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ESSENTIAL HYPERTENSION AND ANXIETY, DEPRESSION AND ANGER

City University of New York PH.D. 1981

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by

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A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

1981

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirements for the degree of Doctor of Philosophy.

2/5/81
date

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Abstract

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by

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This study investigated the possibility that essential hypertensives experience higher levels of anxiety, depression and anger than normotensives. Because of the controversy surrounding the role of subjective distress, the physiological literature focused on how subjective distress is mediated was reviewed. Enough physiological evidence was found to lend support to the hypothesis that hypertensives could be experiencing higher levels of subjective distress. However, despite the fact that a similar position had been taken in the psychosomatic literature, empirical investigations failed to identify significant differences between hypertensives and normotensives. Examination of the scales used in these studies suggested that they were not clearly focused on the variables relevant to the hypertensive personality; thus in order to advance work in this area a new instrument was required and the Subjective Emotional Stress Inventory (SESI) was developed.

The SESI consisted of three scales, an Anxiety scale, a Depression scale and an Anger scale. The scales incorporated

several new features, perhaps the most important of which was the degree of specificity: items were clearly focused on persons or events most likely to be perceived as triggers and/or targets of the three emotions; as a result items examined feelings associated with parents, spouses, bosses, etc. In addition, in order to measure frequency of these feelings more accurately, response alternatives had to move beyond the vague 'Always' 'Often' 'Sometimes' etc. Response alternatives took the form of 'Several times a Day,' 'Every Day,' 'Every other Day,' etc.

Validation statistics were computed on the three scales. Alphas of internal consistency were .82, .70, .85 for the Depression, Anxiety and Anger scales respectively, based on a sample of 101 subjects. Evidence of the scales' sensitivity to subjective distress came from discriminations made between working and non-working college students, college students living and not living with their parents and smoking and non-smoking college students. T-test statistics showed that college students who were not working, college students who lived with parents and college students who did not smoke reported higher levels of subjective distress.

The research sample consisted of 108 subjects of whom 71 were over 26 years and 37 were under 23 years, with just over one-third of each age group being hypertensive or labile respectively. The research design called for seated bilateral blood pressures and heart rates to be taken before, during and after two self-administered questionnaires, the

Psychiatric Epidemiology Research Interview (PERI) and the SESI.

Data analysis consisted of ANOVAs which controlled for age, with covariance on possible confounding variables, sex, ethnicity and weight. The results revealed significant differences between hypertensives and normotensives on each of the three scales of the SESI, Anxiety, Anger and Depression. Of the eight hypotheses associated with the SESI five were supported, indicating that in this sample, hypertensives tended to experience all three types of subjective emotional distress more frequently than normotensives.

No significant differences obtained on the PERI between hypertensives and normotensives, but there were significant differences based on age and ethnicity. The implications of these results for this area of research are discussed.

Acknowledgements

I should like to thank first of all my husband Dr. S. Dayo Olaoye, not only for his unfailing support but perhaps most of all for his dedication to scholarship and excellence that we both share. I also must thank my daughter Temitayo, whose curiosity and boundless ebullience has provided me many a time with much needed energy to push on. To my parents John and Olga Henry I owe a word of thanks and particularly to my aunt, Ivy Roberts, who not only insisted upon, but inspired in me academic excellence from early childhood.

I must need express my appreciation to each member of my committee, Professors Samuel Messick, Chairman, Barbara Dohrenwend, Alden Wessman, Morton Bard and Bert Zippel, for their interest, their help and encouragement with the various details and drafts of this dissertation. The opportunity to work closely with Dr. Messick and with Dr. Dohrenwend, who also served as chairperson initially, are valuable experiences that have made and will continue to make their mark on my academic career.

Several other persons contributed in important ways to the execution and final presentation of this dissertation. I am very grateful to each of them. Dr. Gladys Egri was in large measure responsible for the decision to engage in scale construction. Dr. Godfrey Burns was most helpful in affording me access to hypertensive patients for this study. Gwen Evans and Cynthia Simon were the two nurses, who in taking

pulses and blood pressures, gave generously of their time and skills. Shirley Cooper was a most able coordinator of data collection, and to my typist Susan Kirby much appreciation is extended.

Gratitude also needs to be expressed to friends like Dr. Blanche Blank, Peter Fenton, Joel Morgovsky, Ronald Bonnett and Alison Chambers, who through persistent and timely remembrances helped to keep the fires of friendship glowing; to the faculty and staff of Brookdale Community College appreciation is extended for their understanding and support.

Finally, at some point dissertation work tends to become a quest to find out more and more about less and less, and one feels oneself losing touch with certain larger realities, realities that among other things control levels of vitality and commitment towards one's own life. In maintaining a balanced perspective, and in keeping the sparks of creativity burning, poetry writing became an important vehicle in my life. A final acknowledgement then is made, to all those, seen and unseen, who helped me in spite of many dry spells from time to time to reach deeper levels of understanding and experiences of the following:

I love the delicate dawning
Of fresh thoughts scarcely spawning
The first fledgling forms or notions
Of feelings, longings, yearnings,
Shy diffident, unyielding
To usual manipulation;
Thoughts which in keen and cloistered sequestration,
With tireless time and gentle cultivation,
Weaves from doubt and elusive contradictions,
Profound and poignant integrations.

-- From "On Dawnings"

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CHAPTER I: INTRODUCTION

Statement and Significance of the Problem

The major purpose of this research is to establish whether persons suffering from essential hypertension experience higher levels of subjective distress than normotensives. In particular, attention will be focused on levels of anxiety, depression and anger. Essential hypertension is a medical disorder without known cause afflicting approximately 25,000,000 Americans. Essential hypertension is without cure; current medications can only offer control (Veterans Administration Cooperative Study, 1967, 1972; Gifford, 1974). Nonetheless, hypertension is the most common of the cardiovascular diseases, being the leading complication in deaths caused by myocardial and atherothrombotic brain infarction. Data collected on a random sample of the U.S. adult population ages 18 to 79 years, by the National Health Examination Survey of the Public Health Service, indicated that about 9 per cent of white adults and 22 per cent of black adults had hypertensive disease. The criterion employed was that of the World Health Organization: a diastolic pressure of 95 mm Hg. If a lower cut off point is used, that is a diastolic of 90 mm Hg, the prevalence rates are about 16 per cent for white adults and 30 per cent for black adults (U.S. Dept. of Health, Educ. and Welfare, 1966).

The goal of measuring comparative levels of subjective distress in essential hypertensives is motivated by a need to generate evidence with respect to the possible causal role of subjective distress in essential hypertension. However, teasing out causal threads is always a complex problem and those related to essential hypertension are many and tangled indeed. Nonetheless, the morbidity and mortality statistics quoted above provide this problem with some urgency.

The complexity associated with establishing causality is somewhat reduced by first of all posing the question of whether or not there are physiological pathways which can convert subjective distress into somatic disorders, such as essential hypertension. And second, seeking to establish the probability that these pathways operate frequently. If answers to these questions can be established with some reasonable accuracy from the medical literature, a foundation for pursuing research focused on relative levels of subjective distress in essential hypertension will be created.

It should also be noted that although the psychosomatic literature has posited particular personality characteristics that may be associated with essential hypertensives, empirical investigations (Ostfeld, 1967, 1973) have been unsuccessful in establishing significant differences between hypertensives and normotensives.

Thus the major goal of this research has a double focus, first to clarify the causal role of subjective distress in essential hypertension and second to test the related hypothesis of whether or not essential hypertensives experience higher levels of anxiety, depression and anger. The significance of this research, however, broadens to include the construction of a psychological inventory, designed to measure anxiety, depression and anger. While full justification for its construction is given in the appropriate chapter, scale generation was perceived to be a critical factor in carrying forward the boundaries of knowledge in this area. Ostfeld (1967) reports repeatedly no success in using well standardized scales such as the Minnesota Multiphasic Personality Inventory, the 16 Personality Factor and the Rorschach, to distinguish hypertensives. Thus partial validation of an inventory, as well as its implementation on the population of interest, lend further significance to the present research.

In summary, the problem of examining levels of subjective distress in essential hypertensives, through strategies such as review of the medical literature to clarify the nature of possible causality, and innovative features, such as inventory construction, is capable of filling an important research gap in the psychological literature.

Overview of Study

The current study will review the medical literature in an attempt to establish a firm foundation for psychological research on the relationship of subjective emotional stress to essential hypertension. In particular, the major risk factors and the amount of variance they account for are reviewed. In addition, anatomical and nueral evidence of stress pathways in the body are reviewed and the likely frequencies of their activation. Finally, from the medical literature, the success of current therapeutic drugs are reviewed with respect to the extent that they provide evidence of the over-activity of the central nervous system. In addition to review of relevant medical literature, the psychological literature will also be reviewed: specifically, previous research comparing the effects of environmental stress on hypertensives and normotensives as well as their typical subjective emotional reactions.

Because the need for a new instrument was identified in the review of the psychological literature, the study will be further developed by describing an instrument, the Subjective Emotional Stress Inventory (SESI), designed to focus more clearly on characteristics hypothesized to be related to hypertensives. The research design of the present study is then described, followed by its results, which yield significant differences between hypertensives and normotensives on the variables of interest, anxiety, depression

and anger. The implications and limitations of the study then will be discussed as well as directions for future research.

CHAPTER II: REVIEW OF RELEVANT THEORY AND RESEARCH

Strategy for Reviewing the Literature

A review of the literature related to the problem of levels of subjective distress in essential hypertensives could be limited to studies which simply examined correlational relationships between stress and essential hypertension. However, understanding would be significantly improved by an examination of the major developments regarding the discovery of linkages between the emotions and the endocrine and cardiovascular systems. This is based upon the familiar statistical observation that correlation does not mean causation. Thus in designing a substantive research study, prior review of the major anatomical and physiological links between the emotions and the body can provide critical information with respect to some of the causative factors. This information could not be gained by even the most exhaustive coverage of the correlational literature; thus review of the physiological factors involved will lend interpretative power to the data analysis. As Harrell (1980) points out:

> . . . not only is it necessary for hypertension researchers to keep abreast of findings related to the causal mechanisms that are the focus of their research efforts, but they must also be mindful of findings related to often remote but possibly interacting controlling systems.

Equally germane is Paul Meehl's observation (1978):

> Theories in "soft" areas of psychology (clinical, counseling, social, personality, community and school) lack the cumulative character of scientific knowledge. They tend neither to be refuted or corroborated but instead merely fade away as people lose interest.

Meehl's observation may be well taken, research that is not well anchored is likely to be transient. Thus without some clear perspective from the medical literature with respect to the real role of subjective distress in essential hypertension, null results, if obtained, are likely to be interpreted as an indication that research in this area should be terminated.

Thus the physiological literature will be reviewed briefly in order to show that: (i) Much variance with respect to essential hypertension remains unexplained by risk factors. (ii) Anatomical, neural and humoral evidence point to the possibility that an important role can be assigned to subjective distress in essential hypertension. (iii) Current success of antihypertensive drugs provides persuasive evidence with respect to over-activity of the central nervous system.

Major Risk Factors

Cigarette Smoking

Heavy cigarette smokers, that is persons smoking 20 or more cigarettes daily, have long been observed to die from all causes more rapidly than non-smokers (Pearl,

1938; Roth, 1951; Thomas, 1956; Doyle et al., 1962). Two lines of evidence are important with respect to the effects of cigarette smoking on cardiovascular dysfunction and disease. First, experimental studies measuring the acute circulatory effect of cigarette smoking and second, epidemiological studies which relate smoking habits in defined populations to their later experience of disease.

The measurable factor in inhalation of cigarette smoking that has been most focused upon is amount and rate of nicotine absorption. An effect of nicotine on the cardiovascular system observed by Doyle (1969) and others, is the release of catecholamines from the sympathetic ganglia throughout the body, but notably in the heart and blood vessels. There is consequently a slight increase in heart rate, blood pressure and cardiac output, though muscle blood flow is little, if any augmented. Catecholamine concentration in the brain also increases. However, according to Doyle (1969), these circulatory effects are acute and apparently non-cumulative. He notes no evidence for chronic nicotine intoxication.

The effects of the catecholamines on the metabolism following cigarette smoking is also noteworthy. After cigarette smoking there is vigorous mobilization of free fatty acids and Doyle (1969) finds it conceivable that there may be a cumulative effect on cholesterol metabolism since heavy smokers have been observed to have slightly but consistently higher serum cholesterol concentrations than non-smokers.

With respect to the second line of evidence, epidemiological observations relating cigarette smoking in a defined population to ultimate experience of disease, one of the most interesting studies was conducted by a blue ribbon committee selected by the Surgeon General of the U.S. Public Health Service (Friedman & Rosenman, 1974). This committee after appropriate deliberation reported that although male cigarette smokers have higher death rates from cardiovascular disease than non-smoking males, they were not prepared to state that this statistical association had causal significance: first, the Surgeon General's committee noted that the same studies identified the heavy cigarette smoker as vulnerable to cardiovascular disease, also revealed that pipe smokers and cigar smokers did not appear to suffer a greater vulnerability than non-smokers. This puzzling observation, they thought, could not be attributed to absorption of less nicotine since this substance is as easily absorbed from the mouth as from the lungs. With this question unanswered, the committee was hesitant to assert a causal relationship.

An equally important observation which contributed to the committee's conservative posture was that a substantial fraction of patients with cardiovascular problems do not smoke cigarettes. Thus even if cigarette smoking were a causal agent, it is not the sole one.

The most important fact that needs to be noted for the purpose of this study is that cigarette smoking, although it

may be regarded as a contributory factor, does not by itself, at this time provide an adequate causal explanation of essential hypertension.

Overweight and Obesity

Weight is thought to be related to blood pressure, primarily because of the perceived relationship with cardiac output and blood volume.

It has been estimated that for each pound of adipose tissue, capillaries equal to a mile in length have to be generated. Thus weight of this kind can cause an increase in two factors affecting blood pressure, volume per stroke and force of each stroke. But body weight is not determined only by body fat, but by skeleton and muscle mass, and many essential hypertensives are not overweight. An influential statement of a positive relationship between overweight and hypertension and coronary heart disease published by the insurance industry some years ago has led many people to think of weight as a major causative factor. However, as Blackburn (1976) points out, most of the systematic data in other population studies reveal much lesser degrees of association between overweight and coronary risk than the insurance data.

This may not be surprising when one considers the fact of biochemical individuality. As Lykken (1968) writes:

> A collection of stomachs, livers or hearts obtained from a random sample of normal healthy human beings, may appear so extraordinarily diverse in size, shape and internal structure as to lead the uninitiated to

> conclude that they must have been obtained from several species of animals. Some normal hearts can pump 11 litres of blood per minute, while others only 3; the size capacity and branching patterns of the blood vessels show a similar capacity.

What did some of the systematic studies uncover with respect to weight and essential hypertension? Stamler (1974) agrees that obesity and hypertension are interrelated. In addition, he observes two noteworthy aspects of the weight-blood pressure relationship. His data reveal that (i) the higher the relative weight initially, the greater the tendency to elevated blood pressure by age 50. And (ii) both for those lean in youth and slightly overweight in youth, the greater the gain in weight from young adulthood to middle age, the greater the probability of high blood pressure.

Tyroler and Cassel (1974) have indicated a protective role of weight reduction, that is, they calculated a 40 per cent reduction in essential hypertension if obesity is prevented. Keys (1970), however, in his Seven Countries study, found that the correlation between blood pressure and relative weight or fatness is not large, though of significance--correlations were on the order of .17 to .24. Keys interpreted this to mean that less than 5 per cent of the variance of blood pressure is explained by relative weight or fatness.

Thus for essential hypertensives who are not obese, weight does not provide a definitive solution, and for those

who are, although it may be a significant part of the answer, there remains much variance left unaddressed.

Diet - Saturated Fats

Diet, in particular, intake of cholesterol, has been suspected as a prime cause of cardiovascular disease. Blackburn, for example, reports that

> correlations between average fat composition in the diet with average serum cholesterol levels found in population are strong, where diets are analyzed reliably and chemically and repeatedly during all seasons of the year. High order correlations (0.9) exist between habitual diet fat components and population averages of serum cholesterol level. (1976, p. 128)

Blackburn observes, however, that correlations can be less strong within given cultures because individuals eating the same diet can have different serum lipid levels due to 'intrinsic' individual regulation of lipids. This fact, however, should not negate the main or overall effect of diet on individuals and populations. In addition, another factor which may serve to cloud the issue is the relative homogeneity of the American diet.

The American diet tends to be a high fat, high calorie diet second only to that of East Finland's (Seven Countries Study, 1970). According to Friedman and Rosenman (1974), the average serum cholesterol of the American adult is between 225 - 250 mg./100 ml. While most epidemiologists regard levels of 275 mg./100 ml. or higher as abnormal, Friedman and Rosenman take the position that the setting of this standard was influenced more by current levels of serum

cholesterol among Americans than by a proper consideration of what the levels ought to be. Thus they suggest that serum cholesterol levels above 175 mg./100 ml. are abnormally high (1974).

The significance of cholesterol intake for hypertension lies in the deleterious effect that the presence of cholesterol in blood serum has on the arteries. One question, however, may be asked: Does hypertension occur in the absence of high serum cholesterol levels? Kannel (1974) concedes the position taken by Altschule (1970), that cardiovascular diseases can form in persons with low lipid values. However, it is equally clear to Kannel that both in animals and in man, the process unequivocally accelerates in proportion to blood lipid level.

Studies which examine evidence on the possible causal relationship between ingested cholesterol and on hypertension and other cardiovascular diseases (Keys, 1970; Kannel, 1974) indicate that for most populations studied, there is a relationship. As Kannel (1974) summarizes: "That an association exists between cholesterol and atherosclerosis is incontrovertible. That it is a causal association is more difficult to prove." A somewhat similar position may be taken with respect to hypertension, although a stronger relationship has been developed in the literature between cholesterol and coronary artery disease; while an association exists, a causal role is not indicated, in hypertension.

Diet - Salt Intake

Several pieces of evidence suggest that sodium is at least a contributing factor in essential hypertension. According to Pickering (1968), first, it seems that the sodium-potassium flux in and out of the smooth muscle cells of small arteries may be important in the tone of small arteries and arterioles, and thus in determining arterial pressure. Second, one notes the success of rigorous low sodium diets in reducing arterial pressure. Third, benzothiazide diuretics reduce arterial pressure by reducing peripheral resistance.

A basic question with respect to sodium levels is whether or not there is any evidence that there is a change in sodium metabolism in persons with essential hypertension. Cottier (1960) points out that it has been well established by several recent investigators (Baldwin, Biggs et al., 1958; Birchall, Tuthill, et al., 1953), that patients with essential hypertension show an altered renal excretory response to sodium chloride loading.

From research conducted by Dahl (1967), however, an interesting picture emerges with respect to salt intake in rats. In general, Dahl has accumulated much evidence with respect to possibility that there might be a correlation between amount of salt intake and prevalence of elevated blood pressure in communities. Dahl (1967) proposes that based on clinical and experimental data, pointing in the

same direction, chronic excess salt should be considered as playing an etiologic role in hypertension. Dahl is careful to point out, however, that both in human and in animal experiments, it has been repeatedly observed that some individuals ingested large amounts of salt without developing hypertension.

Two basic methods have been used to measure salt intake in humans, an interview or 24-hour urine values for sodium excretion. The Framingham study rated their subjects' responses in diet interview according to three categories: (i) Low salt intake--never adds salt at the table. (ii) Average intake--add salt if necessary. (iii) High intake--routinely adds salt before tasting. No relationship between blood pressure and these salt intake ratings were found. Hardly surprising, for as Dahl (1967) points out the interviews are "pretty valueless" for epidemiologic purposes. For instance, in Japan, he notes, where "you can't eat" a low salt diet, food is well salted before being brought to table, using these questions everyone would have been on a low salt diet.

Using the second method, 24-hour urine values of sodium excretion for 185 men, were correlated with blood pressure, in the Framingham study, but no evidence was uncovered with respect to a relationship between these two variables.

