levels at half hour intervals during five random work days. The room was traversed with the investigator carrying the meter from one end to the other.

¹¹Minckley, B., "A Study of Noise and Its Relationship to Patient Discomfort in the Recovery Room," Nurs. Research 17:3:247, May-June, 1968.

Subsequently, average readings were computed. As the sound level measurements were taken, the number of narcotics and sedatives administered was also tabulated. The highest levels of noise occurred immediately after an operation was completed and the surgeons and nurses exchanged jocular remarks. Other events which registered "high levels" of noise were the telephone ringing, children crying, vomiting, snoring, and moaning.

Concerning verbal complaints by patients, the only sounds in the high range that patients did not mention were other patients snoring and the ringing of the telephone.

Minckley's hypothesis--that the patients' subjective experience of pain would intensify concomitantly with noise--was well supported by her data. Nothing in her data would reject her argument that noise represents an irritant to the patient who may already be experiencing postoperative pain. An accurate picture of the noise climate does not emerge, however, since Minckley did not indicate the scale used in her measurements.

It is of interest that the studies reported thus far have examined noise effects in departments in which the assumption that "it ought to be quiet" remains an unwritten one. Only in special care units, and from among these, the coronary care unit, have policies been formulated which state that rest and serenity shall be provided the patient. Before examining the few studies that have been carried out in special care units, the environment and philosophy of coronary

care will be discussed briefly.

The Coronary Care Unit As Non-Stressful Environment

Apart from the intensive care unit especially designed for patients with shock, trauma, burns, head injuries, neonates, or multi-system derangements, are the units known as medical intensive care--and within this complex--the coronary care unit, to which patients with heart attacks and other heart disorders are admitted. It is to these units and the cardiac patients housed there that the present dissertation is addressed. It should be noted, however, that small hospitals of from 30 to 150 beds frequently combine surgical and medical intensive care units into what is commonly known as the ICU-CCU. Since the philosophies of care governing the two categories of illness differ greatly, it is usually financial or staffing constraints which force this compromise.

Specifically, the formal policies of most coronary care units state that the unit shall:

- 1) Bring to bear upon the patient's illness medical and nursing care by specialists who have had specific training in cardiac care.^{12,13}

¹²Holt, E., Dolbee, M., and Southwick, J., Coronary Care Units in Small Hospitals, The W.W. Kellogg Foundation, Battle Creek, Michigan January, 1970, page 50.

¹³Weinberg, S., "The Current Status of Instrumentation Systems for the Coronary Care Unit," in Friedberg, C., Ed. Acute Myocardial Infarction and Coronary Care Units, New York, Grune and Stratton, 1969, page 261.

2) employ monitors and other equipment in order to measure, record, or support vital functions of the body.¹⁴

3) segregate the critically ill cardiac patient from patients who are less ill, and to provide an atmosphere of rest and serenity.^{15,16,17,18,19,20}

Several mechanisms are used to reduce stimuli and provide rest for the patient in the coronary care unit. In most, one person--a family member--is allowed to visit the patient for five minutes out of every hour. Families are cautioned not to discuss subjects that the patient might find distressing. Nor do most patients receive their mail for the first forty-eight hours after their admittance to the unit. Television is occasionally permitted, but in many

¹⁴--Report of the Inter-Society Commission for Heart Disease Resources, 301 E. 64th St., Suite 6B, New York, N.Y., 10021, page 101, May 1971 and published in Circulation Vol. XLIII, May 1971.

¹⁵Whipple, G., Peterson, M., Haines, V., Learner, E., and McKinnon, E., Acute Coronary Care, Boston, Little, Brown and Company, 1972, pp. 6-7.

¹⁶Riehl, L., Coronary Nursing Care, New York, Appleton-Century-Crofts, 1971, pp. 11-12.

¹⁷Storlie, F., Principles of Intensive Nursing Care, 2nd Ed. New York, Appleton-Century-Crofts, 1972, page 54.

¹⁸Meltzer, L., Pinneo, R., and Kitchell, J., Intensive Coronary Care: A Manual for Nurses, Philadelphia, Pa., The Charles Press, 1970, pp. 23-24.

¹⁹Nite, G. and Willis, F.N., Jr., The Coronary Patient: Hospital Care and Rehabilitation, New York, The MacMillan Co., 1964, pp. 5-6.

²⁰Modell, W., Schwartz, M., Hazeltine, L., and Kirkham, F., Handbook of Cardiology for Nurses 4th Ed., New York, Springer Publishing Company 1962, pp. 57-59.

CCUs, TV is forbidden to the patient for the entire length of his stay. If a patient requests it, a small battery radio is usually provided him.

The walls of most CCUs are pastel or chalk white. If there are paintings on the walls such as those hung at Rose Memorial Hospital in Denver, Colorado, or at St. Anthony's Hospital in St. Petersburg, Florida, they are softly-colored murals of restful, country scenes. In many units, there are neither clocks or calendars, although this practice has been criticized in recent years as having a depersonalizing effect upon patients. Keilly describes the "average" unit aptly: "The usual design of the intensive care unit results in windowless, colorless, austere, multibed rooms with frighteningly strange monitors, respirators, and other mechanized equipment whose meaning to a marginally rational and critically ill patient may be ambiguous at best."²¹

As far as space is concerned, Weinberg showed that "many critical care areas offer less square footage than is allotted to an elective patient who is not so desperately ill...lacking in privacy, he (the patient) must often be an unwilling witness to patients in extremis receiving desperate and frequently ceremonial treatment."²² While modern units may consist of glassed-in cubicles, most coronary care

²¹Keilly, W., "Critical Care Psychiatric Syndrome," Heart and Lung, 2:1:54, Jan-Feb. 1973.

²²Weinberg, S., "Toward Humane Care for the Critically Ill," Heart and Lung, 2:1:43, Jan-Feb. 1973.

units are converted wards, making marriage of an old environment and the latest in machinery. In all of this, there are present variable degrees of sensory monotony and sensory overload, erratic or interrupted sleep patterns, social near-isolation, unfamiliarity with the environment and prolonged immobilization.²³

Buskirk surveyed all coronary care units in the State of Oregon and found that while many protected the privacy of the patient, many more units consisted of noisy ward accommodations.²⁴

It appears that the philosophy of rest and serenity for cardiac patients is undermined by the characteristic setting of the CCU. The environment is filled with obtrusive auditory stimuli produced by such equipment as monitors, oscilloscopes, fans, respirators, suction equipment, and oxygen apparatus. The patient may get little respite from the noise. The lights may never go out if observation of skin color is required; or, if lights are lowered, the startling sights by day often become monsters in the shadows. And noise that simply irritates by day may shock at night.

A now classic study by Falk and Woods was carried out

²³McKigney confirms this description of a coronary care unit by saying that "it is highly mechanized, unfamiliar and isolated from the general life experiences of the average patient." McKigney, F., "The Intensive Care Syndrome," Conn. Medicine, 30:9:633, Sept. 1966.

²⁴Buskirk, W., Hospital Coronary Care in Oregon, 1974. A Survey Emphasizing Special Training and Responsibilities of Coronary Care Nurses. Unpublished Doctoral Dissertation, University of Oregon, March, 1975.

at, least in part, in a coronary care unit.²⁵ The investigators found that noise levels of 69.8 decibels, linear, in a recovery room and 73.3 decibels, linear, in the acute care unit approximately equal and exceed the threshold for peripheral vasoconstriction. The methodology of this investigation can hardly be faulted. Intensity in the linear scale, band width, tone, the frequency and duration of noise were ascertained.

Noise measurements were taken at the patient's head, close to his ear, at five minute intervals over a period of 24 hours. For each thirty-minute segment, an average intensity was determined. In a second step, the investigators correlated the number of staff present with the noise averages. The noisiest equipment and procedures were as follows: Cardiac monitor at the bedside, 83 db; the cardiac oscilloscope, 83 db; the respirator, 82 db; and the bed scale in operation, 85 decibels. In the recovery room, the noise from the hopper being flushed reached 88 decibels, while the wall suction in use peaked at 76 db. Although these last two measurements were obtained in the recovery room, flush hoppers and wall suction are a standard part of nearly every coronary care unit. It seems obvious that the coronary care unit may be more stressful and a great deal noisier than a man's own home.

²⁵Falk, S., and Woods, N., "Hospital Noise Levels and Potential Health Hazards," New Eng. J. Med. 288:15:774-776. Oct. 11, 1973.

In a different type of study, Wallace used the stress-related hormones (catecholamines) as he sought to index the amount of stress patients with myocardial infarction might be experiencing as a result of exposure to noise.²⁶ Measurements of the kidney excretion of catecholamines were made on the patient population who had already been transferred out of the coronary care unit. Wallace stated that although the cases in his study had been uncomplicated during their acute phases, and had been studied after a prolonged period of convalescence, the intense response of adrenalin and nor-adrenalin to a continuous moderately high level of noise suggests that these patients are particularly vulnerable to sound stress and react with large catecholamine discharges. Furthermore, it is well-known, that the release of adrenalin and nor-adrenalin into the circulating blood can intensify oxygen requirements of the heart muscle.

Wallace suggested further, that sound from buses, airplanes, and traffic outside the window (of the CCU) not to mention the hum and roar of machines used for or around the patient, could aggravate the hypoxia (lack of oxygen) of an already damaged heart muscle and predispose to arrhythmias and heart failure.

Concepts of Treatment of the "Cardiac" Patient

The first concept of treatment relates to the hypothesis

²⁶Wallace, A., "Catecholamine Metabolism in Patients With Acute Myocardial Infarction," in Acute Myocardial Infarction, Julian D., and Oliver, M., Eds. Baltimore, Md., The William and Wilkins Co., pp. 237-242, 1968.

of multicausality, which states, in part, that stress whether environmental, physiological or emotional, has a prominent place in the cause of cardiovascular disease. Therefore, stressors must be considered as factors to be managed during treatment. If noise can be conceptualized as an auditory stressor, the argument can be made that noise must be minimized for the welfare of the patient. However, a consistent problem in units has been that environmental control has been applied to but a fraction of the potential stressors. For example, visitors are restricted, business activities by the patient are forbidden, news and mail are monitored closely, and only selectively is television allowed. At the same time, the number, as well as the noisiness of technical equipment, has increased with the result that most patients are deprived of familiar sounds and bombarded with the unfamiliar. It is not unusual for the patient being admitted to the coronary care unit to have at least eight staff members enter his room within the first ten minutes, and put into operation at least six different pieces of equipment.²⁷

Secondly, the injured heart muscle is always overloaded in relation to its contractile strength (ability to contract forcefully). "The major aspects," write Friesinger, Baker

²⁷It is common for two registered nurses, an inhalation therapist, a ward clerk, an I.V. clinician, an X-ray, laboratory and EKG technician, and finally the physician, in this order to apply monitor leads, start oxygen, begin the admitting sheet, start an I.V. infusion, take a chest X-ray, draw blood for samples, take a 12 lead electrocardiogram, and finally, make a diagnosis.

and Ross "of treatment of the acute stage are relief of pain, reduction of cardiac workload to an absolute minimum, and observation to predict, prevent, or treat any specific complication."²⁸

Reduction of cardiac workload, which means literally minimizing the oxygen demand of the heart muscle, cannot occur in an atmosphere of noise loud enough to elicit the release of adrenalin, or to cause a change in cardiovascular dynamics.

Heart Rate as Cardiovascular Response to Noise

If noise level can be correlated positively with changes in heart rate, whether or not that change is due to catecholamine discharge, a strong argument can be made for soundproofing the environment of the cardiac patient. For if a critical level of noise increases the physiological demands made upon the injured heart, regardless of the underlying mechanism, it follows that an atmosphere of peace and quiet should be obtained. A serious problem in all field studies is that adequate controls are difficult to maintain. For example, there is reason to believe that shocking sights may also elicit a sympathetic response, but beyond the obvious closing of doors and drawing of curtains, there is no efficient

²⁸Friesinger, G., Baker, B., and Ross, R., "Myocardial Infarction," in Harvey, M., Cluff, L., and Johns, R., et. al. Principles and Practice of Medicine, 17th Ed., New York, Appleton-Century-Crofts, 1968.

method of controlling visual stimuli.*

The rationale for selecting heart rate as cardiovascular response in the present study, in addition to the relative ease with which it can be obtained, is that for every cardiac patient a change in heart rate will, at some point be harmful to health. At this critical point--which will vary with each individual--there may be an increase in oxidative processes, or an outright reduction in output (the amount of blood pumped out of the heart per minute).^{29,30,31} Moreover, the effect of a persistent high or low rate, as well as sudden changes in rate, have been implicated as altering the atherosclerotic processes, according to a recently

*In one intensive care unit visited by the investigator, it was possible for one patient to observe fourteen other patients, with their monitors, breathing machines and other apparatus. Curtains surrounding patient cubicles were left open so that observation by staff could be facilitated. Personal visit, The Royal Alexandra Hospital, Edmonton, Alberta, Canada, January 23, 1975.

²⁹When the heart increases its metabolism, the oxidative processes of the myocardium are concomitantly increased. This poses no problem for the normal heart, but hearts made vulnerable by cardiac disease are predictably subject to malfunction in oxidative processes and a reduction in cardiac output. Burton, A., Physiology and Biophysics of the Circulation, Chicago, Year Book Medical Publishers, 1965, page 148.

³⁰Bing, R., "Myocardial Metabolism," Circulation, 12: 1955, pp. 635-647.

³¹Sonnenblick, E., and Skelton, C., "Oxygen Consumption of the Heart, Physiological Principles and Clinical Implications," Mod. Concepts Cardiovascular Dis. Vol. XL No. 3, March 1971, pp. 9-10.

published epidemiological study.³²

It must be pointed out that an increase or a decrease in heart rate in the person with a normal cardiovascular system is not harmful. In fact, such changes comprise a compensatory mechanism which selectively increases blood flow during periods of increased exercise. Some situations in which this occurs are heavy lifting, running, sleeping, reaction to fright, or, for that matter, jumping out of bed in the morning. It is not known whether this function is altered or diminished in patients with myocardial infarction. It is known, however, that the patient with heart disease, especially M.I., possesses an "irritable" heart, and sudden changes in heart rate, particularly while the patient is at rest, are undesirable.

The danger, as Marshall put it, and "the problem of an increased heart rate arises when the heart with all its properties lacks the ability to withstand additional demands made upon its capacity to perform."³³ According to Ayres and Gregory a heart rate of more than 100 results in less time

³²Beakson, D., Stamler, J., Landberg, H., Miller, W., Stevens, E., Soyugeng, R., Tokich, T., and Stamler, R. "Heart rate, an important risk factor for coronary mortality including sudden death--ten year experience of the People's Gas Company," 1958-1968.

³³Marshall, L., "Patient Reaction to Sound in an Intensive Coronary Care Unit," Master's Thesis, Graduate College Medical Center, University of Illinois, Published in Batey, M. Ed., Communicating Nursing Research Western Interstate Commission for Higher Education, Boulder, Colorado, November, 1972. pp. 81-92.

for the arteries which supply the heart with oxygen to fill. The faster contraction rate may also increase the muscle's oxygen requirements. Certain responses in normal circulatory systems operate to increase coronary blood flow appropriately, however, coronary artery disease may limit the response.³⁴

What is being said, essentially, is that at normal heart rate (60 to 90 beats per minutes), the relaxation time between each ventricular contraction is sufficient for the coronary arteries, as well as the cavities of the heart to fill with blood. A fast heart rate, or even a change in heart rate, may reduce filling time, and subsequently reduce the amounts of oxygen and nutrients delivered to the heart muscle. When such a reduction occurs at the same time as an increase in myocardial metabolism, irritability and aberrant heart rhythms result.

In the presence of a tachycardia, the normal heart and cardiovascular system compensate by raising vascular resistance in the peripheral areas of the body, especially to the skin, thereby diverting blood to the heart itself. In patients with coronary artery disease this response may be grossly limited.

In summary: Several investigators have concluded that noise has an effect on the cardiovascular system of normal man. A lesser number of researchers have shown that a

³⁴Ayres, S., and Gregory, J., Cardiology, New York, Appleton-Century-Crofts, 1971. pp. 132, 139 and 230.

critical level of noise has a predictable effect on the heart rate in subjects with normal cardiovascular systems. The question as to whether noise elicits similar responses from hearts that are diseased has not been answered. The present dissertation will ask: Does noise affect the heart rate in subjects with coronary artery disease? If so, how?

The Effect of Noise on Heart Rate: The Marshall Study

Marshall measured patients' reactions to sound in a coronary care unit and used heart rate as the dependent variable.³⁵ More specifically, this investigator explored the relationship between mean pulse rate and various sounds or noises as they occurred naturally in the coronary care setting. By means of a tape recorder, the electrocardiographic signals from an individual cardiac monitor and oscilloscope were passed through a preamplifier, then used to modulate a 3,000 cycle carrier frequency, and the isolated signal recorded on magnetic tape.

During the time the electrocardiographic signals were being collected all sounds and noise permeating the patient's room were being picked up by a microphone which had been placed by the patient's ear. A cable from the microphone was extended to an Ampex tape recorder and the signals were recorded. Although the data were available, and the sounds were categorized as human-produced and non-human produced, there was no attempt to tabulate such physical characteristics

³⁵Marshall, L., Communicating Nursing Research Op. Cit. pp. 88-92.

as intensity, band width, tone or duration. The data were subsequently analyzed by computer with a histogram of mean pulse scatter obtained for each patient in the study.

The findings of the Marshall study indicated that a positive relationship exists between mean pulse rate changes and acoustical stimuli. The relationship was identified by a decrease or an increase in pulse rate.

Lucile Lewis has sharply³⁶ criticized the Marshall study by pointing out that with both acoustical and electrocardiographic data available on tape, the purpose of the study could have been better achieved if changes in heart rate had been correlated with acoustical stimuli.

A second weakness of the study, which Lewis fails to note, is the lack of differentiation between cardiovascular disorders. Several diagnostic classifications could have been utilized: myocardial infarction, suspected myocardial infarction, cardiac surgical patients and those with congestive heart failure. It must be pointed out that in the minds of many practitioners, "cardiovascular problems" includes persons with hypertension and peripheral blood vessel disease. Obviously, the physiological responses of these types of patients could differ greatly from those with coronary artery disease.

³⁶Lewis, L., "Critique of Patient Reaction to Sound in an Intensive Coronary Care Unit," Communicating Nursing Research, Batey, M. Ed. Op. Cit. pp. 93-97.

The present dissertation will define these categories and limit the study population to those persons with coronary artery disease, with or without a myocardial infarction.

While the method of data collection and analysis used by Marshall was extremely sophisticated, the conclusions have had little practical application due to the lack of correlating the independent variable with the dependent in any meaningful way. And the question remains: Does noise have an effect on the heart rate of patients with coronary artery disease?

SUMMARY

Literature supporting the assumption that urban noise is a problem to man was reviewed in Chapter I. The multi-causal hypothesis for cardiovascular disease has been elaborated with special emphasis on the stressor effects of noise on persons who are exposed for more than very short periods of time.

A theoretical framework evolved in which the physical model of stress became the basis for describing the stressor properties of noise upon the cardiovascular system of man. Although the annoying characteristics of noise were mentioned, it was not intended that psychological aspects of noise should be a part of the present study.

Laboratory data were cited as evidence of the sympathetic and pressor effects of noise stimuli measuring greater than 70 decibels. While retaining the argument that noise

is a problem to urban man, the focus has been narrowed to the individual with coronary artery disease as he is admitted to the intensive coronary care unit. Heart rate was accepted as a relevant criterion for the cardiovascular effect. The behavior of heart rate in normal subjects after noise exposure was reviewed and from that review, questions were generated concerning the heart rate response to noise in patients with coronary artery disease. The work of Marshall and Wallace was described in detail, regarding purpose, methodology and conclusions, for these studies form the basis for the present investigation. Important weaknesses in the Marshall study should be corrected in the present dissertation.

Particularly, the intensity of noises common to a CCU will be obtained and correlations with heart rate will be attempted. In addition, the patient population will be specifically defined and limited to those patients with coronary artery disease. All factors known positively to affect heart rate will be tabulated and subgroups will be comprised according to sex, age, hospital, time of data collection, past history of heart disease, last known occupation, family status and place of residence. These, as well as the pharmacological actions of certain cardiac drugs will be discussed in a subsequent chapter, as weaknesses of the study.

Hypotheses For the Present Study

The major hypothesis for the present investigation will be that Noise will affect heart rate in patients with coronary

artery disease. Included in the category "coronary artery disease" are patients with a diagnosis of angina pectoris (chest pain), myocardial infarction, and congestive heart failure, but excluding patients with mitral and aortic valvular disease, hypertension, or peripheral vascular disease.

A second hypothesis will argue that The rate of heart rate response to noise will be significantly greater in patients with myocardial infarction than in patients with other types of heart disease. That is, a greater number of the former will respond to noise stimuli with a change in heart rate.

Myocardial infarction means damage to heart muscle, however, the injured area is not sharply delineated as might be supposed. Accurately described, an infarction includes an area of dead heart muscle, encircled by an area of tissue that is injured, but not necrosed, and finally, by an outer ring of ischemic (oxygen-wanting) muscle. In professional terms these are "zones of infarction." No heart disease renders the heart more vulnerable to stimulants such as coffee, excitement, noise, et. cetera, than does an infarction. For these rather obvious reasons such patients are classified as a separate study group. This continues from the work of Marshall.

The minor hypotheses for the study are first: that the heart rate response to noise will vary with the site of the heart damage. Thirteen years of observation of cardiac patients, coupled with continuous study of the

genesis, pathology, medical and nursing aspects of such patients, underlies the interest of this investigator in the differential responses of patients with varying areas of heart damage. The cause of infarction is a reduction in the supply of blood to any area of the heart muscle. Since the arteries which nourish the heart are many and well dispersed, the anatomical site of infarction is dependent upon the specific arterial branch that is blocked.

A common classification separates inferior and inferolateral infarctions from those that are anterior or anterolateral. It is noteworthy that persons with inferior infarctions tend to exhibit slow heart rates, while those with anterior heart damage generally do not.

The heart rate is initiated, and therefore controlled, by a node of specialized tissue in the right atrium of the heart, with the ventricular contraction rate following from the passage of this impulse down through the right and left ventricular muscles. An inferior infarction often interrupts the blood supply to the cardiac fibers through which the normal sinus impulse must pass, in its transmission through the upper chambers to the lower chambers of the heart. The electrical impulse is literally blocked. An auxiliary pacemaker--so called because it arises when the normal conduction fails, must generate electrical impulses from a site below the heart damage. The ventricles contract as a result of the impulse generation from an abnormal pacemaker.

The question arises as to whether this latter group of patients have the capacity to increase their intrinsic heart rates. The present dissertation asks whether there will be a differential response to noise that can be explained on the basis of the site of the heart damage.

The final hypothesis states that the response to noise stimuli is a function of the gap between ambient and peak levels of noise for any subject. This hypothesis derives from arguments made by Baron and others that ambient noise is not physiologically harmful. Baron fails to take into account the pressure aspects of noise, as discussed earlier in detail, and their insidious effects on the cardiovascular system. Nor is there any indication in Baron's argument what the upper limits of ambient noise might be. It would appear that at some critical intensity "ambient" noise would become continuous loud noise.

Although beyond the scope of the present study, it is acknowledged that the duration of noise may be a factor to be considered in the noise effect. Browning argued that the greater the intensity of noise the less time a subject can safely be exposed. The equal energy basis is that, for example, 90 decibels of noise in the A scale endured for eight hours, is equal in risk to a four hour exposure at 93 dbA, or a 4.8 minute exposure at 110 decibels.³⁷ However, Browning as well as other experts, warned against the dangers

³⁷Browning, C., "Short duration noise exposure and the equivalent energy concept," Pollut. Monitor No. 11 pp. 33-36, 1974.

of extrapolations to minutes or seconds. In fact, Fidell and others held that the noisiness, in perceived noise decibels, of impulsive sounds resembles an energy sensation process to the ear and the phase spectrum is irrelevant.³⁸ These questions remain to be answered definitively in the laboratory for the nature and vulnerability of critically ill cardiac patients prevents exhaustive experimentation in the coronary care unit.

While the noise-duration exposures in the Marshall study are retrievable since they were recorded on magnetic tape, the methodology of the present investigation disallows such tabulation.

Chapter III describes how the study is designed to elicit information to enable one to reject or not reject the hypotheses. Chapter IV will depict the results of the study and discuss the data not to be statistically treated. Chapter V will conclude with suggestions for further investigation and implications for planning in the city.

³⁸Fidell, S., Pearson, K., Gregnette, M., and Green, D., "The noisiness of impulsive sounds," in Bolt, Beranek and Newman, Inc. Canoga Park, California. J. Acoust. Soc. Am. 48:6:1, 1970, pp. 1304, 1310.

CHAPTER III

THE DESIGN OF THE STUDY

Purpose of the Study

This study will attempt to isolate a heart rate effect of noise. Correlations between noise levels and heart rate responses will be analyzed. Noise levels will be measured in decibels A--the scale which most closely approximates the perception of noise by the human ear. Noise levels below or equal to 50 decibels will be categorized as low noise with noise levels to or above 60 decibels termed high noise. Heart rate response will be measured per R to R interval, using a five second time lag cutoff, and extrapolated to beats per minute.

The Study Population

The study population will include all patients who have experienced a myocardial infarction, or who demonstrate evidence of other cardiovascular disease such as chest pain, angina, or congestive heart failure, and who are subsequently admitted to a coronary care unit during the six week period from December 15th, 1974 to January 31, 1975.

An N of at least 25 subjects is sought in order to meet the requirements of statistical testing by regression analysis. The time at which each subject's noise-heart-rate response will be measured will be assigned randomly from choices of time slots distributed across the three hospital

shifts (Day shift, 7:00 A.M.-3:30 P.M.; Evening shift, 3:31 P.M.-11:00 P.M.; and Night shift, 11:01 P.M.-6:59 A.M.).

This aspect of the design is intended as a partial control against unusual noise levels being applied to one or two subjects only, and will thereby reduce the dependency among the observations.

Sampling of the Population

The time at which any particular subject will be monitored will be randomly assigned. An exact number of data times from each shift would be desirable, however, a random sampling of one-hour time slots within the 24 hour day may not yield such an ideal distribution. The following time slots were designated partly because of certain limitations upon the investigator's time, and in other cases a time is included because of the activities that typically occur then. For example, staff activity increases between six to seven in the morning when vital signs and 24 hour summaries are done. One of the busiest times for a coronary care unit is from 7:00 to 9:00 A.M. for the majority of physicians arrive early for their morning rounds. The following table lists the times from which random samples will be drawn.

The following method will be utilized to draw sampling times for a minimum of thirty subjects. For each one-hour period, two squares of paper will be used. This method guarantees that each subject within the population will have approximately the same possibility of being measured at any

given time. Subjects will be represented numerically, with a sample time drawn for each. The time slot drawn for a subject will be referred to as his data collection time and will be recorded as such. If the study population totals more than thirty in number, the least well represented time slot will be arbitrarily assigned to the additional participants.

TABLE I
THE TIME SLOTS FROM WHICH A RANDOM
SAMPLE WILL BE DRAWN FOR
THE POPULATION

Day Shift	Evening Shift	Night Shift
7:00-8:00	5:00-6:00	11:00-12:00
8:00-9:00	6:00-7:00	12:00-1:00 A.M.
9:00-10:00	7:00-8:00	1:00-2:00
10:00-11:00	8:00-9:00	5:00-6:00
11:00-12:00	9:00-10:00	6:00-7:00

Description of the Equipment: Unit A

The equipment to be used in generating heart rate data will include a General Electric Bedside Heart Monitor with the following characteristics:

Electrocardiographic Signal
Amplifier A 3123BB (Model #11KALDL)

Remote Heart Rate Module
A3123CB (Model #11KR3B1)

5 inch Heart Rate Display Scope
A3121A (Model #11KS2A1)

The Remote Heart Rate Module is equipped with a "sample" release button which enables one to obtain an immediate ECG graphic readout from the Electrocardiographic mechanism at the central nurses station. While the heart rate meter displays the heart rate continuously with an accuracy level of 99 per cent, the dial needle oscillates constantly between any two heart beats making it almost impossible to correlate exactly, heart rate with the time of noise exposure without a graphic write-out. Activation of the Sample button initiates a 20 second electrocardiographic tracing when a graph module is a part of the system.