From his cross-cultural studies, however, Dahl (1960) has presented some compelling evidence with respect to the

relationship of salt intake to prevalence of hypertension in five geographic areas. Significant differences exist in prevalence of hypertension among Alaskan Eskimos who consume on the average, less than 4 grams of salt per day and Northern Japanese farmers who consume on the average 26.3 grams per day. Figure 1, below, is reproduced from Dahl's 1960 article describing his results. As may be observed, as salt intake decreases from North to South in Japan, so does the prevalence of hypertension. Also noteworthy is the fact that in Japan, where salt intake is high, since 1951,

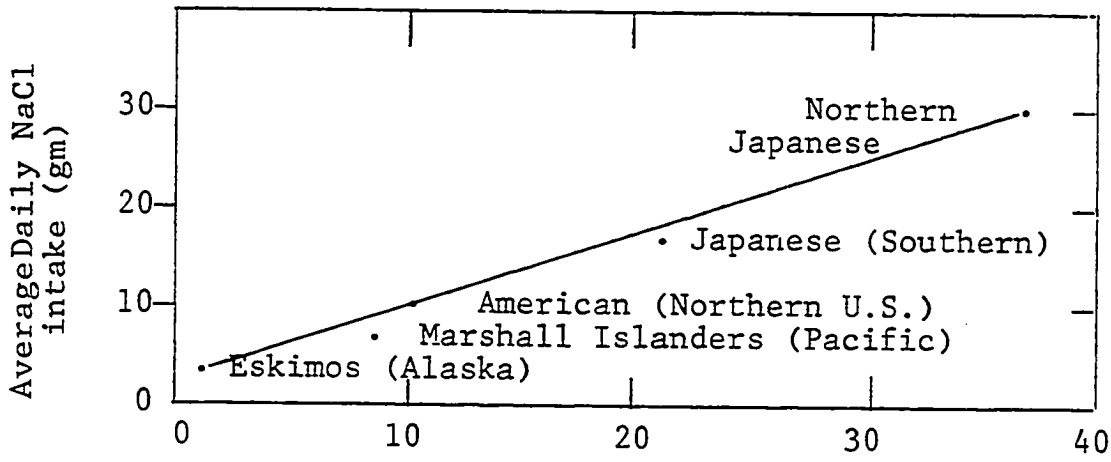

Figure 1

Correlation of average daily salt (NaCl) intakes with prevalence of hypertension

cerebrovascular complications of hypertension have been the leading cause of death.

With respect to the etiological role of salt in essential hypertension, we concur with Dahl's (1960) summary position:

> Among societies or groups habitually consuming low salt diets (perhaps 5 gms of NaCl per person per day or less), essential hypertension will be uncommon. Among societies or groups consuming high amounts of salt (in excess of 10-15 gms per person per day) essential hypertension will be common. Individual susceptibility will determine which one individual in the group will develop the disease . . . It does not mean that salt is not involved, but rather that not only salt is involved. (1960, p. 64)

Diet - Caffeine

Caffeine is a drug used by millions daily. Caffeine generally causes a slight rise in blood pressure associated with increased cardiac output (Goodman & Gilman, 1958). However, evidence of any cumulative effect on blood pressure producing fixed elevations is absent (Kagan et al., 1962).

Neurochemical Mechanisms Which Mediate Subjective Distress and Their Effects on the Cardiovascular System

Neurochemical Mechanisms Controlling the Release of Catecholamines

Walter B. Cannon (1929) was among the earliest investigators to systematically pursue the question of if and how emotions such as pain, fear and rage have any resultant effects on the physiological system. The nature of Cannon's evidence was generally laboratory-experimental, rather than clinical-pathological. Probably the major limitation of his

work was that most of it was done with animals and therefore possibly of limited generalizability. Nonetheless, Cannon's work and that of some of his contemporaries provides an important watershed in the history of research on physiological responses to strong emotions.

At the most fundamental level, Cannon and others were aware of the many changes associated with sympathetic activity and they sought to establish with as much precision as technological advances in the early part of this century permitted, (i) the extent to which adrenalin and noradrenalin are released in moments of great emotion and (ii) the nature and degree of their influence on various parts of the body. Although Cannon (1929) examined the effects of these substances on various parts of the body, this review will be concerned only with the effects on the cardiovascular system.

The most basic piece of evidence with respect to subjective emotional states affecting the cardiovascular system is the presence of an anatomical structure which is able to mediate the relationship. Thus early in his work Cannon (1929) described the general organization of the nerves involved in emotions. The basic division between type of control and sources and speed of innervation are made. Thus the well known contrast was made between smooth muscles and glands which are not under voluntary control and whose responses may be measured in seconds, and the skeletal or striate muscles which are under voluntary control and whose frequency of reaction is more appropriately measured in

thousandths of a second. In addition it was noted that the nerve fibers affecting the muscles have cell bodies which lie within the brain and spinal cord; while the smooth muscle and glands are not directly innervated by the central nervous system, but have an extra set of neurons interposed between the cerebrospinal nerves and the viscera. With respect to the glands however, there is an important exception, the adrenals; they are not indirectly innervated.

The second piece of anatomical data that is relevant to this research problem is the division of the autonomic or the non-voluntary division of the nervous system into the thoraco-lumbar or sympathetic division and the cranial and sacral or parasympathetic division. The most striking difference between these two divisions is the wide distribution of fibers from the sympathetic division as compared to the limited distribution of fibers from the parasympathetic.

Equally striking is the relatively antagonistic or opposing effects of the two systems. The sympathetic system is able to reach all parts of the body that are necessary for alert posture: sympathetic fibers lead to the pupils of the eyes where they can cause dilation; fibers also go to the heart where they can cause increased heart rate; they go to the peripheral arteries and arterioles of the skin where they can cause constriction; fibers from the sympathetic region also travel the entire length of the gastro-intestinal canal where they can inhibit digestive activity; sympathetic fibers also reach the genito-urinary tracts where they can cause

contraction of the smooth muscle of the internal genital organs and relaxation of the bladder. And, finally, sympathetic fibers reach the liver where they can cause release of various starches stored there.

The last anatomical note to be made here concerns the adrenal gland, composed of an outer portion called the cortex and a central portion called the medulla. Of critical importance is the substance which can be extracted from the medulla known as adrenalin. This substance is noteworthy since even in small amounts, it affects structures innervated by the sympathetic division in the same manner as if they were receiving impulses. The effects are produced by direct action on the organs themselves, thus the effects may be observed after particular structures are removed from the body and kept alive artificially. Also as noted before, the adrenals are exceptional glands in that they are connected by preganglionic fibers to the sympathetic division, thus the anatomical structure allows for the possibility that stimulation of the sympathetic division initiated by nervous discharges can automatically be augmented and prolonged by the chemical effects of secretions from the adrenal medulla.

Having established the presence of an anatomical structure which could mediate effects of subjective distress via the sympathetic division, a necessary but insufficient cause, two problems need to be addressed: (i) the physical source or origin of strong emotions and (ii) whether or not they result in the activation of the sympathetic division and the adrenal glands also.

Cannon and several of his contemporaries (Bard, 1928; Bechterev, 1887; Pieron, 1928) engaged in a series of experimental studies designed to shed light upon the problem of the source or origin of the type and quality of behavior recognized to constitute the emotions. The vigor of their research was stimulated at least in part by the claim of James (1884) and Lange (1885) that the type and quality of behavior we describe as emotions originates in the viscera and/or innervated muscles. This did not seem at all reasonable to Cannon and other workers, whose evidence had led them to believe that the seat of emotions lay in the brain. An important question for them, however, was in what part of the brain did these mechanisms reside. In the newly developed cerebral cortex, or in the more ancient part of the brain, the brain stem.

Cannon reasoned that since emotions such as fear and anger are associated with survival, that is with attack or flight from danger, it seemed likely that the center for these emotions, though held in check by a dominant cortex, might be located in the more ancient brain stem. Accordingly Cannon and Britton (1925) undertook to investigate whether or not these emotional centers in the brain stem would be capable of displaying typical emotional activity if the cerebral cortex were removed. Decortication experiments were performed on cats and Cannon and Britton observed what they labelled 'sham rage' in the cats upon recovery from the operations. The pseudo-emotional phenomena included thrusting

and jerking of restrained limbs, arching of the back, displaying of claws and snarling and attempts to bite. Cannon and Britton noted that these were all actions controlled by the skeletal muscles. In addition, they noted as more typical and longer lasting the effects of the sympathetic nerve impulses on the viscera. These included dilation of the pupils, rapid heart beat, erection of tail hairs, high blood pressure, outpouring of adrenalin and increase of blood sugar, up to five times the normal concentration. This pseudo-emotional state would continue at times for two to three hours.

Thus Cannon may be credited with establishing several critical features concerning the problem of physiological responses to stress: not only did he find anatomical structures that can mediate powerful changes throughout the body, but he also observed emotional centers in the brain that control these responses. In particular, the reflexive and dual control features were identified and the physiological conditions that they are likely to bring about. Cannon (1929) went on however to ask a further question, given the fact that the control of the body by the emotions is supposed to be of great utilitarian value, under what circumstances does this relationship become dysfunctional, that is, for example, over stimulating the cardiovascular system and causing physiological damage?

For Cannon (1929), the answer lies at least in part in the reflexive nature of the emotions. He observed, for

instance, that persistence of an adequate stimulus for a reflex action is sufficient cause for persistence of action. He noted that if an irritant affects the eye, tears flow; if the tears are able to wash the irritant away, they cease to flow. Cannon argued that similar conditions govern the more complex responses associated with intense emotional experiences. The persistence of powerful emotional stimuli may be explained by (i) memory and (ii) vicarious associations created through classical conditioning. Thus Cannon wrote: "Our emotional reflexes, like the salivary reflex, become complicated by the conditioning of indifferent stimuli" (1929, p. 260).

In summary, then, Cannon presented a plausible explanation for persistent disorder of particular bodily functions in strong emotional reactions--the persistence of the stimuli which elicit the reactions.

Neurochemical Mechanisms Controlling the Release of Corticoids

Having reviewed Cannon's contributions with respect to the possible relationship between subjective emotional distress and the release of adrenalin, two critical questions arise: First, has the work of Cannon and his colleagues stood the test of time? And second, what advances have been made since then? In response to the first question, Cannon's contributions have held up over time, his animal experiments have pointed the way to fruitful research on man and continue to be the focal point of much methodological and theoretical

refinement. As Selye (1978) notes it may even be merely poor fortune that Cannon was not given a greater honor for his work, since Sir Henry Dale in England and Otto Loewi in Austria each earned a Nobel prize for demonstrating that both noradrenalin and acetylcholine are produced at nerve-endings and that it is through these antagonistic nerve secretions that the brain and the nerves exert their many actions.

In response to the second question--advancements in this area of research since Cannon--the work of Hans Selye comes to mind. For the last twenty-five years or so, Selye's name has been associated with the concept of stress; his research and publications in this area have virtually created a medical revolution. However, our concern with Selye is limited: basically we need to know how his work differs from, complements or contradicts the work of Cannon and his colleagues, and beyond that, whether or not he has made further contributions to the solution of the current research problem.

Selye's contribution of greatest significance to the current research problem is the discovery of a relatively independent or a second stress pathway leading to the release of hormones from the adrenal glands also. Thus after Cannon we can speak of the nervous mediation of stress reactions and since Selye we can also refer to the hormonal mediation of stress reactions. Thus although Selye's work differs from Cannon's, it is similar in an important manner, and certainly

at least with respect to the current research problem, complements Cannon's work.

Interestingly enough, both Cannon's and Selye's pathways originate in the hypothalamus and reach to the adrenal glands. However, Cannon's work focused on the secretions of the adrenal medulla, while Selye's focused on the products of the adrenal cortex. Selye's pathway is often referred to as the hypothalamus-pituitary-adreno-cortical axis. From Selye's research this sequence was uncovered: stressors act on the hypothalamus first, and cause the release of corticotrophin (CRF); this in turn acts on the pituitary gland close by and causes the release of any or all of three hormones: adrenocortiotrophin hormone (ACTH); somatrotrophic hormone (STH); and thyrotrophic hormone (TTH). While the last hormone, TTH, acts on the thyroid gland, the other two hormones, ACTH and STH, act on the adrenal cortex. Here these hormones serve to stimulate different types of corticoids. ACTH stimulates anti-inflammatory corticoids, or as Selye (1978) also refers to them, glucocorticoids, since they raise blood sugar. STH, in contrast, stimulates the pro-inflammatory corticoids or as Selye calls them, mineralocorticoids--they influence mineral metabolism, generally increasing sodium retention and excretion of potassium.

As with most medical research, most of Selye's work was performed on animals. The basic experimental strategy he pursued in establishing his hormonal stress pathway was that

of removing a gland, then injecting the animal with the hormone peculiar to that gland and observing whether or not the problem created is corrected. Not only was he able to establish evidence of his proposed hormonal pathway by this method, but it has been repeatedly confirmed and developed. Thus Persky (1962) notes that secretion of the adrenal cortex, hydrocortisone, stimulated by the pituitary hormone ACTH, circulates in the blood plasma and metabolizes to produce at least 11 different steroids. One group of these steroids are referred to as hydroxycorticoids and another group as the 17-ketosteroids. According to Persky (1962), elevated levels of hydrocortisone are found in the blood plasma of acutely disturbed or anxious patients; in addition he claims that the levels can be reliably increased by stress interviews. Persky, Grosz, Norton and McMurtry (1959) also observed that hypnotic induction of anxiety produced marked elevation in plasma hydrocortisone. And Handlon (1962) was able to produce a significant decrease in plasma 17-hydroxycorticosteroid levels in normal subjects through relaxing hypnotic suggestion or by having subjects watch a "bland" motion picture. Finally, Schwartz and Shields (1956) also noted elevated levels of hydroxycorticoids in medical students just after taking an important final exam.

The studies briefly reviewed above are but a few of the many studies which show how Selye's line of research has developed and continues to develop. Later reviews will show

the importance played by the technological development in the 1950's of plasma and urinary analysis capabilities on this area of research.

At a fundamental level, Selye has established an important line of research. It is now time to examine some relevant issues raised by Selye which may contribute to the solution of this research problem. In general, as a result of the major contributions of Cannon (1929) and Selye (1978), more personality research can become "psychophysiological" as Stern (1964) defines the term, that is, where physiological measures are used as dependent variables.

With respect to forging links between personality and physiology, then, Selye presents two interesting concepts: first, the notion of direct vs. indirect pathogens, and second, the notion of heterostasis. Selye (1978) finds it important to differentiate between direct and indirect pathogens. He points out that a direct pathogen would be operating if one immersed one's hand in boiling water or a strong alkali--the hand would be damaged regardless of any vital tissue reaction to these agents. In contrast, the problems which arise as a result of exposure to pollen or cat's dander and to which an individual may respond with an intense immunologic defense reaction are not directly caused by the external agent, but by the excessive adaptation mechanisms. In Selye's words, "It is such indirect pathogens that are the main causes of the so-called 'stress-diseases'

or 'diseases of adaptation,' such as high blood pressure, heart attacks, peptic ulcers, migraine headaches . . ." (1978, p. 84).

To some, Selye's statement just quoted above may seem quite premature, since although he has supplied some of the missing links, he certainly has not supplied all. However, he might be pointing in the right direction: while Cannon (1929) used the principles of conditioning to supply us with a psychological model of emotional overaction or distress, Selye (1978) has presented its parallel at the purely physiological level. As medicine and psychology become more refined, it might be anticipated that they will uncover various categories of disease causation. Thus with Cannon (1929) and Selye's (1978) line of thinking, a relatively new category of diseases have been distinguished.

The second concept that Selye presents that is of particular relevance to this research problem is that of heterostasis. Cannon (1929) introduced the notion of homeostasis, and demonstrated the presence of mechanisms which contribute to the maintenance of this state. Selye (1978) argues, however, that when the body is subjected to unusually heavy demands, the conditions presented by homeostasis are inadequate and the "thermostat of defense" must be raised to a higher level. This Selye (1978) refers to as heterostasis, the new steady state that is established by the stimulation of normally dormant defensive tissue reactions.

Heterostasis for Selye (1978) also generally involves artificial intervention in order to achieve the new balance between, for instance, catatoxic and syntoxic enzymes.

Since the turn of the century, however, physiologists have uncovered many feedback mechanisms throughout the body, and the endocrine system was no exception. Thus it has been repeatedly observed, first by the physiologist Dwight Ingle (1950), that if large amounts of cortisone are administered, the adrenals involute, because higher concentrations of corticoids in the blood depress the secretion of ACTH by the pituitary glands. Through this feedback mechanism the body is able to maintain steady corticoid concentrations in the blood. Thus it is because of this self-regulation that some investigators wondered if Selye could really make the claim that at times--albeit stress--that a real increase of corticoid levels could develop. However, Selye points out that in 1940, he was surprised by the observation that during stress, this moderator system is bypassed. According to Selye, alarm signals released from various cells of our tissues during stress can stimulate ACTH secretion, even when corticoid concentration in the blood reaches its highest attainable levels. For Selye (1978), this made much sense, for if the feedback mechanism was absolute, we would be unable to mobilize levels of corticoid concentrations in the blood necessary to maintain life during stress.

Finally, as was pointed out, there are two all-embracing pathways in the body which can contribute to a functional

common denominator--the nervous system and the blood vessel system. However, Selye (1978) observes that while cells, for example, may transmit messages by releasing parts of protein molecules in the blood, it is equally possible that the alarm signal is not a substance, but rather the lack of one. Furthermore, in instances where actual messages are transmitted, a single substance or even a single deficiency may not have the monopoly of acting as an alarm signal. Different substances may carry the same message, as long as these varied messages could be interpreted and tallied by one of the organs of adaptation, for example the pituitary. For Selye (1978), then, this is the frontier of stress research, identifying the nature of signals which cause the body to prepare and/or remain on the defensive, identification of the first mediators of stress.

Correlations of Catecholamine and Corticoid Secretions with Subjective Distress

In attempting to bring the work of Cannon and Selye up to date, the reviews and research of John W. Mason (1972) are perhaps the most comprehensive. This is the case because Mason, early on, became aware of the limitations of studying a single endocrine system in isolation and was able to pursue this complex line of investigation because of the recent availability of new biochemical methods for isolating the metabolic effects of hormones as well as for studying endocrine regulations. Although much of Mason's research concentrates on the physiological influences of the pituitary-

adrenal-cortical system, he has conducted research on the other major system, the sympathetic-adrenal medullary system, and stays abreast of developments in this area. Thus a comprehensive review of recent progress will focus on Mason's reviews and contributions, especially since an examination of interhormonal relationships and the interdependent functioning of the endocrine system is a better approximation of the physiological reality.

Research on the sympathetic adrenal medullary system lay dormant for many years after Cannon's important contributions, however, major biochemical discoveries opened up the field once more for study. In particular, Euler (1946) and Holtz, Credner and Kronberg (1947) demonstrated the natural occurrence of epinephrine in the body and shortly afterwards its role as an adrenal medullary hormone and chemical transmitter of the adrenergic nerves. Knowledge of two separate hormones made necessary the reassessment of adrenal-medullary function, particularly the question of whether the two hormones are regulated separately or whether they respond in a unitary fashion. In the cat the two hormones are almost equal, while in man noradrenalin predominates. They occur in different cells, which can be separately activated by hypothalamic stimulation, hypoglycemia and psychological strain. Blascko and Welch (1953) report that both in the adrenals and at the sympathetic nerve endings, adrenalin and noradrenalin exist in granules; these granules synthesize, store and release the catecholamines.

Differential effects of noradrenalin and adrenalin have been investigated at two main levels--the physiological level and the psychological level. At the physiological level, our major concern is the differential effects of adrenalin and noradrenalin on the circulation. These have been summarized very carefully by Pickering (1968). Some of his observations are based upon infusions of 10 - 40 µg per minute in adult human subjects, other observations, as will be noted, are from the work of other investigators:

(i) Equal amounts of each hormone result in much stronger subjective sensations with adrenalin: with the latter, there is a curious feeling of expectancy or apprehension, followed by hyperventilation, palpitations and tremor. With similar doses of noradrenalin these feelings are absent.

(ii) Adrenalin produces a sustained rise of systolic pressure with unchanged or lowered diastolic pressure, preceded by a transient rise and fall of both values. Noradrenalin, however, produces a rise in both pressures.

(iii) With adrenalin, the heart usually quickens and extrasystoles are not infrequent. With noradrenalin, the heart slows through vagal action and auriculo-ventricular dissociation may occur (Bancroft and Knozett, 1949; Barnett and others, 1950).

(iv) Adrenalin increases significantly cardiac output, noradrenalin produces no change (Goldenberg and others, 1948).

(v) Adrenalin increases muscle blood flow, the increase being large for a few seconds and later doubles. Noradrenalin produces little change.

(vi) Both substances blanch the skin, greatly decreasing skin blood flow.

(vii) Both adrenalin and noradrenalin decrease renal blood flow without greatly influencing glomerular filtration rate (Barclay, Cooke & Kenney, 1947; Barnett et al., 1950), but in both skin and kidney, adrenalin is more active than noradrenalin.

(viii) Adrenalin doubles liver flow while noradrenalin leaves it unaltered (Bern, Billing & Sherlock, 1951).

(ix) Adrenalin increases cerebral blood flow; noradrenalin decreases it slightly (King, Sokoloff & Weschsler, 1952). DeLargy et al. (1950) examined the effects on the circulatory system of mixtures of adrenalin and noradrenalin. They found that in equal parts, adrenalin dominates but balance of opposing effects are achieved with mixtures containing three to eight parts of noradrenalin for each part of adrenalin.