The electrocardiographic signal will be obtained from a General Electric Electrocardiographic Machine (Model #10321-P-10) which is equipped with an automatic 12 second Memory Mode to give a total write-out of 20 seconds of ECG tape. The specifications include:

Input impedance 300k ohms

Sensitivity 0.5 volts/cm.

Frequency response DC to 100 Hz (3db point)

Paper speed 25 mm/second.

Table'II depicts the variance in total tape emitted, stylus warmup time, and the amount of readable ECG strip.

The variation in amount of the ECG sampling is minute (.33 sec.), and is unlikely to have any appreciable effect on the measurements of heart rate after noise exposure.

TABLE II
 AMOUNT OF READABLE ECG TAPE IN SECONDS FROM
 SIX SAMPLINGS OF THE GENERAL
 ELECTRIC MEMORY MODULE

Stylus Warmup	Total Sample	Readable ECG	Total ECG Tape
.80	19.20	18.40	20.00
1.00	18.80	17.80	19.80
.80	18.00	17.20	19.60
.80	18.40	17.60	19.20
.84	19.20	18.40	20.04
.84	19.20	18.36	20.04
SD = .007 sec.		SD = .33 sec.	

Characteristics of the Noise Recording Equipment

A Bruel and Kjoer Impulse Precision Sound Level Meter Type 2209 (Model #2204) will be utilized to measure noise intensity in decibels A. The battery operated device is capable of taking measurements in the A,B,C or D and linear scales. An advantage of this model of noise level meter is that a "slow" or "manual hold" circuit for the needle position allows extremely accurate reading of low and peak levels of noise.

Locational Description: Unit A

Four private rooms, hereafter called A B C and D, make up the coronary care portion of the 10 bed Unit A. These rooms are located on the second floor of a block and brick structure which was completed three years previous to the present study. Rooms A B C and D will be monitored for noise

level from time to time throughout the period of data collection with no particular emphasis on the pattern of use for individual subjects. (The rooms may be identified on Page 75 as A B C and D with room C as the model.)

The furniture, the electrical and mechanical equipment in each of the rooms is identical, with the exception that a warm air vent is situated in the ceiling of Room A. The noise level meter (1) will be placed upon a room-wide, built-in commode at the immediate head of the patient's bed. (2) This places the microphone of the noise level meter not less than two, but no farther than three feet from the ear of the patient. The ECG oscilloscope (3) is located immediately adjacent to the sound level meter and will provide for a minimum time lag for activation of the sample switch. The Sample button (4) on the face of the bedside monitor will be used to activate a 20 second ECG tracing at the central nurses' station (5). These tracings are retrieved for analysis of the heart rate in the manner previously described.

Description of the Equipment: Unit B

A Memory Module is unavailable for data collection in Unit B. The instruments to be used are an Electronics for Medicine Heart Rate Monitor, (Model #ERI-5 11808), and an Electronics for Medicine Instantaneous Pickup (Model #WNI-6). Specifications are as follows:

Input impedance 10 meg.

Sensitivity .5 volts/cm.

Frequency response DC 0.2 to 200 Hz.

Paper speed 25 mm/sec.

Because the electrocardiographic recording makes use of a hot stylus against a wax coated ECG paper, stylus warm-up time is a part of every standard electrocardiogram. The following warmup times (delay times) were noted over a sequence of five trials. Each trial consists of several ECG samples so that every score is a mean. Table III shows this data.

TABLE III
 DELAY TIMES CAUSED BY STYLUS
 WARMUP FOR FIVE ECG SAMPLES
 IN ONE PATIENT

Trial	Delay in Seconds
1	.32
2	.265
3	.253
4	.231
5	.207
Mean Delay Time	.255
SD = .0014 seconds	

Locational Description: Unit B

In Unit B the heart rate monitors, as well as the write-out modules are located at the foot of each patient's bed.⁽¹⁾ This arrangement forces the investigator to stand at the foot

of the bed in order to activate the write-out device. The noise level meter will be placed on the bedside stand which is, in turn, situated about two feet from the head of the patient.(2) The face of the noise level meter is always in direct view of the investigator.

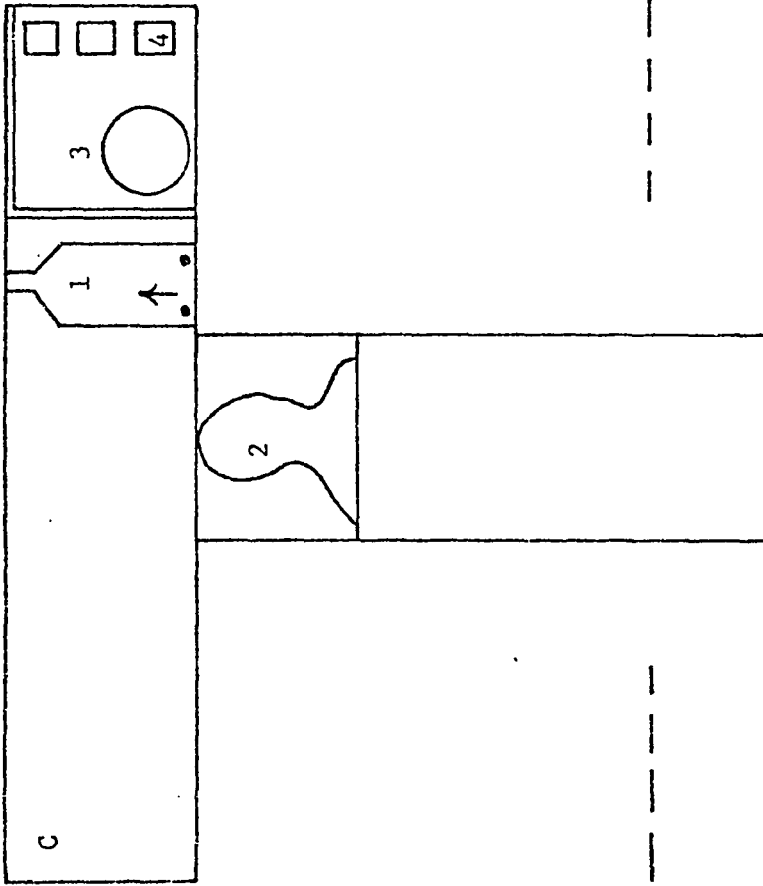
Eye-hand reaction time was estimated to range from .10 to .15 of a second when the trigger finger was left on the Sample button at all times. Thus it is recognized that two delays must be taken into account in a determination of heart rate response to noise, i.e. a delay in information flow called the "stylus warmup" and a second, eye-hand reaction delay. However, the sum of these two delays is less, or equal to the physiological reaction time of heart response (after Landis)¹. An eye hand reaction time of from .10 to .15 seconds when added to the mean stylus warmup time is still well below the time required for the heart to respond to any stimulus, and particularly, to noise. The instant start write-out device is considered an acceptable means for measuring a heart rate response to noise in Unit B.

METHODOLOGY

The operator will be stationed at the head of the subject's bed (Unit A) or at the foot of the bed (Unit B), close to the monitoring equipment. One finger will remain on the Sample button at all times during data collection. In the

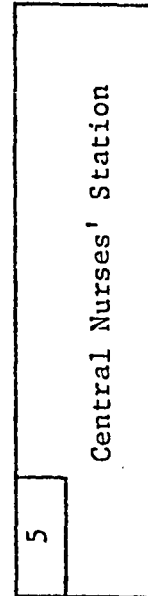
¹Landis, C., and Hunt, W., The Startle Pattern, New York, Farrar and Rinehart, Chapter 2, 1931.

D



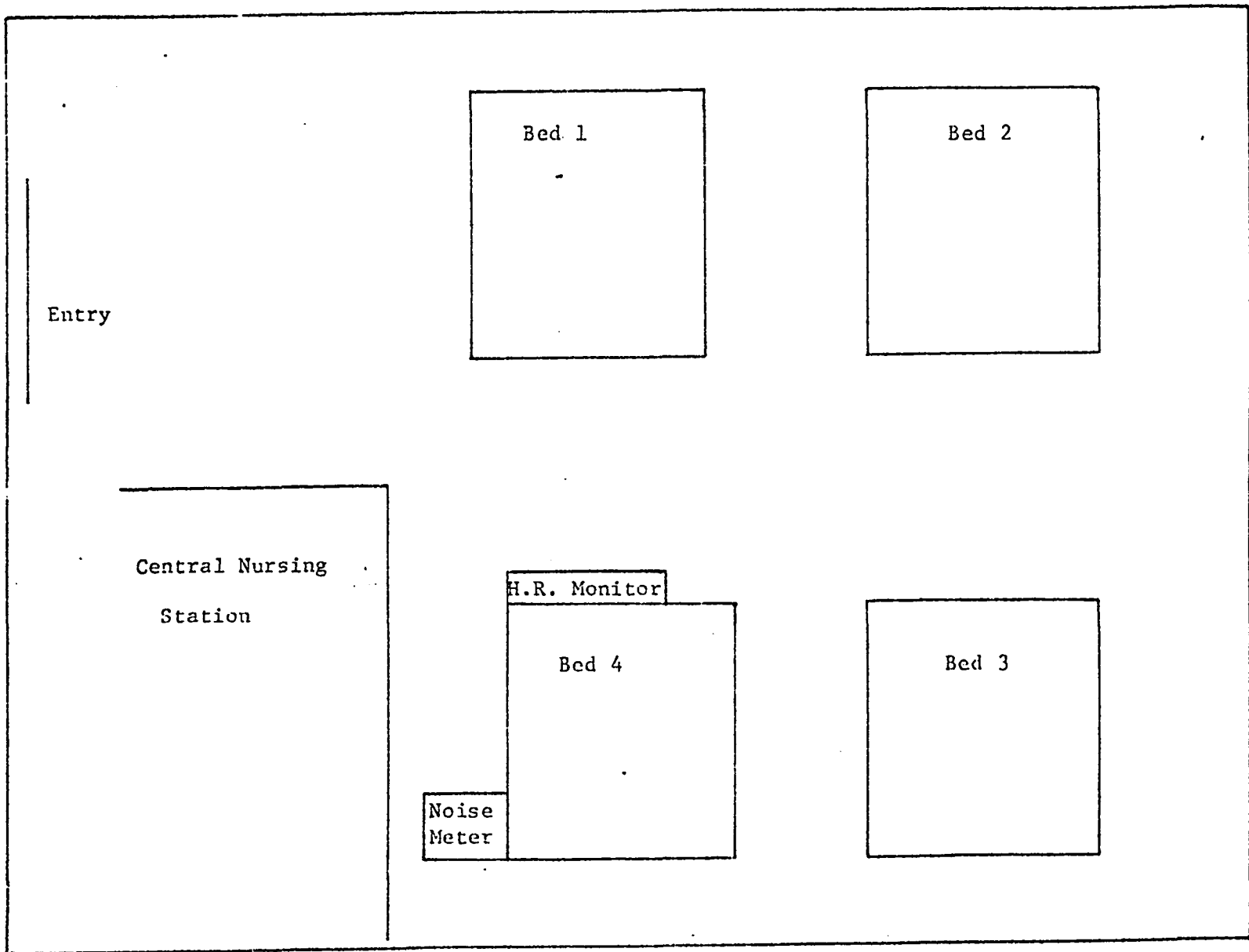
B

A



Door

U N I T A



UNIT B

event a subject asks for information about the procedure, a brief explanation will be given in the interest of assuring the patient. When, after a period of time, the noise level meter registers no greater intensities than 45 through 50 decibels, three to five samples of heart rate will be drawn. These samples will subsequently become the patient's base or resting heart rate.

If, during collection of the base rate, impulsive noise occurs, all samples accumulated before the noise occurred will be discarded and collection will begin again. Similarly, when the noise level meter registers 60 or more decibels, the Sample switch will be activated and a continuous twenty second tracing will be obtained. The samples for subjects in Unit A will include the 12 second memory feature, thus the remaining 7 seconds will be analyzed for heart rate change in response to noise.

For subjects in Unit B, where no memory loop is available the sample continues for twenty seconds. It is necessary to subtract the lag time for the HRR which, according to Landis, ranges from .3 to .5 seconds. The readable strip for Unit A will be 7 seconds. For Unit B the sample is approximately 19 seconds in length. Subsequently the heart rate under noise conditions is obtained by measuring any two R to R intervals and extrapolating this information in milliseconds, to heart rate per minute.

It is expected that in the course of an ordinary hospital day, each subject would be exposed to intervals of

high noise, as herein defined. A mean heart rate during exposure to high noise conditions will be generated for each subject by averaging the heart rate obtained during three discreet exposures. The following two tables depict this information using fictitious data.

TABLE IV
HEART RATE AT LOW NOISE FOR FIVE
TRIALS IN ONE SUBJECT,
FICTITIOUS DATA

Sample	Noise in dbA	Heart Rate
1	34	64
2	38	64
3	36	68
4	33	66
5	35	60
X=35.2		64.4

SD Heart Rate = 7

TABLE V
HEART RATE AT HIGH NOISE FOR FIVE
TRIALS IN ONE SUBJECT
FICTITIOUS DATA

Sample	Noise in dbA	Heart Rate
1	64	66
2	70	64
3	77	70
4	82	72
5	64	66
X=71.4		67.6

SD Heart Rate = 8.6

The gap between mean noise levels for the subject shown is 36.2 decibels and the mean heart rate response score is 3.2 beats per minute. The mean of low noise, or basal conditions, subtracted from the mean of high noise becomes the noise differential for that subject. For example, $X N_L - X N_H =$ the noise differential. And the mean heart rate response at low noise subtracted from the mean heart response at high noise becomes the heart rate response score for each subject. For example, $X HRR_L - X HRR_H =$ heart rate response score. Unlike the noise differential score which must, by definition, be a positive number, the heart rate response score may be zero, a plus or a minus quantity. A noise effect is said to occur when a change of heart rate is present regardless of the direction of the change. Figure 1 shows the noise level and the heart rate response for several subjects. All scores are means and each subject is represented only once. The data are fictitious.

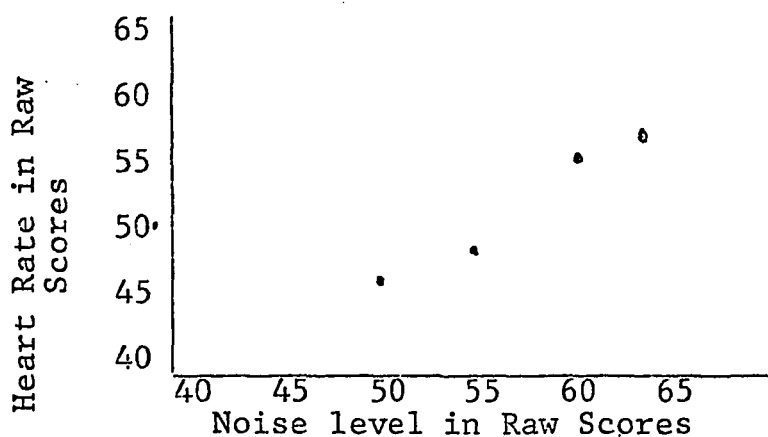


Figure 1. The effect of noise on heart rate by site of heart damage.

Figure 2 regresses the Noise Differential against the Heart Rate Response Score. The data are fictitious.

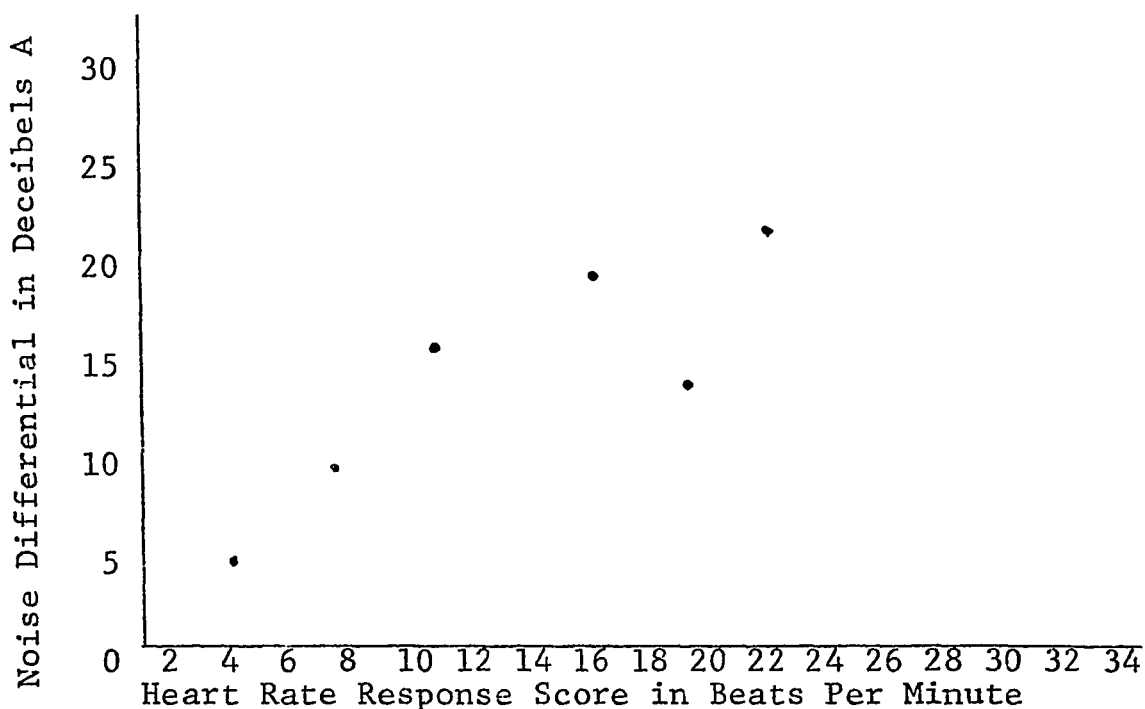


Figure 2. Noise gap related to heart rate response.

Statistical Analysis of the Data

An ideal statistical tool for analysis is not forthcoming. This is due in part to the methodological limitations placed upon the collection of data on critically ill patients. A basal heart rate that is averaged over several samplings of the heart rate at rest cannot be correlated, sample by sample, with subsequent changes in heart rate after noise-exposure, even when all known variables have been controlled. Moreover, the interest of this dissertation lies with establishing the presence or absence of a heart rate response to noise exposure. The "noise effect" may take the form of an increase or decrease over basal rate as described above.

A pair of mean scores (sequential scores for loud noise) will be generated for each subject, that is heart rate at low noise (basal as X_a and heart rate at high noise, X_b in beats per minute and extrapolated to rate per minute. Directionality of the response is not relevant to the major hypothesis i.e. that noise will effect heart rate. Based upon the work of Marshall² and citing Croxton,³ a nonparametric test, the sign test, will be applied to determine the presence of an effect. The lack of an outcome (no response) will be assigned a negative and the presence of a change in rate will be assigned a positive. Because of the manner in which H_0 is stated the signs may be arbitrarily assigned.

The power efficiency of the sign test is about 63 per cent for large N s but becomes increasingly more efficient when used with small samples. Using the sign test, Marshall showed a linkage between noise and heart rate in her study of cardiovascular patients.

It must be emphasized that a more efficient parametric test is required before conclusions can be drawn about the magnitude of the noise effect where the presence of a noise response implied nothing about the magnitude of that response. Therefore statistical analysis to determine whether the heart rate changes that occur to subgroups within the total population will be carried out by means of the Students t test

²Marshall, L., Loc. Cit.

³Croxton, F. Elementary Statistics, New York; Dover Publications, 1953.

adapted for use with paired and sequential samples on the same population.

Such a test is expected to give information about the significance of heart rate changes following noise exposure within specific subgroups. Where t is

$$t = \frac{\bar{X}_a - \bar{X}_b}{\sqrt{\frac{\sum D^2 - \frac{\sum D^2}{N}}{N(N-1)}}$$

Campbell and Stanley argue for the use of a t for dependent means that takes r between X_a and X_b into consideration.⁴ However their arguments are aimed primarily at designs in which only one presentation of the treatment condition (in this case noise) is introduced. Thus the chance sampling error of occasions might contribute to what is thought to be a significant difference between means. The present design will utilize three separate samplings of heart rate at low noise to generate a mean basal heart rate, and three samplings of heart rate response to noise, over three independent occasions. Glass and Stanley indicate that the most efficient formula for t would be

$$t = \frac{\bar{X}_a - \bar{X}_b}{\sqrt{\frac{S_1^2}{N_1} + \frac{S_2^2}{N_2} - \left[2r_{12} \frac{S_1 S_2}{N} \right]}}$$

⁴Campbell, D., and Stanley, J., Experimental and Quasi-Experimental Designs for Research, Chicago, Illinois. Randy McNally, 1966, page 45.

where X_1 is the mean heart rate at low noise and X_2 is the mean heart rate at high noise on the same subjects. While a greater magnitude of r would reduce the standard error, it seems important that random paired samples without large standard deviations be used.⁵

The data as generated by the present study does not meet the criteria for utilizing such a formula, therefore the former t for sequential scores will be used.

A t test for independent samples will be applied to data relevant to hypotheses two and three, that is where the mean heart rate response between independent subgroups is to be analyzed. Where t is

$$t = \frac{\bar{X}_b - \bar{X}_a}{\sqrt{\frac{s^2}{N_a} + \frac{s^2}{N_b}}} \quad \text{and degrees of freedom} = N_a + N_b - 2$$

where \bar{X} in each group represents a mean response score or the difference between heart rate at low and heart rate at high noise.

Finally a simple regression analysis will be carried out to determine whether the magnitude of the heart rate response can be correlated with the magnitude of the noise gap between low and high noise. Thus the heart rate response score which evolves from the difference in heart rate at

⁵Glass, G., and Stanley, J., Statistical Methods in Education and Psychology, New Jersey, Prentice-Hall Inc. 1970. Section 14:4 Inferences about using dependent samples.

noise low and high noise, is represented by the Y axis and the noise gap, N_h minus N_l is represented on the X axis. It is anticipated that the regression will be used for the entire population rather than on subgroups within the population since many of these are very small.

DATA NOT TO BE STATISTICALLY TREATED

Procedures which are commonly carried out within the coronary care unit or upon the patient who is hospitalized there will be monitored to ascertain their noise emissions. Documentation of noise levels for procedures and equipment will be carried out for each unit. These will include:

1. Telephone ringing
2. Telephone ringing while on "high" tone
3. Voice paging
4. Bed curtains being drawn
5. Bed rails being raised or lowered
6. Side rails being pulled up or down
7. Positive pressure breathing machine
8. Hopper or toilet being flushed
9. Water running in the patient's room
10. Gastric suction in use
11. Tracheal suction in use
12. Ambient noise of continuous bedside monitor
13. Wash pans being readied for bath
14. Laundry being placed in hampers

A number of activities that involve the staff at the bedside of the patient will also be monitored for noise level. These

include:

1. Doctor at the patient's bedside
2. Visitor at the patient's bedside
3. Electrocardiogram being taken
4. Delivery of the pharmacy supply cart
5. Change of shift report
6. Visitors asking questions at the desk
7. Supervisor-nurse report
8. Inhalation therapist at bedside

Each measurement will be approximately 20 seconds in duration and three such measurements will be made for each activity before an average noise emission level for that activity is documented. It is anticipated that the measurements will be spread over any number of data days due to the limits upon the amount of time the investigator can spend in each coronary care unit. For example, if a patient is being maintained on a respirator but the alarm is not activated during the time of routine data collection, that information will be collected whenever the investigator is present in the unit and the pressure alarm does sound. Such measurements of noise emissions will not be correlated with heart rate thus they may be made at any time during the six weeks data collection period.

However, it must be noted that exposure to noise levels of 70 or more decibels results in peripheral vasoconstriction. For that reason the number of procedures that consistently emit noise at or above the 70 dbA limit will be tabulated.

The purpose is to ascertain how many times during a hospital day a patient might be exposed to dangerously loud levels.

Lastly, an ambient noise level will be tabulated for each one-hour period during a 24 hour day. No unusual noise level fluctuation will be included and should such impulsive noise occur, for example, a cart accidentally banging against the wall, a window breaking or a Code 99 call, the entire sample will be discarded and another obtained under more "normal" noise conditions. The 24 hour noise profile will be depicted to show the peaks and ebbs for each unit.

The comments and complaints of patients or their visitors will not be solicited, however, if offered to the investigator spontaneously they may be included in the Discussion.

Limitations On The Study

The major weaknesses of the study arise from experimentation with patients in the coronary care unit who are critically ill. Humane considerations prevent the interruption of ongoing medical and nursing care, therefore equipment must be small in size, quiet, and stored out of the way when not in use. Ideally heart rate would be monitored over a long period of time and a scatter of heart rates over x number of quiet trials for each subject would be generated. The result would be a more nearly accurate basal heart rate for each subject. However limitations of time and finances make the ideal unfeasible for this investigation. The methodology will be adjusted and epochs of a few seconds,

as described previously, will be used. Also, the investigator is aware of the danger of extrapolating R to R intervals in milliseconds to beats per minute. While it is an acceptable method to use in the day to day care of these patients and widely used by practitioners, it is less than ideal in a research endeavor.

Ideally, a sound level meter with an automatic write-out device and band width analyzer located just outside the subject's room would have been employed. Cables would then lead to an output jack at the bedside microphone. In this manner any fluctuation above ambient noise level (as previously set) would automatically activate the electrocardiographic write-out. The data could then be analyzed and exact correlations made.

Early in the planning, a Revox Model A 700 professional quality, two track tape recorder was to be used to simultaneously record noise level, from a microphone at the patient's head, and the ECG tracing from an output jack at the bedside monitor. The noise level signals and the 1 millivolt electrocardiographic signal would be recorded together on magnetic tape. Data reduction could then proceed by means of a two channel write-out of any suitable manufacture. Although some initial work had been done using this methodology, the consultant withdrew from the study and the equipment became unavailable.

Care must be taken that the subject is not inadvertently exposed to additional stress through data collection.

The noise measurement equipment is minimal when compared to the array of technological devices already in operation inside the patient's room. Conversation with a subject will not be encouraged, however, in the event a patient initiates small talk each occurrence will be evaluated separately. It is hoped that the subject will be at rest or asleep when the epochs of heart rate are collected for a basal rate.

The full intent of the study cannot be shared with members of the staff who give direct care to these patients. Most persons trained in coronary care are aware of the philosophy of rest and quiet as it applies to these patients so that awareness on the part of the caretakers would naturally tend to create a "quiet" environment. The setting for data collection is the coronary care environment as it exists even though that environment may be far from the ideal.

It is well known that heart rate is controlled and affected by numerous neuro-humoral influences. Given the limitations of time and money posed by this study it is impossible to control for all of these. However, it is the intent to recognize and identify the various drugs and physiological conditions, other than noise, that will affect heart rate. Among these are: severe pain, anxiety, of which noise may be a cause, sleeplessness, acid-base or electrolyte imbalance, activity level, body temperature, complete heart block, and other vagal and sympathetic influences. Also, certain drugs such as cardiotonics, anti-arrhythmics and sedatives are known to have some influence

on the heart rate. These factors will be discussed with the conclusions drawn from current literature in the field.

Factors Other Than Noise That May Affect Heart Rate

External temperature will be the first of these factors to be discussed. In an epidemiological study of climate and heart disease, Burch and Giles found that a hot and humid environment appeared to correlate positively with the incidence of heart disease. They conclude that deaths from cardiac disease "literally soar after the onset of hot weather."