In summarizing the effects of these two substances, Pickering (1968) observes some important contrasts:

(i) Whereas adrenalin redistributes blood and increases cardiac output, noradrenalin has a fairly generalized constrictor effect on the blood vessels, except on the coronaries which it dilates, thus its pressor effect is due wholly to

a rise in peripheral resistance, while with adrenalin increased systolic pressure is due to the rise in cardiac output.

(ii) These different types of actions, Pickering (1968) thinks, may be correlated with the function of noradrenalin as the chief transmitter of the sympathetic nervous impulse and its reflex control of the circulation. On the other hand, adrenalin as the dominant secretion of the normal adrenal gland, prepares the body for emergency.

Having reviewed the effects of adrenalin and noradrenalin on the circulation, research investigating the psychological correlates of adrenalin and noradrenalin will now be examined.

Redgate and Gellhorn (1953) demonstrated the selective release of adrenalin and noradrenalin by stimulating different hypothalamic areas in the cat. And Folkow and Euler (1954) were able to do the same. Goodall (1951), having studied the catecholamine content of the adrenal glands of various African mammals, postulated that aggressive animals have higher concentrations of noradrenalin than do typically nonagressive animals. In addition, Mason (1972) notes that Cohen and Silverman (1959), in their review of earlier studies done in the psychosomatic field, suggested that anger, hostility or aggressive impulses were associated with blood pressure elevations, which Mason (1972) thinks might be construed as indirectly suggesting the association between noradrenalin release and these emotions.

However, reliable methods for measuring hormones were not available until the 1950s. Lund (1949, 1950) developed the chromatographic-fluorimetric method by which to measure adrenalin and noradrenalin in the plasma. Methods were also developed by Weil-Malherbe and Bone (1952, 1957). Methods for measuring adrenalin and noradrenalin in the urine were developed by Euler and his associates (Euler & Floding, 1955; Euler & Hellner, 1951; Euler & Lishajko, 1959).

The 1960s, not surprisingly, saw the completion of many studies relating various psychological factors to urinary catecholamine responses. These included responses to "aggression-provoking," "anxiety-provoking," bland and amusing films (Levi, 1964b, 1965). Urinary catecholamine levels were significantly lowered in response to the bland films. Catecholamine responses were observed in response to various stressful experiences: piecework (Frankenhauser & Kareby, 1962; Levi, 1961, 1963) as opposed to salaried work (Levi, 1964a); scholastic examinations (Bogdonoff, Estes et al., 1960; Bogdonoff, Harlan et al., 1959); sleep deprivation (Metz, Schaff & Grivel, 1960); emotional reactions to dental treatment (Schmid, Suss & Zicha, 1964); in physicians performing cardiac catheterizations (Ira, Whalen & Bogdonoff, 1963) and others.

The information with respect to the effect of psychological factors on sympathetic-adrenal medullary system, as developed by research in the 1960s, is based largely upon urinary analysis by chromatographic-bioassay method. As

Mason (1972) points out, the research of the 1960s clearly established that the sympathetic-adrenal medullary is highly sensitive to the influence of psychological factors. This conclusion can be based on two types of observations: (i) the stimulus-response relationships and (ii) the approximate direct quantitative correlations between intensity of emotional response and magnitude of catecholamine response. Thus Cannon's (1929) earlier observations are substantiated by more recent biochemical and bioassay methods.

Having reviewed the current status of the catecholamine levels in psychological response, we can now turn to a relevant question Mason posed in 1968: How many endocrine systems are responsive to psychological factors? Mason's main contributions are focused on the pituitary-adrenal cortical system. Although psychological influences on the adrenal medulla had been observed much earlier by Cannon, the pituitary-adrenal cortical system had been studied more extensively in relation to the influence of psychological factors. Selye's research, some of which was reviewed earlier, provided the impetus for many related endocrine studies. As with research on the sympathetic-adrenal medullary system, the discovery of chromatographic methods for biochemical measurement of cortical hormones marked a new era in this area of research; in particular the relatively specific and reliable measures of 17-hydroxycorticosteroids (17-OHCS) in blood and urine.

Mason (1972) notes that unfamiliar or "first experience" situations have been associated with substantial 17-OHCS elevations in man, indicating that this is a particularly potent stimulus to the pituitary-adrenal cortical system. Elevated plasma 17-OHCS levels associated with the experience of hospital admission in elective surgery patients were reported by Price, Thaler and Mason (1957). Sabshin et al. (1957) also observed plasma 17-OHCS elevations in patients on the first day that they were brought to a laboratory for psychoendocrine experiments.

Anticipation of uncomfortable states is also a stimulus associated with elevated adrenal cortical activity: Hetzel, Schottstaedt, Grace and Wolff (1955) described a case in which the fearful anticipation of exposure to the cold was associated with greater urinary 17-OHCS response than the cold exposure itself.

As might be expected, a proportion of the studies relating psychological responses to increased adrenal cortical activity have utilized experimentally induced stress. Use of motion pictures has been explored by some workers as a means of altering emotional states. Limited success has been reported: Euler, Gemzell et al. (1959) failed to find significant increases in mean plasma 17-OHCS levels in subjects observing censored violent movies; they did observe mild elevations in urinary 17-ketosteroid levels. Similar findings were observed with small groups watching war movies. There was a marked decrease in plasma 17-OHCS

levels which consistently occurred during the viewing of Disney nature films.

Hypnotic induction of emotional distress has also been attempted. Persky Grosz, Norton and McMurtry (1959) found that induction of hypnotic trance significantly reduced plasma 17-OHCS levels in both sexes, but subsequent induction of anxiety raised the levels in women by 75 per cent, but did not affect levels in men.

The other laboratory attempt to differentiate emotional responses in association with adrenal cortical activity is the use of interpersonally threatening situations. Korchin and Herz (1960) report two experimental tasks in which "shame" and "disintegrative" threats might be perceived by normal college students. Elevated plasma 17-OHCS responses resulted in both situations, with the disintegrative threats showing a greater response.

However, while laboratory situations generally have confirmed the sensitivity of 17-OHCS levels to psychological factors, and in particular that levels may be raised or lowered psychologically; Mason (1972) thinks that results have been somewhat disappointing. He points to the small changes in 17-OHCS levels evoked and the substantial range of individual differences observed. With respect to the latter, Mason suggests that either most subjects are able to maintain effective defenses against contrived stimuli presented, or the stimuli presented so far have not been of sufficient intensity.

Having reviewed studies which have been primarily concerned with the problem of a relationship between acute emotional responses and concomitant pituitary-adrenal cortical responses, we will examine studies conducted on normal human subjects who have been subjected to chronically trying situations.

Friedman, Mason and Hamburg (1963) in long term studies of the parents of leukemic children, made during the course of their child's illness, noted a number of marked adrenal cortical elevations associated with emotional responses to life events, but the persistence of characteristic individual differences in chronic mean basal urinary 17-OHCS levels over periods of months or years was an even more striking finding.

The question which now needs to be posed is: what does this review of the psychoendocrine relationships have to contribute to the problem of the etiology of essential hypertension? At the most fundamental level, it has been established that psychological stimuli, in particular emotional stimuli, do lead to increased levels of certain hormones in the system, as can be determined by plasma and urine analysis. But do hormones function in unison, with similar direction, duration, intensity and configuration of response curves? From Mason's work (1972), there seems to be evidence of a complicated pattern of organization: in response to psychological stimuli, if direction of hormonal change is used as a criterion, Mason (1972) distinguishes two general response groups: the levels of 17-OHCS,

adrenalin and noradrenalin, thyroxine and growth all increase initially; in contrast, levels of estrogens, androgens and insulin decrease. Mason also points out that there are sometimes significant differences in duration of response, following a single 72-hour avoidance or restraining session, adrenalin responses may be 2 days in duration, while thyroid responses lasted as long as three (3) weeks.

Evidence of the participation of psychological factors in endocrine regulation is overwhelming, and while this definitely complicates the servo-mechanism formulation of endocrine regulation, increasing knowledge of brain functions is revealing instances of higher level regulatory mechanisms superimposed on lower level mechanisms. Thus Malmo (1972) draws attention to the fact that the carotid sinus reflex, under normal circumstances serves a homeostatic function, guarding against excessively high blood pressure. However, this reflexive system is frequently overridden by cortical-hypothalamic and mid-brain mechanisms, for example, when an individual is working on a task under high motivation. Uvnas (1960) has provided a review of these brain mechanisms.

In summary, understanding and explanation of psycho-endocrine functioning is naturally complex, but must of necessity include consideration of cortical-hypothalamic and mid-brain mechanisms.

The Role of the Renin-Angiostensin-Aldosterone System in Essential Hypertension

Prior to 1970, most studies of the sympathetic nervous system in hypertension in patients classified them as having primary or secondary forms of hypertension. Recently, since about 1972, it has become possible to further sub-classify patients with primary or essential hypertension into groups with low, high or normal plasma renin levels, or those with low, high or normal plasma volume. These recent classifications are largely the contributions of Hans S. Brunner and John H. Laragh. Laragh (1977) advances the thesis that essential hypertension is not a single entity but a multiform disorder with variable history and prognosis. However, in all forms the characteristic hemodynamic abnormality is a persistent elevation of blood pressure in the arterial tree associated with increased vascular resistance, particularly at the level of the smaller arteries and arterioles. Laragh (1977) is concerned with the increasing evidence that all hypertension is related to abnormalities impeding normal homeostatic control of arterial blood pressure. In particular, his focus is on the renin/angiostensin/aldosterone hormonal system which regulates blood pressure by determining both volume, sodium balance and vasoconstrictor components. Laragh argues further that because of this very fundamental role all hypertensive disorders ought to exhibit either primary or reactive disturbances in the system reflected

by abnormalities in the hormones themselves or in their substrates, that is, in sodium or potassium balance.

The two main organs involved in the above mentioned system are the kidney and the adrenals, organs already identified in hypertensive states of various origin. However, since both of these organs are generally intact in essential hypertensive patients, evidence of dysfunction is sought in the renal-adrenal hormonal system, normally operative in blood pressure control. The three principal components of this system are first, renin, found in the kidney but once released into the circulation, renin works enzymatically on a non-pressor alpha globulin to split off angiostensin I, also inactive itself. However, hydrolysis of angiostensin I by converting enzymes then releases the highly pressor-active substance angiostensin II.

Angiostensin II is the most powerful pressor substance yet identified. Its vasoconstricting powers have been demonstrated on smooth muscle preparations and in intact animals and in man. Intravenous injection in all species studied evokes a steep rise in arterial pressure. When compared by weight with norepinephrine, angiostensin II is ten to twenty times as vasoconstrictive.

The second property of angiostensin II is its stimulating effect on the adrenal cortex. Experimentally, Laragh (1977) reports injections of angiostensin II cause a prompt and sustained increase in secretion of aldosterone. Related

evidence suggests that angiostensin II may be a major regulator of aldosterone secretion.

A third property of angiostensin II is its direct effect on the kidney, causing a modification in the kidney's capacity for salt excretion, in some circumstances causing salt retention and in others natriuresis. Laragh (1977) also points out that these several functions of angiostensin II only require minute amounts of the hormone.

With respect to the effects of aldosterone, it is the most potent mineralcorticoid secreted by the adrenals, its principal action being to induce reabsorption by the distal renal tubules, in exchange for potassium, the excretion of which is promoted. Like angiostensin, aldosterone is normally present in plasma in very low concentrations.

What, however, is the connection between the renin-angiostensin-aldosterone system and essential hypertension? Laragh found evidence of dysfunction in the direction of abnormally high or abnormally low plasma renin activity in a substantial number of essential hypertensive patients. On this basis, Laragh (1977) has suggested subclassification of essential hypertensives. He claims that this is more than just a new taxonomy as the sub-types have clinical implications. Specifically, a taxonomy based on renin activity, according to Laragh, could permit simpler and more specific drug therapy for a fraction of patients. Low renin patients seem to have more of a volume component than high or normal renin hypertensive patients. Thus the former are sometimes

able to normalize their blood pressure on diuretics alone, while the latter may respond to anti-renin therapy alone, with drugs like propranolol.

However, Laragh (1977) is careful to point out that classification of essential hypertension in terms of the renin-angiostensin-aldosterone system should not preclude a continuing search for other contributing factors. For example, he continues, although specific defects of the nervous system, central or autonomic have not been identified, the fact that many anti-hypertensive drugs appear to work by suppressing nervous system activity is reason enough to continue searching in this direction also.

The Role of the Sympathetic Nervous System in Essential Hypertension

Medical investigators are still hesitant about according sympathetic nervous activity a significant causative role in essential hypertension. Important research conducted by Austin Doyle (1977) raises the question whether sympathetic stimulation in hypertension could represent an exaggerated response of the hypertensives' circulation to normal sympathetic activity rather than an increase in sympathetic activity per se. If the latter were true, Doyle argues this should be reflected in an increase in circulating norepinephrine that also would be reflected in increased metabolism and excretion. But while some investigators, starting with Von Euler, report increased urinary catecholamine excretion

in the majority of hypertensives, others fail to find consistent alterations in norepinephrine excretion or its analogues except in hypertension associated with pheochromocytoma. Of course, part of the problem was the limited methodology available before the 1950s, as well as the fact that some studies of hypertensives were conducted while the patients were on anti-hypertensive therapy. But a well controlled study conducted by Doyle (1977) in which untreated hypertensive patients were compared with healthy university students, urine was collected from both groups, while they remained fasting, recumbent and undisturbed. Mean excretion rates of both norepinephrine and epinephrine were significantly higher in hypertensive subjects.

The above differences, however, were still regarded as poor estimates of sympathetic activity because of the problems associated with measurement of urinary catecholamines. As had been pointed out earlier, measures of circulating catecholamines provide far more accurate indicators. Chemical assays based on the flourescence techniques had been introduced in the 1950s but Doyle (1977) regarded them as neither specific or sensitive enough for quantifying catecholamine concentrations in blood samples of reasonable size. A radioenzymatic assay developed by Engelman and his coworkers (1970), utilizing a double-isotope derivative method, provided the specificity and sensitivy that Doyle perceived to be necessary. When Engelman et al. applied their assay clinically, they found mean values for total plasma catecholamines

twice as high in hypertensive patients as in a comparable normal group. Subsequently others reported similar results (Doyle, 1977).

Doyle (1977) also reported his observations using the radioenzymatic assay: Thirty-one patients, none of whom had received medication for their hypertension or showed evidence of renal or adrenal disease, were admitted to the hospital and kept at rest for several days. During this time they received a standard sodium supplement--100 m Eq Na/day. On the third morning, a cannula was inserted into a forearm vein and plasma samples taken. Doyle (1977) notes that by this time seven of the thirty-one patients no longer appeared to have significant hypertension, that is diastolic pressures had dropped below 95 mm Hg. These were therefore classified as labile. When compared with the definitely hypertensive, catecholamines were clearly increased in the latter. And when norepinephrine and epinephrine were measured separately, almost all the catecholamine increment was accounted for by norepinephrine.

Doyle (1977) also observed that in the definitely hypertensive group, there was a close correlation between circulating norephinephrine levels and resting diastolic pressures. This for Doyle implied that the level of sympathetic activity might be an important determinant of the height of blood pressure.

Doyle (1977) being very thorough-going found the observed elevations of circulating norepinephrine difficult to interpret. An important possibility was that the increase

was a function of a decrease in reuptake of norepinephrine by sympathetic nerve endings. Doyle had available relevant methodology that allowed for the empirical testing of this possibility.

Release of stored norepinephrine involves several steps. However, much of the released norepinephrine never reaches the effector cell but is take up again and stored by the sympathetic nerve endings. The particular point that Doyle (1977) makes is that only a small fraction of locally released norepinephrine escapes the recapture mechanism and diffuses into the circulation, as free norepinephrine, where it would be reflected in plasma norepinephrine assay or in urinary excretion. Thus neither provide a true index of the functionally active pool of catecholamines under normal or abnormal conditions. Excess norepinephrine could be the result of dysfunctional reuptake mechanisms.

In order to clarify this issue, Doyle (1977) examined the enzyme catalyzing the final step in norepinephrine synthesis within the synaptic vesicle--dopamine-beta-hydroxylase (DBH). When a nerve stimulus causes release of norepinephrine from the vesicle, DBH is not subject to reuptake. After release, it enters the circulation, where its circulating half of life is considerably longer than that of norepinephrine. Also in animal experiments, DBH has been shown to increase with sympathetic stresses and reduced by chemical sympathectomy. For Doyle, then, circulating DBH levels would provide a better index for evaluation of sympathetic activity.

Using a DBH assay recently developed by Rush and Geffen, Doyle (1977) undertook to restudy under the same standardized conditions, the patients who had previously shown elevated plasma catecholamines: thus only patients whose blood pressure remained high during three days of hospitalization were classified as definitely hypertensive. Thirty-seven hypertensive patients were compared with 14 normotensive volunteers. Blood was taken for estimation of plasma catecholamines and DBH. Plasma samples were assayed independently in separate laboratories for norepinephrine and DBH. Doyle (1977) reports that in the hypertensive group a clear relationship obtained between blood pressure and both norepinephrine and DBH. Doyle is satisfied that he has presented important evidence relating to excessive sympathetic stimulation as a possible cause of essential hypertension.

Implications of Success of Therapeutic Drugs for the Possible Role of Subjective Distress in Essential Hypertension

Of relevance to the possible etiological role of stress in essential hypertension is the long observed success of sympathectomy and more recently, the consistent success of "chemical sympathectomy." The commonly used antihypertensive drugs come in three types, the diuretics, the vasodilators and the sympathetic blockades. The basic mechanisms by which these three types of antihypertensive agents work

to reduce blood pressure will be reviewed briefly, as well as the relative importance of the sympathetic blockades.

Diuretics. Currently the precise mechanism by which the thiazides exercise their therapeutic action is unclear. The theory receiving most support is the sodium-loss theory, meaning that the antihypertensive effect is dependent on the loss of sodium produced by the drug. Freis (1960), however, reviewed research which showed that diuretic agents reduce blood pressure responsiveness to norepinephrine and other pressor amines. For him this suggested that diuretics decrease reactivity to the unknown pressor principles operative in essential hypertension also.

It is also interesting to note that when diuretic drugs are used alone, there will always be about 45 per cent of essential hypertensive patients whose blood pressure will not be lowered enough to prevent vascular complications (Tobian, 1977). For these patients, additional drugs are needed, generally sympathetic blockades of one type or another. Thus one would agree with Grollman (1960) that the effects of sodium restriction on blood pressure are only symptomatic, that is, they influence the manifestation of the disease without affecting the fundamental disturbances responsible for the disorder.

Vasodilators. From Gifford's observations (1974) hydralazine is the only agent currently available for oral use that reduces blood pressure by dilating arterioles.

The action of hydralazine, however, is relatively symptomatic. Plummer (1960) reviewed much evidence showing that the site of action of hydralazine is peripheral: it interferes with the hypertensive or constrictor effects of several peripherally acting pressor substances, such as serotonin, epinephrine, norepinephrine and pitressin. In fact, the action of the vasodilators in Plummer's words, "point to resultant dampening of sympathetic vasoconstrictor activity and by the same token, to a degree of chemical sympathectomy."

Thus not only does the successful use of hydralazine in the treatment of essential hypertension not offer any competing causal factors, it supports the hypothesis that sympathetic overactivity might be a causal factor in essential hypertension.

Sympathetic Blockades. Plummer (1960) notes that although nervous activity has not been demonstrably implicated as an etiological factor in essential hypertension, it has been repeatedly observed for some time now that pharmacological suppression of this system offers therapeutic benefits. Gifford (1974) observes that currently, after diuretics, the most commonly used antihypertensive agents block sympathetic activity at one or more sites. The major sites at which sympathetic inhibiting drugs, administered orally, exert their hypotensive action are: (i) the central and peripheral nervous system; (ii) ganglionic blockades;

(iii) neuroeffector transmission blockades; (iv) false neurotransmitters and (v) receptor blockades.

With the exception of the diuretics, all of the effective hypotensive agents mentioned above acted on the sympathetic nervous system or the neurohumors which they secrete. However, even diuretics may be acting indirectly on the sympathetic nervous system through their reduction of plasma volume: Freis (1960) notes that reduction in plasma volume, normally achieved by diuretics, alters blood pressure responsiveness to norepinephrine infusion.

Thus all three types of antihypertensive agents act directly or indirectly on the sympathetic nervous system or the neurohumors it generates. The basic issue is clear: surgical and chemical sympathectomy have both proved effective in lowering blood pressure in essential hypertension. What is less clear is the related issue of the various side effects associated with sympathetic blockade at various sites. Thus the availability of antihypertensive agents serves not only to furnish treatment for essential hypertension, but also to provide persuasive evidence of the critical role of sympathetic nervous system in the genesis of the hypertensive state.

Comparisons of Psychological Reactions of Hypertensives and Normotensives

The role of psychological factors in essential hypertension is the main concern of this research. As must be clear by now, the problem is one of staggering complexity, and evidence relating to the possible causal relationship of emotional factors has been reviewed not only at several levels, but also from varying vantage points. In this section of the review, attention is focused upon the plausible but unproven association between particular psychological traits and the presence of essential hypertension.

Experimental data reviewed earlier showed how acute psychological stress can initiate sudden and transient elevations in blood pressure. What is still to be established is, if and how repetitive or continuous psychological stress leads to sustained elevations of blood pressure. Research related to this problem can be divided into two main categories: one type of investigation has focused upon populations exposed to disastrous events; these populations' blood pressures have been measured after the event and unusually high prevalences of blood pressure have been observed over time.

The second type of investigation represents the main focus of research in the United States. Studies focus on personalities of hypertensive patients with and without comparison groups. Methods generally employed were interviews with concomitant recordings of cardiovascular functions.

Environmental Stress

The first category of studies vary, in terms of their geographic location. However, they provide some data with respect to psychological responses in whole populations exposed to disturbing events. Probably among the most well known of the older studies was the Ruskin, Beard and Schaffer study (1948) which reported sustained diastolic blood pressure levels of over 95 mm Hg for one to two weeks in 56 per cent of the persons exposed to the Texas City explosion in 1947. Likewise, J.D.P. Graham (1945) found that in 27 per cent of soldiers who had seen protracted combat in the African desert in World War II, showed diastolic pressures of over 100 mm Hg for several months thereafter.

In general these studies provide data relating to the hypothesis that essential hypertension is initiated by environmental stress. Evidence concerning disturbing life events preceding the onset of essential hypertension in patients, however, is harder to come by. One fundamental problem is that changes in blood pressure take place without patients' awareness, so there is difficulty in making a connection. Additionally, there is the other problem of deciding whether these events, which are presumably stimuli which evoke the disease, are the same as for all the other illnesses in which this sort of temporal correlation is relied on. Reiser, Brust and Ferris (1951) reported that in 41 of 80 patients for whom the time of onset of the disease was known, significant psychological stimuli preceded

the onset of hypertension. They were careful to point out that the "dynamic factors in the personality structure of the patients' reaction pattern to related experiences, are such that, the life events would have special emotional meaning to the patient." When "symptoms" become the focus of attention the situation changes dramatically. Reiser, Brust and Ferris found correlations between events and "symptoms" in 119 of 182 patients according to Graham (1972). The "symptoms" reported by Reiser et al. included what they described as the "usual symptoms associated with hypertension, mainly headaches, weakness, muscle pains, dizziness, difficult breathing and 'blurred vision.'" Wolf, Cardon Shepard and Wolff (1955) also report correlations between disturbing life events and onset of hypertension.

Although data presented here is interesting and plausible, it is inconclusive: first it is clear that essential hypertension is not always preceded by a stressful life event; neither do all persons exposed to similar traumatic life situations respond with a significant increase in sustained blood pressure. In addition, the stressor may very well be only the precipitator of hypertension, rather than the cause. This differential predisposition for either genetic, nutritional or other reasons, would help explain why all persons do not respond to environmental stress with increased pressure. In fact, in recent studies by Lovell (1960), Maddocks (1961), Poindexter (1953), it has been pointed out that differences in genetic endowment, body

bulk, nutrition, prevalence of anemia, and race may be important determinants of differences in blood pressure. Evidence with respect to the contribution of various nutritional factors, e.g. salt, cholesterol and particular personal habits, e.g. smoking, have been reviewed earlier in the study. It was made clear that these factors do affect blood pressure. Thus, environmental stress could be a precipitator in some instances, but the possibility also remains that if events are traumatic and/or prolonged enough, they could play a substantial role in causing essential hypertension.

Subjective Emotional Stress

A second line of investigation, pursued more frequently than the first, has a fairly consistent thematic approach: psychological conflicts are regarded as being more characteristic of hypertension than others. According to Ostfeld (1967) these conflicts are generally of two types: (i) a conflict between passive, dependent longings and aggressive, competitive impulses; and (ii) a conflict between chronic anger, close to consciousness awareness and a need to repress these feelings for fear of loss of love and esteem. If Ostfeld has presented an acceptable hypothesis, one can observe here that at least two basic emotions are involved in the hypertensives' subjective struggle: anger and anxiety. Various studies have tried to differentiate between these two emotions; attempts have been made to see if elevations

in high blood pressure generated by these two emotions are differentially caused: for instance, cardiac output as well as peripheral resistance may be associated with the first emotion but only peripheral resistance may be causing elevated blood pressure in the second case.

Studies attempting to establish either anger or anxiety as a central psychological factor in essential hypertension will be reviewed first, then studies which have evidence of both emotions will be reviewed.

With respect to anger most investigators will agree that a significant aspect of the possible problem that hypertensives may have with anger is with its expression. Two basic ways of dealing with anger are generally identified in the literature, "anger-in" and "anger-out." The former is traditionally thought to be more characteristic of hypertensives. Thus Graham (1972) writes that "hypertensives have hostile impulses that they do not act on." More important, however, are the frequent clinical observations that elevations in blood pressure occur in association with unexpressed or suppressed anger (Hambling, 1952; Wolf et al., 1955). The evidence is based on self-report of these feelings during periods of elevated pressure. A striking anecdotal case with documentation is reported by Wolf, Pfeiffer, Ripley, Winter and Wolff (1948): they noted that a patient's blood pressure fell from 165/100 to 125/85 after he had beaten up his brother-in-law! This might suggest that the expression of hostility can reduce blood pressure. A more recent related

study by Hokanson, Burgess and Cohen (1963) generated some experimental evidence which lent some support to this position. Only systolic pressures were measured in this study, though. College-age subjects who were counting backwards by twos were harassed and interrupted by an experimenter. They showed a significantly greater increase in systolic blood pressure than did control subjects, who were not frustrated--15.6 mm Hg versus 4.4 mm Hg. It was also found that when some of the frustrated subjects were later permitted to administer electric shocks to the experimenter or other persons as part of a "guessing game," the pressure dropped most in those who shocked the experimenter himself--to a level representing an increase of 7.3 mm Hg over the baseline. Blood pressure dropped least in those who were given no opportunity to shock anyone--to a level representing an increase of 12.5 mm Hg over the baseline. Blood pressure levels dropped to intermediate levels in those who shocked the persons other than the experimenter.

Kaplan et al. (1961) also report a related study: they observed rises in systolic pressure, mean 6.7 mm Hg, and in diastolic, mean 2.1 mm Hg, in hypertensive subjects when asked to talk for 5 minutes. The systolic rise was statistically significant, the diastolic was not. However, there were significant differences between hypertensives and normotensives in both pressures, in particular because the normotensives had a mean fall in diastolic pressure. Rises in pressure occurred while the subjects were speaking in a

manner judged to be hostile, thus the tentative conclusion was that hostility may be a correlation of rise in blood pressure. Additionally one hypertensive patient who had been studied intensively, providing a total of 13 verbal samples, showed significant correlations between his verbal samples and both systolic and diastolic pressures.

If these data are regarded as having limited validity, a fundamental conclusion may be drawn: that is, continuous or repeated exposure to people or situations which threaten an individual and subsequently lead to angry or hostile impulses, which in turn are blocked, can lead to temporary elevations in blood pressure.

With respect to the capability of anxiety to increase blood pressure temporarily, one of the most frequently observed situations occurs when blood donors are about to have venipuncture. Blood donors, especially those who stated that they were "nervous," also those who faint later, show elevations in blood pressure (Graham, Kabler & Lunsford, 1961). This change also occurs in persons who develop the common emotional vasovagal faint in other situations. Of course, in these situations, self-descriptions of "nervous" are equated to being anxious and the assumption is made that anxiety is more likely to be experienced when one is about to undergo venipuncture, as opposed to anger.

A related problem that has drawn some attention is whether or not anger and anxiety cause elevation of blood pressure by somewhat different processes. In particular,

some investigators think that they may have found evidence that anger increases cardiac output. Two factors, however, suggest that accurate information on differential contributions of anger and anxiety to elevated blood pressure may be premature: the instruments used in these studies do not allow clear-cut physiological interpretations: ballistocardiographic tracings do not provide very reliable data with respect to measures of peripheral resistance and cardiac output (Graham, 1972). Second, the results of the various investigations tend to contradict one another. However, a few studies are worthy of review for the possible leads which they might suggest.

The first study to be reviewed was conducted by Moses, Daniels and Nickerson (1956), using a ballistocardiograph for hemodynamic investigations. Their observations led them to conclude that (i) large elevations of blood pressure (160-200/100-130 mm Hg) are associated with rage and resentment and are mediated by increased peripheral resistance with normal stroke volume and heart rate; (ii) minor elevations of blood pressure (140-160/90-100 mm Hg) are associated with "anxiety with minimal overt expression," and are mediated by increased peripheral resistance. However, minor elevations of blood pressure found in association with overtly expressed anxiety are mediated by increased stroke volume and heart rate, that is, increased cardiac output, without change in peripheral resistance. Although these findings are of interest, additional limitations of the study are its use of

only ten stubjects and lack of specification of the various patterns.

From the studies just reviewed, while there seems to be only tentative findings regarding possible differential mediation of elevated blood pressure by anger and anxiety, it does seem clear that both anger and anxiety are capable of raising blood pressure. This raises the related issue of how realistic it is to separate anger from anxiety in life situations. Hambling (1952) and Binger (1951) have mentioned a relationship between anger and anxiety in hypertensives. Basically the idea is that hostile feelings (anger) tend to lead to feelings of fear (anxiety) about retaliation. Ackerman (1950) also describes hypertensives in a similar fashion: they "exhibit a pervasive sense of trapped, helpless exposure to the danger of aggressive injury, usually in the context in which the threat seems to derive from the very person on whom the patient depends for security." If Ackerman offers a relevant insight his statement reduces to:

(i) Hypertensives perceive themselves to be in an alien or even dangerous environment.

(ii) The environment is essentially an interpersonal one.

(iii) The persons who cause feelings of being "helpless or trapped" and to whom hypertensives may want to direct aggressive impulses are generally people upon whom they are also dependent.

The presence of these three factors generally leads to a repeated cycle of negative emotions: need for self-assertion which expresses itself in hostile or aggressive feelings and/or actions; which in turn lead to fear of retaliation and/or retaliation. Then follows dependent longings and/or feelings of inferiority, which ultimately in turn give way to the reactiviation of the need for expression of hostility and/or aggressive feelings or actions instead of self-assertion.

This cycle of internal feelings helps to explain how two of the most powerful negative emotions are often inextricably linked and also extend for considerable periods of time. None of the studies presented have attempted to deal with this more complex aspect of the problem. Hardly surprising, since ethical restrictions limit the intensity and the period of anxiety or anger induction. The method most often used by investigators employs the use of questionnaires that tap personality traits. What is remarkable about these studies, however, is the lack of replicated significant correlations with hypertension despite the consistent success of studies which relate short-term anger or anxiety to elevated blood pressure. A typical example is the research of Ostfeld, who has systematically used some of the best known psychological inventories in attempts to find traits which may be associated with essential hypertension.

Ostfeld and his associates' research in this area include several published studies. In one, 18 essential hypertension subjects were compared with 15 renal hypertension subjects and 18 normotensives. All subjects were similar in age, sex, race, education and employment and marital status. The Minnesota Multiphasic Personality Inventory and the Rorschach were administered to each of the participants, who were also subjected to "distressing" interviews. In addition, most of the hypertensives were followed for several months through at least one difficult life event. Ostfeld (1959) reports that there were no important differences in psychological test scores of the renal hypertensives, essential hypertensives or the normotensive subjects. He also observed that in both groups of hypertensive subjects, blood pressure rose with approximately equal frequency and magnitude during untoward life events or distressing interviews. No comparisons of normotensives and hypertensives under these conditions are mentioned.

In a second study, Ostfeld and Lebovits (1960) compared a small group of persons with essential hypertension with normotensives who were similar with respect to age, sex, race, occupation and education. MMPI scores were obtained from each group as well as multiple blood pressure measurements over several months. Ostfeld reports that in both the normotensive and hypertensive groups there was a significant positive correlation between blood pressure variability and scores on measures of emotional lability. Thus the MMPI

scores of hypertensives whose blood pressure varied widely resembled the scores of normotensives with labile blood pressure. Thus Ostfeld writes, "Twice, therefore, we failed to find psychological test score patterns characteristic of persons with elevated blood pressure" (1967, p. 323).

Despite these failures, Ostfeld and his associates thought it necessary to keep on trying: "The limited interpretation that can be placed on these results indicated to us the advisability of further studies employing reproducible and objective personality measurements on larger and more representative samples" (1967, p. 323). Ostfeld then reports on subsequent efforts, part of which were published in Lancet (Ostfeld & Paul, 1963). In this study, a cohort of 2,000 men aged 40 to 55 years were randomly selected. They were all employed in the same manufacturing plant. Although the main purpose of the study was to identify multiple risk factors in coronary heart disease, data collected in the first two years of the study relate to the problem of high blood pressure. Casual, single, left arm blood pressures were obtained by physicians using a mercury manometer. Each subject also completed a family history which was reviewed with him by a medical secretary. Arm girth measures were taken and two psychological questionnaires, the MMPI and the Cattell 16 Personality Factor Questionnaire, were completed by each man. Ostfeld describes the advantages of these inventories; they had been "standardized on mid-Western

American populations, machine scorable, relatively standard interpretation and free of observer bias" (1967, p. 324).

With respect to data analysis, three major examinations of the data were made. First, all men in the sample whose blood pressure, both systolic and diastolic, exceeded the mean by more than one standard deviation and all men whose pressures, both systolic and diastolic, were more than one standard deviation below the mean, were identified. Each man in the high blood pressure group was matched with a man in the low pressure group so that no pair differed by more than three years in age or 2.5 cm in arm girth. This resulted in 77 pairs--all that could be matched. The mean MMPI and 16 Personality Factor scores for both groups yielded identical scores for 10 of the 16 Cattell factors and differences of 1, for the other 6 factors. Scores on the MMPI yielded a similar pattern. Thus Ostfeld was unable to report any significant statistical or psychological differences between the two groups.

In a second approach to the data, all men with a positive family history of hypertension were identified. The group with a positive family history of hypertension was divided into those whose blood pressure equalled or exceeded 160 systolic or 95 diastolic and those whose pressures equalled or were less than 140 systolic and 90 diastolic. The mean MMPI and 16 PF scores were compared. With a remarkable similarity, 10 of the 16 PF factors were identical and the other 6 factors differed by 1 point. (For what it is worth,

the similarity to the previous results were numerical only, that is, the 10 identical factors were not the same factors in the two analyses.) Results with the MMPI also revealed no significant differences.

Undaunted, Ostfeld performed a third analysis: this time attempts were made to investigate more closely the possible relationship between psychological conflicts and high blood pressure. A clinical psychologist skilled in the use of the MMPI and familiar with the literature relevant to hypertensives, defined three patterns of the MMPI scale scores: (i) a pattern reflective of conflict over expression of aggression; (ii) a pattern reflective of expression of hostility without conflict; and (iii) a pattern reflective of unconflictful passive-dependent characteristics. All men in the entire cohort whose MMPI score configurations met these specifications were identified: 28 men were identified as conflicted; 31 men as aggressive; and 18 men as passive-dependent. The mean blood pressures of these three groups were compared and not only did they have nearly identical blood pressures but they were under 140/90 mm Hg. Mean blood pressures were 138/88 mm Hg, 136/88 mm Hg and 135/85 mm Hg for each group respectively. Standard errors were 4.5 mm Hg or 3.9 mm Hg for systolic pressures and 2.5 mm Hg, 1.8 mm Hg and 2.7 mm Hg for the diastolic pressures.

Ostfeld (1967) thinks that there are three major limitations to these data and the analyses: First, the data are cross-sectional rather than prospective; second, it is

possible that the two tests employed do not assess the relevant psychological variables; and third, the data analysis method, comparison of group means, may be obscuring differences that other techniques might reveal. So Ostfeld continues, "Further analysis of the data at hand and the prospective use of these questionnaires are planned."

No further review of Ostfeld's work, however, is planned. The sentence of greatest relevance and which sums up the fundamental flaw in this particular research endeavor is the observation that possibly the two tests employed do not assess the relevant psychological variables. In the introduction to this work, the grave limitations of current psychological inventories were noted and the need for construction of an inventory was indicated. The research of Ostfeld and his associates serves to underscore this point. Taken at face value his results contradict rather than develop in any consistent manner the psychophysiological studies relating temporarily induced stress, experimentally and naturally, to elevations of blood pressure.

For two decades Caroline Thomas and associates at John Hopkins have been investigating the precursors of hypertension and coronary artery disease using medical students at that university. In a long term prospective study, 1200 subjects have been followed; not only their health was monitored but that of their parents as well. The study is still in process and conclusive data are not yet available. The study is mentioned here, however, because again attempts

were made to use a well-established and popular psychological test to help shed light on the problem of personality characteristics of hypertensives: Rorschach protocols of 200 male students were analyzed blind for hostile or aggressive impulses. The sample had been grouped according to parental history of hypertension and coronary artery disease. This yielded 69 with positive parental history, 35 with questionable histories and 96 with negative histories. Thomas (1967) points out that the data were analyzed from multiple points of view; the eight types of hostility and aggression measured by the Rorschach were in some instances combined to yield more complex and meaningful categories. Yet nothing of statistical significance was found.

Thomas (1967), however, goes on to report division of subjects into groups according to whether either, neither or both parents were affected by hypertension, regardless of coronary disease. Discriminant function analysis is being employed, using six somatic variables and three psychological ones: anxiety, depression and anger. The latter came from a "habits of nervous tension questionnaire." Thomas (1967) reports that while the anxiety, depression and anger scores did not contribute significantly to distinguishing between subjects based on parental histories of hypertension, they were of significant aid in discriminating between groups of subjects with different parental histories with regard to coronary disease. Thomas (1967) writes, "even when used as

a single variable, the anxiety score showed differences which were significant at the $p < .001$ level" (p. 338).

The precise significance of the latter statement is not altogether clear--why coronary disease and not hypertension? Is it that in the former the latter is almost always present and together represent a more well-defined form of cardiovascular problems? Thomas (1967) does not indicate. However, it does seem that when more appropriate scales are used, less puzzling results are obtained. Cattell (1960) has published an anxiety scale but apparently it did not occur to Ostfeld (1967) that its use might be relevant to his work, employing it either instead of, or along with the 16 Personality Factor questionnaire. It should be clear, then, that the lack of evidence with regard to personality traits of hypertensives may have much to do with the inappropriateness of the scales and inventories that have been used in this area of research. The inventory constructed for use in the current study will address itself to what would seem to be more direct and systematic elicitation of psychological variables relevant to hypertension.

CHAPTER III: THE SUBJECTIVE EMOTIONAL STRESS INVENTORY (SESI)

Introduction

The psychological literature reviewed in the previous chapter revealed that the absence of significant findings may have been related to the fact that the psychological characteristics assessed were not those of particular relevance to hypertensives. Progress in this area of research would seem to require that there be very specific focus on at least three emotional states, anxiety, anger and depression. The focus on depression was based on the Freudian hypothesis that depression is anger turned inward and would therefore be an important form of subjective distress. As was pointed out in the introductory chapter of this study, a decision was made to engage in scale construction because of noted inadequacies in current inventories focused on normal emotional functioning. Inadequacies include the poor coverage of negative states or traits such as anger or anxiety as well as unsatisfactory resolutions of item construction problems.

Despite the many limitations of self-report data, psychological scales and inventories continue to be developed (ZIPERS, Zuckerman, 1977; S-R Inventory, Endler and Okada, 1975; the STAI, Spielberger, Gorsuch and Lushene, 1970). Probably because self-report scales possess the singular advantage of gaining information directly from the person experiencing the various phenomena, they continue to be

perceived as useful. Any other observer perforce is limited to reporting apparent interpretations of a person's experience based on behavior and verbal responses. Of course as one moves from the normal to the psychopathological end of human functioning, self-report data become conceivably less valid and also less reliable than professional observations and interpretations. However, the converse is also likely to be true, as we move from the psychopathological to the normal end of human functioning, self-report data is likely to be more valid than observers' interpretations. If the latter can be assumed, then self-report data from relatively normal subjects is of particular quality and importance. Also the specificity and the accuracy that can be elicited at the item level should also be positively correlated with normality.

While the above presents at a most general level reasons for constructing a new inventory, there are several other specific reasons why scale construction appeared to be necessary. First, there were problems with the content domain covered by other inventories designed for use by normal persons. More specifically, there seemed to be a reluctance to address the sources of anger, anxiety and depression which fall within the normal person's experience. Also the issue of states versus traits admits of a more elegant solution than those presented thus far. Another reason for scale construction was related to item format and construction. It was thought that a systematic format could aid in improving memory and accuracy of the data.

A corresponding change in response alternatives was also perceived to be necessary. Finally, the implications of these changes for the scoring system and the interpretation of results also had to be explored. Each of these reasons presented in brief above will now be examined in greater detail.