The reason appears to be related to the increased workload imposed upon the heart since the responsibility for transporting large quantities of heat from the central area of the body to the surface lies primarily with the cardiovascular system. Large increases in what Burch and Giles refer to as "thermal load" whether because of fever, physical exertion, or environmental temperature, must increase the cardiac output (amount of blood pumped by the ventricles many times over). A fast heart rate commonly results as the heart works harder to pump large quantities of warm blood to the dilated vessels of the skin where excess heat is lost.

⁶Burch, G., and Giles, T., "The Burden of a Hot and Humid Environment on the Heart," Mod. Concepts Cardiovasc. Dis. XXXIX No. 8 August, 1970, pp. 115-120.

⁷Ansari, A., and Burch, G., "Influence of hot environment on the cardiovascular system: Clinical study of 23 patients at rest," Arch. Int. Med. 123:371-378, 1969.

On the other hand, Raven and his associates found that heart rate also increased significantly when their subjects were exposed to ambient temperatures of five degrees centigrade for a period of two hours.⁸ The literature lends little to support or refute the Raven findings.

When body temperature rises an increase in heart rate will occur at increments of approximately 10 beats per minute for each additional degree Fahrenheit. Regardless of the process (infection, inflammation, endocrine dysfunction, or any other), the assumption holds that the oxidative processes of the body are increased.⁹

Most coronary care units are temperature-controlled the year round. Room temperature is maintained within very narrow limits (i.e. from 68 to 70 degrees Fahrenheit). However patients with myocardial infarction or congestive heart failure tend to develop hyperthermia, usually in the range of 98 to 101 degrees Fahrenheit and unassociated with external room temperature. According to Storlie, and Rombilt and Fowler the patient's temperature may be expected to return to normal within two to three days if there are no complications. Nor is there evidence that the feedback functions of the

⁸Raven, P., Iaso, N., Dahms, T., and Horvath, S., "Compensatory cardiovascular response during an environmental cold stress, 5C.," in Variables of physiological parameters of unacclimatized males during a two-hour cold stress of 5 C. Int. J. Biometeor. 14:309-320, September 1970.

⁹Best and Taylor's Physiological Basis of Medical Practice, Ninth Ed. Eds. John Brombeck, Baltimore, The William and Wilkins Company, 1973, pp. 3:1-24.

baroreceptors and chemoreceptors are diminished by external or body temperature. The capacity of the heart to respond to neurogenic and humoral stimuli is not necessarily influenced by temperature in these patients.^{10,11}

Heart Rate In Relation To Age

Heart rate appears to vary in a predictable fashion with aging. The average heart rate in humans is about 70 beats per minute with a wide range between individuals. According to Fuchs-Schmuck the variation over a 24 hour day in an individual is minimal.¹² The stable heart rate diminishes, however, progressively from birth to adolescence and increases again in old age. In normal persons there is a marked stability in heart rate over time.¹³

Diurnal Influence On Heart Rate

Diurnal influence on heart rate has been investigated mostly in relation to sleep and wakefulness. In what is, perhaps, the most sophisticated study, Glagov et. al. tabulated the 24 hour heart rates of 100 normal men over a seven month period of time. The mean heart rates at sleep ranged

¹⁰Storlie, F., Principles of Intensive Nursing Care, 2nd Ed. New York, Appleton-Century-Crofts, 1972. pp. 50-51.

¹¹Rombilt, D., and Fowler, N., "Physical Signs in Acute Myocardial Infarction," Heart and Lung Vol. 2 No. 1 January-February 1973, pp 74-80.

¹²Fuchs-Schmuck, "A Study of Pulse Behavior in Relation to Industrial Noise," Op. Cit. page 5.

¹³Storlie, F., Principles of Intensive Nursing Care, Op. Cit.

from 52 to 93 beats per minute for different subjects, while the awake rates (without exercise) ranged from 74 to 93 beats a minute. The individual means over the seven month period were extremely stable although there were wide variations between individuals.¹⁴ There is no indication in the report that the population was controlled for age, except that all were adults. Although Freidman's primary interest with heart rate was in patients with cardiovascular disease, his conclusions were remarkably similar to Glagov's.¹⁵

Plasma catecholamine levels are known to exert a direct effect on heart rate, however investigators have been unable to correlate diurnal variations in catechol levels with changes in heart rate. Aronow et. al. found no relationship between the concentration of plasma catecholamines and systolic time intervals.¹⁶

¹⁴Glagov, S., Rowley, D., Cramer, B., and Page, R., "Heart rate during 24 hours of usual activity for 100 normal men," J. Applied Physiol. 79:6:799-805. December, 1970.

¹⁵Freidman, M., Rosenman, R., and Brown, A., "The continuous heart rate in men exhibiting an overt behavior pattern associated with increased incidence of clinical coronary artery disease," Circulation 28:861-866, 1963.

¹⁶Aronow, W., Harding, P., and De Quattro, V., et. al. "Diurnal Variation of Plasma Catecholamines and Systolic Time Intervals," Chest 63:5:722-726, May, 1973. "Systolic time intervals" refer to the amount of time it takes for the ventricles to be depolarized (after which they contract) since the ventricles, not the atria are considered to be the physiological pumps for circulating blood throughout the body. The temporal aspect refers to the amount of time required for this depolarization process and can be obtained by measuring the QRS deflection of the electrocardiogram. Time is measured in milliseconds in the direction of the arrow, from the beginning of the deflection to the end.

And, in a classic study of circadian crests, Halberg and Folliers studied children "transversely." Measurements of heart rate, temperature, urinary volume and cortisol excretion were obtained for 36 children. Phasic reactions occurred which varied among children, but temperature and heart rate peaked between 12:00 o'clock noon and 6:00 P.M. Only one child's heart rate crested between 6:00 P.M. and midnight and five children had heart rate crests between 6:00 A.M. and noon. Cortisol began to rise at the beginning of the sleep period and in the majority of children the crest appeared just before waking time.¹⁷ It must be pointed out that there is much disagreement concerning the effects of light and darkness on endogenous body rhythms. Even if agreement were possible, the question of the effect of light and darkness on the heart rate may not be a valid one for patients in the coronary care unit, since the lights are never entirely extinguished.

In some units lights are dimmed at about 10:00 P.M. but in many others the lights burn brightly day and night. Therefore no effort will be made to control for diurnal variation in heart rate in the present study. Whether or not the heart rate varies over time (after Glagov) has little bearing on the hypotheses for this study since the interest here is with the capacity of the heart to alter its rate as response

¹⁷Halberg, F., and Folliers, C., "Variability of physiologic circadian crests in groups of children studied "transversely.", J. of Pediatrics 68:741-746, and published in Medical Arts and Sciences 24:3:45-64, 1970. Loma Linda University.

to noise.

The Effect Of Activity On Heart Rate

In the individual with a normal heart the rate can be shown to vary with exercise. Best and Taylor observed that man, at rest, exhibits a heart rate of approximately 81 beats a minute whereas that same subject performing heavy work (the degree being determined by treadmill testing) may increase his heart rate to 163 beats per minute.¹⁸ At the transition from rest to exercise pulse rate may increase in accordance with the workload being performed. The faster heart rate with heavy exercise is attributed to reflexes mediated through the carotid sinus and baroreceptors.¹⁹ The reduced arterial blood pressure which triggers the baroreceptor-carotid response results from vasodilatation in the active, working muscle. Such activities as eating, getting upon the bedside commode, sitting upright in bed, bathing, or ambulation, normally cause a reflex increase in heart rate which is slight. However, the majority of patients with cardiovascular disease, especially myocardial infarction are restricted to absolute bed rest. In many cases, the patient is not allowed to feed

¹⁸Best and Taylor, Chapter 3. Op. Cit. pp 1-12.

¹⁹The carotid and aortic bodies are specialized groups of cells, located in the bifurcation of the carotid artery, and in the aortic arch of the ascending aorta. Their specialized functions include sensing minute changes in the pressure of blood flow as it passes through the aorta, also in changing ratios of carbon dioxide/oxygen in the circulating blood. Corday, E., and Irving, D., Disturbances of the Heart Rate, Rhythm and Conduction, Philadelphia, W.B. Saunders Company, 1969. pp 17-20.

or toilet himself, or even turn from side to side without the assistance of the nurse. When the cardiovascular patient is engaged in such activity, transient increases in basal heart rate are expected. In addition, small meals of liquids, or soft foods are provided in order to minimize the energy expenditure (workload) of the heart.

The present study will control rigidly for activity by collecting data when the patient is not engaged in any activity. He must be at rest, or sleeping when both basal and noise responses are monitored.

The Effect Of Pharmacological Agents On Heart Rate

Of far greater significance to heart rate are the effects of rate-altering agents. There is a profuse literature on the subject, so that of necessity this discussion will cover only those agents most commonly administered to patients with cardiovascular disease.

Quinidine and pronestyl hydrochloride are two drugs whose effects on the electrocardiogram are well documented. Generally the desired actions are slowing of the heart rate or suppression of ectopic impulses.²⁰ This means that an electrical impulse which arises from any site other than the sinus node (normal pacemaker for the heart) is an abnormal finding. That focus can be obliterated with quinidine or pronestyl. A regular sinus rhythm is often reestablished after the administration of quinidine. Pronestyl hydrochloride

²⁰Ayers, S., and Gregory, J., Op. Cit. Page 278.

exhibits similar effects on the heart except that hypertension (low blood pressure) is a greater risk with the latter drug.

Lidocaine (xylocaine hydrochloride) is perhaps the most widely used of the anti-arrhythmic agents. Lidocaine has been shown to exert little effect on the velocity of electrical conduction between the atria and the ventricles.²¹ The prime action of lidocaine is suppression of irritable foci in the ventricular muscle.²² Thus far there is no evidence that the ability of the myocardium to react to neurogenic or hormonal influences is compromised with the use of this drug. The heart can be expected to retain the capacity to alter its rate as a response to stimuli.

Rosen et. al. investigated the effects of diphenylhydantoin (Dilantin) on the heart rate of man and concluded that it is a potent rate altering drug.^{23,24} Dilantin has the propensity to decrease the rate of firing of the sinus node,

²¹Rosen, R., Lau, S., Weiss, M., and Damato, A. "The effect of lidocaine on atrioventricular and intraventricular conduction in man," Amer. J. Cardiol. 25:1, 1970.

²²When inadequate amounts of oxygen are being delivered to the myocardial cells irritability results and, in the ventricles in particular, ectopic foci arise. Thus the impulses initiated by the ectopic ventricular cells arise earlier in the cardiac cycle than do those of the sinus node and the heart rate is controlled by the ventricle. Cardiac output is altered.

²³Rosen, M., Lisak, R., and Rubin, I., "Diphenylhydantoin in cardiac arrhythmias," Amer. J. Cardiol. 20:674, 1967.

²⁴--Editorial, "Cardiac Arrhythmias and Sodium Diphenylhydantoin," JAMA 201:2:128, 1961.

which is to say, to decrease the heart rate. In fact, sinus bradycardia is a calculated risk with the administration of Dilantin. There is reason to believe that the capacity of the heart to respond to sympathetic nervous stimulation is somewhat compromised by the use of this agent in that cardiac and/or sinus arrest is a risk of administering Dilantin intravenously.

Propranolol (Inderal), another rate altering agent, has been observed to decrease the rate of discharge of the sinus node in selected patients exhibiting a fast heart rate. Sinus tachycardia, and digitalis induced arrhythmias are the principle indications for its use.²⁵ Whether propranolol significantly compromises the capacity of the myocardium to respond to sympathetic stimuli is not known, however its recent popularity as an agent to reduce heart rate in patients with angina (transient chest pain) suggests that there may be some diminution of this capacity.

The cardiotonics (digitalis group) are well known for their inotropic and chronotropic effects on heart muscle.²⁶ Digitalis will slow the heart rate in persons with normal hearts as well as those who have cardiovascular disease. Burch and Winsor stated that sinus bradycardia is an important

²⁵Turner, J.R.B., "Propranolol in the treatment of digitalis-induced and digitalis-resistant tachycardias," Amer. J. Cardiol. 18:450, 1966.

²⁶An inotropic agent enhances the strength of contraction. Chronotropic, refers to rate. A drug with chronotropic properties would be expected to alter the heart rate.

side effect of digitalis administration.²⁷

Pain medications such as morphine sulfate and meperidine hydrochloride (Demerol) are not associated with changes in heart rate, unless hypotension is a side effect of the medication. In any event when these drugs are administered in therapeutic doses it is not expected that the capacity of the myocardium to alter its rate would be changed.

The tranquilizer drugs exert their main physiological effects on the sensorium and any change in heart rate would occur secondary to a sudden drop in blood pressure. Although chlordiazepoxide hydrochloride (Librium), and diazepam (Valium), as well as hydroxine hydrochloride (Vistaril) are routinely administered to cardiovascular patients, the doses are minimal. Most of the patients admitted to a coronary care unit will be maintained on one or more of these agents.

Vasodilators form another category of cardiac drugs in common use. While Lesch and Gorlin have shown that the vasodilator group has no direct effect on heart rate, it is common for a patient to respond to their use with a brief reflex tachycardia.²⁸ A slow heart rate due to increased venous return is a less common sequelae.

Many cardiovascular patients are maintained on a combination of two or more of the drugs that have been discussed.

²⁷Burch, G., and Winsor, T., A Primer of Electrocardiography 6th Ed. Philadelphia, Lea & Febiger, 1972 pp 61-62.

²⁸Lesch, M., and Gorlin, R., "Pharmacological Therapy of Angina Pectoris," Mod. Concepts Cardiovasc. Dis. Vol. XLII No. 2:5-10 February, 1973.

The effects of an agent, when used alone, are well documented, however when a complex of pharmacological agents are administered together, the physiologic action of any one of them is obscured.

The present study acknowledges the problems inherent in attempting to isolate a noise effect from the contribution made by the various groups of drugs. This study does not control for this factor; except to the extent that subclassifications of patients on specific medications will be analyzed. This posture is due, in part, to the investigator's interest in the heart and its rate, not as a stable quality, but in the capacity and the propensity of the myocardium to react to noise stimuli. No other study, known to this investigator, has demonstrated that heart rate changes in persons with cardiovascular disease may be dependent, at least in part, upon the noise load imposed upon the patient. Nor has any other study controlled for as many naturally occurring variables in the field setting as is proposed here.

Specificity Of The Population

Finally, a weakness of the study may lie in the particular population to be studied. Two private hospitals and their coronary care units will be utilized for data collection. The socioeconomic status is expected to be middle class with a majority of the subjects, Caucasian. Data generated from a larger population, and reflecting a more diverse spread of ethnic and class parameters, might result in results significantly different than the present study.

DEFINITION OF TERMS

Ambient Noise: The background noise, or prevailing noise in a general area, often in the absence of a noise of particular interest. For the purposes of this study "ambient noise" is the prevailing noise in the field setting, i.e. the coronary care units, as measured by a noise level meter.

Amplitude: The maximum value or the peak of a sound wave.

Band: A segment of the frequency spectrum, that is an octave, a half octave or a third octave.

Basal Heart Rate: The heart rate as measured at total rest. For the purpose of this study basal heart rate is measured in the absence of any activity (observable) in the subject.

Catecholamine: A compound of a catechol and an amine, which elicits the sympathetic nervous system response. Examples of catecholamines are epinephrine and nor epinephrine.

(Adrenalin and Nor Adrenalin)

Cardiotonic Drug: A drug that exhibits a tonic effect on the heart, that is, it strengthens myocardial contractions. Examples of cardiotonics are digitalis and its many derivatives.

Coronary Care Unit: A special place within the hospital where patients with myocardial infarctions, congestive heart failure and heart rhythm abnormalities are admitted. Surgical intensive care (SICU), Medical intensive care (MICU), or simply Intensive care (ICU) are not synonymous with the coronary care unit (CCU).

Compensatory Mechanisms: Complex feedback systems which alter one or more bodily systems to accommodate to a change in

another system. For example, a drop in blood pressure, resulting in a decrease in the amount of blood flowing past the baroreceptors; or an increase in the carbon dioxide level of the blood flowing past the various chemoreceptors will trigger the cardioaccelerator center to increase the heart rate.

Critically Ill: A state of health in which the patient's vital signs i.e. blood pressure, heart rate, temperature and respirations are unstable. Care cannot be planned in advance for more than a very short period of time and continuous observation is necessary.

Decibel: A means of denoting the ratio of two quantities when the range of possible values is very great. A Bel, of which the decibel is but one-tenth, can be described as the number of tenfold increases the lower (smaller) quantity must be given to equal the higher, for example $\log_{10} \frac{P_1}{P_2}$

in which one multiplies by ten to obtain decibels.

Frequency: The number of times a vibrating system completes a repetitive cycle. In sound, frequency is expressed as cycles per second and abbreviated Hz or Hertz.

Heart Rate: The number of times the heart contracts (beats) per minute. For the purposes of this study, heart rate will be calculated in its smallest quantitative measurement, the R to R interval. Electrocardiographically, the R wave is inscribed when the electrical impulse has traversed the ventricular myocardium. The mechanical event--contraction--

follows immediately. Thus each successive R to R interval represents rate and can be tabulated in milliseconds.

Hospital Day: A 24 hour day made up of three functional shifts: 7:00 A.M. to 3:00 P.M.; 3:00 P.M. to 11:00 P.M. and 11:00 P.M. to 7:00 A.M.

Intensity: The rate of energy flow per unit area expressed as a decibel ratio of watts/m². Note: The sound pressure in decibels is more or less numerically equal to the sound intensity level when sound pressure levels are read on a weighted A scale network on the noise meter. A weighting reduces the meter's response to very low and very high frequency sound in order to closely simulate the response of the human ear.

Level: The value of a quantity in decibels.

Loudness: The perceived intensity of a sound by the human ear.

Myocardium: The muscular portion of the heart.

Myocardial Infarction: Synonyms: heart attack, and coronary occlusion. Abbreviated as MI. For the purposes of this study the three classical diagnostic criteria will obtain, 1) electrocardiographic changes indicative of MI, 2) elevated serum enzyme levels and 3) intractable chest pain. Note: Where the evidence of MI is inconclusive or debated, the diagnosis as used by the attending physician will be used.

Noise: Sound that is unwanted and or exerts an untoward effect on the human body. See conceptual framework for a complete discussion.

Normal Hearing: As used for this study, normal hearing will denote that the subject can hear a normally voiced conversation in soft tones and further, that an examination of the chart shows him to have no hearing impairment.

Oxidative Process: Refers to metabolism and the usage of oxygen and nutrients by the myocardial cells. The increased need for and utilization of oxygen (oxygen uptake) by the heart is considered deleterious to the patient with a myocardial infarction.

Perceived Noise Level: Abbreviated as PNdB. The sound pressure level of between one-third of an octave and an octave of random noise at 1000 Hz which is considered by "normal" people to be equally noisy to the sound in question.

Pitch: An aural assessment of sounds so that they can be ranked on a scale from high to low. Pitch is primarily dependent upon frequency.

Post Infarction Day: The first, second or any subsequent day after the occurrence of an infarction. Patients are more prone to develop rhythm disturbances, or changes in heart rate in the first 48 to 72 hours after a heart attack.

Response, Heart Rate: The reaction of the heart, through increasing or decreasing its rate, to noise stimuli. When any two consecutive R to R intervals are shortened the heart has increased its rate, when two R to R intervals have lengthened, the heart has decreased its rate.

Rest, or Bed Rest: An ill-defined term often applied to forced restrictions on the activity of patients with heart attack. The intent is to reduce the workload of the heart by decreasing activity and mental strain to a minimum. Bed rest is the more specific term in that it denies the patient feeding, toileting, bathing or dressing himself.

Rural Residence: Outside the limits of a city. Country is used here as the absence of the city.

Site of Infarction: The specific anatomical area of the heart muscle that has been damaged by an infarction. For example, anterior, anterolateral, or inferior.

Sound Level Meter: An instrument designed to measure the frequency weighted value of sound pressure level. It converts pressure fluctuations into voltage fluctuations, which are then rectified and amplified. The voltmeter, which is calibrated in decibels measures, in most meters, the root mean square value of the sound rather than the peaks.

Vasoconstrictor Drugs: Drugs that bring about a vasoconstrictor response i.e. causing a narrowing of the peripheral blood vessels. Reaction of the sympathetic nervous system to certain stimuli elicits a vasoconstrictor response, as well.

Weighting Network: Circuits built into many sound level meters which can be utilized to reduce the response of the meter to very low or very high frequencies. In the notation of decibels and postscript, linear, A, B, or C should be used. For example, 60 dbA.

CHAPTER IV

THE RESULTS OF THE STUDY

The concept that noise is a problem to man has been put forth as the basis and a rationale for investigating the effects of noise upon the cardiovascular system. Heart rate is used as the cardiovascular response in this investigation. Patients admitted to the coronary care unit with diagnosed or suspected myocardial infarction are thought to comprise a most vulnerable population, although their resting and awake heart rates are similar to those found for persons with normal hearts.¹ Two coronary care units within a large metropolitan area were utilized to provide data for the study.

The major hypotheses for the field study are as follows:

- 1) Noise will affect heart rate in subjects with cardiovascular disease.
- 2) The heart rate response will be significantly greater in subjects with myocardial infarction than in those with other and chronic types of heart disease.

The minor hypotheses are:

- 1) The rate of the heart rate response will vary with the anatomical site of heart damage.
- 2) The degree of heart rate response is a function of the gap between ambient and peak noise levels experienced by

¹Friedman, M., Rosenmann, R., and Brown, A., "The continuous heart rate in men exhibiting an overt behavior pattern associated with increased evidence of clinical coronary artery disease," Circulation, 28:861-866, 1963.

each subject.

The population consisted of 38 patients who were admitted to the coronary care unit of one of two metropolitan hospitals between the dates of December 16, 1974 and February 1, 1975. The range of diagnoses was typical of patients admitted to coronary care units generally, and included congestive heart failure, chest pain, angina, and myocardial infarction.² In the cases of suspected heart attack, chest pain was the most commonly found admitting diagnosis, followed by shortness of breath, dizziness and nausea. Patients whose final diagnosis was myocardial infarction were characterized by elevated enzyme levels,³ electrocardiographic changes,⁴ or both. The following table depicts the age and sex distribution of the total population.

²Chest pain may be a tentative diagnosis when it is uncertain whether the origin is the heart and coronary circulation. For example, influenza, pleurisy, inflammation, and many other conditions can cause chest pain which is unrelated to CAD. Angina is a term used to describe chest pain which is known to result from inadequate coronary artery circulation. Myocardial infarction, is a closed diagnosis made after electrocardiographic and laboratory evidence is positive for necrosis of the heart tissue.

³Serum enzyme levels refer to certain compounds that are released into the circulating blood when tissue cells such as those in the myocardium have been destroyed. These include serum oxaloacetic glutamic acid, creatinine phosphokinase, and lactic dehydrogenase, as well as several less well documented ones.

⁴Electrocardiographic changes include at a minimum, a current of injury pattern (noted by an elevated ST segment) and a Q wave of diagnostic significance.

TABLE VI
AGE AND SEX DISTRIBUTION OF THE
STUDY POPULATION
N = 38

Males	33-80	59.3	67	19
Females	54-70	63.2	65	19
Totals	33-80	61.2	68	38

Twenty of the patients were admitted to Unit B, a unit which has open ward-type accommodations. Four beds separated by approximately two feet between each, house either male or female patients. Privacy is provided by means of draw curtains which are strung on rods reaching to within a few inches of the ceiling. A central nursing station with slave monitors and a utility room with a flush hopper complete the unit. Unless the curtains are drawn between beds, each patient is in the immediate sight and hearing of all other patients in the unit. A heavy door separates Unit B from the adjoining corridor and a large medical ward. Just outside this door are benches for the use of friends and families of the patients.

During the time of data collection from Unit B, thirteen men and seven women were admitted for various heart ailments. Eleven of those admitted were subsequently diagnosed as having had a heart attack. Since the ambient noise

levels were higher for every day and night sampling period in Unit B, it is of interest to note whether a differential response to noise could be obtained across the two study units. The following table shows the ambient levels of noise for night and day over a 24 hour period in each of the units.

TABLE VII
 AMBIENT LEVELS OF NOISE IN DECIBELS A
 OVER TIME FOR TWO CORONARY CARE
 UNITS

Unit	7:00 A.M.-7:00 P.M.	7:00 P.M.-7:00 A.M.
A	53	50
B	62	54
Recommended	45	40

Figure 3, page 113, is a graphic representation of this data on an hour by hour basis. Only for two samplings does the ambient level on noise in Unit A exceed that for Unit B. (11:00 P.M. and 6:00 A.M.). In addition, Unit B was characterized by wall suction apparatus which measured 78 to 82 dbA when in use, and beds which required cranking to be raised or lowered. This latter procedure resulted in noise levels well in excess of 70 dbA. Preparation for the morning bath, specifically arranging the metal basins on the side stand, elicited noise levels ranging from 68 to 72 decibels even when the pans were not being clanged together. However, the noisiest procedure that was practiced routinely in Unit B,

was the frequent emptying of soiled laundry into large paper bags--the latter being located at the foot of each patient's bed. Noise levels greater than 80 dbA were elicited from several points in the Unit when the bags were opened and laundry placed within. Other procedures and their noise emissions are listed in Table 8. It should be noted that all but one of these exceed the acceptable and recommended noise limits, as cited previously.

TABLE VIII
NOISE LEVELS ELICITED BY ACTIVITIES
AND PROCEDURES COMMONLY CARRIED
OUT IN UNIT B

Source of Noise	Range in decibels A
Water running	54-60
Hopper being flushed	60-60
Heart rate alarm	41-42
Bed rails up or down	62-66
Curtains being pulled	60-64
Telephone muted but ringing	45-49
Inhalation therapist checking his equipment	53-67
Visitors at the desk waiting	52-58

Unit A has all electric beds, however a manipulation of bed level elicited noise levels ranging from 62 to 64 decibels and raising or lowering bed rails, 71 to 74 dbA. Sixty-three to 70 decibels of noise occurred when the technicians were

adjusting electrocardiographic leads on the patient. The only additional apparatus that measured above the threshold for peripheral vasoconstriction (70 dbA) was the pressure alarm associated with the positive pressure breathing machine (Bennett MA₁). Since a life very often depends upon the nurse's reaction to the pressure alarm, the noise is very loud and irritating. Band width data were not generated from this study, however such information would perhaps, explain the high annoyance value assigned the ringing of the pressure alarm. Table 9 lists the noise level measurements of certain other equipment and procedures that are carried out regularly in Unit A.

TABLE IX
NOISE LEVELS ELICITED BY ACTIVITIES
AND PROCEDURES COMMONLY CARRIED
OUT IN UNIT A

Source of Noise	Range in decibels A
Water running	55-56
Hopper being flushed	64-68
Heart rate alarm (from desk)	45-58
Wall suction being used	60-62
Respirator cycling	53-54
Pharmacy cart being emptied	66-66
Nurse at bedside	62-65
Visitors at desk waiting	57-70

Generally higher ambient levels of noise were obtained

in Unit B for all but two periods of the day, and, in addition, equipment and procedures were noisier. The question arises then, of whether a differential heart rate response could be obtained between patients in these units. However, an examination of the data suggests that any differences that may exist between patient responses in Unit A or B may be masked by the intervening variables of sex, age, and type of MI. There were marked differences in the distribution of the latter three variables between Units A and B.

Thirteen males, (69 per cent) of the total population, were admitted to Unit B, while only 7, or 38 per cent of the females were admitted to that Unit. Moreover, the type of myocardial infarction differed between the two units. Tables 10 and 11 show these distributions.

TABLE X
AGE AND SEX DISTRIBUTION OF PATIENTS
ADMITTED TO TWO CORONARY CARE
UNITS

Unit	Males	Range	Females	Range
A	6	44-68	12	50-74
B	13	33-80	7	51-73
Totals	19	33-80	19	50-74

TABLE XI

DISTRIBUTION OF PATIENTS WITH ANTERIOR
AND INFERIOR MYOCARDIAL INFARCTION
BETWEEN TWO CORONARY CARE UNITS

Unit	Inferior	Anterior	Totals
A	1	6	7
B	8	3	11
Totals	9	9	18

The major hypothesis states that noise will affect the heart rate in persons with cardiovascular disease. Figure 4 shows the heart rate at low noise and under high noise conditions of the study population. N=37.