Content Domain: Anger, Anxiety, Depression

Probably the best known and well-used anger scale is the Buss Hostility-Guilt Inventory (1961). The scale was carefully constructed and the content domain focused on hostility, aggression and guilt. Two types of hostility were identified: resentment and suspicion. Five types of aggression were identified: assault, indirect, irritability, negativism and verbal. A single guilt category was added. A major concern in constructing the inventory was the minimization of defensiveness in responding. Aware that "social desirability accounts for much of the variance of a normal's response to inventories," attempts to reduce this were made primarily through three item-writing techniques:

(i) Buss chose to assume that socially undesirable states already existed, and then asked how a relevant emotion was expressed, e.g. "When I really lose my temper, I am capable of slapping someone"; "When I get mad, I say nasty things." Buss argues that in these items the loss of temper is assumed the subject is asked only whether he expresses it.

(ii) Buss also applied the technique of providing justification for the occurrence of aggression: "Whoever insults me or my family is asking for a fight"; "People who continually pester you are asking for a punch in the nose." According to Buss, when the item provides a rationale for aggression, the subject's defenses and guilt reactions are reduced and "he does not necessarily answer in the direction of social desirability."

(iii) Buss also employs the use of idioms: "If somebody hits me first, I let them have it"; "When I am mad at someone, I will give him the silent treatment." Buss points out that idioms have a high frequency usage and therefore should be readily accepted and admitted.

Buss used factor analysis in validating the structure of the inventory's scales. He used Thurstone's centroid method to extract factors generated by male and female responses. Rotation yielded factor loadings generally between .48 and .66. However, Buss chose to regard factors of .40 loadings as meaningful. This allowed him to keep the "Indirect" scale. Scale reliabilities, test-retest after five weeks were also acceptable. With the exception of negativism they varied from .61 to .78. Test-retest reliability for the inventory was .82. Buss's inventory employs True and False response alternatives.

Despite the rigor, outlined above, that was used in the construction of the Buss Hostility-Guilt Inventory, it was regarded as unsatisfactory for use in this study for several

reasons: At the most general level the Buss inventory is offensive: the items describe for the most part people who are not only very angry, but also quite violent. The focus is scarcely on the type and range of anger that has been described as typical of hypertensives. One characteristic quality, for instance, is that hypertensives have difficulty expressing anger; a frequent reason being possible loss of valued support. Thus two important foci are, items tapping ability and freedom to express anger, as well as the context in which it tends to occur, for instance in the presence of people in authority.

In addition, although Buss attempted to address the social desirability issue in a systematic fashion, he failed to address it at an important level: implicitly throughout the inventory, expression of anger is regarded negatively. A polarity exists between expression of and non-expression of anger. There is little or no opportunity to express oneself self-assertively. Thus items tend to be either "I often feel like a powder keg ready to explode" or "I am always patient with others." The more subtle and more relevant issue of inappropriate versus appropriate expression of anger is for the most part unaddressed.

A second factor of importance that was not treated in a systematic fashion was frequency of anger. First the True and False response format reflects merely the expression rather than the frequency of the expression. Additionally, adverbs such as always, never, often are used inconsistently

in items, throughout the inventory. Thus the information yield is of limited value.

A third factor of importance that is not addressed is the discrimination between experiencing feelings of anger and the expression of these feelings. Some discrimination is made however, with respect to manner of expression--e.g. assault, indirect, irritability, negativism.

A fourth factor of relevance is the lack of specific reference to cause or focus of anger. All references in the inventory are vague--"When someone"; "When people"; "If somebody," etc. Thus little data is collected as to who or what in a person's general or interpersonal environment act as stimuli, eliciting an angry response and who or what is frequently the target of angry expressions.

The above are the main reasons which led to the construction of a new anger inventory, rather than use of the Buss Hostility-Guilt Inventory. Three basic assumptions guided the construction:

(i) Everyone experiences anger feelings and expresses anger in some manner.

(ii) While there is free-floating anger, much of it is specifically triggered and focused.

(iii) Feeling and expression of anger may be appropriate or inappropriate.

With respect to the content domain, five strategic facets were identified:

(i) Triggers - stimuli which repeatedly elicit anger.

(ii) Mode of expression - manner or manners in which an individual expresses anger.

(iii) Intensity - moderate versus more intense expressions of anger.

(iv) Duration - typical period time in which an individual tends to remain angry.

(v) Mode of recovery - what happens after feelings and/or expression of anger; how does the individual seek to regain a more pleasant frame of mind. (See Figure 2)

In addition there is an awareness of Joel Davitz's (1969) four dimensions of emotion. Two dimensions are considered in Bradburn's (1969) work--happiness and level of activation. To these Davitz adds the dimensions of Relatedness and Competence. While knowledge of these dimensions did not cause any expansion in the five major facets of the content domain, they served to help direct and anchor item generation.

Issues related to the construction of items for the anger scale will be discussed in the following section. Meanwhile it is necessary to examine issues with respect to the content domain of depression. Probably the best known and most widely used depression inventory is the "Inventory for Measuring Depression" by A.T. Beck (1961). He constructed an inventory with 21 sub-categories, covering various moods, attitudes and behaviors. Within each subcategory he used the Guttman technique to build in degrees of progression. Thus a typical category consists of four

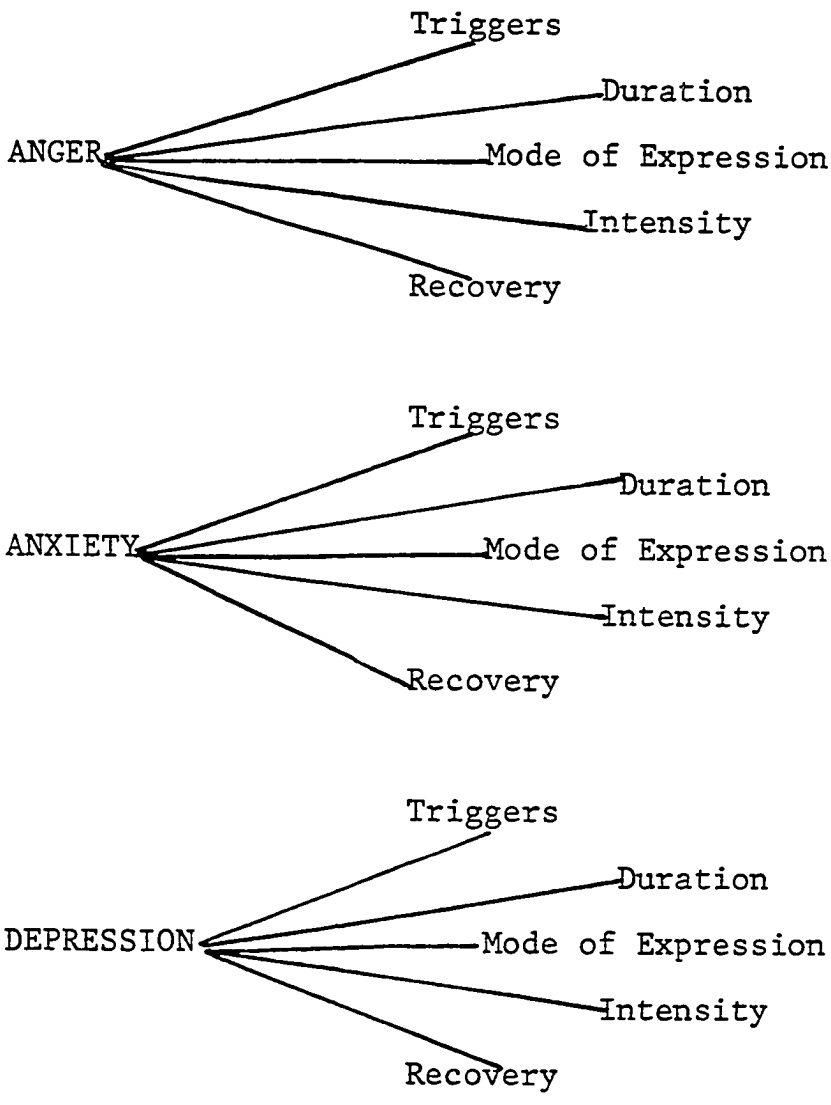

Figure 2

Facets of content domain.

to five items. Categories include Mood, Pessimism, Sense of Failure, Lack of Satisfaction, Guilty Feelings, Sense of Punishment, etc.

A single category includes items ranging from "I do not feel sad"; "I feel blue or sad" to "I am blue or sad all the time and I can't snap out of it"; "I am so sad or unhappy that it is very painful"; "I am so sad or unhappy that I can't stand it."

The categories included in the Beck Depression Inventory represent a comprehensive coverage of the various aspects of the depression domain--attitudes, feelings and behaviors. The major problem is related to the fact that all the items are located in time but not in terms of the situations that instigated them. No data are collected on possible triggers or stimuli related to depression, though limited information with respect to relative frequency is obtained. Perhaps, however, the most powerful factor influencing the decision not to use the Beck Depression Inventory was the lack of correspondence that would obtain in scale format and scoring.

Similar types of reasoning partly applied to the creation of an anxiety scale. The Taylor Manifest Anxiety Scale, despite wide usage, was used on several occasions by this author, and observed to be so highly confounded with social desirability that little or no discrimination resulted. Edwards (1957) has documented many such instances. The State-Trait Anxiety Inventory (STAI) constructed by Charles Spielberger, Richard Gorsuch and

Robert Lushene (1968), promised to be a significant improvement over the Taylor Anxiety Scale, partly because of Spielberger's extended research in this area and also because it seemed to address itself systematically to the state/trait issue. On careful examination, however, the STAI proved to be disappointing: the Buss Hostility-Guilt Inventory has 75 items, the Beck Depression Inventory has 21 categories with at least four (4) items per category, yielding over 84 items. In sharp contrast to both of these inventories, the STAI has 40 items. The 'state' items typically focus on a person's current feelings, "at this moment": "I feel calm," "I feel upset," "I feel anxious," "I am tense." The 'trait' items ask the respondent to indicate how he "generally feels." This is the only difference between the state and trait items. Thus many of the items measuring traits are worded in precisely the same manner as that state items: "I feel pleasant"; "I feel like crying"; "I feel blue." Some items differ slightly: "I am losing out on things because I can't make up my mind soon enough"; "I wish I could be as happy as others seem to be."

The four response alternatives differ, however, for 'states' and 'traits.' For 'states' they are "not at all," "somewhat," "moderately so" and "very much so." For 'traits' they are: "almost never," "sometimes," "often," and "almost always."

Basically, the same facets and dimensions delineated for the anger inventory earlier are assumed to apply to the

other two inventories. Thus conspicuous by absence from the STAI are triggers of anxiety, manner of expression, degree of intensity, duration of anxiety and mode of recovery. Note also that these are facets that are considered relevant to the state/trait issue. Spielberger et al. (1970) in the manual state that "since most of the existing anxiety scales measured trait anxiety, a large number of the items embodying content of proven relationship to the most widely used anxiety trait scales were rewritten in a manner that would permit each item to be used as a measure of both A-State and A-Trait." Thus it seems that maybe Spielberger et al. had a very limited focus of the anxiety domain in mind when creating the STAI. It was anticipated that in order to be discriminating with respect to habitual levels of anxiety in hypertensives an instrument covering a more representative sample of the anxiety domain is needed. In addition to the factors noted to be absent above, Davitz's four dimensions of emotion are not adequately tapped in the STAI. Figure 2 presented diagrammatically a map of the content domain for each of the three emotions, anger, anxiety and depression.

Perhaps the content domain of still another inventory is relevant to this study on hypertensives, that of the Jenkins Activity Survey (JAS). As Jenkins et al. (1967) state, in describing the development of their scale, "the content of many of the Jenkins Activity Survey items refer to style and pace of life and to occupational goals . . ." The scales constructed for use in the present study have a

contrasting focus. Instead of just limiting themselves to behavior or activity, they also focus on the emotions which give rise to particular activities and behaviors. Thus although the content of the Jenkins Activity Survey is related, it has a somewhat different focus from the one which guides this study.

Item Format and Construction

Probably the most important question addressed in a consideration of item format and construction is the type of data that the instrument should yield--in particular, the degree of specificity and the amount of accuracy. It is generally agreed, at least in scientific endeavors, that the more of both that can be attained, the better the instrument is serving its purpose. Thus items which through their format allow the content domain to be broken down meaningfully, with maximal specificity and accuracy, contribute to a more thorough coverage of the domain.

The degree of specificity obtainable is controlled by two main factors: (i) the item structure and focus, and (ii) the response alternatives. The contributions of both will be considered in turn.

Certainly one of the most fundamental challenges in scale construction is the elicitation of specific and accurate data, that is at the same time relevant and meaningful. In constructing new inventories for anger, anxiety and

depression, how was this challenge handled? The first factor that we will focus on is accuracy.

While accuracy of a very high order is not yet within our reach, certain approximate levels are within our grasp. If as stated in the section on content domain, anger, for example, is experienced by most persons, and that, further, the same applies to the other two emotions in question, then their frequency and relative frequency become critical variables of interest. Thus the stem for all items in each inventory is "How often..." Respondents are constantly asked "How often..." For those used to traditional questionnaires, this format may strike them as a little unusual at first, but they soon get accustomed to the level of accuracy required. For others unexposed to traditional questionnaires, it strikes them as natural and some appreciate the value in it.

Another dimension related to accuracy and to all three emotions under investigation is that of duration. Item format consistently addresses the frequency of duration of each of the three emotions also. This is in marked contrast to all the inventories which were considered--they all failed to pose the question and tended to generalize prematurely from information asked about mere occurrence.

Still another factor with respect to accuracy and item format that contrasts with the prior inventories is systematic discrimination between experience of particular feelings and the expression of these feelings. It may be recalled that one hypothesis with respect to why hypertensives may be

experiencing abnormally high levels of subjective distress, may be related to their inability to express anger. That is they are unable or unwilling to address and/or redress personal violations or abuse of varying types and degrees. Thus if this hypothesis is to be put to a real test, some method by which the problem is consistently and clearly defined must be employed. Thus item format in the three new inventories probes systematically, frequencies of emotional feelings as well as corresponding frequency of expression of these feelings.

If, however, the assumption made earlier with respect to the ubiquitousness of emotions holds, it becomes incumbent upon any measuring instrument to provide as many discriminations as possible between normal and unusual levels of these emotions. Frequency has been identified as a very relevant factor here, but the variable of intensity also should be assessed. Thus item format should include systematic discrimination of normal levels of intensity as compared to higher but non-psychopathological levels of these emotions. In contrast to the prior scales, the current emotional inventory systematically seeks to monitor intensity of the three emotions in question.

There are at least two other issues that need to be addressed regarding item format: the problem of triggers or "causes" of the particular emotions, and the related problem of jogging memories. Ackerman (1950), referred to earlier, offered a three-part formulation relevant to the

emotions of hypertensives:

(i) Hypertensives tend to perceive themselves to be in an alien or even dangerous environment.

(ii) The environment is generally an interpersonal one.

(iii) Generally these people who are perceived to be the cause of their unhappy state are people towards whom aggression cannot be directed because the hypertensive is generally dependent upon these persons. If Ackerman is moving in the correct direction, his statement means that other people play a large role in triggering or "causing" particular emotional states in hypertensives. Of course, an important question is what kind of people? In answer to this, Ackerman also offers a clue: the people to whom the hypertensive would be most sensitive, would be persons in authority in relation to them and with whom they are often forced to interact. Thus item format should in addition to the other aspects discussed before, also focus quite specifically on particular people in the hypertensive's life space. Items should be concerned with quality of relationships with spouses, parents, employers, siblings, etc.

At a general strategic level, the inventory should also have a built-in flexibility, which allows respondents to indicate and reveal data about persons in their lives, with whom they have much negative interaction. This is necessary because it may be awkward for a single inventory to cover all the individual possibilities that are nonetheless making consistent and significant contributions. Thus the strategy

employed with this scale is that of asking respondents to list the persons in their lives with whom they interact most, or who have the greatest influence on their lives. They are then asked to check and make sure that these persons are represented on the inventory; if not, they may make substitutions or additions.

The second related factor, referred to earlier, was that of jogging memories. Undoubtedly, the use of specific referents such as spouses, employers, and parents helps to immerse the respondent in concrete situations and relations where feelings are more readily recalled. Another factor related to ease of recall is the arrangement of the items. The random arrangement of items does not facilitate recall of information. While the goal of randomization of items is generally aimed at hopefully eliciting information without the subject's awareness of the structure of the domain and hopefully without his manipulation of the data, it is doubtful whether this strategy is really helpful. Honesty on the respondent's part has to be invited and elicited: first the respondent accepts the invitation, then the instrument is structured in such a manner as to facilitate recall. If the respondent rejects the invitation to cooperate, then arranging the instrument's items in a random fashion, thereby hoping to trick the respondent into answering honestly, is probably for the most part ineffective.

A copy of the inventory is presented in Appendix I, but an example of how all the above stated factors have been

woven into a clear item format is worthwhile. Items from the anger inventory will be used for demonstration purposes. The anger inventory consists of 83 items. The first 40 items are arranged in deliberate clusters of four. This clustering allows for recording of (i) frequency, (ii) intensity, (iii) discrimination of feelings versus expression of feeling, and (iv) focus on specific triggers and/or targets of emotions. A typical cluster takes the following form:

HOW OFTEN -

1. Does your father make you <u>feel angry</u> (e.g. by not giving you something you think you should have?)

2. Do you <u>express anger</u> to your father (e.g. having an argument over something about which you disagree?)

3. Does your father make you <u>feel very angry</u> (e.g. so angry you feel as if you are going to explode inside?)

4. Do you <u>get very angry</u> with your father (e.g. have a quarrel, yelling or shouting and leave hating each other?)

It may be observed that all items ask about frequency; also that the first two items focus on moderate levels of feelings, while the last two focus on intense levels of the emotion. The difference between the first and the third items and the second and the fourth items should also be noted. The former focus on feelings while the latter focus on expression. It may also be noted that all four items have a single trigger or target--father. Finally, the use of examples should be noted.

Use of examples is the second feature of the built-in flexibility referred to earlier: descriptions of anger are offered as examples only. Again, because the inventory cannot completely cover the vast emotional domain, respondents are reminded that these descriptions are just offered as a basis for comparison with their own experiences, in an attempt to help them focus and recall more clearly.

A question which probably comes to mind now is, what is the focus of the remaining items in the anger inventory? Their format is similar in that they focus on frequency, but the subject of the items changes from trigger or targets of the emotion to various patterns of coping. Thus five items ask how long this emotion tends to last--duration. Some items focus on the somatization of emotions, that is, for example, the frequency of association of anger and headaches or backaches. Others focus on the deliberate release and expression of emotion through jogging or similar exercise, and/or through appropriate confrontation. In addition some items address themselves to the kind of affect or emotions that surrounds or follows angry feelings and expressions. Thus in summary the item format contributes to eight major goals of the inventory:

(i) To establish reported frequency of feelings of anger, anxiety and depression.

(ii) To establish reported frequency of the expression of these feelings.

(iii) To establish reported duration of these feelings.

(iv) To identify common triggers and targets of these emotions.

(v) To identify the degree of flexibility or the lack of it in dealing with the emotions in question.

(vi) To identify appropriateness or lack of it in handling the three emotions.

(vii) To identify affect and attitudes about feeling and expression of these emotions.

(viii) To identify denial of these three emotions.

Response Alternatives

Closely related to some of the issues raised in the previous section on item format is the role and contribution of response alternatives. The critical issues in scale construction which arise in relation to response alternatives are: (i) specificity, (ii) accuracy, and (iii) memory jogging. These considerations which are critical to a successful psychological instrument are in part controlled by the response alternative mode. The inventories that were examined for possible use in this study--the Buss Hostility-Guilt Inventory, the Beck Depression Inventory, the Taylor Manifest Anxiety Scale and the State Trait Anxiety Inventory--used in most instances the True and False response mode. The one exception was the STAI, in which the state response alternatives were, Not at all, Somewhat, Moderately so and Very Much so; and the trait response alternatives, Almost never, Sometimes, Often and Almost always. These responses

allow for some minimal assessment of intensity with respect to the states and some minimal assessment of frequency with traits.

Probably of greater significance, however, is whether the response alternatives referred to above offer the greatest possible specificity and the highest level of accuracy available. The True and False format elicits data of two types: (i) they inquire as to whether an individual has ever experienced a particular emotion or expression--mere occurrence; or (ii) there is a subtle invitation to the respondent to regard the behavior as typical of them--over generalization.

What does consideration of more varied response alternatives yield? They seem to afford slightly better approximations of frequency or intensity. The basic problem here is, what does, for example, 'almost always' or 'often' mean? What are the referents? And, are these referents comparable between respondents? There is no precise answer to the first question. And it is often agreed with respect to the second, that referents will differ among individuals--thus one individual may interpret 'often' to be once a week, while another may interpret it to be once a day.

The question arises then as to whether a set of response alternatives can be generated that would be a clear improvement on those just discussed. A basic problem with the response alternatives just discussed is that they are overly generalized, and insufficiently differentiated.

Respondents are encouraged to report their habitual biases regarding frequency of particular emotions. But response alternatives which ask the respondent to be more specific should be a more accurate reflection of his actual experience and behavior. Thus an improved set of response alternatives would be: Several times a Day; Every Day; Every other Day; Once a Week; Once a Month; Two or Three times a Year; Never or Not Applicable. Note that there are seven, as opposed to two or four, response alternatives, allowing for more accurate reflection of the various frequencies which do occur. Also since respondents are asked to organize their responses around specifically recalled instances, this permits an investigator the opportunity to establish labels such as 'often' or 'sometimes' in a more meaningful and valid fashion. Finally, use of concrete referents are better memory tracers and triggers than the less specified responses.

Validation of the SESI

Validation on College Students

A sample of 196 college students from St. Peter's College and Brookdale Community College in New Jersey, most of whom were between 17 years and 25 years (N = 126), were used for validating the Subjective Emotional Stress Inventory (SESI). All were members of Introductory Psychology classes and received some credit for completing the SESI. While complete validation and standardization of the SESI is a task well beyond the scope of the current research

project, certain minimal requirements with respect to reliability and validity had to be met before research could proceed further. In particular, not only would coefficients of internal consistency need to be generated for each of the scales, but sufficient evidence that each of the three major scales, Depression, Anxiety, and Anger, were indeed different from each other, needed to be presented also. In addition, with respect to validity, each of the scales would have to demonstrate ability to discriminate between people who were in more and less stressful situations.

At this stage of its development, each item on the SESI is assigned scores based on frequency of feelings or behaviors only. No weights are assigned in relation to intensity of emotions. Thus if a respondent reports "feeling angry everyday," e.g. having arguments, and another respondent reports "feeling very angry everyday," e.g. wanting to hurt someone, each respondent is assigned the same score. Thus current scores provide conservative estimates of the strength of the three emotions measured, anxiety, depression and anger. Detailed examples of scoring numerical value and grouping of items for subscales are given in the Appendix.

The SESI in its original form consists of 18 rationally based subscales, with approximately six subscales for each of the three major scales, Depression, Anxiety and Anger. Means, standard deviations and alphas for the Depression scale are presented in Table 1. The overall alpha for the entire Depression scale based on 101 college students is

TABLE 1

RELIABILITY

SESI Means, Standard Deviations and Alphas* Based on College Students

DEPRESSION SCALE

N = 101 Ss

α = ·82

Subscales	Mean	SD	Items n	Alphas
Feelings - Moderate	17.5	13.2	17	0.90
Feelings - Intense	17.0	13.0	17	0.90
Duration	5.1	3.6	4	0.70
Positive Coping	18.5	10.8	6	0.83
Negative Coping	11.9	7.5	7	0.77

* Cronbach alphas (1951) provide measures of internal consistency by computing what amounts to be the average of all split-half reliabilities. They provide an index of the homogeneity of items in a particular scale.

= .82. Alphas for the individual subscales may be observed to be satisfactory. Duration of Feelings of Depression had the least number of items (N = 4), and the lowest alpha (α = .70). The number of Ss indicated above will be the same for the alphas computed on the three major scales, because while 196 Ss completed the Anger scale, only 101 Ss completed all three scales.

Similar statistics for the Anxiety scale are presented in Table 2. The overall alpha for the entire scale is = .70. Alphas for the individual subscales may be observed to be satisfactory, with Duration of Feelings of Anxiety, obtaining the lowest, α = .58, this subscale has the lowest number of items, N = 4.

Finally, the reliability statistics for the Anger scale are presented in Table 3. The overall alpha for this scale is α = .85. Again, the Duration subscale, with the least number of items (N = 5) obtained the lowest alpha, α = .73.

Despite the satisfactory alpha coefficients referred to above, changes had to be made to each of the scales, because upon examination of the intercorrelation matrix (see Appendix II), several of the subscales were observed to correlate highly with each other, too high, to justify separate classifications. Thus within the three major scales, subscales which correlated between r = .70 and r = .93, with each other were combined to maximize clarity and meaningfulness. This resulted in the Depression subscales being combined into one major scale. The Anxiety subscales were

TABLE 2

RELIABILITY

SESI Means, Standard Deviations and Alphas Based on College Students

ANXIETY SCALE

N = 101 Ss

α = •70

Subscales	Mean	SD	Items n	Alphas
Feelings - Moderate	30.9	32.2	11	0.90
Feelings - Intense	28.9	31.2	12	0.90
Expression	13.7	9.0	12	0.81
Duration	5.7	3.1	4	0.63
Positive Coping	10.9	5.7	4	0.58
Negative Coping	10.1	5.4	4	0.61

TABLE 3

RELIABILITY

SESI Means, Standard Deviations and Alphas Based on College Students

ANGER SCALE

N = 101 Ss

α = ·85

Subscales	Mean	SD	Items n	Alphas
Feelings - Moderate	42.8	63.4	12	0.91
Feelings - Intense	40.8	61.4	12	0.91
Expression - Moderate	42.0	62.4	12	0.91
Expression - Intense	40.2	61.0	12	0.91
Duration	6.0	3.8	5	0.73
Positive Coping	13.6	9.4	13	0.84
Negative Coping	9.5	7.2	9	0.81

combined into three larger scales: (i) Frequency of Anxiety Feelings and Expression; (ii) Negative Coping with Anxiety; (iii) Positive Coping with Anxiety. Finally the Anger subscales were combined into two large scales: (i) Frequency of Feelings of Anger and Expression and (ii) Coping with Feelings of Anger.

Presented in Table 4 are the intercorrelations between the new subscales. One notes the relatively high correlations between Feelings of Depression and Negative Coping with Anxiety, r = .67. This may be explained by the similarity of activities and behaviors which individuals tend to engage in when experiencing these two states, e.g. drinking, smoking, etc. However future work on these scales will pay careful attention to the possibility that they may be redundant. In particular the question of whether this correlation is a sampling fluctuation error or whether it is a representative statistic will be closely examined. Also given the possibility of real redundancy, items will be reexamined and revised and limited to those which discriminate more between depression and negative coping with anxiety.

Perhaps of great theoretical interest is the correlation between Depression and Negative Coping with Anger, r = .48. Negative Coping with Anger focuses on a significant proportion of withdrawal behavior as well as some aggressive behaviors. What this correlation may provide some support for is Freud's (1917) notion that depression is anger turned inward. One may also note the contrasting low

TABLE 4

Intercorrelation Matrix: New Structure of the SESI Based on College Students

N = 101

	Feelings of Depression	Feelings of Anxiety	Negative Coping with Anxiety	Positive Coping with Anxiety	Feelings of Anger	Coping with Anger
Feelings of Depression	=					
Feelings of Anxiety	.18	=				
Negative Coping with Anxiety	.67	.11	=			
Positive Coping with Anxiety	.25	.01	.20	=		
Feelings of Anger	.02	.58	-.01	.08	=	
Coping with Anger	.48	.03	.48	.23	.05	=

correlation of Depression and Expression of Anger, r = .02. Finally, also worthy of note is the correlation between Negative Coping with Anger and Negative Coping with Anxiety, r = .48. This suggests, as might be expected, that there is a negative coping factor. However, substantive and rational issues are more clearly analyzed if they remain separate. Table 5 shows that for the given numbers of items alphas for the new subscales are satisfactory. Fewer items lead to lower alphas, by increasing the length. With similar items alphas may be improved.

In Table 6 intercorrelations between the three major scales are presented. The goal being to generate evidence with respect to whether or not the scales are sufficiently different from each other to justify separate classification. Inspection of the matrix reveals low correlations: Between Anxiety and Depression the correlation is only r = .39; between Depression and Anger, r = .26; and between Anxiety and Anger, r = .25. Thus minimal reliability requirements for the SESI were satisfied, in terms of both internal consistency and differential classifications.

Another consideration made in deciding the continued structure of the SESI was the length of the inventory. A critical factor contributing to the length of each SESI scale is the specification of triggers or targets, individuals within each person's life who are likely to be triggers and/or targets of, for example, anger. Figure 3 shows the percentage of students using specified numbers of triggers/targets on the

TABLE 5

RELIABILITY

SESI Means, Standard Deviations and Alphas,

New Structure, Based on College Students

N = 101 Ss

Subscale	Mean	SD	Items n	Alphas
	DEPRESSION			
Feelings of Depression	54.4	35.6	49	.95
	ANXIETY			
Anxiety Feelings	65.0	32.2	34	.85
Negative Coping with Anxiety	10.1	5.4	7	.52
Positive Coping with Anxiety	10.9	5.7	6	.52
	ANGER			
Feelings of Anger	146.7	63.9	48	.91
Coping with Anger	29.1	16.0	27	.88

TABLE 6

Correlation Matrix of the Three SESI Scales

Based on College Students

N = 101 Ss

	Depression	Anxiety	Anger
Depression	=		
Anxiety	.39	=	
Anger	.26	.26	=

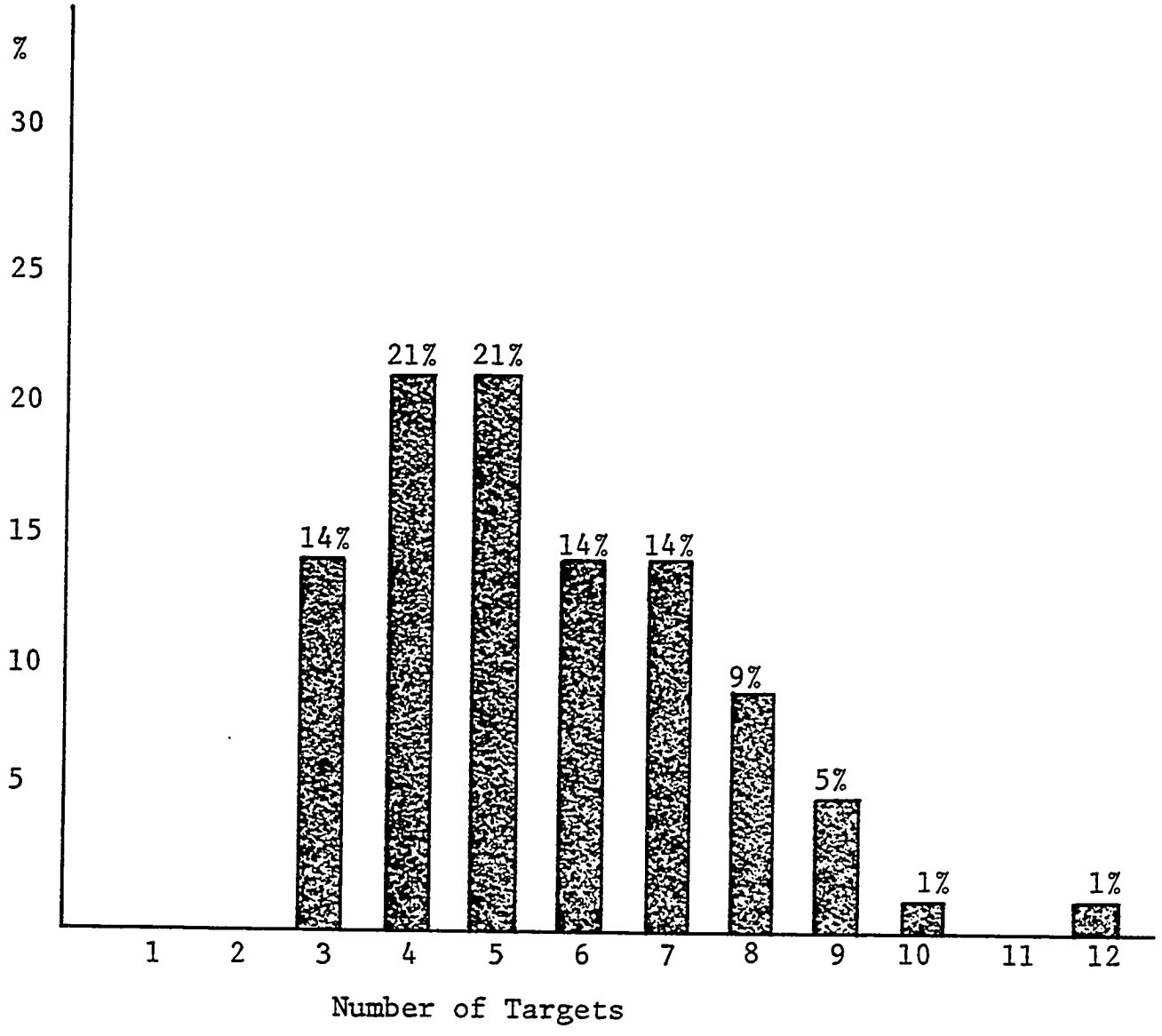

Figure 3

Number of Targets Used by 100
College Students

N = 101

anger scale. The maximum number specified on a SESI subscale is twelve, on the Anger scale. As may be observed, nearly half (42%) of the students identified eight or nine triggers/targets. An additional 14 percent identified ten trigger/targets and 28 percent more identified six or seven trigger/targets. Thus the vast majority of Ss used most of the trigger/targets specified, thereby justifying continued usage.

Another issue which arose was the relationship of the number of persons in an individual's life who could be triggers/targets, and the frequency and/or intensity of subjective distress. This is essentially an empirical problem and it was hypothesized that there would be no relationship between the two variables, that is, people will vary with respect to not only the number of people in their lives, but also with respect to the number of people who become triggers of or targets for their anger. Since there was no prior evidence for this, however, correlations were computed between number of triggers/targets and frequency of depression, anxiety and anger. Table 7 shows that there is a low negative correlation between frequency of feelings of anxiety and number of targets. However there is a low positive correlation between number of targets and coping with anger. The other correlations did not achieve significance. Thus only one significant positive relationship was found, the general trend being non-significant.

The next issue to which attention must be turned is that of the external validity of the SESI. While consistency measures proved adequate, does the SESI have discriminating

TABLE 7

Correlation between Number of Targets and Frequency of Subjective Distress in College Students

N = 101 Ss

	Depressed Feelings	Anxiety Feelings	Coping Negatively with Anxiety	Positive Coping with Anxiety	Anger Feelings	Coping with Anger
Number of Targets	.13	-.20*	.08	.11	-.02	.19*

* $p < .05$

capability with respect to stressful and nonstressful situations? To test this capability, data were analyzed according to particular categories that were hypothesized to be stressful and less stressful. Ss were asked to report whether or not (i) they lived with parents; (ii) whether or not they worked; (iii) whether or not they smoked. It was anticipated that (i) and (ii) would be stressful, that is, if Ss lived with parents or if they were not working. (Many Ss had a well defined need for at least part-time work.) The outcome for smoking was less well defined, smokers having a varied typology that encompasses negative and positive characteristics. However, it was hypothesized that there would be differences between smokers and non-smokers.

In Table 8 results of comparisons between Ss living with parents and those who were not are presented. Using the new structure of the SESI described earlier, where six new subscales were formed, significant differences were obtained on the Depression, Anxiety and Anger scales. On the Depression scale, with an N of 169, $t = 2.28$, $p = .02$, indicating that students who were still living with their parents, N = 109, had significantly more periods of feeling depressed than those who were not living at home, N = 60. On the Anxiety scale, two subscales out of three yielded significant differences. First, Ss living with their parents reported significantly more frequent periods of feeling anxiety than those who were not, $t = 3.20$, $p = 0.002$. In addition, the same students reported significantly more frequent use of

TABLE 8

VALIDITY

Differences between College Students Living with Parents (LP) and Those Who Do Not (NLP), on the SESI

N = 169 Ss

Scales		Mean	SD	N	t	2-tailed Prob.
Depression	LP	39.9	45	109	2.28	0.02*
	NLP	26.4	31	60		
Anxiety Feelings	LP	37.8	37	109		
	NLP	21.5	28	60	3.20	0.002**
Anxiety Coping	LP	12.5	14	109		
	NLP	10.5	12	60	0.99	0.3NS
Coping Positively	LP	5.9	6	109		
	NLP	4.2	5	60	1.95	0.05*
Anger Feelings	LP	145.7	56	109		
	NLP	117.2	71	60	2.70	0.008**
Anger Coping	LP	30.2	15	109		
	NLP	29.0	16	60	0.49	0.6NS

* $p < .05$

** $p < .01$

positive coping strategies, t = 1.95, p = 0.05. Finally students living at home also reported having significantly more frequent periods of feeling and expressing anger than those living away from home, t = 2.70, p = 0.008. Thus the hypothesis was supported and the SESI demonstrated discriminating capability.

Perhaps more striking are the differences presented in Table 9, comparing working students (N = 110) and non-working students (N = 56). Significant differences encompass the three major scales of the SESI. On the Depression scale, t = -3.59, p = 0.001, indicating that non-working students reported feeling significantly more frequent periods of depression. Similar findings obtain for Frequency of Feelings of Anxiety, here t = -3.90, p = 0.000, in the same direction. With respect to Duration and Negative Coping with Anxiety, non-working students reported having these experiences more frequently, t = -2.41, p = 0.02. Finally on Frequency of Feelings of Anger, the non-working students reported having significantly more frequent periods than the working students, t = -3.00, p = 0.004. Once more the hypothesis was supported and the SESI demonstrated discriminating capability.

Finally differences between smokers (N = 62) and non-smokers (N = 102) were observed. Table 10 presents these findings. The first observation which may be made is the almost consistent trend showing lower negative affect scores for smokers. The Depression scale yielded a non-significant

TABLE 9

VALIDITY

Differences between Working (W) and Non-Working (NW) College Students on the SESI

N = 166 Ss

Scales		Mean	SD	N	t	2-tailed Prob.
Depression	W	26.3	36	110	-3.59	0.001***
	NW	51.7	47	56		
Anxiety Feelings	W	24.4	31	110		
	NW	47.7	39	56	-3.90	0.000***
Anxiety Coping	W	9.1	13	110		
	NW	16.3	14	56	-3.21	0.002**
Coping Positively	W	4.6	6	110		
	NW	6.8	6	56	-2.41	0.02*
Anger Feelings	W	125.2	50	110		
	NW	159.4	77	56	-3.00	0.004**
Anger Coping	W	30.0	15	110		
	NW	29.1	16	56	0.36	0.7NS

* $p < .05$

** $p < .01$

*** $p < .001$

TABLE 10

VALIDITY

Differences between Smoking (S) and Non-Smoking (NS) College Students on the SESI

N = 164 Ss

Scales		Mean	SD	N	t	2-tailed Prob.
Depression	S	30.8	43	62		
	NS	39.0	40	102	1.23	0.2NS
Anxiety Feelings	S	24.0	30	62		
	NS	38.0	37	102	-2.63	0.01**
Anxiety Coping	S	12.3	16	62		
	NS	12.0	12	102	0.14	0.8NS
Coping Positively	S	4.2	5	62		
	NS	6.1	6	102	-2.14	0.03*
Anger Feelings	S	124.1	56	62		
	NS	145.5	65	102	-2.23	0.03*
Anger Coping	S	31.5	14	62		
	NS	29.2	16	102	0.95	0.3NS

* $p < .05$

** $p < .01$

difference but favored smokers. The Anxiety scale yielded two significant differences in the same direction. First on the frequency of feelings, $t = -2.63$, $p = 0.01$, indicating that smokers reported significantly fewer periods of anxiety than non-smokers. Second, on Positive Coping with Anxiety, $t = -2.14$, $p = 0.03$, smokers again reporting that they had fewer times when they found it necessary to cope with anxiety. Finally still favoring smokers, there were significant differences on Frequency of Feelings of Anger, $t = -2.23$, $p = 0.03$. Thus although a particular direction was not stated, the SESI differentiated between smokers and non-smokers. With respect to this finding, it's possible that smokers were more likely to be working and/or living away from home, which could account for the direction of these differences. On another level, was the subjective distress identified a function of personality traits associated with a small section of the sample? Or was the subjective distress largely situational, associated, for example, with 'not working' or 'living with parents.' In order to answer this question, the data were subjected to a 2 x 2 x 2 crosstab analysis. Results showed that with smoking controlled, distribution of subjects who were working or not working, and who were living with parents or not living with parents, balanced out almost to parity, indicating that the subjective distress identified was for the most part associated with situational factors. No sex differences were found (females, N = 144; males, N = 52) on any of the three scales of the SESI.

As may be observed the anger coping scale failed to discriminate across the three groups. What this seems to indicate is that combining the positive and negative coping scales because they were highly correlated may have masked differential factors, which cancelled each other out when combined. Thus while 'controlling anger' was not significantly correlated with depression, 'withholding anger' was highly correlated, $r = .52$, $p < .001$.

With respect to the reliability and validity of the SESI, adequate evidence concerning its capability on both of these criteria was found. Now that capability to discriminate subjective distress during stressful periods had been demonstrated, it was time to go on to what could be a more rigorous test, that of discriminating between hypertensives and normotensives.