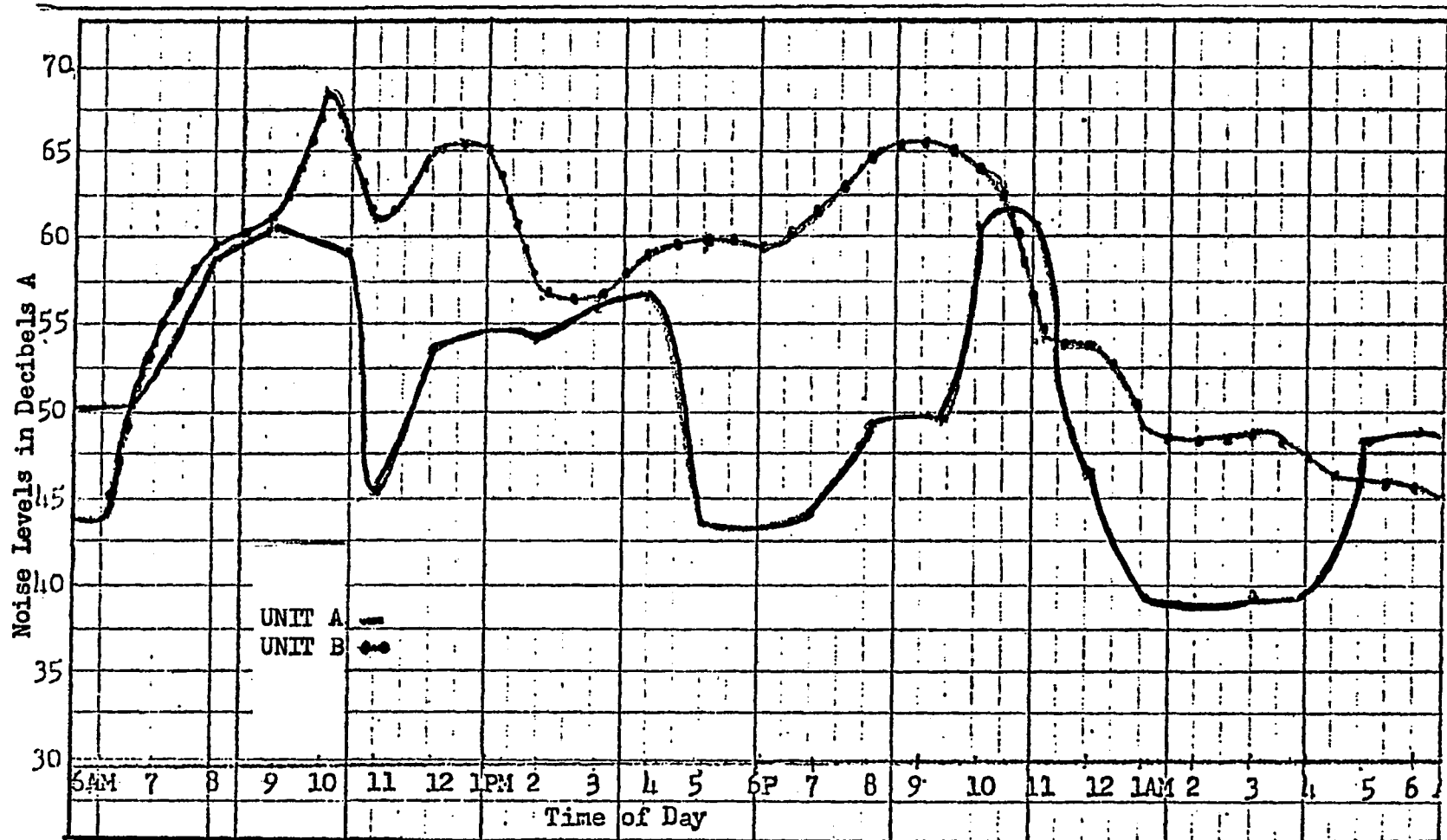


Figure 3. A graphic representation of the mean noise level for each one-hour period during a typical 24-hour day in Unit A or Unit B

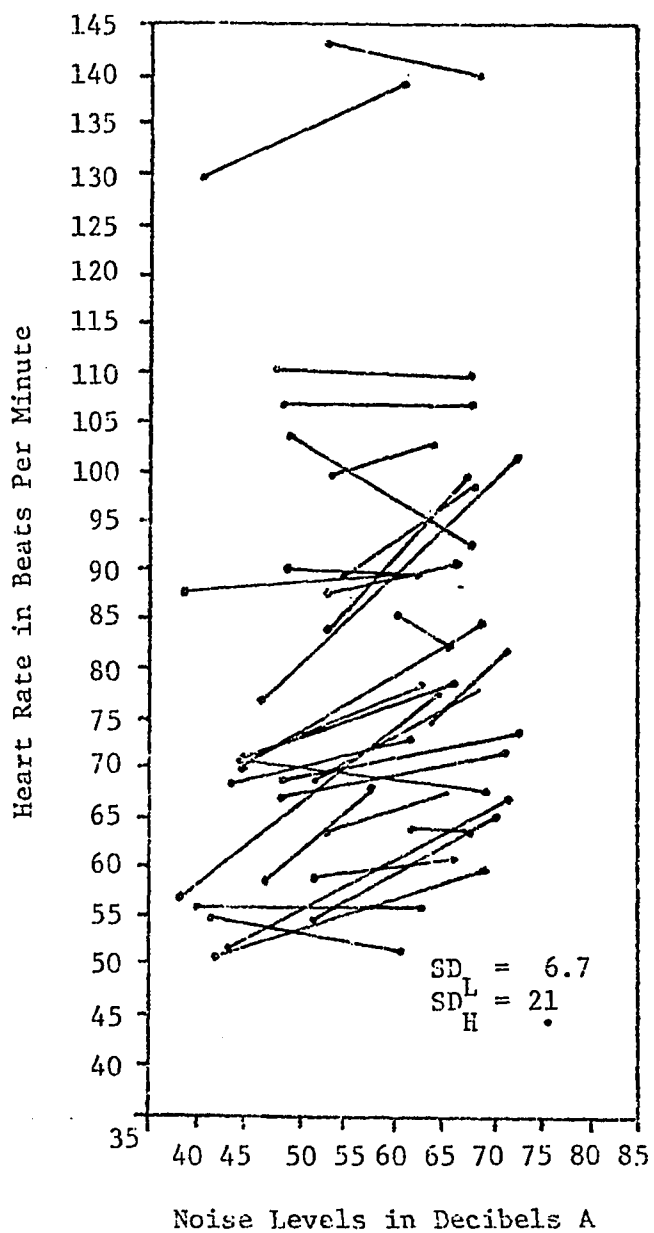


Figure 4. Mean heart rates related to noise levels in 37 subjects with cardiovascular disease

$$t \text{ (df=36) } = 5$$

$$\underline{P} = <.001.$$

TABLE XII

AVERAGE PULSE UNDER LOW AND HIGH NOISE CONDITIONS: THE
DIFFERENCE BETWEEN THESE MEANS, AND THE PER CENT
OF CHANGE IN 37 SUBJECTS HOSPITALIZED IN TWO
CORONARY CARE UNITS

Subject	Pulse at Noise L	Pulse at Noise H	Change	Per cent of Change
1	55	57	2	3.6
2	75	82	7	9
3	58	68	10	17
4	100	103	3	3
5	69	74	5	6
6	90	90	0	0
7	86	83	-3	3.5
8	84	100	16	19
9	55	52	-3	5.4
10	96	96	0	0
11	71	70	-1	1
12	107	107	0	0
13	79	79	0	0
14	73	79	6	8
15	111	110	-1	.9
16	59	61	2	3.4
17	56	56	0	0
18	64	68	4	6
19	64	64	0	0
20	68	73	5	8
21	51	60	9	18
22	89	99	10	11
23	88	91	3	3.4
24	56	56	0	0
25	71	79	8	11
26	55	65	10	18
27	88	90	2	3.4
28	52	67	15	29
29	57	79	22	40
30	70	85	15	21.5
31	130	140	10	8
32	104	93	-11	10.5
33	71	68	-3	4
34	69	78	9	13
35	77	102	25	32.5
36	144	141	-3	2
37	67	72	5	7.4
X	77	82	= or -	6
SD	6.7	21		

The heart rate changes as demonstrated by the study population were greater than could be expected by chance ($P = <.001$) thus the hypothesis that noise will affect heart rate in subjects with cardiovascular disease was not rejected.

Although a hypothesis regarding a differential response to noise according to sex was not included in the study design, it became apparent during the analysis of the data, that such a difference may exist as an extraneous finding. It is a fact that females react differently to electrocardiographic stress tresting than do males, in that the number of false positives for heart disease is significantly greater for females.

Eighteen males aged 33 through 80 years, were admitted to the two coronary care units during the period of data collection. Figure 5 shows the heart rate response to specified noise levels of this group, subject by subject.

Nineteen females, aged 54 through 74 years, were admitted to Unit A or B during the six week period of data collection. Figure 6 depicts the heart rate data for this group.

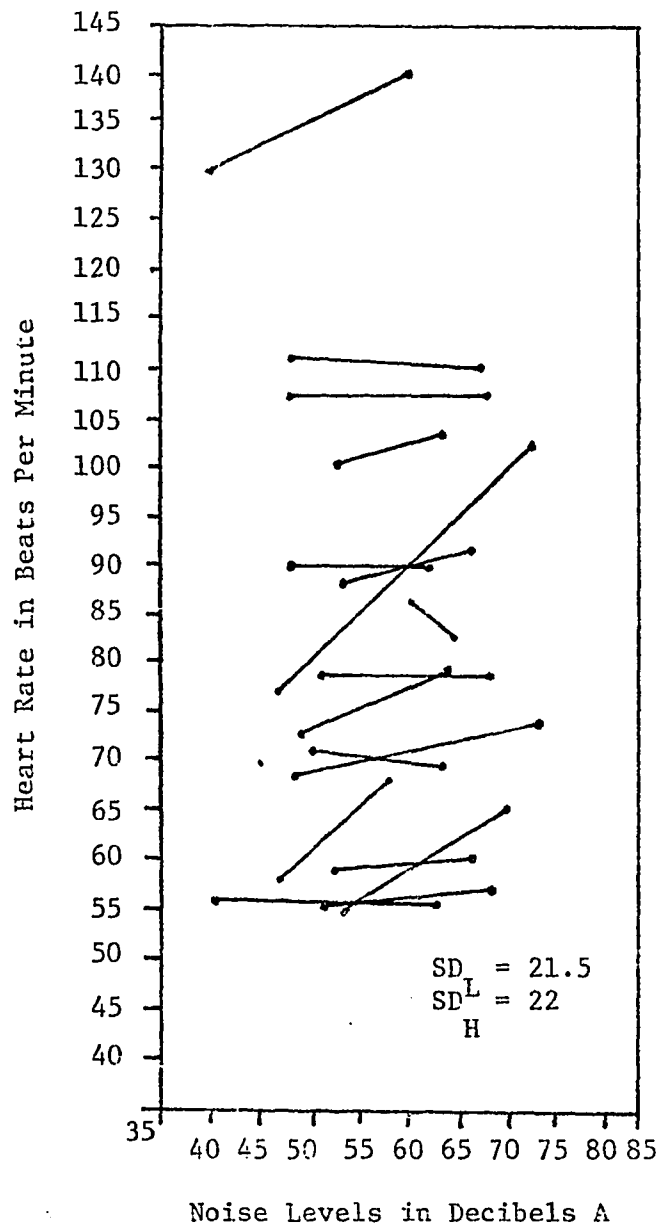


Figure 5. Mean heart rates related to noise levels in 18 males of all ages admitted to Unit A or Unit B

$t=2.63$ $p<.02$

$df=17$

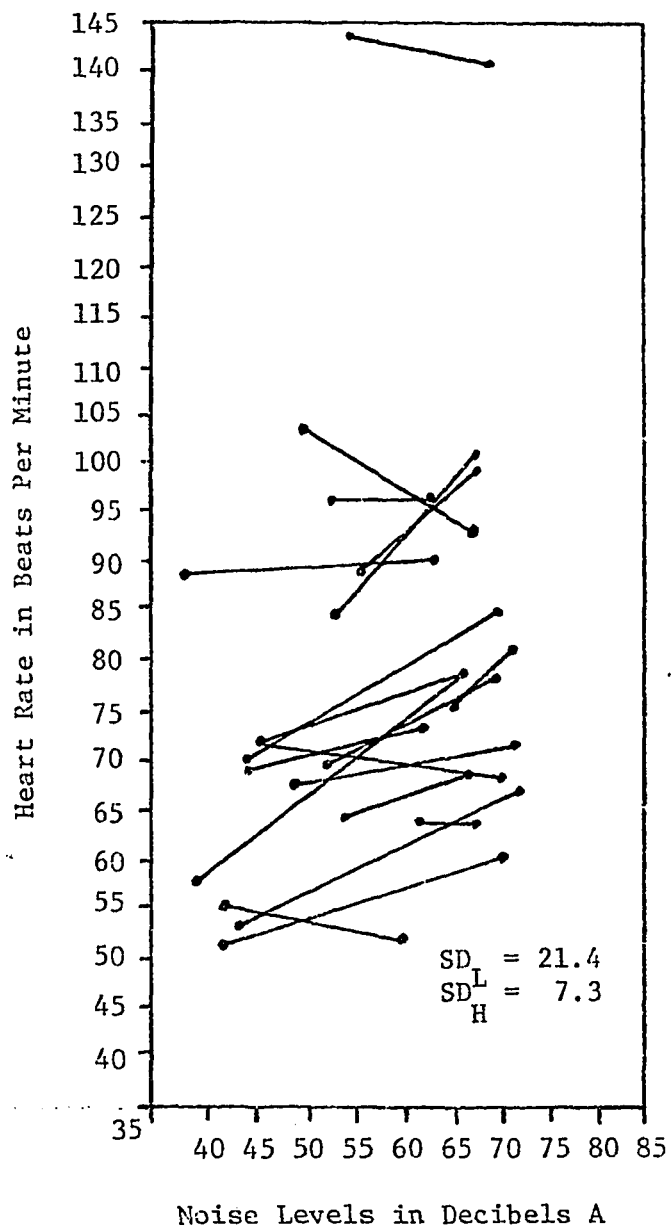


Figure 6. Mean heart rates related to noise levels in 19 females admitted to Units and B

$$t (df=18) = 3.70$$

$$P = <.01$$

While a significant response was obtained for the males in the population (N=18), the significance may have been influenced by the intensity of the response in but a few subjects. The fact that five of the 18 males did not demonstrate any heart rate change further supports the above statement. Figure 7 compares the data for males with that of the female subgroup.

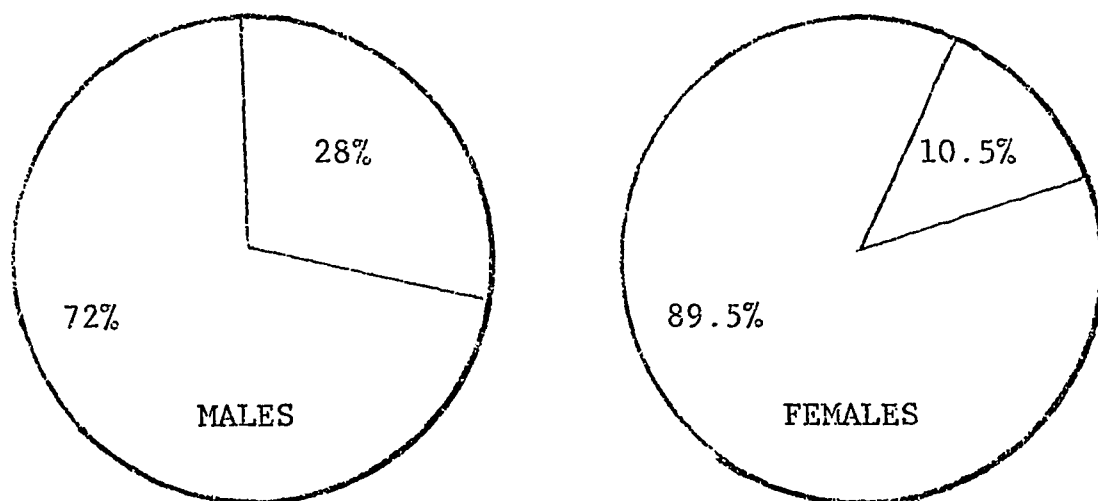


Figure 7. A comparison in per cent of the heart rate change in 18 males and 19 females.

The results for the male group were not significant by the sign test, but the Student's t gave a significant level .02.

The female population (N=19), demonstrated a greater rate of heart rate response and those responses were of greater intensity. The female population appeared to be less influenced by scores at the tails, and the standard deviations both for low and high noise response were less than for the male group. The HRR of the females was significant by the sign test ($\underline{P} = < .01$) as well as by the t, ($\underline{P} = < .01$).

TABLE XIII
 HEART RATE RESPONSE DIFFERENTIAL
 ACCORDING TO SEX IN 37 SUBJECTS
 ADMITTED TO TWO CORONARY
 CARE UNITS

Sex	Positive HRR	Negative HRR	Totals
Male	13	5	18
Female	17	2	19
Totals	30	7	37

$$\chi^2 = 2.26$$

$$df = 1$$

$$P = <.20 - >.10$$

The probability for a Chi square of 2.26 occurring by chance is more than 10 times in one hundred but less than twenty. Therefore no conclusions can be drawn from these findings. It is suggested, however, that a difference does exist between the heart rate response to noise--an assumption that requires a larger sample for verification.

Further analysis of the HRR data for males indicates that as N is reduced, for example, in the desegregation of the data, the statistical significance of the response decreases. The HRR of males admitted to Unit A (N=5), was not significant at any level of confidence. The following figure shows the heart rate response to noise conditions for 13 males admitted to Unit B.

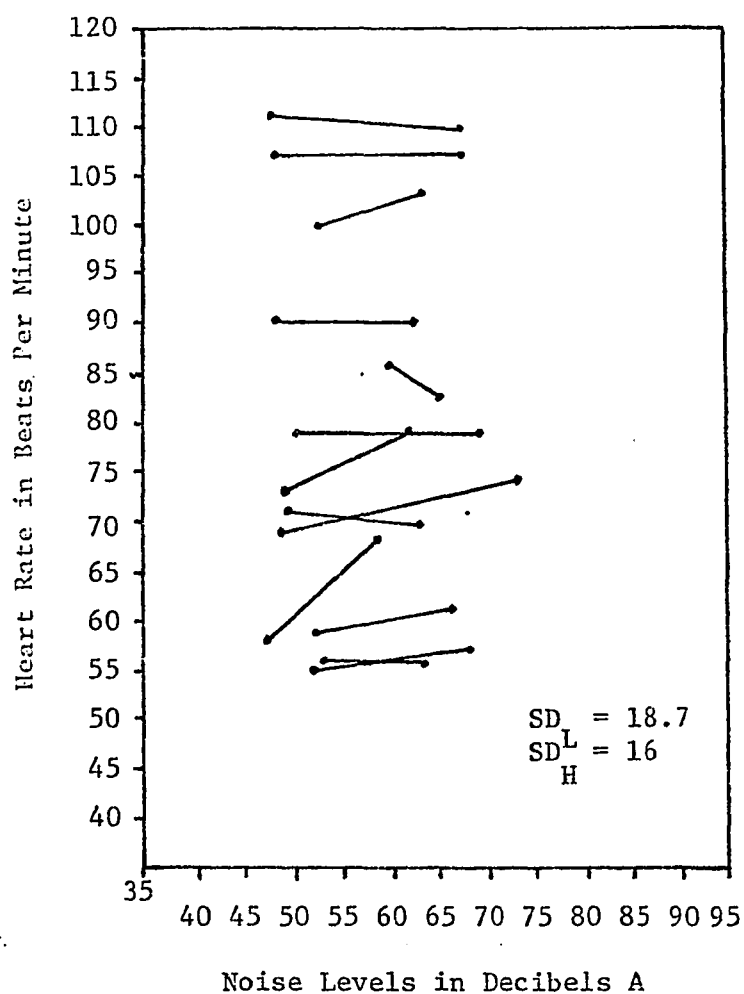


Figure 8. Mean heart rates related to noise levels in 13 males admitted to Unit B

$$t \text{ (df=12)} = 2.50 \quad \underline{P} = <.05$$

It is suggested that the significance of the HRR in males is dependent, to some extent, upon the scores at the extremes, as well as the size of N. Variance in age, compared to the females, did not appear to contribute to the lesser significance of the heart rate response to noise by males.

The female population (N=19), was desegregated according to unit and age. Figures 9 and 10 depict this data. The

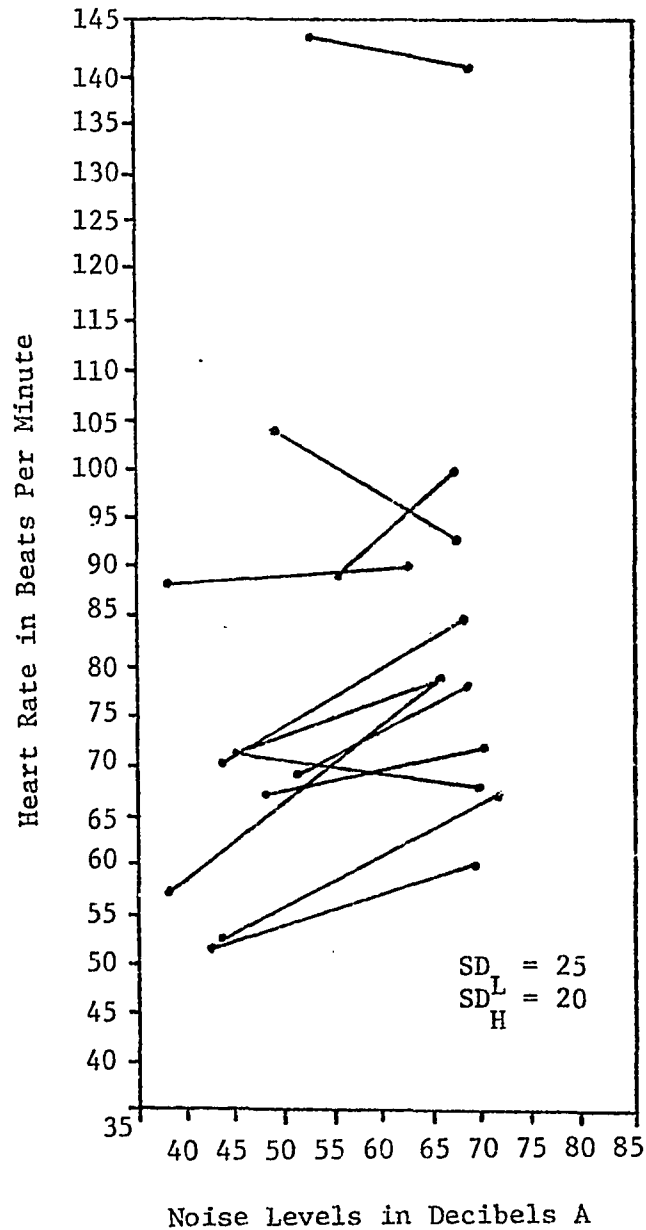


Figure 9. Mean heart rates related to noise levels in 12 females admitted to Unit A

$$t \text{ (df=11)} = 2.14 \quad \underline{P} = >.05 - <.10$$

greater significance in the younger age bracket (less than 60 years) suggests that age-relatedness may be a valid factor to be considered in the heart rate response to noise by females. It should be pointed out that an N of 4 in Unit A

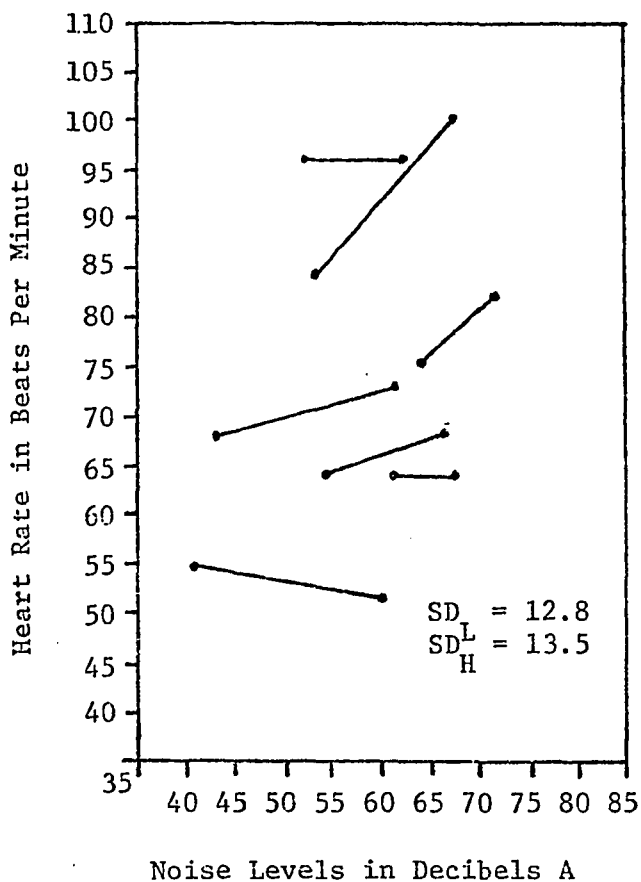


Figure 10. Mean heart rates related to noise levels in 7 females admitted to Unit B

$$t (df=6)=2.96 \quad \underline{P} = <.05$$

and an N of 3 in Unit B are too small of a sampling to carry out an analysis for each unit.

For females, 60 or more years of age, significant heart rate responses were obtained for a population of eight who were patients in Unit A. Only 4 females, aged 60 or more years were admitted to Unit B during the entire data collection period, and one of these displayed a continuous tachycardia of 107 beats per minute. The t test was non-significant for this latter group. Figure 11 shows the heart rate

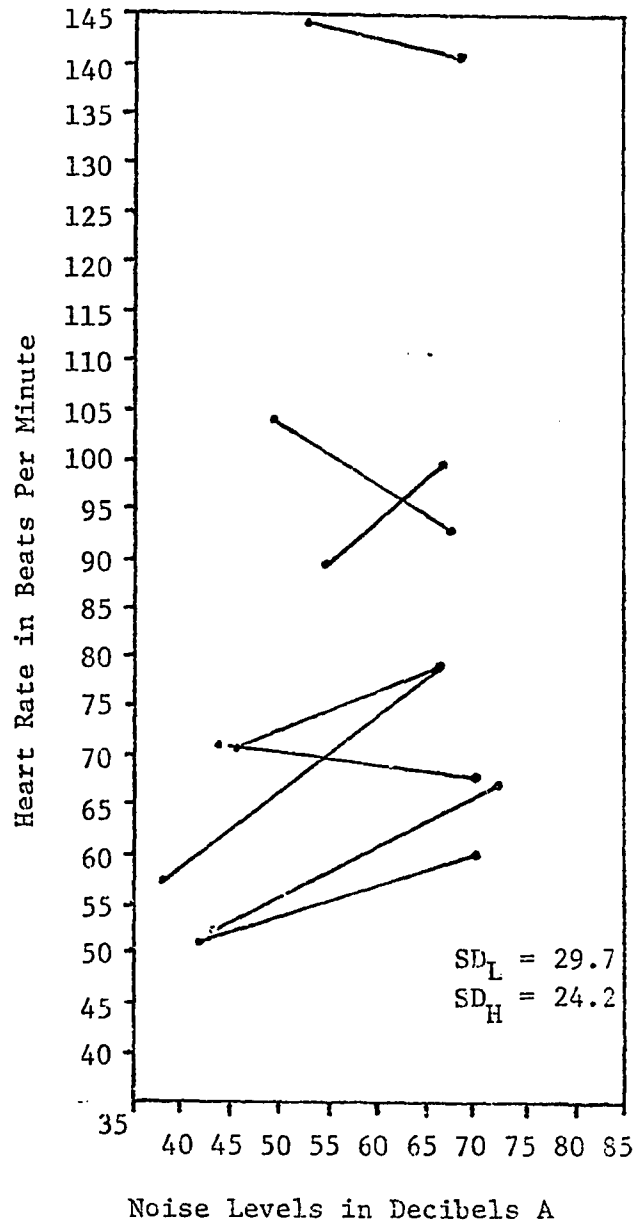


Figure 11. Mean heart rates related to noise levels in 8 females, 60 years of age or older and admitted to Unit A

$$t (df=7) = 2.73 \quad \underline{P} = <.01$$

response to noise for 8 females in the older age group who were admitted to Unit A.

The second major hypothesis states that the heart rate response will be significantly greater in subjects with

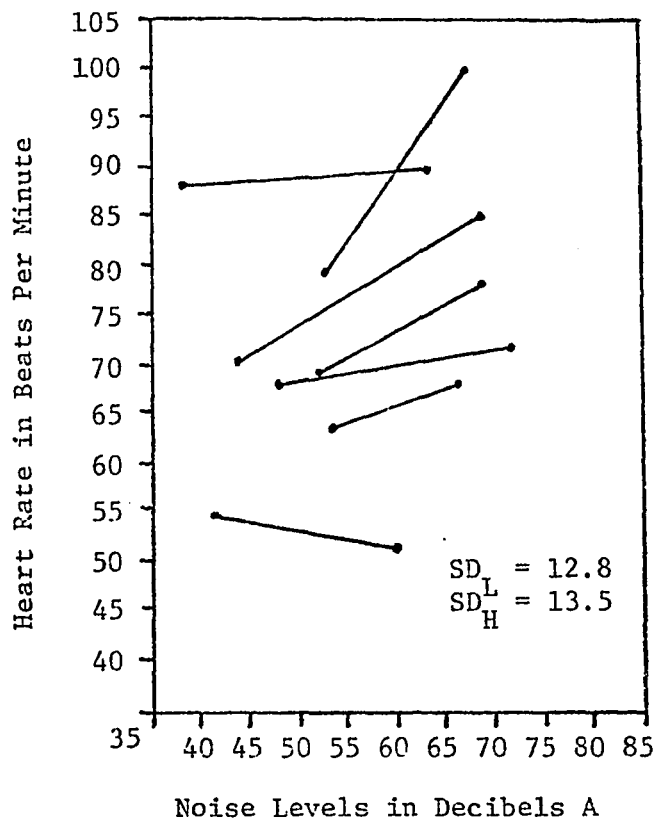


Figure 12. Mean heart rates related to noise levels in 7 females, less than 60 years of age and admitted to Units A or B

$$t \text{ (df=6)} = 2.96 \quad \underline{P} = <.05$$

myocardial infarction than in participants with other and chronic types of heart disease. Figure 13 depicts graphically the heart rate response to noise of the population admitted to the CCU with cardiovascular disease, but subsequently diagnosed as not having experienced a heart attack.

Figure 14 shows the same data for 18 subjects whose final diagnosis was myocardial infarction.

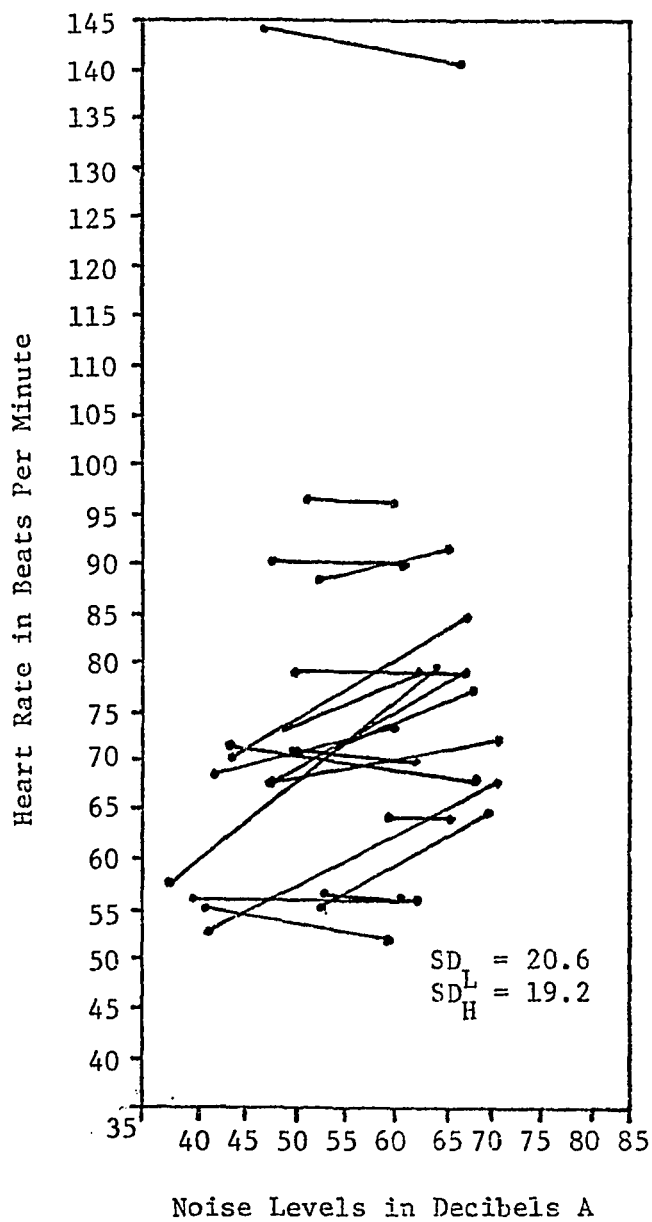


Figure 13. Mean heart rates related to noise levels in 19 subjects, both sexes, all ages, admitted to Units A or B but without a myocardial infarction

$$t (df=18) = 2.86 \quad \underline{P} = < .02$$

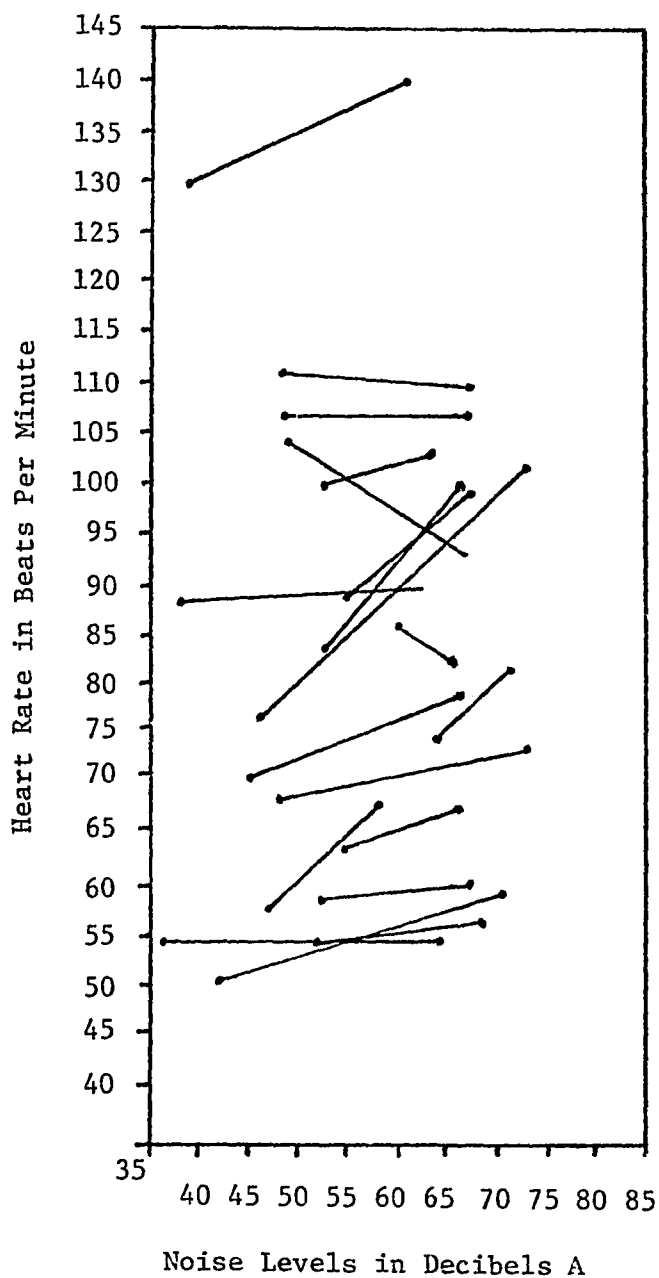


Figure 14. Mean heart rates related to noise levels in 18 subjects admitted to Units A or B with a diagnosis of myocardial infarction

$$t \text{ (df=17)} = 3.91 \quad \underline{P} = <.01$$

$$\begin{aligned} SD_L &= 23.6 \\ SD_H &= 20.3 \\ SD_L &= 17.8 \\ SD_H &= 14.6 \end{aligned}$$

Six, or 32 per cent of the population without MI, had no heart rate response to noise, while only one subject in the group with myocardial infarction (N=18) did not demonstrate a heart rate response to noise. This comparison is depicted in the following figure.

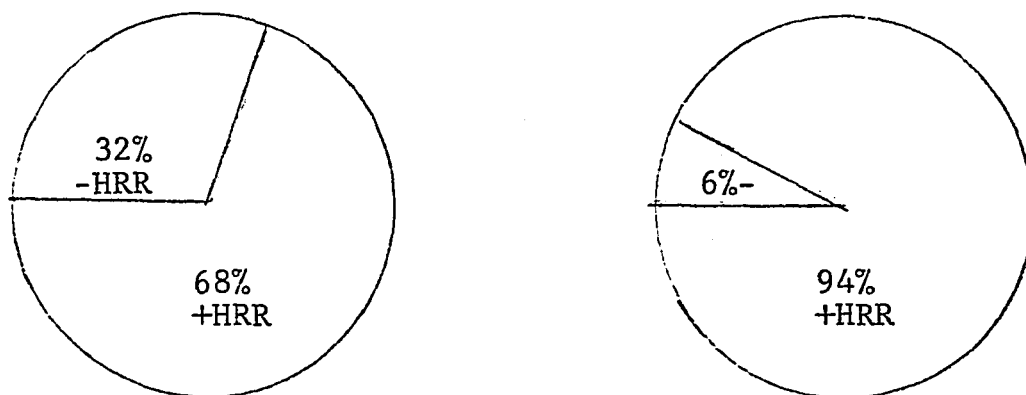


Figure 15. A comparison of the heart rate response to noise of 19 patients without and 18 patients with a myocardial infarction.

The power of significance differs with the cardiovascular disease, the non MI group having a $P = .02$ for a t ($df=18$) $= 2.86$, and the subjects with a positive diagnosis for myocardial infarction attaining a $P < .01$ for a t ($df=17$) $= 3.91$. A chi square of 4.06, P is equal to .05 shows that such a difference is not likely to have occurred by chance. However, analysis with a more powerful tool, the t test for independent means, resulted in a significance level of slightly greater than .05 but less than .10. It is suggested that the difference between the levels of significance obtained with these two methods would likely be reduced if the N were increased to twice its present size. It appears that the second hypothesis: that is that the heart rate response

between patients with MI and those with other types of heart disease will not differ except by chance can be rejected.

TABLE XIV

CHI SQUARE APPLIED TO HEART RATE RESPONSE
TO NOISE FOR 18 SUBJECTS WITH, AND 19
SUBJECTS WITHOUT MYOCARDIAL INFARCTION

Subgroup	Positive HRR	Negative HRR	Totals
Infarction	17	1	18
Non Infarction	13	6	19
Totals	30	7	37

$$\chi^2=4.06$$

$$df = 1$$

$$\underline{p} = .05$$

Desegregation of the data for the population without myocardial infarction reveals some groups with significant responses to noise conditions. Figures 16 and 17 depict this data graphically.

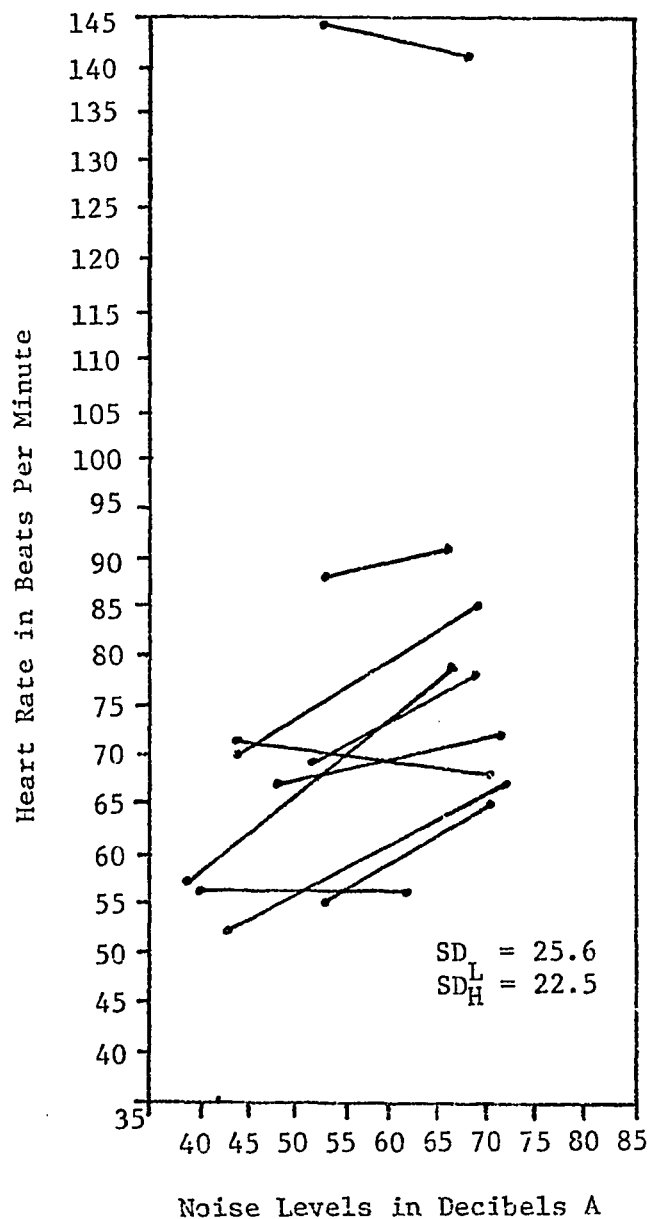


Figure 16. Mean heart rates related to noise levels in ten subjects with cardiovascular disease, without myocardial infarction, admitted to Unit A

$$t \text{ (df=9)} = 3.18 \quad \underline{P} = <.02$$

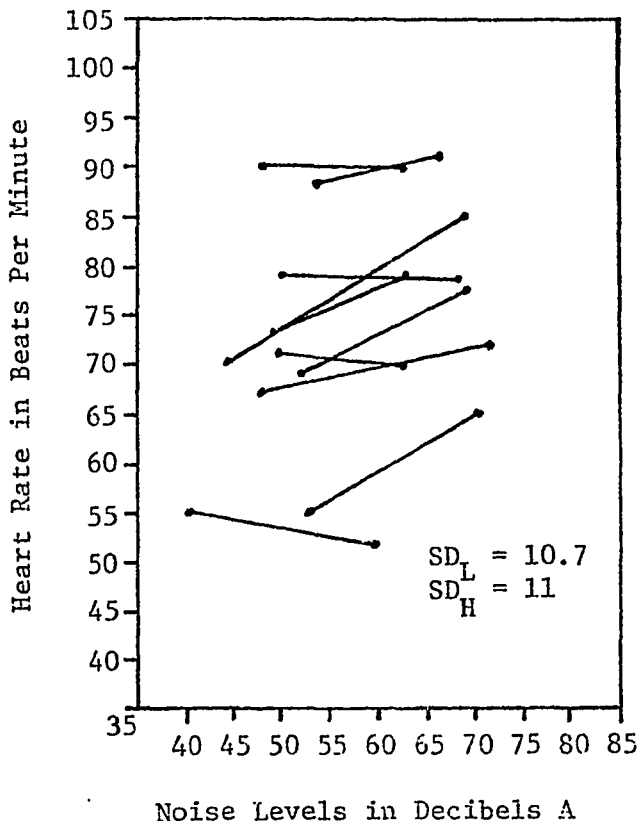


Figure 17. Mean heart rates related to noise levels in 10 subjects with cardiovascular disease, without myocardial infarction and less than 60 years of age

$$t (df=9) = 2.75 \quad \underline{p} = <.05$$

The third, a minor hypothesis states that there will be a differential response to noise according to the site of the myocardial infarction. Stated in different terms, the heart rate response to noise will vary depending upon the anatomical site of the heart damage, that is between patients with inferior or anterolateral wall damage. Figure 18 shows the HRR to noise for nine subjects admitted to Unit A or B with a positive diagnosis of inferior or inferolateral infarction.

Desegregating the inferior infarction group by age, it is noted that those subjects 60 years of age or older all experienced a positive heart rate response to noise. The group less than 60 years of age (N=4) also showed a response to noise, however the N is considered too small to obtain a significant t. Figure 19 shows this data for comparison.

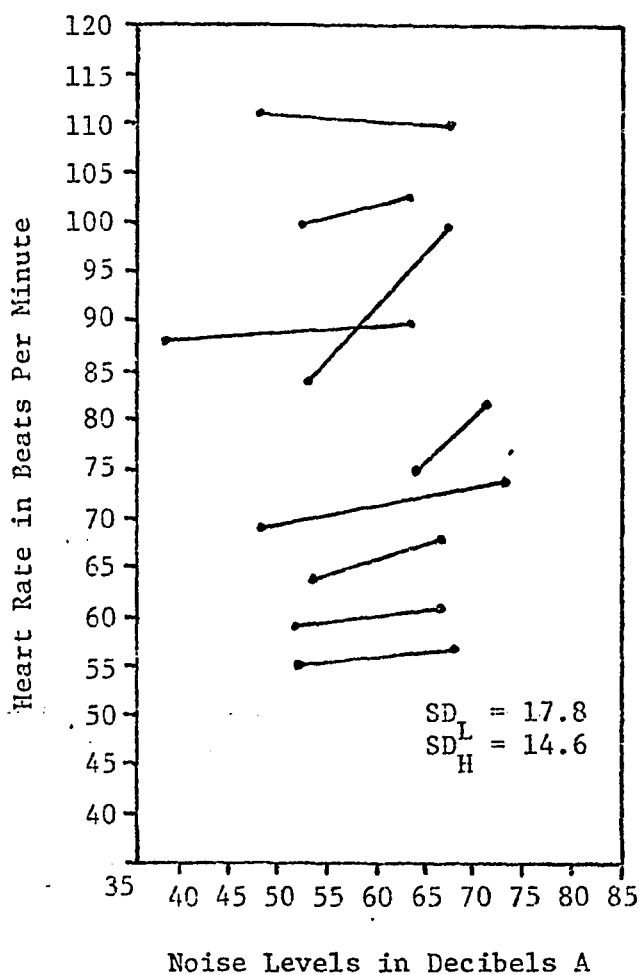


Figure 18. Mean heart rates related to noise levels in 9 subjects admitted to Unit A or B with a diagnosis of myocardial infarction

$$t (df=8) = 3.27 \quad \underline{P} = < .02$$

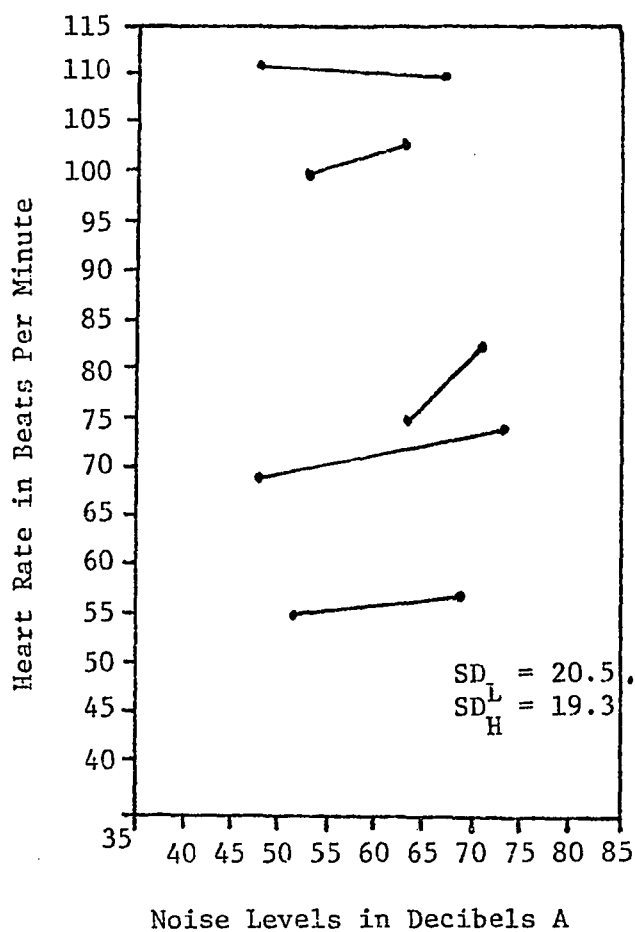


Figure 19. Mean heart rates related to noise levels in 5 subjects admitted to Units A or B with a diagnosis of myocardial infarction and 60 or more years of age

$$t (df=4) = 2.96 \quad \underline{P} = < .05$$

The group with a positive diagnosis of anterior or anterolateral infarctions demonstrated significant heart rate responses to noise. The age-related difference was most notable in persons with anterior infarctions and 60 or more years of age. Figure 20 and 21 depicts this data for nine subjects with a diagnosis of anterior infarction, seven of whom are more than 60 years of age.

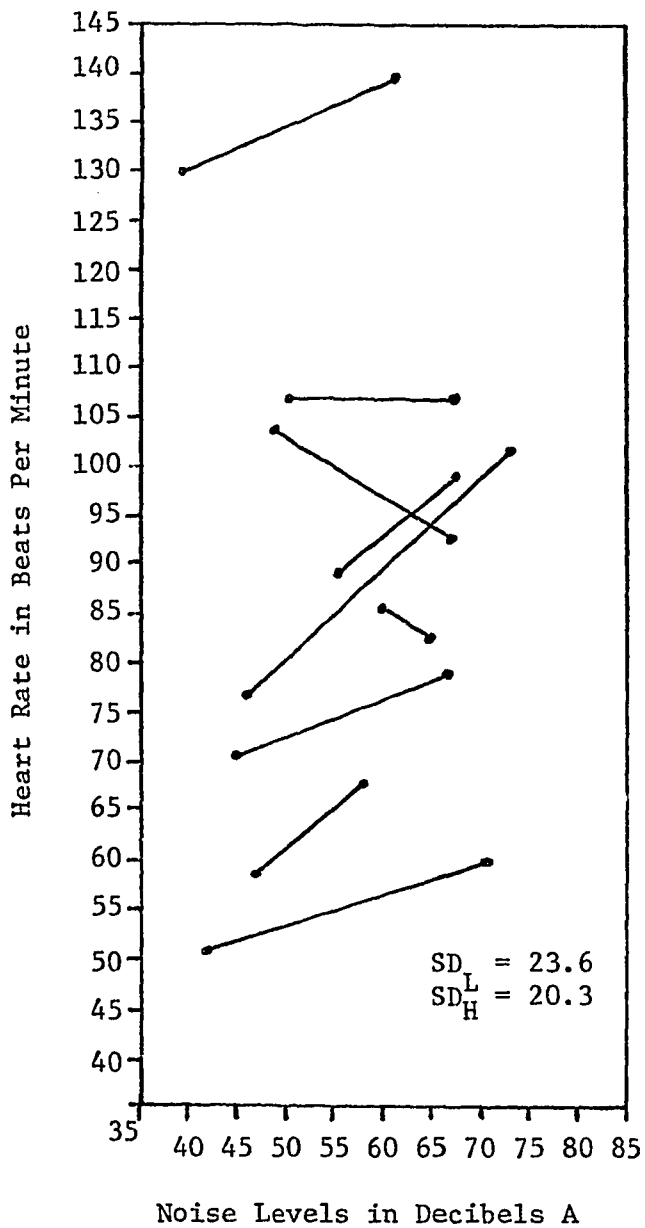


Figure 20. Mean heart rates related to noise levels in 9 subjects with a diagnosis of anterior infarction

$$t (df=8) = 2.83 \quad \underline{P} = <.05$$

The subgroup with anterior infarction less than 60 years of age (N=2) was of insufficient number to apply a statistical test. One of the two subjects did not demonstrate a heart rate response to noise.

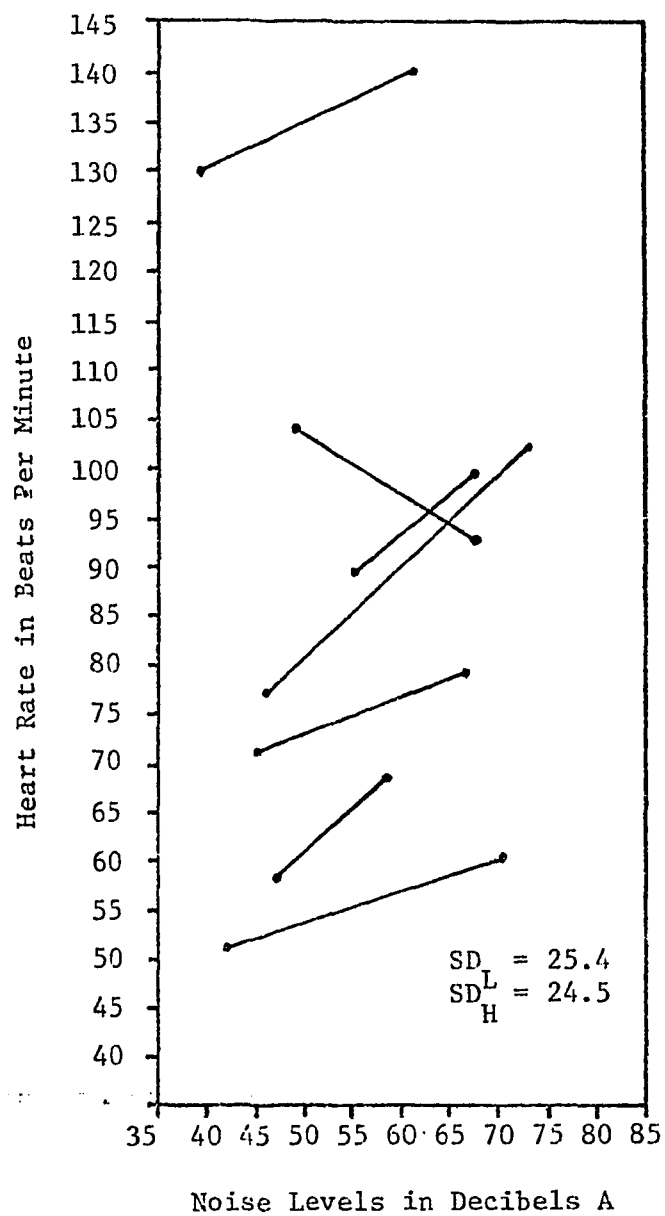
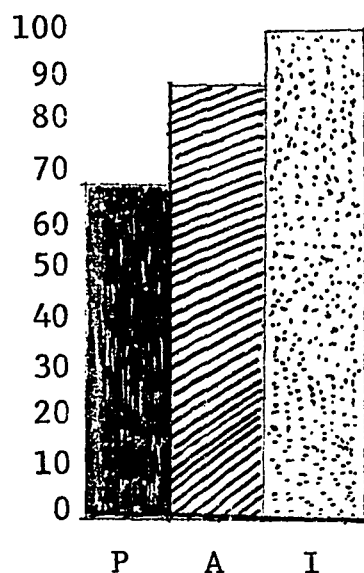


Figure 21. Mean heart rates related to noise levels in seven subjects with a diagnosis of anterior myocardial infarction and 60 or more years of age

$$t \text{ (df=6)} = 3.92 \quad \underline{P} = < .05$$

A comparison of the two subgroups of patients with a myocardial infarction, inferior (N=9), and anterior (N=9), shows that 100 per cent of the former responded to noise with a change in heart rate, whereas 89 per cent of the latter did. The following figure shows this comparison.