Validation on Normotensives and Hypertensives

Administering the SESI to a new sample, the essential hypertensives and normotensives, comprising the main focus of this study, also afforded the opportunity to see what rational-factorial structure would obtain. Specifically, one was interested in seeing if there would be replication of the previous structure. The correlation matrix for the new sample may be found in Appendix III. This sample consisted of 108 subjects of whom 37 were between 17 and 23 years and 71 between 26 and 65 years; 41 were hypertensives and 67 were normotensives; the proportion of Black and Caucasian subjects approximated parity.

When compared to the previous correlation matrix, a similar structure may be observed. Thus there was replication of the earlier rational-factorial structure. Subscales that had meaningful correlations ranging from r = .63 to r = .95, or had common variances from 40% to 90%, were combined. This resulted in a reduction from eighteen to eight subscales: in each of the three major scales, Depression, Anxiety, and Anger, the two levels of frequency of feelings (moderate level and intense level) correlated highly with each other, as in the previous sample. They were each combined. In the case of the Anger scale, frequency of expression also seemed to be part of the frequency of feelings cluster; they had correlations of similar order. Thus eight subscales over the three scales were reduced to three. What was left was the Duration and Coping subscales for each of the three scales. They were combined within scales if they correlated .63 or over. This resulted in the other five scales. The eight subscales are: (i) Frequency of Feelings of Depression (DEFEEL); (ii) Duration and Positive Coping with Feelings of Depression (DECOPP); (iii) Frequency of Feelings of Anxiety (AXFEEL); (iv) Expression of Anxiety and Negative Coping with Depression (AXXPR); (v) Duration of Anxiety and Negative Coping (AXCON); (vi) Coping Positively with Anxiety (COPP); (vii) Frequency of Feelings and Expression of Anger (AGFEEL) and (viii) Duration of Feelings of Anger and Negative and Positive Coping (AGCOP). These are in contrast to the six subscales which obtained in the

previous matrix. Reasons for this difference are presented later in this section. Reliabilities for each of the eight subscales are presented in Table 11; they may be observed to be satisfactory.

An intercorrelation matrix between these subscales (N = 108) is presented in Table 12. Inspection will show that some of the correlations are high. Explanations will be offered for those between r = .43 and r = .73. In addition, comparisons will be made with the previous intercorrelation matrix based on data from 101 college students.

When the high correlations are compared to previous correlations, one notes major similarities. First, the correlation between Feelings of Depression and Negative Coping with Anger. In the more recent matrix, r = .43, in the previous matrix (Table 4), r = .48. Thus not only is this good replication but also more support for the Freudian notion that relates these two variables.

Second, attention was drawn in Table 4 before to the correlation between Feelings of Depression and Negative Coping with Anxiety (r = .67); this was explained by pointing to the similar activities and behaviors which tend to characterize these states. In Table 12, Expression of Anxiety and Negative Coping with Anxiety are separated, but as would be anticipated, they both correlate with Feelings of Depression, r = .69, r = .64 respectively. Again, one observes replication.

The third similarity occurs between Feelings of Anxiety and Feelings of Anger. In the earlier matrix, r = .58, in

TABLE 11

RELIABILITY - SESI

SESI Means, Standard Deviations and Alphas

Based on Hypertensives and Normotensives

Depression	Anxiety	Anger
α = .93	α = .82	α = .95

N = 108 Ss

Subscales	Mean	SD	Items-n	Alphas
DEPRESSION - feelings	40.3	43	34	0.96
DEPRESSION - coping	28.9	23	17	0.91
ANXIETY - feelings	56.1	36	21	0.87
ANXIETY - expression	18.2	21	14	0.91
ANXIETY - negative coping	20.5	14	12	0.79
ANXIETY - positive coping	12.1	9	7	0.73
ANGER - feelings	138.7	87	48	0.95
ANGER - coping	33.8	34	27	0.95

TABLE 12

Correlation Matrix of SESI Subscales Based on Hypertensives and Normotensives

N = 108 Ss

	Feelings of Depression	Coping with Depression	Anxiety Feelings	Expression of Anxiety	Positive Coping with Anxiety	Negative Coping with Anxiety	Feelings of Anger	Coping with Anger
Depressed Feelings	=							
Coping with Depression	.32	=						
Anxiety Feelings	.26	.02	=					
Expression of Anxiety	.69	.33	.40	=				
Positive Coping with Anxiety	.37	.30	.21	.30	=			
Negative Coping with Anxiety	.64	.57	.24	.73	.45	=		
Anger Feelings	.36	-.02	.55	.34	.05	.24	=	
Coping with Anger	.43	.20	.28	.36	.14	.34	.38	=

the current matrix, r = .55. The 30 per cent to 34 per cent of common variance in question may be explained by the fact that anger and anxiety tend to be both high energy states. So high energy persons could be providing the common variance observed here.

The current correlation matrix, as may have been observed, has eight subscales in contrast to the previous matrix which has six. The essential difference is that a decision was made to separate out positive coping scales. However, as inspection will show, there are relatively high correlations between Coping Positively with Depression and Coping Positively with Anxiety and Negative Coping with Anxiety-- r = .57 and r = .45 respectively. The 20 per cent to 32 per cent common variance could be explained by a great need for coping, which is sometimes positive and sometimes negative.

Finally, the highest correlation, r = .73, needs to be addressed. This correlation occurs between Negative Coping with Anxiety and Expression of Anxiety. This 53 per cent common variance is hardly surprising; the more anxiety one experiences, the higher the need will be for coping.

Thus successful replication of structure on two samples may be claimed for the SESI, as well as meaningful correlation clusters. Attention is now turned to whether discriminability between the three major scales also replicated. Intercorrelations between them are presented in Table 13. The correlation between the Anger scale and the Depression

TABLE 13

Correlation Matrix of the Three SESI Scales

Based on Hypertensives and Normotensives

N = 108 Ss

	Depression	Anxiety	Anger
Depression	=		
Anxiety	.51	=	
Anger	.25	.29	=

Anger scale is low, r = .25, likewise between the Anxiety scale and the Anger scale, r = .29. The correlation between the Anxiety scale and the Depression scale, however, is higher, r = .51. These scales were regarded as sufficiently distinct to warrant continuation of analyses; the largest amount of common variance being 26%. Also correlations between all the scales were much less than the reliabilities of each scale, thus the homogeneity among scale items was greater than the differences between the scales, thus correction for attenuation would be less than one. The alphas for these three scales are: Depression α = .93, Anxiety α = .82 and Anger α = .95.

Also of interest in this sample was an issue examined in the earlier sample, that of the relationship between the number of persons in an individual's life who could be triggers/targets and the frequency and/or intensity of subjective distress. Table 14 shows that for this sample, that is, the hypertensives and normotensives, relevant correlations did not achieve significance. Thus here, as before, no evidence of a significant relationship was found. Ascertaining the absence of this relationship was of importance because it was necessary to see if the number of targets was a critical contributor to scores on subjective distress. If this were so, it would have been necessary to partial out this source of variance.

TABLE 14

Correlations between Number of Targets and Frequency of Subjective Distress in Hypertensives and Normotensives

N = 108 Ss

	Depressed Feelings	Coping with Depression	Anxiety Feelings	Expression of Anxiety	Anger Feelings	Coping with Anger
Number of Targets	.13	.05	-.07	-.02	-.13	.11

CHAPTER IV: RESEARCH DESIGN

The extended literature review in the early part of this work showed an interesting contradiction: despite the steady accumulation of data from Cannon (1927) to Doyle (1977) suggesting a possible relationship between subjective distress and essential hypertension, there is a conspicuous lack of positive research findings demonstrating a relationship between personality characteristics and essential hypertension. The work of Ostfeld and his associates (1967) is notable in this respect. Nonetheless, experiments involving induction or monitoring of acutely stressful situations have yielded consistent elevations of various related catecholamines and corticoids (Mason, 1972). If the present research can be anticipated to yield anything different than that reported by similar studies, changes will have to be through the use of more appropriate instruments. To this end none of the traditional scales or inventories--the Minnesota Multiphasic Personality Inventory (MMPI), the Sixteen Personality Factor Questionnaire (16PF) for example--were utilized; instead the inventory described in the previous section, designed to bring relevant variables into clearer focus, was used. The major purpose of this research design then was to put to as rigorous and economical a test as resources allowed the hypothesis that hypertensives experience more subjective emotional distress than normotensives. Although the design

examined correlational patterns rather than causal data, clarity at this level of analysis is needed, and can provide an important foundation for future work.

The basic data structure allowed for a 2 x 2 analysis of variance with controls for age and covariances computed on possible confounding variables. More specifically, the goal was to test for main effects with respect to hypertension on several measures of subjective distress and life stress. Matching was used as a control where possible, but analysis of covariance was computed on ethnicity, weight and sex. Although the research design requires that several physiological measures be taken on each subject, their analysis will be limited to t-tests, to verify differences between 'experimental' groups and their controls.

Sampling

Expectations concerning availability of subjects at an outpatient clinic were not fulfilled; however with respect to the 'experimental' group, care was taken to select persons with essential hypertension as opposed to those with secondary hypertension. Outpatients at a metropolitan hospital clinic were perceived to be ideal subjects. If hospital population and records had allowed, essential hypertension subjects would have been further subdivided according to two main criteria: (i) length and/or severity of hypertension and (ii) type of hypertension. With respect to the former, categories were differentiated between labiles and mild hypertensives, diastolics between 90 and 110 mm Hg and moderate to

severe hypertensives, that is diastolics over 110 mm Hg. With respect to type of hypertension, if hospital records had allowed, patients would have been further classified according to (i) renin levels and (ii) plasma volume. These classifications would recognize, as revealed in the review of the literature, that essential hypertensives are a heterogenous rather than a homogeneous group. Despite well-laid plans for working with a metropolitan outpatient hospital and attendance there for several months, data collection had to extend itself to a nursing station at a community college and a metropolitan apartment building, thus classification by renin levels and plasma volume were not possible. Further details are described in the section "Verification of Experimental Classification."

While these additional sites helped to provide the samples of interest, charts allowing for detailed information on severity and type of hypertension were not available. Thus information is limited to identification of essential hypertensives without other related medical diagnoses such as kidney dysfunction and diabetes mellitus. No information was forthcoming with respect to renin or plasma volume classification, though subjects generally would be able to indicate how long they had hypertension and what kind of medication they were taking.

Observed Variable and Outcome Variables

The observed variable in this study is the presence of hypertension, that is, persons who have blood pressure

reading of over 140/90 mm Hg. The nature of the research problem does not allow the direct manipulation of the observed variable, thus alternative strategies were employed. In particular, individuals with the required characteristics were sought. Ideally, the "best" form of the observed variable would be found in untreated hypertensives. However, the limited resources of this study did not permit the extensive screening that this would entail. Thus in order to obtain a hypertensive sample as well as a control normotensive group, treated hypertensives from an outpatient clinic were used. This raises the issue of the appropriateness of using treated hypertensives in this study.

Probably the most relevant consideration here is that hypertensives under treatment have their hypertension controlled rather than cured. This means that study of "treated" hypertensives is not inappropriate. Thus although ideal conditions have not been established with respect to the observed variable, the actual conditions are useful in that they still are able to provide a relevant form of the observed variable.

The outcome variables in this study are several. At the most general level, they are: (i) number of stressful life events; (ii) frequency of feelings and expression of anger; (iii) frequency of feelings and expressions of anxiety; (iv) frequency of feelings and expression of depression; (v) levels of blood pressure and heart rate before, during and after the interview.

The first measure offers an assessment of the number of stressors in the individual's life. The second, third and fourth measures offer an assessment of subjective emotional distress, which may be related to the stressors. The fifth measure is that of heart rate and blood pressure, the latter being technically the same as the observed variable. However, blood pressure can be viewed from several vantage points. With respect to the observed variable, blood pressure is used as a general or average measure. With the outcome variable, blood pressure is measured in terms of moment to moment fluctuations as a function of focusing on stressful life events and related subjective emotional responses. The question might arise as to whether the relationship between blood pressure as observed variable and blood pressure as an outcome variable would not be so highly correlated as to make a study unnecessary, but this is precisely the point of the study, the relationship as revealed by research conducted so far is unclear but of great interest.

Therefore it is hypothesized in this study that there may be some reliable differences between the experimental and control group in systolic and/or diastolic blood pressures in responding to the interview. Measures of blood pressure are being taken before, during and after the interview because from the literature review, any of these three points may prove to be sensitive.

Measures of heart rate as monitored by the pulse were also taken in order to better interpret the nature of the

blood pressure response: that an increase of heart rate accompanied by an increase in systolic blood pressure could be interpreted as increased blood pressure resulting from increased cardiac activity. On the other hand, increase in blood pressure with no increase in heart rate could suggest increased blood pressure resulting from increased peripheral resistance. The capability to monitor such differences increases significantly the meaningfulness and possible usefulness of the data. In addition, given the incomplete nature of hypertension treatment, further monitoring of hypertensive responses can provide relevant information that could ultimately lead to more specific and more complete treatments.

Possible Confounding Factors and Controls

There are four factors which are perceived as affecting level of blood pressure and which should therefore be controlled. The control strategy to be employed is that of matching. Thus the 'experimental' or hypertensive group was matched with the control or normotensive group with respect to range of age, range of weight and height, and ethnicity. Matching of subjects was not done by the investigator, since this would negate the single blind procedure.

In order to gain a better understanding of the many dynamics at work, some additional characteristics of the hypertensive and normotensive groups were monitored. These were: number of cigarettes smoked per day; parents with

hypertension or related disorders; diet--amount of salt intake. Also, as mentioned before, attempts were made to monitor medication in the hypertensive group.

Experimental Conditions

Experimental conditions can be classified into two main categories--external factors and internal factors. With respect to the former, Wenger (1962) pointed to five environmental variables which relate substantially to physiological results:

(i) Time of testing

(ii) Initial room temperature

(iii) External temperature at time of testing

(iv) Lowest relative humidity

(v) Highest barometric pressures in the 24-hour period before testing.

With respect to these factors, efforts were made to make sure that time of testing and room temperature remained uniform across groups. Control of external temperature, relative humidity and barometric pressure was considerably more difficult to achieve. Data were collected in the late fall. Appointments were rescheduled if days were extremely inclement.

Internal factors are basically of two kinds and are more difficult to measure than the external variables. The first is the subject's definition of the situation and the second is the subject's level of activation or motivation.

With respect to the former, efforts were made to structure a particular mental set and to reinforce it throughout the interview session. Also, in order to maximize the power of the experimental condition, the beginning and the end of the interview had to be clearly defined. Thus in keeping with this structure, the 'before' blood pressure was taken before any instructions or explanations were given. The 'during' blood pressure was taken while subjects were filling out the anger scale from the Subjective Emotional Stress Inventory (SESI). And the 'after' blood pressure was taken while chatting with or after debriefing subjects, when that proved to be necessary.

The motivation or activation variable was assessed by taking note of gross behaviors and postural cues, as well as two or three questions about perceived level of alertness.

With respect to possible carryover effects, in particular, boredom or fatigue, the order of questionnaires was arranged in a manner designed to minimize these effects. The Psychiatric Epidemiology Research Interview (PERI), a stressful life events questionnaire, was administered first; that is, after taking blood pressure and pulse and general orientation of the subjects. The PERI, while based on the concept of "life change units" like the popular Holmes and Rahe scale, is far more specific, and in this manner clearly superior. Focus on stressful life events provided an appropriate mind set for the Subject Emotional Stress Inventory which followed. Both the PERI and the SESI were self-

administered. Together with the taking of bilateral blood pressures and pulses, administration varied from one hour to one and a half hours for each subject.

Hypotheses

As stated in the Introduction, the empirical focus of this research was on comparable levels of subject distress experienced by hypertensives and normotensives. Both the literature review and inventory generation have been focused on explaining or facilitating this process. Below are the ten hypotheses that focused this research. The first eight are concerned with depression, anxiety and anger as they are measured on the Subjective Emotional Stress Inventory. The other two are concerned with frequency of stressful life events as measured by the Psychiatric Epidemiology Research Interview.

Hypothesis I:

Essential hypertension subjects will show significantly higher levels of frequency of feelings of depression when compared to normotensive controls.

Hypothesis II:

Essential hypertensive subjects will show significantly higher levels on duration of feelings of depression and manner of coping when compared to normotensive controls.

Hypothesis III:

Essential hypertensive subjects will show significantly

higher levels of frequency of feelings of anxiety when compared to normotensive controls.

Hypothesis IV:

Essential hypertensive subjects will show significantly higher levels of frequency of expressing anxiety when compared to normotensive controls.

Hypothesis V:

Essential hypertensive subjects will show significantly higher levels of frequency of positive coping with feelings of anxiety when compared with normotensive controls.

Hypothesis VI:

Esssential hypertensive subjects will show significantly higher levels of duration of feelings of anxiety and negative coping when compared to normotensive controls.

Hypothesis VII:

Essential hypertensive subjects will show significantly higher levels of frequency of feelings of anger and frequency of expression of feelings of anger than normotensive controls.

Hypothesis VIII:

Essential hypertensive subjects will show significantly higher levels of duration of feelings of anger and manner of coping when compared to normotensive controls.

Hypothesis IX:

Essential hypertensive subjects will show significantly higher levels of number of negative life stressors when compared to normotensive controls.

Hypothesis X:

Essential hypertensive subjects will show significantly lower levels of number of positive life stressors when compared to normotensive controls.

Subjects

A total of 108 subjects completed the experimental sessions in time to be part of the current data analysis. There were four subjects, all over twenty-six (26) years, who started, but chose not to complete the inventories. Of the 108 Ss, 37 were between 17 years and 23 years and 71 were between 26 years and 65 years, creating the basic separation based on age. Across these two groups, 52 Ss were Caucasian and 50 were Black, 6 were Hispanic.

Within the two groups, the major breakdown was between hypertensives (N = 27) and normotensives (N = 43) for the older groups, and labiles (N = 14) and normotensives (N = 23) for the younger groups. In both cases, normotensives numbered considerably more than their counterpart group. With respect to the younger groups, it may be seen in Table 15 that there are inequalities: a preponderance of females (64%), a larger number of Caucasian labiles (71%), though weight is less unevenly distributed. In Table 16, inequalities may be observed also; here the diagnosed hypertensives are compared with their controls. Preponderance of females obtain once more (74%), a larger number of Black hypertensives (63%); but perhaps the most striking of all, is the large percentage

TABLE 15

Comparisons of Characteristics of 'Labiles' and Their Controls

Characteristics		Labiles %*	Controls %**
Sex:	Female	64% (9)	70% (16)
	Male	36% (5)	30% (7)
Race:	Black	29% (4)	35% (8)
	Caucasian	71% (10)	65% (15)
Weight:	Desirable and Underweight	79% (11)	87% (20)
	Overweight	21% (3)	13% (3)

* N = 14

** N = 23

TABLE 16

Comparisons of Characteristics of Hypertensives and Their Controls

Characteristics		Hypertensives %*	Controls %**
Sex:	Female	74% (20)	64% (28)
	Male	26% (7)	36% (16)
Race:	Black	63% (17)	57% (25)
	Caucasian	37% (10)	43% (19)
Weight:	Desirable and Underweight	30% (8)	73% (32)
	Overweight	70% (19)	27% (12)

* N = 27

** N = 44

of people in the hypertensive category who are overweight (70%) when compared to the controls (26%). With these inequalities, while examining the effects of hypertension on specified dependent variables, it would be necessary to not only control for age, but to compute covariance for sex, ethnicity, and weight also. This was done throughout the analysis and is described in the "Results" section which follows.

Verification of Experimental Classification

The conditions which obtained while selecting subjects make it necessary to verify classifications. The original intent of the study, as described earlier, was to screen and select all subjects from a metropolitan outpatient clinic. This did not work out as had been planned, in part because of the limited type of patients who could be persuaded to cooperate. Specifically, it was observed that many of the patients were over 50 years and some had multiple diagnoses, which disqualified them for this study. The decision was then made to expand data collection to include a metropolitan apartment building and a community college campus. This expansion was designed to gain access to more people between the ages of 30 years to 45 years, people who were working or otherwise pursuing an active lifestyle and who had no known health problems in the case of the normotensive controls and whose only health problem was hypertension in the case of

the hypertensive subjects. This action fulfilled our expectations and resulted in a sample of 108 subjects.

In the course of this action, an unexpected observation was made: originally interest had been focused on gaining subjects 25 years and older, because it has been observed that essential hypertension tends to manifest itself after this age. However, because young people were willing to volunteer, the decision was made to let them participate. The unexpected observation was the physiological response of some of these young subjects to the experimental condition.