P = general population 68%

A = anterior infarction group 89%

I = inferior infarction group 100%

Figure 22. A comparison in per cent, of the heart rate response to noise in nine subjects with anterior infarction and 9 subjects with inferior infarction and the general population.

A cursory examination of the data suggests that there will be no significant difference in the heart rate response of patients with myocardial infarction that could be explained on the basis of differences in the site of heart damage. The Chi Square of .55 ($X^2=.55$) was non-significant. The hypothesis was rejected.

Due to the rate-related sequelae that often follows an inferior infarction, this latter finding was unexpected. Most of these patients exhibit a slow sinus heart rate and many experience complete heart block. None of the patients with a diagnosis of inferior infarction included in the study were in any type of heart block during the time of data collection.

A number of additional variables were of interest to the investigator although it was anticipated that the Ns would not be large enough for tests of significance to be applied to most of them. Among these are: past history of heart disease; patients presenting to the hospital with angina pectoris (chest pain) but without an infarction, the effects of certain potent drugs, and occupational differences.

It is well known that a past history of heart attack or angina pectoris increases the risk of death for the patient with a myocardial infarction. That is, a greater number of persons survive an initial heart attack, than do their second, third or a subsequent infarction. This is to be expected for with each infarction a certain amount of heart muscle can be expected to die, thus the pumping force of the remaining myocardial fibers tends to decrease in many cases, or myocardial irritability increases. The present study does not seek information on premature beats or "irritable" heart rhythms rather the emphasis lies with the propensity of the heart to change its rate under conditions

of noise.⁶ The results of the present study regarding a positive history of heart disease and noise are inconclusive due in part to the small N and secondly to the compounding variables of age and sex. Table 15 displays an age, sex and medical profile of nine patients with a diagnosis of anterolateral infarction.

TABLE XV

AGE, SEX AND PAST MEDICAL HISTORY OF NINE
SUBJECTS WITH ANTERIOR MYOCARDIAL
INFARCTIONS

Number	Age	Sex	Hx Diabetes	Hx Hypertension	Hx CHD*
1	67	F	0	0	0
2	74	F	0	x	x
3	61	F	0	0	0
4	67	F	x	x	x
5	68	M	0	0	0
6	66	M	0	0	0
7	64	M	0	0	0
8	55	M	0	0	0
9	54	M	0	0	x

*CHD, coronary heart disease

Only one male and two females with a present diagnosis of anterior myocardial infarction had a past history of heart attack or other cardiovascular disease (CHD). The mean age

⁶In the present study tabulation of heart rate and noise data began and terminated only when there were no early, premature and irritable heart beats present. Had a tachometer been available, premature beats could have been included in samples of heart rate since this device has the capability of storing 24 hours of continuous heart rate.

of the female population is 69.2 years, six years greater than the mean for women in the total study population.

The mean mase for males in the subgroup with anterior or anterolateral infarction was 61.4 years, 2.1 years more than for men in the total study population.

Table 16 shows a similar profile for nine patients with a diagnosis of inferior or inferolateral myocardial infarction.

TABLE XVI

AGE, SEX, AND PAST MEDICAL HISTORY OF NINE
SUBJECTS WITH INFERIOR MYOCARDIAL
INFARCTIONS

Number	Age	Sex	Hx Diabetes	Hx Hypertension	Hx CHD*
1	56	F	0	0	0
2	51	F	0	0	0
3	54	F	0	0	x
4	71	F	NK**	NK	NK
5	53	M	0	0	0
6	75	M	0	0	0
7	80	M	0	0	x
8	68	M	0	x	0
9	62	M	0	0	x

* CHD coronary heart disease

**Not known

A past history of heart disease appeared to be related to the heart rate response to noise. The subgroup was equally divided between the sexes, however, a significant heart rate response was evident only in the females (N=5). Of those with a past history of coronary heart disease, but less than 60 years of age (N=4), two were female. Figure 23, 24 and 25 depict the age and sex related data for the subgroup with a past history of cardiovascular disease.

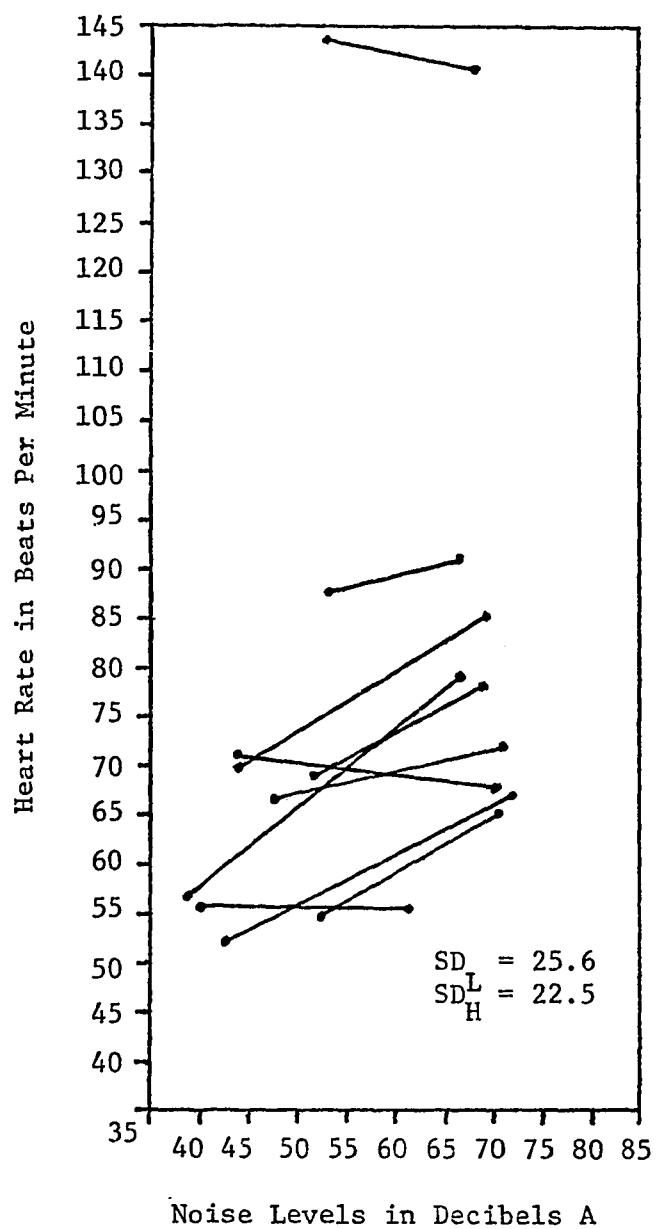


Figure 23. Mean heart rates related to noise levels in ten subjects with a positive history of cardiovascular disease

$$t \text{ (df=9)} = 3.18 \quad \underline{P} = < .02$$

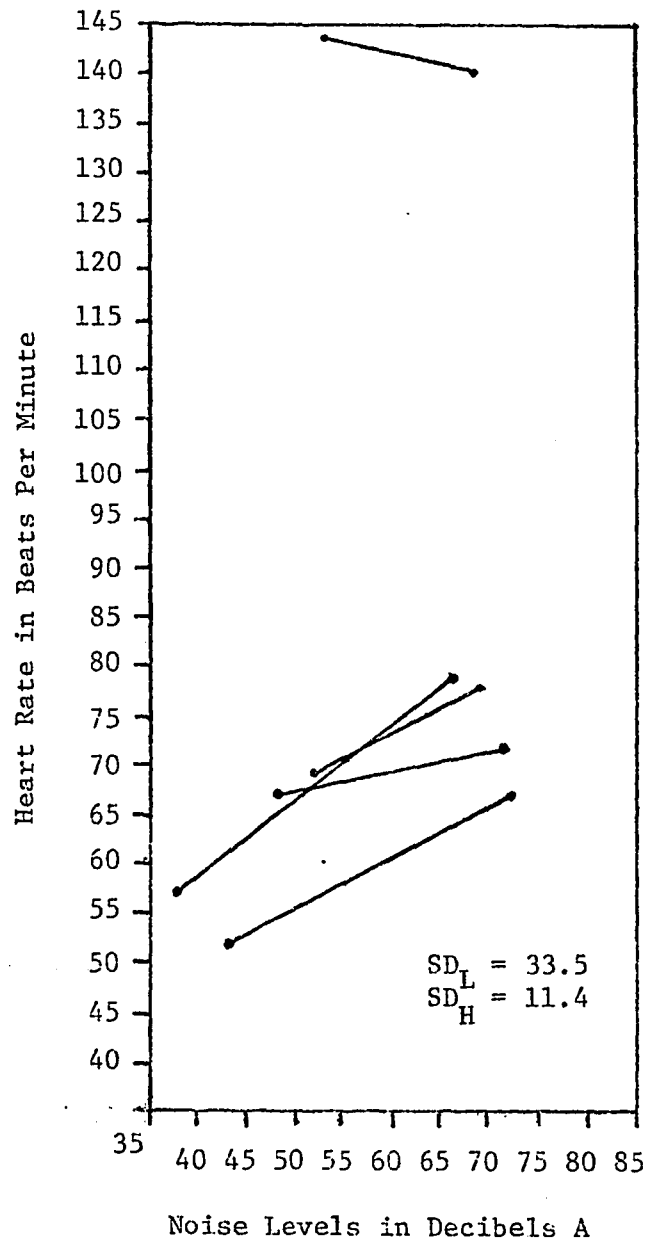


Figure 24. Mean heart rates related to noise levels in five female subjects with a positive history of cardiovascular disease

$$t (df=4) = 2.77 \quad \underline{P} = .05$$

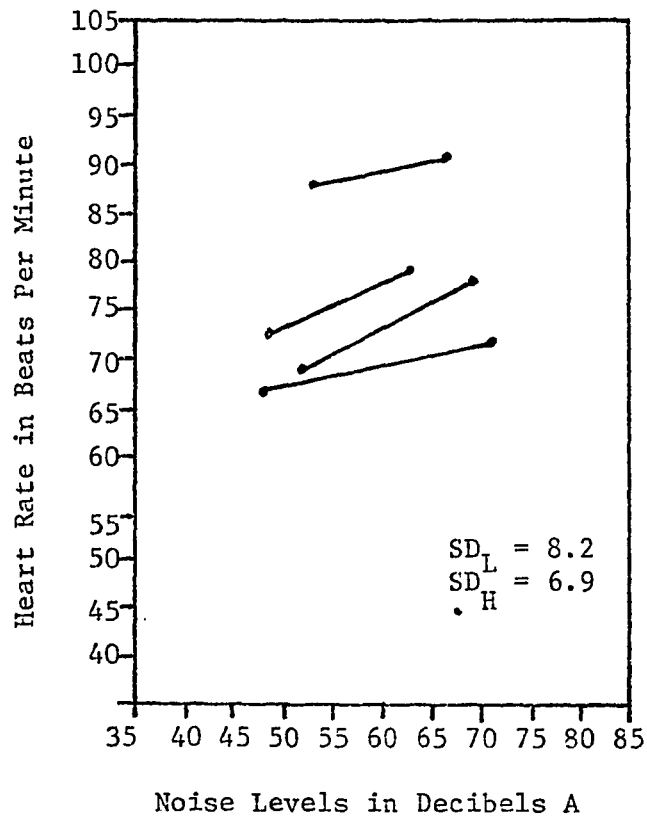


Figure 25. Mean heart rates related to noise levels in four subjects with a positive history of cardiovascular disease and less than 60 years of age

$$t (df=3) = 4.52 \quad \underline{P} = >.02$$

Concerning patients admitted to the hospital with angina (N=14), both sexes and all ages, only two subgroups, "Unit A," and "Female," demonstrated a significant HRR to noise. The following figure shows the data for seven patients admitted to Unit A with chest pain but who subsequently were diagnosed as not having had an infarction.

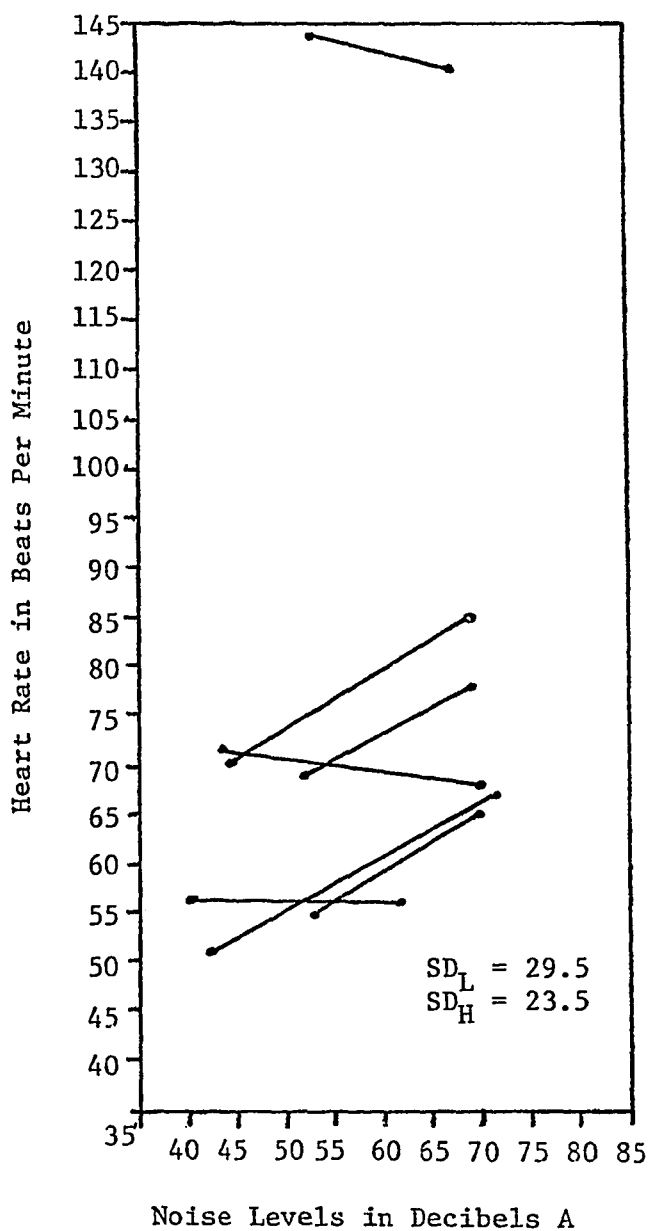


Figure 26. Mean heart rates related to noise levels in seven subjects with anginal pain and admitted to Unit A

$$t (df=6) = 2.68 \quad \underline{P} = < .05$$

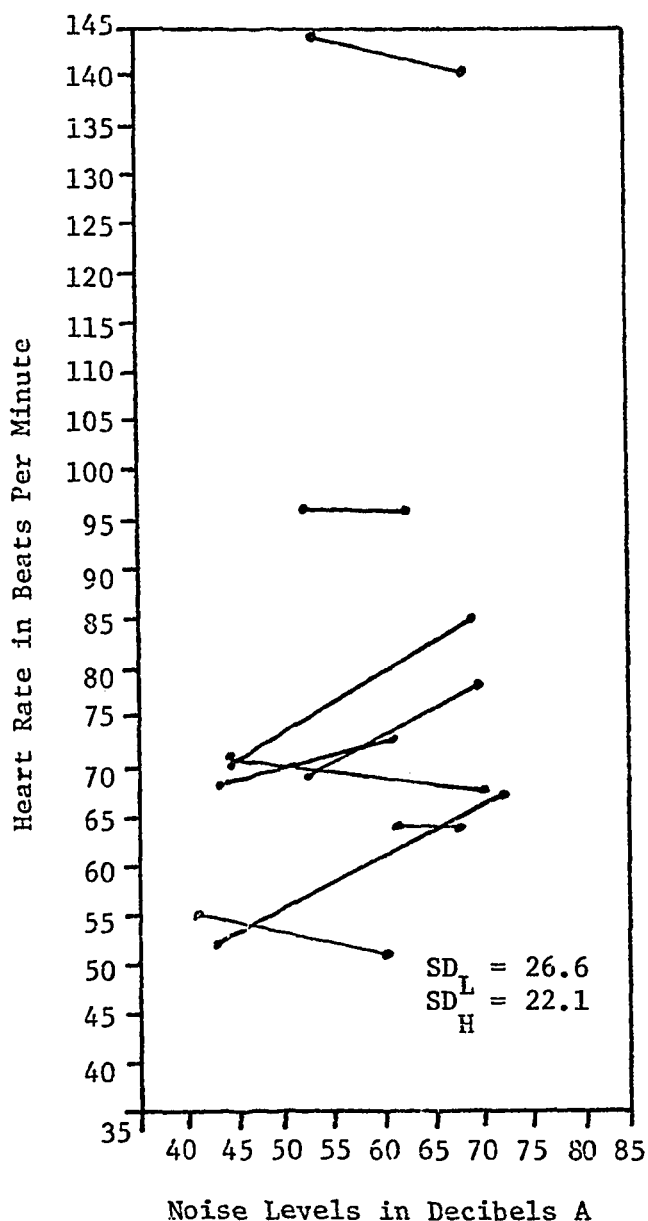


Figure 27. Mean heart rates related to noise levels in nine female patients with anginal pain admitted to Units A or B

$$t \text{ (df=8)} = 2.44 \quad \underline{P} = < .05$$

Popular belief holds that certain occupations, particularly those of an administrative or business nature are highly stressful, and further, that persons in these occupations are more vulnerable to heart attack. The present study

does not address the question because of the small N, and the fact that an extremely wide range of occupations are represented in the population. More importantly, almost half of the subjects had been retired for several years before their present heart problem occurred.

Ten males and six females stated that they had retired from their former occupation or profession. Some interesting aspects were revealed during discussions with patients about their work. One of the male "retirees" had spent his entire life as a transient, and now, in the coronary care unit, referred to himself as a retired transient. Another man, aged 75 years, who had been a physician for nearly 50 years declared, "I am never retired!"

Many of the females had difficulty answering questions about occupation, for those who had not worked outside the home felt they had nothing to retire from. One of these, a farm wife in her late seventies, who had raised eight children to adulthood, told the investigator that she had never worked a day in her life. The following table lists the last known occupation, by class, of the subjects in the study.

TABLE XVII

DISTRIBUTION OF THE STUDY POPULATION
BY LAST KNOWN OCCUPATION

Occupational Class	Number	Per Cent
White collar	9	23.7
Blue collar	14	36.8
Housewife	15	39.5
Totals	38	100

Eighteen persons, slightly less than one-half of the total population, had a final diagnosis of myocardial infarction. Figure 28 shows the distribution of these subjects according to their occupational class.

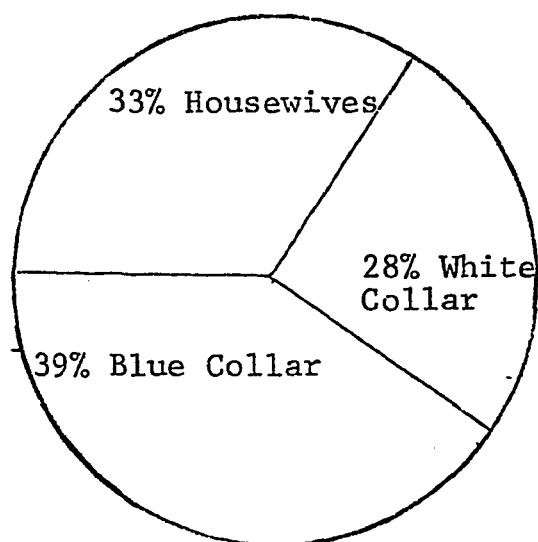


Figure 28. The distribution, in per cent, according to last known occupation of 18 subjects with a diagnosis of myocardial infarction.

The Effect Of Drugs On HRR To Noise

Nine patients were taking no drugs at the time of data collection and seven subjects were being maintained on a single medication such as a sedative or tranquilizing agent. The remainder of the population (N=22) were being given various combinations of the agents listed in Table 18. There were insufficient numbers in the subgroups for statistical analysis.

TABLE XVIII

DRUGS AND COMBINATIONS OF DRUGS BEING
ADMINISTERED TO 37 PATIENTS IN THE
CORONARY CARE UNITS OF TWO
METROPOLITAN HOSPITALS

<u>Drug</u>	<u>Number of Subjects</u>
Pain medication only	6
Tranquilizer only	1
Pain medication and tranquilizer	4
Tranquilizer and antiarrhythmic	2
Tranquilizer, pain medication and vasodilator	2
Pain medication and digitalis	1
Pain medication and rate depressant	1
Pain medication and vasodilator	2
Pain medication, vasodilator, digitalis and diuretic	1
Diuretic only	2
Diuretic and sleeping medication	1
Rate stimulant only	2
Rate stimulant and digitalis	1
Rate depressant and digitalis	2
Rate stimulant, diuretic and pain medication	1

Of the 37 subjects in the total population, six persons were being given a digitalis derivative. These were of various types: Digoxin, Lanoxin and Cedilanid. The route of administration was either intramuscular or intravenous.

Digitalis is a potent rate-altering drug which, in most cases is administered to slow a ventricular rate response to atrial fibrillation or to decrease a sinus tachycardia. Figure 29 shows the heart rate response to noise of five subjects being maintained on a digitalis derivative.

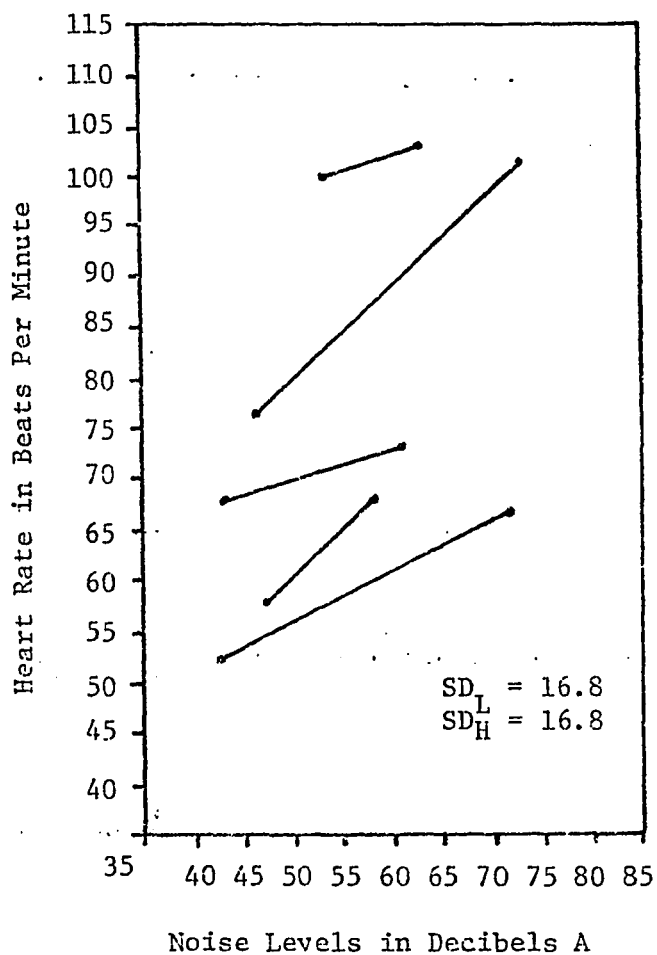


Figure 29. Mean heart rates related to noise levels in five subjects being given a digitalis drug

$$t (df=4) = 2.34 \quad P = > .05 - < .10$$

Rate-altering drugs constitute another possible intervening variable, which could exert a depressant action upon the entire heart. Generally, these agents are administered to the patient who exhibits symptoms of an irritable myocardium, for example, premature ventricular beats. Included in this group of pharmacological agents are quinidine and quinidine-like drugs, such as pronestyl hydrochloride (Pronestyl). One subject was taking quinidine, two persons, Pronestyl, one of these in combination with digitalis.

Propranolol hydrochloride (Inderal) was being administered to one subject and another patient was receiving diphenylhydantoin (Dilantin) and lidocaine--both potent rate depressant agents. The following figure depicts the heart rate responses to noise of these five patients.

A separate class of rate-altering agents are those whose principal effect is to increase a slow heart rate, such as sinus bradycardia. Three subjects were being given rate-stimulating drugs, one of these was taking atropine sulfate; another, ephedrine and a third was being maintained on isosorbide dinitrate (Isordil). All of these subjects were male. Because of the very small N, a statistical test was not applied to the data. It should be pointed out, however, that two of the males are also within the myocardial infarction group which, as a whole demonstrated a significant HRR to noise conditions.

The final hypothesis states that the heart rate response will show a positive relationship to noise gap.

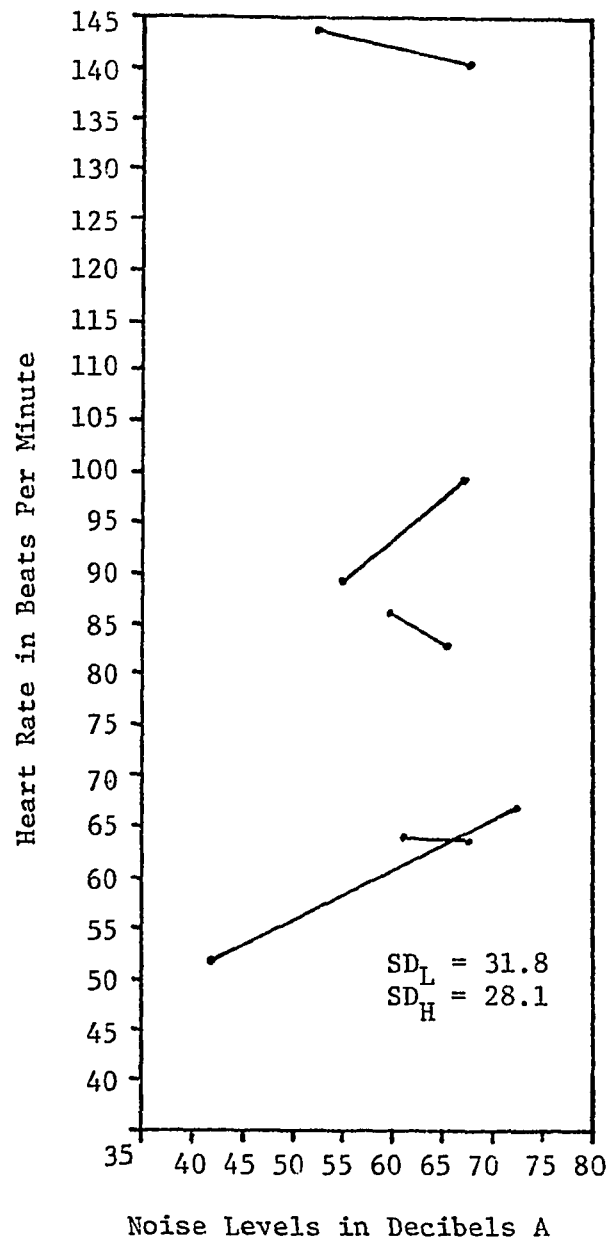


Figure 30. Mean heart rates related to noise levels in five subjects being given rate-depressing agents

$$t (df=4) = 2.50 \quad \underline{P} = >.05 - <.10$$

Several questions follow: Is the degree (not the simple occurrence of) of HRR a function of the difference between "low" and "high" noise conditions? Are the variables correlated to the extent that when noise gap is known heart rate change can be predicted? What degree of variance in the HRR can be explained by the variation in noise conditions?

A linear regression model $Y = a + bX$, was applied to the data with the heart rate as the dependent variable and the noise gap as the independent variable. Four additional variables were utilized, age, sex, presence of a myocardial infarction, and a positive history of heart disease.

For the total population ($N=37$, $df=N-2$), the correlation between noise and heart rate was .45 ($r=.4528$, $r^2=.2050$). An F of 4.12 was obtained for this data and the significance was at the 5 per cent level, ($P=.05$). Figure 31 shows this data.

A significant response to noise was also obtained for the subgroup of males and females over 60 years of age ($n=20$, $df=n-2$) and a correlation of .51 resulted ($r=.5073$, $r^2=.2574$). Variation in noise conditions could explain slightly less than 26 per cent of the variation in heart rate response in these persons.

Those subjects with a past history of heart disease ($n=10$, $df=n-2$) attained a correlation of .74 ($r=.7373$, $r^2=.5436$) and a F value of 5.32. The data were significant

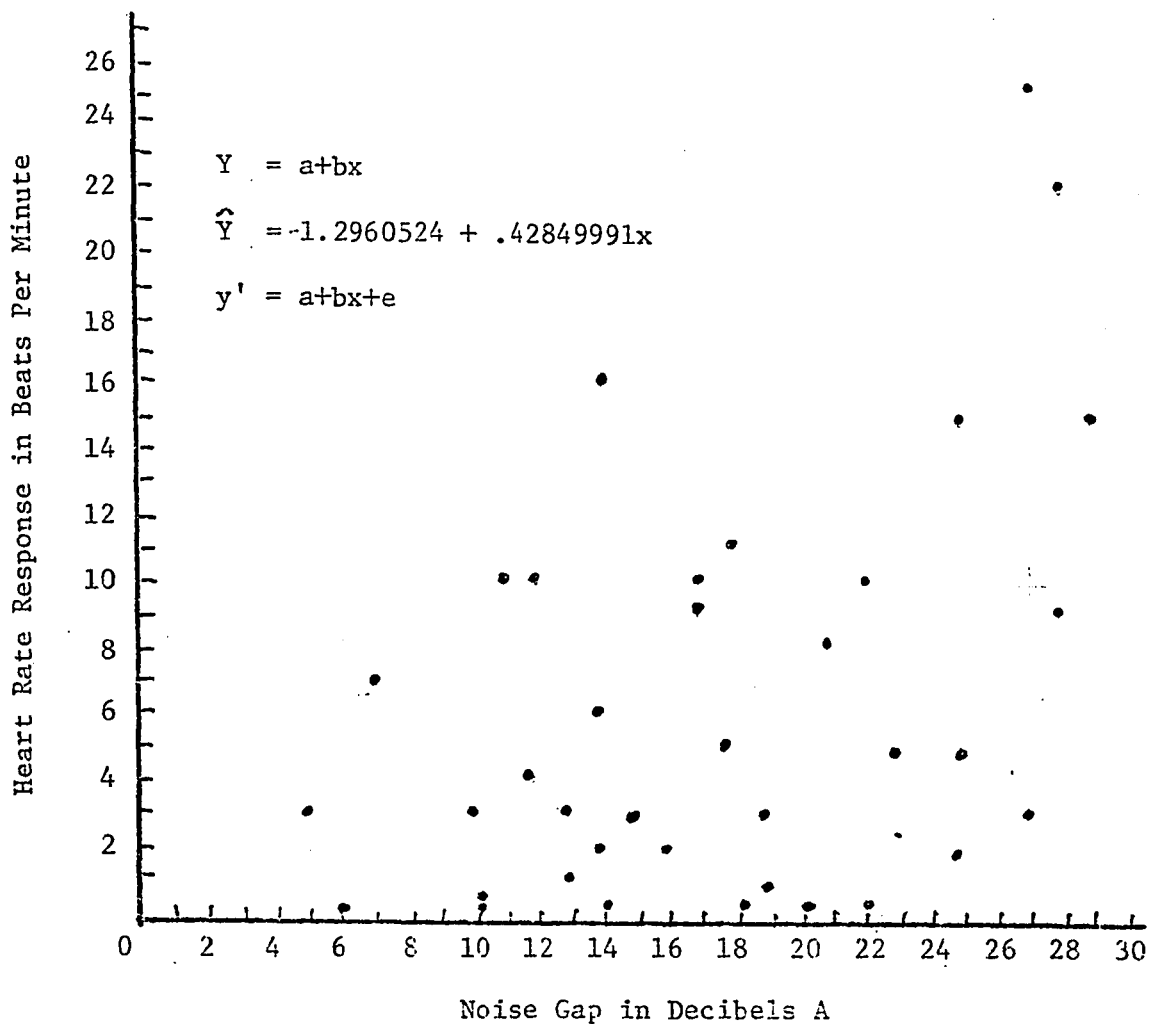


Figure 31. Regression line fitted to the data for a simple correlation between noise gap in decibels A and heart rate response for 37 subjects with cardiovascular disease admitted to two coronary care units

$$r = .45 \quad r^2 = .2050$$

$$F = 4.12000 \quad (df=N-2) \quad \underline{P} = .05 \quad \text{M.A.D. } 4.443$$

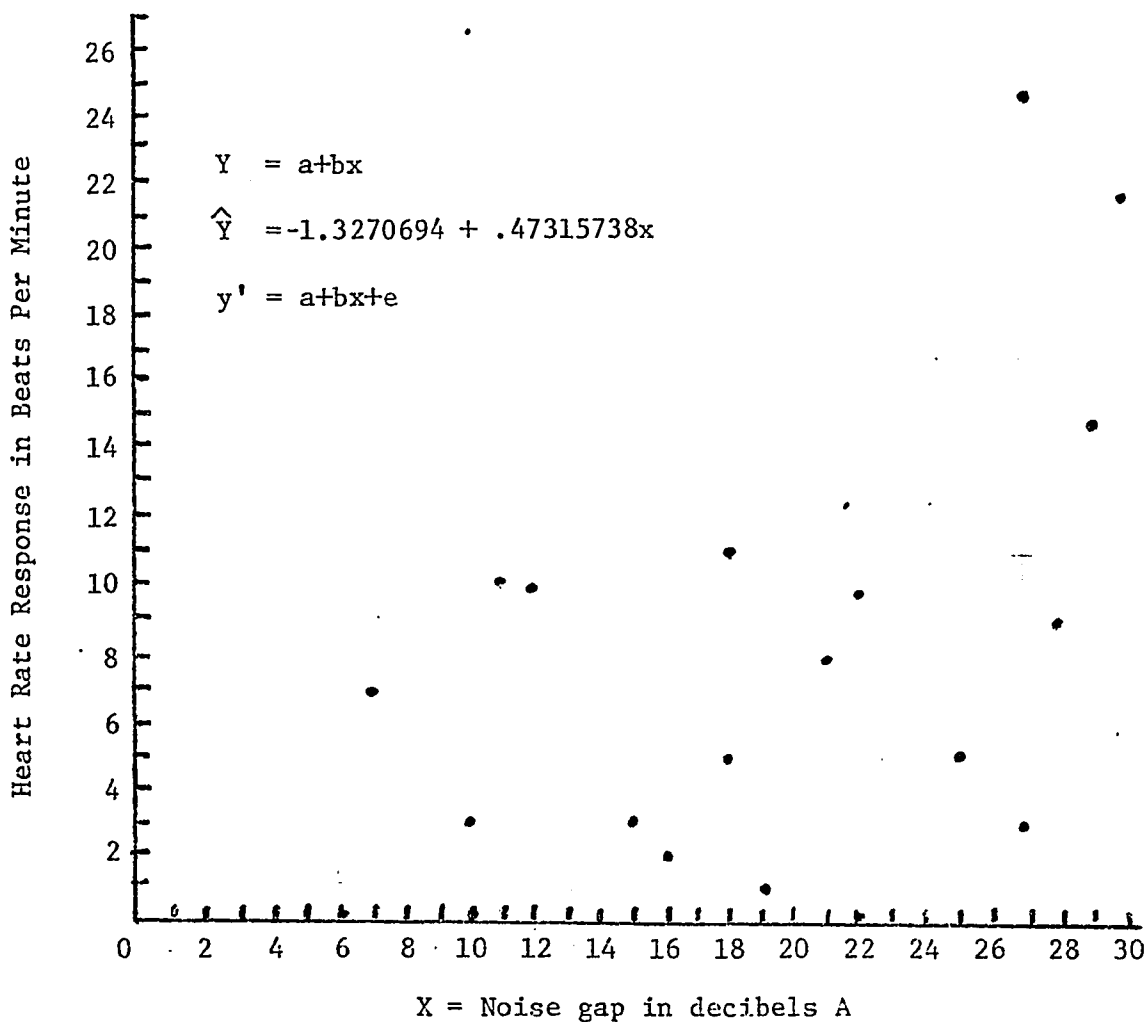


Figure 32. Regression line fitted to the data for a simple correlation between noise gap in decibels A and heart rate response for 20 subjects with cardiovascular disease admitted to two coronary care units, but 60 or more years of age

$$r = .51 \quad r^2 = .2574$$

$$F = 4.41000 \quad (df=N-2) \quad \underline{P} = .05 \quad \text{M.A.D. } 4.775$$

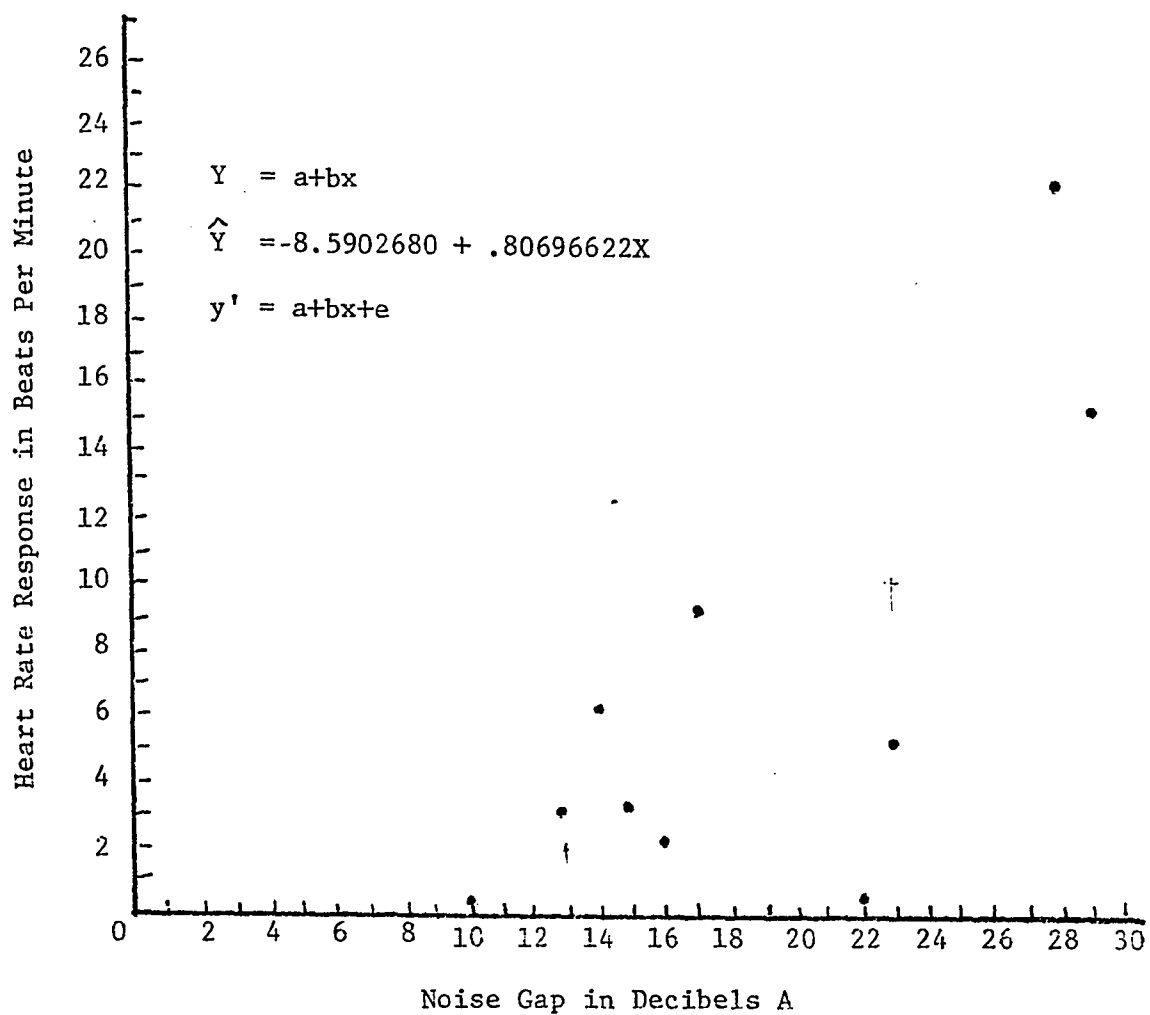


Figure 33. Regression line fitted to the data for a simple correlation between noise gap in decibels A and heart rate response for 10 subjects with a positive history of coronary artery disease and admitted to two coronary care units

$$r = .74 \quad r^2 = .5436$$

$$F = 5.320000 \quad (df=N-2) \quad \underline{P} = .05 \quad \text{M.A.D. } 3.393$$

at the 5 per cent ($P=.05$) level.

Finally, a multiple regression model ($y=a+b(X-\bar{X})$) yielded a significant correlation between noise and heart rate for the total study population ($R=.5368$, $R^2=.2882$). These variables appeared to be able to explain only about 29 per cent of the variation in HRR in 37 subjects.

The net regression of noise on heart rate response reached slightly less than 21 per cent ($r=.4528$, $r^2=.2051$). The probability of attaining these results by chance alone was less than five times in a hundred ($P= .05$). These data are shown in Figure 34.

It is interesting to note that the effect of noise on heart rate, i.e. the correlation, attained much higher levels when the various subgroups were analyzed separately, than in the multiple regression analysis. The simple linear regression model may be better suited to the analysis of the data in that the scores for each subject were a mean of the means ($\times 3$) for each subject. One possible explanation is that degrees of freedom are sacrificed in the transition from a simple to the multiple regression analysis. Since many of the significance levels were at borderline between no significance and significance, the few degrees of freedom sacrificed in the multiple regression resulted in much lower correlations.

The analysis points out that much of the variation in the heart rate response is not explained by the variation in noise levels experienced by these patients.

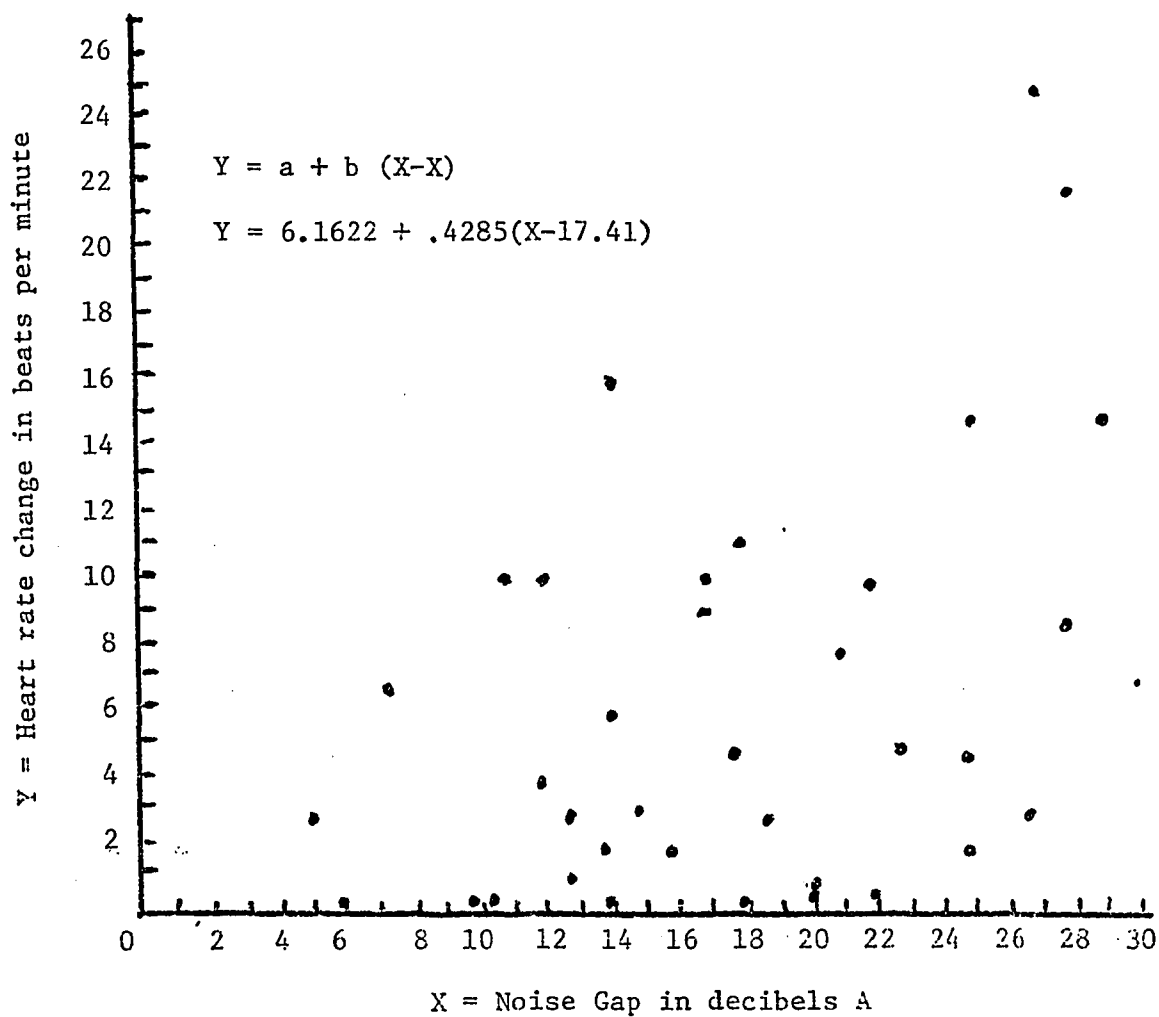


Figure 34. Regression line fitted to actual data for noise gap correlated with heart rate response scores in 37 subjects with cardiovascular disease and hospitalized in two coronary care units

$$R = .5386 \quad R^2 = .2882 \quad \text{S.E. } 5.671$$

$$r = .4528 \quad r^2 = .2051$$

$$F = 2.51 \quad \underline{P} = .05$$

Chapter V presents the conclusions and recommendations for further analysis of the effect of noise on heart rate, as well as upon the health of urban man.

CHAPTER V

CONCLUSIONS AND DISCUSSION

Introduction

The question of noise as a potential health hazard to urban man has been raised. The literature has been searched with a threefold purpose in mind; first, to establish that the urban environment is a very noisy place, and secondly, to point up the difference in the incidence of coronary artery disease between urbanized and rural areas of the United States.

Having established these premises, specific physiological responses to noise were identified through an extensive study of the available research.

No attempt was made in this dissertation to include explanations regarding the urban-rural, rate differentials offered by the various disciplines. For example, from the point of view of sociologists, industrialization,¹ social change,² and social upheaval³; should be considered as possible causes. Other investigators have sought to show a relationship between urban residence and CAD, based almost wholly

¹Tyroler, H., and Cassel, J. "Health consequences of culture change-II." The effect of urbanization on coronary heart mortality in urban residents. J. Chron. Dis. 17:167, 1964.

²Syme, S., Hyman, M., and Enterline, P., "Some social and cultural factors associated with the occurrence of coronary artery disease," J. Chron. Dis. 17:277, 1964.

³Sauer, H., "Epidemiology of Cardiovascular Mortality-Geographic and Ethnic," Am. J. Pub. Health 52:1:94-105, January, 1962.

on differences in dietary intake,⁴ or on life style.⁵

While it has been shown conclusively that noise along busy freeways, or on downtown thoroughfares often reaches dangerously high levels,⁶ less emphasis has been placed on the study of within-structure noise. Of the work that has been done, the major portion deals with industry or office noise,⁷ and noise around or inside airports.⁸

In-hospital noise levels, particularly noise within special care units, is the major interest of the present investigation.

Summary Of The Present Study

The present study builds upon the work on in-hospital noise carried out by Falk and Woods,⁹ Minckley,¹⁰ and Marshall.¹¹ The following questions are those of special interest

⁴Hirst, A., Prapont, P., and Gore, I., "A Comparison of Atherosclerosis of the Aorta and Coronary Arteries in Bangkok and Los Angeles," Am. J. Clin. Path. 38:2:162, August, 1962.

⁵Rosenman, R., Friedman, M., and Straus, R., et. al., "Coronary heart disease in the western collaborative group study," JAMA 195:86, 1966.

⁶Beranek, L., Noise and Vibration Control, New York, The McGraw-Hill Book Company, 1971. Chapter 18, pp 575-577.

⁷Burns, W., Noise and Man. London, John Murray, 1968, page 215.

⁸Cohen, A., and Ayer, H., "Some Observations of Noise at Airports and in the Surrounding Community," Am. Ind. Hygiene Assoc. 25:139, March-April, 1964.

⁹Falk, S., and Woods, N., *Ibid*, Chapter II.

¹⁰Minckley, B., *Ibid*. Chapter II.

¹¹Marshall, L., *Ibid*. Chapter II.

to the present investigator. What are the average noise levels inside of the urban hospital, specifically within their coronary care units? It is to these departments that patients are admitted under the most critical of circumstances, either complaining of chest pain having already experienced a heart attack, or with other cardiovascular crisis such as congestive heart failure.

Does noise represent a health hazard to these patients? Can a response that is cardiovascular in nature be elicited when these patients are subjected to conditions of noise?

The literature provided ample evidence that patients are capable of identifying those noises inside the hospital that bother them most. Such complaints have been documented many times. However, the theoretical base for the present study utilizes a physical model of stress, with several concepts evolving further from that model. A reaction to noise-stress that is mediated through the sympathetic nervous system is hypothesized. Also it was suggested that high levels of noise represent a continuous stress to urban man, and nowhere more so than to patients who are exposed to noise daily in the routine operation of the urban hospital. When exposure to noise results in a change in one or more physiological functions, it can be said that a reaction to noise has occurred. Examples of such reactions are: elevation of the serum, or urine catecholamines,¹² elevated blood pressure,¹³

¹²Wallace, A., Ibid. Chapter II.

¹³Rosen, S., Ibid. Chapter I.

vasoconstriction,¹⁴ or a change in heart rate.^{15,16}

The literature is explicit in relating noise to stimulation of the sympathetic nervous system and a resulting increase in circulating catecholamines. Increases, or in fewer cases a decrease, in heart rate, as well as instability of the myocardium, are secondary to the increase in adrenalin and nor-adrenalin in the blood stream. The exact mechanisms whereby circulating catecholamines exert their effect on ventricular vulnerability remain unclear.¹⁷ Heart rate was identified as the most feasible cardiovascular response to noise, in that heart rate can be accurately monitored in critically ill patients, and the generation of data requires little equipment other than that found in most coronary care units.

DISCUSSION OF THE HYPOTHESES

The major hypothesis postulates a heart rate effect of noise exposure in patients with known cardiovascular disease. The formulation of the major hypothesis resulted from many years of interest in the prognostic significance of heart rate change, by the investigator. Further, recent studies

¹⁴Hildebrandt, G., Ibid. Chapter I.

¹⁵Landis, C., and Hunt, W., Ibid. Chapter I.

¹⁶ReVelle, C., and ReVelle, P., Sourcebook on the Environment: The Scientific Perspective, Boston, Houghton Mifflin Company, 1974, p 165.

¹⁷Verrier, R., Thompson, P., and Lown, B., "Ventricular vulnerability during sympathetic stimulation: role of the heart rate and blood pressure," Cardiovasc. Research 8:602-610, 1974.

addressing the "vulnerable heart" of the patient with myocardial infarction, added to the general interest in heart rate.

Secondly, it is obvious that intensive care units are noisy environments due to the plethora of mechanical devices in continuous operation, as well as the concentration of professional and ancillary staff found in such units.

Of the study population (N=37), 30 subjects, or 81 per cent, demonstrated a positive heart rate response to noise. The results were significant ($P = <.001$) and the major hypothesis was not rejected. It is important to note that every subject was at absolute bed rest during the time of data collection, both at low noise and during noise conditions. That is, none were sitting up, ambulating, bathing, eating or performing any other activity. A few subjects were asleep, but the majority were simply "resting."

Although no hypothesis was made concerning differences based on sex, it became apparent at the initial examination of the data that such a difference may exist. Women react differently to electrocardiographic stress testing than do males in that females demonstrate more false positives than do men. Moreover, there is a greater tendency for women to acquire prolapse of the mitral valve than their male counterparts. The reasons for these findings are obscure.

The significance level of the HRR by males was $<.02$ compared to $<.01$ for females. The t test for independent

means resulted in a significance level of slightly more than .10, however, a less exacting test, the chi square showed that such differences could occur by chance more than ten times in a hundred ($P = >.10 - (.20.)$)

The age distribution between the groups (male and female) is one possible explanation for the difference in their heart rate response to noise. The ages of the men ranged from 33 to 80 years, with the mean age, 59.3 years, compared to 63.2 years for the females. Thirteen men were housed in Unit B with only five being admitted to Unit A.

The age range for women was 54 to 74 years, with 12 of the females being admitted to Unit A and only 7 entering Unit B. The difference between the HRR to noise of men and women may be masked by their unequal distribution between a unit with consistently high levels of ambient noise (B) and one that was relatively quiet (A).

Unit A is a carpeted CCU with four single enclosed patient rooms and a wide space between the patient area and the nurses' station. Unit B is a converted four bed ward with draw curtains the only means of separation both between patients and from the central nurses' station. The 24 hour noise profile showed that Unit A had consistently lower levels of ambient noise, the exception being between the hours of 9:30 and 11:00 P.M. This latter finding may be due to the manner in which evening nurses in the two units dealt with patients' families. The staff in Unit A encouraged the families of patients to remain at the bedside for extended

periods of time during the evening. Family members of the less critical patients were allowed to participate in their evening care.

This was not the case in Unit B where a strict visitors' code was enforced equally by the nursing personnel on all shifts.

The thirteen males who were admitted to Unit B showed a significant heart rate response to noise ($P = <.05$), while the five men in Unit A did not.

The twelve females admitted to Unit A, the less noisy environment, showed a significant HRR to noise of slightly greater than .05, ($P = >.05$ but $<.10$) compared to the 7 women admitted to Unit B whose significance level was somewhat greater than $>.02$ but less than $<.05$. It would appear that some real differences underlie the responses obtained for subjects in a noisy, compared to a less noisy environment. However, the total N is small, the range of data, narrow, so that the level of significance obtained can do no more than suggest that such differences may exist.

The older women, that is 60 years or more of age, that were admitted to Unit B ($N=4$) did not have HRRs that were significant, whereas the older females admitted to Unit A ($N=8$) demonstrated a highly significant heart rate response to noise ($P = <.01$). None of the women admitted to Unit B and over 60 years of age had experienced an infarction and one subject had a consistently fast heart rate (107 per minute).

Seven females in the study were less than sixty years of age. Of these, two had a diagnosis of inferior wall infarction, nevertheless, regardless of the Unit, or diagnosis, all had a significant heart rate response to noise.

Regarding hypothesis number one, the results of this study show a significant HRR to noise by the total population of 37, and further, tend to suggest that the response is more widespread and of greater intensity in females, and in males more than 60 years of age.

The second major hypothesis stated that the heart rate response to noise would differ significantly between patients with myocardial infarctions and subjects with other chronic types of cardiovascular disease. Wallace showed that patients who had experienced heart attacks responded to noise with large catecholamine discharges and went on to suggest that traffic and air travel noise could aggravate the already damaged myocardium. While there are obvious references to circulating adrenalin and heart rate, the work of Wallace does not explicitly identify the steps leading from stimulation of the sympathetic nervous system, to increased catecholamine levels, to the physiological outcomes such as a change in heart rate.

A comparison of the mean heart rate responses of patients with an MI ($N=18$ $P = < .01$), and patients without a myocardial infarction ($N=19$ $P = < .02$) resulted in a chi square that was significant at the $P = < .05$ level. However the t test for independent means--a more efficient measure--

resulted in a P of slightly greater than .05. There appeared to be a real difference between the HRR to noise of these two groups, with the greater rate and intensity of response being demonstrated by patients with a myocardial infarction. This finding supports the work of Wallace, Marshall and others who concluded that the individual with a myocardial infarction has a highly vulnerable heart. Noise may be a special hazard for him.

Myocardial infarctions vary, in their size, their anatomical site in the heart and in their prognosis for recovery. It was of interest to the investigator to know whether a differential response to noise could be explained on the basis of the site of the damage. Would the rate and the intensity of HRR to noise differ in persons with inferior heart damage? Would greater HRR to noise be obtained in subjects with an anteriorly situated infarction? The third hypothesis, a null hypothesis, stated that no such differences would be found.

Quite by chance the population with myocardial infarction was equally divided with 9 patients having a diagnosis of anterior or anterolateral infarction and an equal number (9) having had inferior or inferolateral infarctions. A significant HRR to noise was obtained for the total population with the anterior infarction subgroup having a significance of $P = < .05$, and the group with inferior heart damage attained a significance level of $P = < .02$.

The t test for independent means was non-significant, as was the chi square of $X^2 = .55$. There appeared to be no

difference between the HRR to noise between these two groups of subjects. The null hypothesis was, therefore, not rejected.

It must be noted that, unexpected as are these latter results, no patient in the group with inferior infarctions had a heart rhythm that was complicated by heart block at the time of data collection. The one male patient with a complication of complete heart block was dropped early in the study. Quite different results might be obtained if similar measurements of HRR to noise were to be made on patients whose heart rhythms are not paced from the sinus node. In that sense the population of patients with inferior infarctions seems atypical when compared to those one finds in a general survey of CCU populations.

As might be expected the subgroup of patients with infarctions but 60 years of age or older had a significant HRR to noise, ($P < .05$). Little practical meaning can be attached to this finding since the entire group, regardless of age, had a significant HRR to noise.

Some interesting facts came out of an examination of the characteristics of the MI group. The mean age for males was 61.4 years, 2.1 years more than that for the male population as a whole. The mean age for females with a heart attack was 69.2 years, six years older than the mean for all women in the study, and 7.8 years older than the mean age for men with infarctions.

The study population is typical of that described elsewhere in the literature in that women experience heart attacks

at an older age than do males, and that most heart attacks occur to persons of older age.

Ten subjects, five males and five females, had a past history of heart disease, either a previous myocardial infarction (8), congestive heart failure (1) or a serious arrhythmia (1). The age range in this group was similar to that for each sex in the entire study. It was of interest to the investigator to know whether these persons of both sexes, all ages, admitted to either Unit, with or without a present infarction, would respond to noise in a significant manner. The t resulted in a significance level of $<.02$. When the group was desegregated according to age, the significance remained. Four persons were less than 60 years old, however, their HRR to noise culminated in a t of 4.52 ($df=3$) and a P of $>.02$ but less than .05.

It becomes clear that older persons, especially those with a past history of heart disease will respond to noise. This finding has important implications for planning the location of institutions housing the elderly, as well as for the control of noise within those structures.

Presenting angina (chest pain) is another variable typical of any population with cardiovascular disease. Fourteen subjects, none of whom were subsequently diagnosed as having had a heart attack, were admitted to the Units with a complaint of chest pain. Only men and women admitted to Unit A ($N=7$) and the females ($N=9$), attained t s that were significant. The probability level reached $<.05$ for each

group. A close examination of the characteristics of groups with chest pain showed them to be predominantly female (N=9), and more than 60 years of age (N=9). Thus the variables of sex and age probably contributed to the significance for patients with anginal pain.

The only group with significance apparently unrelated to sex or age was that with myocardial infarction. Such a finding, demonstrates again, the sensitivity of the injured myocardium to noise.

Rate-altering agents--even those that depress the heart rate seemed to have little effect upon the capacity of the heart to respond to noise. Six patients were receiving a derivative of digitalis, with the goal of therapy to reduce the ventricular rate. A borderline level of significance ($P = >.05$ but $<.10$) was obtained for this group.

Five persons were being given a potent antiarrhythmic agent, either quinidine, lidocaine (xylocaine), or diphenylhydantoin (Dilantin). However, the heart rate response in this group reached a level of significance slightly greater than .05. An analysis of variance was not carried out with this data due to the small populations.

The final hypothesis stated that HRR would show a positive relationship to noise gap. This does not address the presence or lack of a HRR, rather it asks whether the extent of the response varies in a positive manner with the intensity of the noise gap. The expectation is that the greater the difference between low and high noise conditions, the

greater the heart rate change. When the hypothesis was drawn, it was expected that an N approaching 50 would be used, however, technical and financial constraints made this goal unreachable, and a population of 37 was accepted.

The results of a simple linear regression of noise on heart rate showed a significant relationship between the two variables. ($F=4.12$ $df=36$), $P=.05$. The correlation was not as high as was expected (.4528), so that a little less than 21 per cent of the variation in heart rate ($r^2=.2050$) could be explained by the variation in noise gap.

A significant response ($F=5.32$, $P=.05$) was also obtained for 20 patients who were more than 60 years of age. The correlation between HRR and noise gap reached .51 ($r=.5173$) with approximately 26 per cent ($r^2=.2574$) of the variation in heart rate being explained by the variation in noise gap.

The only other subgroup to attain a level of significance between HRR and noise gap was the population ($n=10$) with a positive history of cardiovascular disease, previously described as largely female and more than 60 years of age. The correlation reached .73 for these subjects ($r=.7373$, $F=4.41$, $P=.05$). Slightly more than 54 per cent ($r^2=.5436$), of the variation in HRR appeared to be explained on the basis of the variation in noise gap.

A multiple regression analysis for the population ($N=37$), was also significant ($F=2.51$, $P=.05$), although the correlation between heart rate response and noise gap was

low ($R=.53682$, $R^2=.2882$). The net regression of noise on heart rate after the other variables were dropped, was also significant ($F=4.12$, $P=.05$), and again, the correlation was minimal ($r=.4528$). Slightly less than 21 per cent of the variation in heart rate could be explained by the variation in noise gap ($r^2=.2051$).

Although higher correlations would seem necessary before definitive statements can be made, there was nothing in the results of the regression analysis to disprove what the remainder of the data show. However, generalizations based on these findings must be made cautiously for the N s were consistently small and the range of the data narrow. It is quite possible that only a small segment of a multi-shaped curve was examined and other variables not identified herein could account for an equal amount of variation in heart rate. Nevertheless findings relating noise to heart rate response and age (over 60 years), sex (females), to myocardial infarction, and to a past history of heart disease seem consistent throughout the study.

The 7 subjects who did not demonstrate a HRR to noise were largely male (5 of 7), younger persons (4 of 7), and had no prior history of coronary artery disease (5 of 7). Only one of these had a diagnosis of myocardial infarction.

Implications For Health Planning: Locational Decisions

In 1972 heart attack claimed the lives of 683,100 people in the United States, and left living an estimated 4,000,000

persons with a past history of heart attack or angina pectoris. Most of these people are in their older years of life.¹⁸ Moreover, a summary of the present data on CCU populations in general, shows that a substantial majority of the persons admitted there are 60 years of age or older.

An increase in life span in Western man over the past few decades has practical implications for age-related illnesses. Increasing numbers of Americans can be expected to develop coronary artery disease as a result of atherosclerosis.

Certain age related changes have implications for health care, especially for convalescence and the quality of life after recovery from a heart attack. In the Unit, the older person is more prone to develop complications such as pulmonary or congestive heart failure. Physical changes, for example, a decrease in cardiac output or aberrations of heart rate and rhythm are common sequelae of myocardial infarction in the aged.

Significant heart rate response to noise, as evidenced by the present study for several subgroups of older patients, has implications for health planning in the urban setting. Locational decisions for nursing homes and convalescent hospitals should take noise level into account. It seems almost obvious that acute care hospitals, especially coronary care units should be shielded from noise.

Research on noise is not a recent phenomenon, however

¹⁸--Heart Facts, 1975, The American Heart Association, 44 East 23rd Street, New York, N.Y. page 5.

noise climate has not been considered as important as economic factors in making locational decisions for health care facilities. This posture is probably due to the lack of specific data linking continuous adrenergic arousal to changes in cardiovascular and other physiological mechanisms--mechanisms which ultimately alter the workload of the heart.

In general, planners have based their decisions on economic considerations, particularly on accessibility. Accessibility, in the case of the Chicago Health Services Survey--means a maximum travel time of 30 minutes for all consumers. Frieden and Peters expanded the definition of "accessibility" to include cost of care and waiting time.¹⁹

The importance of accessibility cannot be denied, however, the quality of care, which in turn, may be directly related to such factors as environment, may be of equal import. If, as is often the case in large cities, acute care hospitals must be erected in the central business district, protection from noise pollution should be guaranteed early in the planning process. The use of thick partitioning walls, insulated structure, green belts, and foilage barriers can minimize noise reception by the patients. Taylor argued that the cost of such protection is not formidable.²⁰

Economic considerations should never be used to justify

¹⁹Frieden, B., and Peters, J., "Urban Planning and Health Services: Opportunities for Cooperation," in Levey, S., and Loomba, N., Health Care Administration A Managerial Perspective, Philadelphia, J.B. Lippincott, 1973. pp 309-335.

²⁰Taylor, R., Ibid. Chapters 3, 13 and 14.

construction of hospitals or convalescent homes near recreational areas, activity centers, playgrounds, ball or racing parks.

Nor, from the point of view of this practitioner, is it sufficient to build structure upward, "above noise level" for in so doing the most unnatural kind of environment for the ill and the elderly is created. When the patient becomes the real focus of hospital planning, locational decisions will reflect greater attention to the noise environment.

Hospital Design and Structure

Historically, hospitals were designed for the convenience of the staff, particularly for the physician. This view is usually implicitly stated, as for example, in the following excerpt by a medical editorial writer, "the intensive care unit should...be located within easy access of the recovery room, the surgical suite, the emergency, and the x-ray department."²¹

The only reference to auditory aspects was a comparison between solid wall partitions "which have sound-dampening advantages" and glass partitions, "which aid in patient observation." It is clear to experienced practitioners that unless each room is sound-proofed, the patient in this "easy access" unit will be exposed to groaning and crying out from the ER, the clacking of rapid-series x-rays, as well as the squeaking of stretcher wheels by day and by night.

²¹--Features to consider in designing an intensive care unit, Editorial, Hospitals JAHA 42:18, June 1, 1968.

In one of the most comprehensive of planning guides Hudenberg payed much attention to ventilation, lighting, flooring, workspace for physicians, bed space, and the location of electrical equipment, with no reference whatsoever to the prevention or control of noise.²² More recently, Clipson and Wehrer, discussed Planning For Cardiac Care²³ with minimal reference to the sonic environment. Their model depicts the heart patient as the center and object of integrated systems or "three dimensional activities." In the work of Clipson and Wehrer, a great deal of importance is placed on monitoring, resuscitative and life support systems. Much less emphasis was given to the thermal, luminous or sonic environment. (Appendix B).

In fact the authors conceded that "These three levels (activity systems and physical environment) are not of equal importance in defining priorities for planning. The activity centered approach tends to account for the bulk of the requirements...Finally, a smaller group of requirements for design can be seen as environmental in origin."

Their model, relating the organization, placement within the hospital, and use of space in the CCU, is intended for hospital planners and architects. Design decisions are

²²Hudenberg, R., Planning the Community Hospital, New York, The McGraw-Hill Book Company, 1967. Chapter 6, "The Patient Housing System."

²³Clipson, C., and Wehrer, J., Planning For Cardiac Care A guide to the planning and design of cardiac care facilities. Ann Arbor, Michigan, The Health Administration Press, 1973.

based upon cost and access. However the results of the present study, as well as others, showed that the sonic environment directly effects the welfare of the patient with coronary artery disease, especially myocardial infarction. Based upon the results of this study the following suggestions are made:

1) Locational decisions regarding acute care hospitals and nursing homes should take noise factors into account. Particularly such institutions should not be constructed in close proximity to "high noise" areas, unless measures to guarantee noise proofing are a part of the planning process. Such measures would, most surely add to the cost factor, and may result in altering locational decisions.

2) Regulations regarding speed, automobile horns, loud mufflers, revving of engines and the like, should be reinstated and enforced in the vicinity of the acute care hospital or nursing home.

Recommendations Specific To Coronary Care Units

1) The CCU should not be situated in close proximity, beside, below or above, a commercial delivery area, kitchen, storeroom or laundry. If, in fact, the existing unit is exposed to noise from these areas, the walls of the CCU should be soundproofed to insure noise exposure of no greater than 45 decibels A at any time of day or night.

2) CCUs should be planned well away from support areas such as the emergency room, x-ray department, laboratory, or surgery. If this cannot be done, sound-proofing as noted in

1 should be carried out.

3) General foot traffic (staff and visitors) should be directed well away from the CCU.

4) All floors around and within the coronary care unit should be carpeted.

5) Patient areas in the coronary care unit should be single rooms. No patient with a serious cardiac disorder should be exposed to the sounds (or sights) of other critically ill patients.

The Economics Of Sound-Proofing

The cost of sound-proofing existing facilities, as well as new structures was discussed with several practicing architects and builders, one of whom had completed three intensive coronary care units in the Phoenix area in the last few years. All agreed that sound-proofing was necessary and must be aimed at both external (airborne) and internal (airborne and structure borne) noise. It was agreed also that complex problems arise when a multitude of sources must first be identified, then evaluated economically and esthetically, before corrections are initiated.

According to one source²⁴ a complete frequency analysis is required to identify the characteristics and cause of each noise source. The cost of acoustical tile, of double glazing windows, of lining heat and air ducts with noise-absorbent materials were all discussed at length but no one was

²⁴Interview, Rossman, A., PhD. Noise Consultant, The Auditorium Research Company, 4601 E. McDowell Road, Phoenix, Arizona, September 13, 1976.

willing to estimate the cost. One source argued that heavy carpeting, heavy, double drapes over glass, and double glazed windows are the most obvious corrections for excessive noise. He indicated that in tests made by their firm, it was shown that the decibel level could be reduced from 60 to 50 dbA, in most buildings, with these measures.

The experts were quick to agree that there is absolutely no mechanical problem in completely sound-proofing a hospital, but the problem as they saw it was one of cost.²⁵

When the investigator pointed out that she had been unable to document noise levels equal to or below the recommended levels for hospitals in many states, except at 3:00 A.M., the resource person pointed out some additional difficulties in sound-proofing modern construction. Lighter materials and movable walls, have replaced the wood and brick double walls of the old hospital. As ReVelle and ReVelle pointed out,²⁶ large areas of glass are replacing solid walls in an effort to maximize eye contact from the nurse to patient. All agreed that with the advantage of continuous observation comes the disadvantage of continuous noise. The result of wide, open spaces, sliding doors, and glass panels has been not only a decrease in the capacity of the building to shield patients from externally generated noise,

²⁵Interview, Hinshaw, D., Assoc. Professor of Architecture, College of Architecture, Arizona State University, Tempe, Arizona, September 2, 1976.

²⁶ReVelle, C., and ReVelle, P., Sourcebook on the Environment: The Scientific Perspective, Ibid. pp 303-306.

but also diminution in the ability to insulate internally generated noise. The cost of keeping noise below 40 to 45 decibels in such an environment would most certainly be passed on to the consumer.²⁷

Selection Of Equipment For The CCU

Most manufacturers have the capability of noise proofing their products. Much of the medical equipment in current use could have been almost silent given the proper alterations in their design. It is suggested here that manufacturers of hospital equipment utilize a code of maximum noise emission for each article of technical equipment. This would not be greatly different from the maximum leakage codes for electrical devices, which are rigidly enforced in most coronary care units.

When a planned coronary care unit must utilize existing facilities, for example, the converted four bed ward, the ideal may not be immediately obtainable. However, noisy equipment can be placed as far as possible from the patient's ear, thus guaranteeing a reduction in noise reception in accordance with the inverse square law.*

Programs Of Staff Education

The measures suggested in this dissertation would undoubtedly result in a reduction of noise in most CCUs, however,

²⁷Interview, Sydnor, R., of Varney, Sexton, and Sydnor, Associates. 221 Camelback Road, Phoenix, Arizona, September 16, 1976.

*Sound intensity is inversely proportional to the square of the distance the noise source is from the listener. Taylor, Op. Cit. page 110.

unless a continuous educational program is maintained for personnel, the results may be less than ideal.

Staff education is important, considering that in the present study conversations between the nurses, between doctors and nurses, or among staff and family members contributed to excessively high noise levels in both units.

An awareness of the effects of noise, on the part of the hospital staff, is required if the results of noise reduction are to be lasting. The older patient, the patient with myocardial infarction, or the person with a past history of heart disease is vulnerable to loud noise. As indicated by the results of the present study, a heart rate response to noise when the patient is at rest or asleep, is an overt expression of myocardial vulnerability.

Suggestions For Further Study

The suggestions for further scientific inquiry are of two types: those dealing with the general population, and those that examine the effects of noise on hospitalized patients. There remains a need for definitive data regarding the long term effects, if any, of noise exposure. For example, it is not sufficient to state that cities are noisy environments therefore cardiac health is impaired. The fact that higher rates of coronary artery diseases and myocardial infarction are found for large cities gives little indication as to causes.

Prospective studies which utilize large urban popula-

tions, and which systemtically plot noise levels over time against parameters such as blood pressure, heart rate, catecholamine excretion, cholesterol levels and size of the myocardium are needed. Particularly, populations living near airports, freeways, or industrial areas should be studied.

Cardiovascular factors, as described above might also be measured over time and in relation to noise exposure in persons housed in convalescent and nursing homes. This latter may be vulnerable population, since by virtue of their advanced age, many would have a past history of heart disease. It is anticipated that a vast number of variables, in addition to noise, would have to be taken into consideration.

A third suggestion is that an inquiry might measure the weight assigned to the sonic environment by various specialists in urban planning, for example, city, industrial and health planners. While the present study utilized a relevant planning literature, there was no attempt to collect primary data about planners and the processes used to set priorities for decision making.

It seems obvious that additional studies are needed in which a relationship between the course of illness of patients and their sonic environment is sought. The plethora of variables encountered in the field setting are difficult to control for, and no more so than in units housing critically ill patients. However, given fewer financial and time constraints, precise documentation of the effects of noise on these patients could be ascertained.

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APPENDIX A

University of Oregon Medical School



DEPARTMENT OF MEDICINE / DIVISION OF CARDIOLOGY

June 28, 1974

Mrs. Frances Storlie, R.N., M.S.
3811 N. E. 41st Street
Vancouver, Washington 98663

Dear Mrs. Storlie:

Your proposed doctoral topic sounds most interesting and should provide us with considerably more information about an area about which we know very little, the effects of sensory-deprivation or sensory-overload on physiological parameters in acutely ill patients. Dr. Donald Kassebaum is Head of the Medical Intensive Care Units at the Medical School Hospital and University Hospital North (Multnomah Hospital) and I am sure he would be most interested in the direction of your studies and willing to support your project through access to the MICU's and consultation. Since he is responsible for those areas, he would be the appropriate person to contact regarding permission to carry out the studies.

Regarding your specific questions, I am not familiar with Hildebrandt's method of PAQ, although heart rate and respiration are certainly two of the most basic physiological parameters and should give some representation of response to the physical environment. We now have facilities for continuous monitoring of heart rate and ECG on magnetic tape and it might be interesting to do this in selected patients and observe their response in relation to known variations in light and activity around them. Since we use these instruments for research purposes as well as clinical observation, I think it might be possible to arrange this without significant cost or difficulty. In a similar way, we have an Electronics for Medicine recorder in each MICU and it would be possible to rig this for plethysmographic measurements of respiration, although it would be excessively expensive to record over a protracted period of time.

I would agree that more extensive physiological measurements, such as EEG, which would be very interesting, might be too complicated and expensive to undertake. In the past, Dr. Paul Blachly did cardiac output and blood volume determinations after valve replacement and, although this was fairly complex and time-consuming, we noted a definite relationship between agitation in the patient related to postoperative hallucinations, discomfort, et cetera and rather extensive changes in the hemodynamic parameters.

Dr. Blachly has been interested in responses by patients to stress situations in the postoperative unit for a long time and has done a number of other psychometric studies. I think he would be an excellent person to contact regarding his thoughts about interviewing patients concerning their perceptions of noise, traffic, discomfort, lighting, et cetera. He might be willing to participate himself or assign a trainee in psychiatry or psychology to developing a standard approach toward evaluating the patient's perception of his environment.

Page Two
Mrs. Storlie
June 28, 1974

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Good luck in your project and let me know if I can be of any assistance.

Sincerely,

A handwritten signature in cursive script that reads "Frank Kloster". The signature is written in black ink and is positioned above the typed name and title.

Frank E. Kloster, M.D.
Professor of Medicine
Head, Division of Cardiology

FEK:hf

DEPARTMENT OF THE AIR FORCE
SCHOOL OF HEALTH CARE SCIENCES, USAF, (ATC)
SHEPPARD AIR FORCE BASE, TEXAS 76311



24 June 1974

Frances Storlie R.N.M.S.
3811 N.E. 41st Street
Vancouver, Washington 98663

Dear Ms Storlie

I am very pleased and happy that you have contacted me for information and assistance in your endeavors to complete a study for your dissertation for PhD in Urban Studies from Portland State University.

I would like to recommend that you read my presentation (pp 81-92) and its critique (pp 92-97) in the fifth series entitled "Communicating Nursing Research". If it is not in your university library, a copy may be purchased for three dollars from:

Western Interstate Commission for Higher Education
P. O. Drawer "P"
Boulder, Colorado 80302

My article is a condensed version of my 122 page thesis and the constructive critique by Dr. Lewis identifies some of the pitfalls that you might wish to avoid. The article describes the instruments used for measurement.

On first reading your interest in the environment, it would appear that you are attempting to cover many parameters which may affect patient heart rate and rhythm. Perhaps these many variables (noise, traffic, lights, odors) are considered appropriate for the doctoral level of study. It might appear that you would like to study the universe, not just a segment.

You can measure sound levels within the rooms using special equipment which you might obtain from an environmentalist. Several questions come to my mind: How do you plan to control the amount of persons (traffic)? Into and past the room? What if patient goes into cardiac arrest and there is a need for more persons in the room? Do you plan to control the times and length of use of artificial lights? Are you thinking of this as sensory alteration? How will you know if patients have the ability to smell? Will you have air samples taken for analysis of certain gases and odors?

I would like to recommend that you discuss your interest in this area with a physiologist, an environmentalist, psychologist and an authority in acoustics.

If I may be of further assistance, please do not hesitate to write ¹⁹⁶
me. I will be transferred to the Philippines within the near future.
My new address will be: Colonel L. A. Marshall
USAF Hospital Clark (PACAF)
APO San Francisco 96432

and effective after 11 August 1974. In fact, I would appreciate feed-
back concerning progress on your study.

Much Success

A handwritten signature in cursive script that reads "Louise A. Marshall". The signature is written in black ink and is positioned above the typed name.

LOUISE A. MARSHALL, Colonel, USAF, NC
Chief, Department of Nursing

University of the Witwatersrand, Johannesburg



Department of Medicine

Medical School 197

Hospital Street, Johannesburg, South Africa

Telephone 724-1561

Ms. Frances Storlie, R.N.,
3811 N.E. 41st Street,
Vancouver,
Washington 98663,
U.S.A.

Your reference

Our reference

Date

15th October, 1974.

Dear Ms Storlie,

Thank you for your letter of September 24th, and the very interesting problems you pose. The possible effects of noise on a heart rate had never entered my thoughts until your letter arrived. I have given the matter considerable thought, and feel that your approach is the correct one. Irrespective of the underlying disease processes or other parameters affecting the heart rate, I would have thought that once you have a basic rate that is all the control you really need. Indeed, I would not even exclude complete A-V block, for it is possible that the idioventricular rate may also be affected. Are you also approaching this on an experimental basis, i.e. to say do you have a noise-making apparatus which you subject your patients to?

I regret that my information is obviously of such little use, but please feel free to write to me at any time in the future, and I will do my best to be of some assistance. I wish you all the luck with your doctoral thesis.

With very best wishes.

Yours sincerely,

A handwritten signature in black ink, appearing to read 'L. Schamroth', written over a horizontal line.

L. SCHAMROTH
PROFESSOR OF MEDICINE



Department of Veterans Affairs

Ministère des Affaires des anciens combattants

Please quote no / Citer le no

Westminster Hospital,
Box 5701, Terminal A,
London, Ontario.
N6A 4S2

January 30, 1975.

Miss Frances Storlie, R.N.,
3811 N.E. 41st Street,
Vancouver, Washington. 98663

Dear Miss Storlie:

I wish to acknowledge your letter concerning the study of noise and its effect on heart rate.

A number of years ago Dr. K. Ingham who worked with me was doing a study using a cardiometer which plotted the beat-to-beat cardiac interval so that one could follow, in the average rate per minute, the changes within the fraction of a minute.

Our interest was entirely in the analysis of a startle response and not a measurement of noise per se. We found that this measurement, even people walking in the hall, would produce a change in cardiac rate so that we ended up by having the patient in a sound proof room. Our problem was to get some sort of method to startle that was not in the nature of a condition reflex and what we had was a door bell under the bed which we could ring and study their cardiac response.

I would gather from your letter that this is probably not of particular interest to you in your current study.

Yours sincerely,

G. Edgar Hobbs, M.D., F.R.C.P. (C)
Chief of Service, Psychiatry

GEH*AMW

APPENDIX B

DATA COLLECTION SHEET

I. Epoch Number	40dba	HRR ₁	60dba	HRR _h	II. Subject # _____
1.					Diagnosis _____
2.					Site of Infarction _____
3.					Time of Sampling _____
4.					Room Number _____
5.					Medications _____
6.					Time of Sampling-Patients post 24 hours infarction _____

Means	N ₁	HRR ₁	N _h	HRR _h	Sex _____	Age _____
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$XN_1 + XN_h =$ noise differential ()

$XHRR_1 + XHRR_h =$ Heart Rate Response Score ()

Residence _____ Occupation _____

III. Subjects with M.I. Post 24 hours infarction

1.				
2.				
3.				
4.				
5.				
6.				

Xs	N ₁	HRR ₁	N _h	HRR _h
----	----------------	------------------	----------------	------------------

$XN_1 + XN_h =$ noise differential ()

$XHRR_1 + XHRR_h =$ Heart Rate Response ()

IV. Lowest Noise Level to which Patient Was Subjected _____

Comment _____

Heart Rate _____

Highest Noise Level to which Patient Was Subjected _____

Comment _____

Heart Rate _____

Past Medical History _____

PATIENT NUMBERDATA SAMPLING TIME

1.	11 to 12 Noon
2.	6 to 7 P.M.
3.	5 to 6 P.M.
4.	10 to 11 A.M.
5.	9 to 10 A.M.
6.	9 to 10 A.M.
7.	11 to 12 M
8.	12 M to 1 A.M.
9.	10 to 11 A.M.
10.	9 to 10 A.M.
11.	9 to 10 A.M.
12.	9 to 10 A.M.
13.	10 to 11 A.M.
14.	5 to 6 P.M.
15.	7 to 8 A.M.
16.	5 to 6 P.M.
17.	9 to 10 P.M.
18.	9 to 10 P.M.
19.	5 to 6 P.M.
20.	12 M to 1 A.M.
21.	8 to 9 A.M.
22.	5 to 6 A.M.
23.	5 to 6 P.M.
24.	5 to 6 P.M.

PATIENT NUMBERDATA SAMPLING TIME

25.

7 to 8 A.M.

26.

8 to 9 P.M.

27.

8 to 9 A.M.

28.

5 to 6 P.M.

29.

9 to 10 P.M.

30.

10 to 11 A.M.

APPENDIX C