The research design called for taking seated bilateral blood pressures as the very first part of the experimental condition. This was followed by administration of the PERI and the SESI. Seated bilateral blood pressures were also taken during the administration of the Anger scale on the SESI. Finally seated bilateral blood pressures were taken when the SESI had been completed, subjects had been thanked and given a sliding key chain, containing a mirror and which had the inscription "Enjoy each day . . . It's yours! Thank you." This delighted them without fail.

The physiological response which caught our attention is demonstrated by examining the blood pressures of an 18 year old female subject. Initial blood pressure reading in the left arm were 130/100 mm Hg. Left arm blood pressures taken during the Anger scale reflected a marked increase, 160/120 mm Hg. Finally, left arm blood pressures at the end of the session were 130/92 mm Hg. Right arm blood

pressures followed a similar pattern, 114/90 mm Hg initially, 150/120 mm Hg during, and 140/98 mm Hg on completion. It probably should be underscored here that the experimental condition was non-stressful. Blood pressures were taken and inventories were administrated in an informal group setting. Individuals were often acquainted with each other or introduced if they were seated at the same table. The eighteen year old subject referred to earlier, for example, attended the session with a friend. Apple juice was available for those who cared but generally subjects were discouraged from drinking coffee or smoking, since these could artificially inflate blood pressures.

A decision was made to continue interviewing younger subjects and create as a special category those whose blood pressures went beyond 140/90 mm Hg at any point during the experimental session. These subjects will be referred to as 'labiles' to differentiate them from diagnosed hypertensives. When the decision to stop data collection was made, it turned out that the 'labiles' were all between 17 and 23 years. Thus four groups emerged, the older diagnosed hypertensives, over 26 years, and their controls, and the younger undiagnosed labiles and their controls.

In Table 17 mean bilateral blood pressures, Before, During and After, for the older diagnosed hypertensive group compared to their controls are presented. As may be observed, all differences are very significant.

TABLE 17

Comparisons of Mean Bilateral Blood Pressures of Hypertensives and Their Controls

		Hypertensives	Controls	df	t
BEFORE:	Syst $\overline{BP}X$	153.26	119.37	40	8.07***
	SD	19.5	12.4		
	Diast $\overline{BP}X$	100.00	76.0	41	7.31***
	SD	15.0	10.0		
DURING:	Syst $\overline{BP}X$	145.56	114.56	36	6.22***
	SD	23.6	13.3		
	Diast $\overline{BP}X$	98.44	74.6	54	7.76***
	SD	12.6	12.3		
AFTER:	Syst $\overline{BP}X$	144.22	112.02	36	6.88***
	SD	22.3	15.1		
	Diast $\overline{BP}X$	96.5	77.1	44	5.76***
	SD	15.1	11.2		

Hypertensives N = 27

Controls N = 43

*** $p < .001$

In Table 19 mean bilateral blood pressures, Before, During and After, for the younger labile group compared to their controls are presented. Again the differences between these two groups may be observed to be very significant.

The same information is diagrammed in Figures 4 and 5, for both groups and their controls. The consistent significant differences in the case of the younger groups, justified in part, keeping these undiagnosed labiles as a separate group.

Also of relevance are the heart rates of the experimental and control groups. As shown in Table 18, there were significant differences in heart rates between hypertensives and their controls on the Before and After measures, hypertensives had higher heart rates. A similar pattern may be observed in Table 20. Although the differences were not significant, higher heart rates and greater lability were recorded for the labiles. The parallels obtained between hypertensives and labiles on heart rate provide further justification for the experimental classification.

TABLE 18

Comparisons of Mean Heart Rates of Hypertensives and Their Controls

		Hypertensives	Controls	t
BEFORE:	Heart Rate $\bar{X}$	86	77	2.55*
	SD	14	15	
DURING:	Heart Rate $\bar{X}$	81	77	1.39
	SD	11	9	
AFTER:	Heart Rate $\bar{X}$	81	75	2.75**
	SD	9	7	

Hypertensives N = 27

Controls N = 43

* $p < .05$

** $p < .01$

TABLE 19

Comparisons of Mean Bilateral Blood Pressures of Labiles and Their Controls

		Labiles	Controls	t
BEFORE:	Syst $BP\bar{X}$	130.1	114.4	3.29**
	SD	16.0	10.0	
	Diast $BP\bar{X}$	86.6	72.5	4.01***
	SD	10.0	10.7	
DURING:	Syst $BP\bar{X}$	128.4	111.9	3.74***
	SD	14.6	9.8	
	Diast $BP\bar{X}$	83.3	69.8	4.13***
	SD	13.3	8.7	
AFTER:	Syst $BP\bar{X}$	126.0	111.5	4.38***
	SD	10.4	8.4	
	Diast $BP\bar{X}$	87.7	73.7	4.49***
	SD	10.4	6.6	

Labiles N = 14

Controls N = 23

** $p < .01$

*** $p < .001$

TABLE 20

Comparisons of Mean Heart Rates of 'Labiles' and Their Controls

		Labiles	Controls	t
BEFORE:	Heart Rate $\bar{X}$	85	78	1.36NS
	SD	19	10	
DURING:	Heart Rate $\bar{X}$	76	76	0.11NS
	SD	17	9	
AFTER:	Heart Rate $\bar{X}$	80	76	0.82NS
	SD	14	12	

Labiles N = 14

Controls N = 23

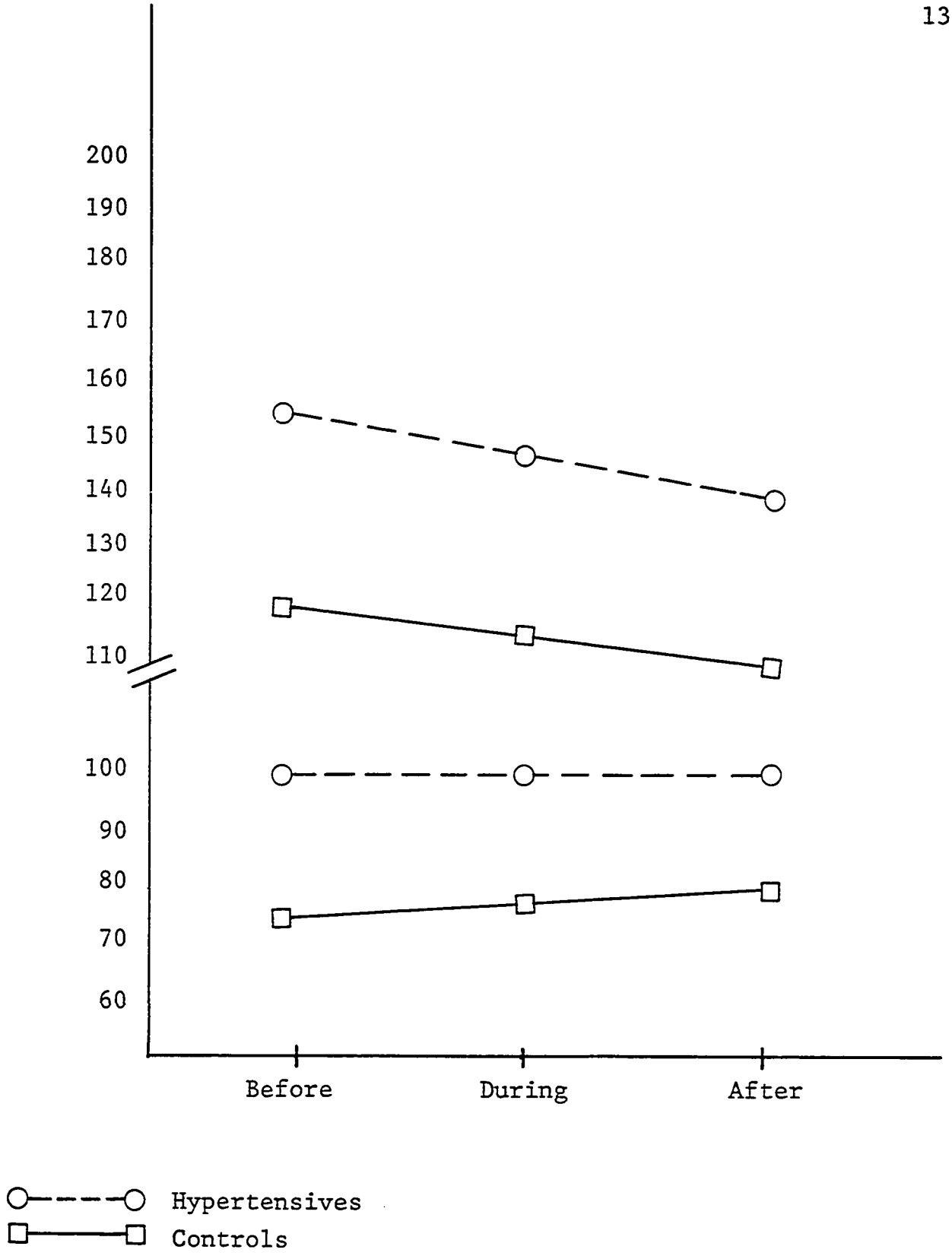

Figure 4

Mean Bilateral Blood Pressures for Hypertensives and Their Controls

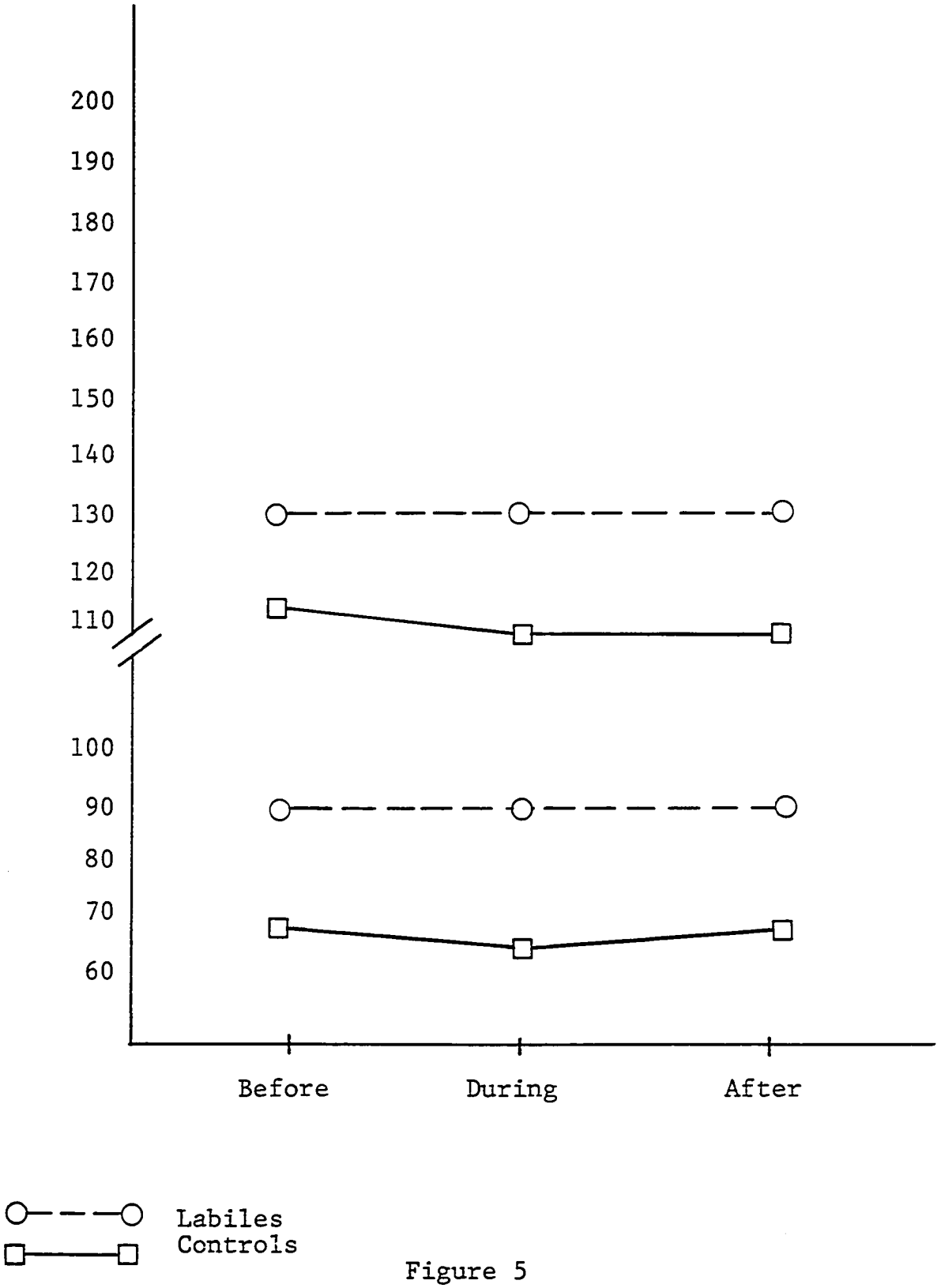

Figure 5

Mean Bilateral Blood Pressures for 'Labiles' and Their Controls

CHAPTER V: RESULTS

This research study has been concerned with testing hypotheses related to possible differences between hypertensives and normotensives on several measures of subjective distress and life stressors. Analysis of variance, the hierarchical approach by SPSS (Nie et al., 1975) was the basic statistic used for analyzing the data. This particular approach was chosen because it capitalized optimally on the unequal cell frequencies and the need to examine several other variables simultaneously. In each case, a two-way ANOVA was computed controlling for age, with three covariates--sex, ethnicity and weight.

Hypothesis I was concerned with significantly higher levels of frequency of feelings of depression in hypertensives. Relevant data are presented in Table 21. By inspection it may be seen that neither age nor hypertension achieved significance in relation to Frequency of Feelings of Depression. When covariates are examined, none achieved significance either, but it may be observed that weight approached significance, $p = 0.07$. Not shown in the table is the correlation between Frequency of Feelings of Depression and weight: $r = -.20$, $p = 0.04$. In this sample, there seems to be some relationship between greater weight and increased reports of depression. Thus there was no support for Hypothesis I.

TABLE 21

Analysis of Variance of Frequency of Depression

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	7982.305	3	2660.77	1.45	0.23
Ethnicity	194.870	1	194.87	0.11	0.75
Sex	1500.156	1	1500.16	0.82	0.37
Weight	6287.277	1	6287.27	3.42	0.07
Main Effects:	5085.387	2	2542.69	1.39	0.25
Age	942.266	1	942.26	0.51	0.47
Hypertension	4143.121	1	4143.12	2.26	0.14
2 Way Interactions	5.891	1	5.89	0.003	0.95
Age Hypertension	5.891	1	5.89	0.003	0.95

Hypothesis II was concerned with significantly higher levels on Duration of Feelings of Depression and Positive Coping in hypertensives. By inspection of Table 22 it may be seen that hypertension as a main effect achieved significance, $F = 9.71$, $p = 0.002$, indicating that hypertensives reported significantly longer periods of Feelings of Depression and need for Positive Coping than normotensives. However, there are two types of hypertensives, the labiles or undiagnosed and the diagnosed hypertensives. Controlling for age addresses itself to the differences between these two groups, but in an effort to further clarify the subjective distress profiles of the diagnosed and the undiagnosed, T-tests were computed between their scores on the SESI. Results showed that on all the SESI scales, there were no significant differences, indicating that the labiles were responding in a manner similar to hypertensives. Thus, despite diagnostic status, main effects for hypertension are indeed just this (see Figure 6). Hypothesis II was supported.

Hypothesis III was concerned with significantly higher levels on Frequency of Feelings of Anxiety in hypertensives. By inspection of Table 23, it may be observed that once more hypertension as a main effect achieved significance, $F = 4.87$, $p = 0.03$. Perhaps even more striking is the level of significance achieved by age, $F = 9.08$, $p = 0.003$. As might be expected there is also a significant interaction between age and hypertension, $F = 10.60$, $p = 0.002$. This is diagrammed in Figure 7. It should be noted that there is an unusual

TABLE 22

Analysis of Variance of Coping with Feelings of Depression

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	38.081	3	12.694	0.05	0.98
Ethnicity	6.209	1	6.209	0.02	0.87
Sex	18.837	1	18.837	0.08	0.78
Weight	13.035	1	13.035	0.05	0.82
Main Effects:	3037.339	2	1518.669	6.16	0.003**
Age	644.911	1	644.911	2.62	0.109
Hypertension	2392.428	1	2392.428	9.71	0.002**
2 Way Interaction	252.854	1	252.84	1.026	0.31
Age Hypertension	252.854	1	252.84	1.026	0.31

** $p < .01$

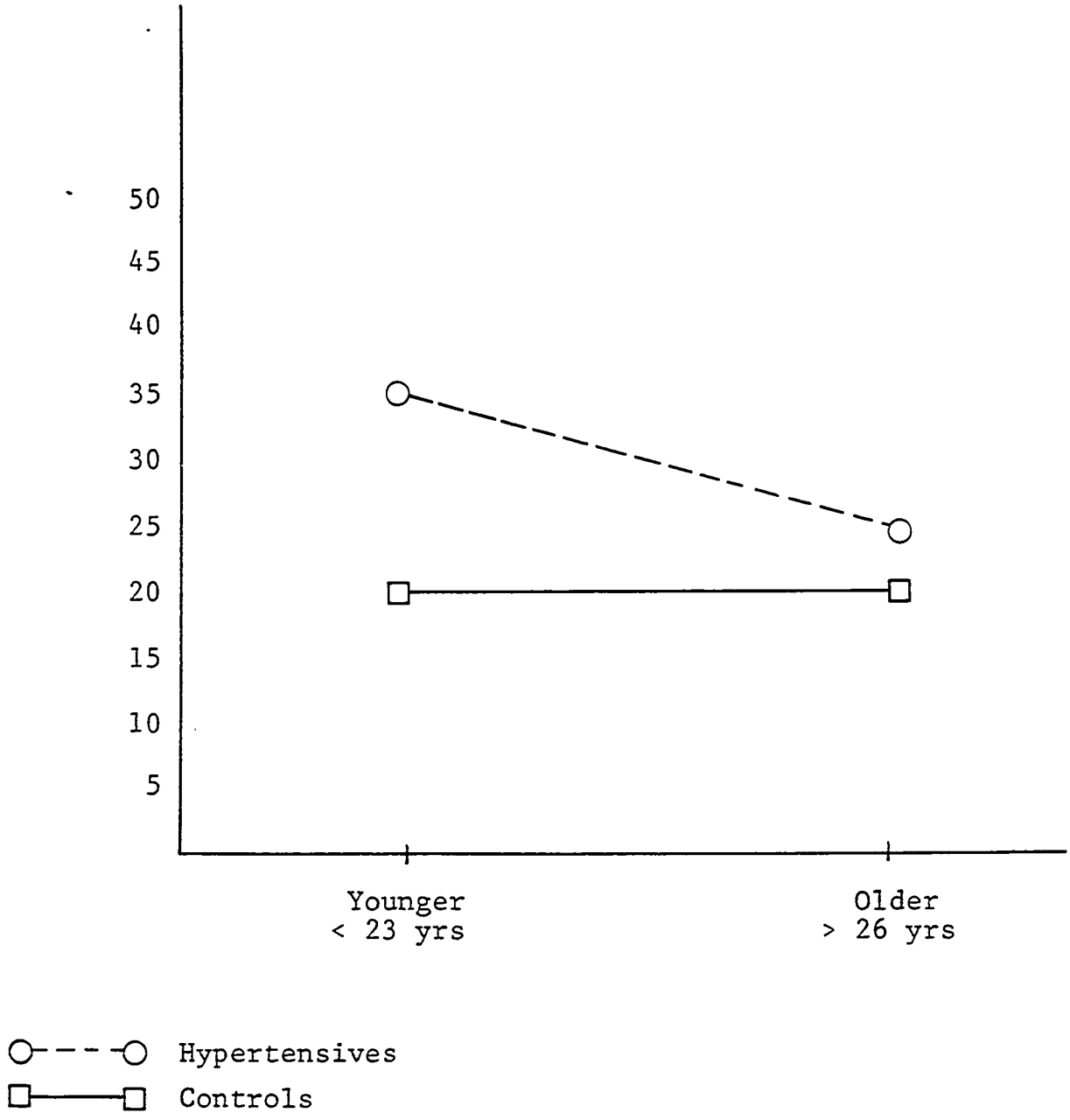

Figure 6

Comparisons of Hypertensives and Their Controls on Coping with Depression

TABLE 23

Analysis of Variance of Frequency of Feelings of Anxiety

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	944.726	3	314.90	0.263	0.852
Ethnicity	157.601	1	157.60	0.132	0.718
Sex	787.000	1	787.00	0.657	0.420
Weight	0.125	1	0.13	0.000	0.992
Main Effects:	15349.789	2	7674.89	6.41	0.002**
Age	10870.523	1	10870.52	9.08	0.003**
Hypertension	4479.266	1	4479.27	4.87	0.03 *
2 Way Interactions:	12706.484	1	12706.48	10.61	0.002**
Age Hypertension	12706.484	1	12706.48	10.61	0.002**

* $p < .05$

** $p < .01$

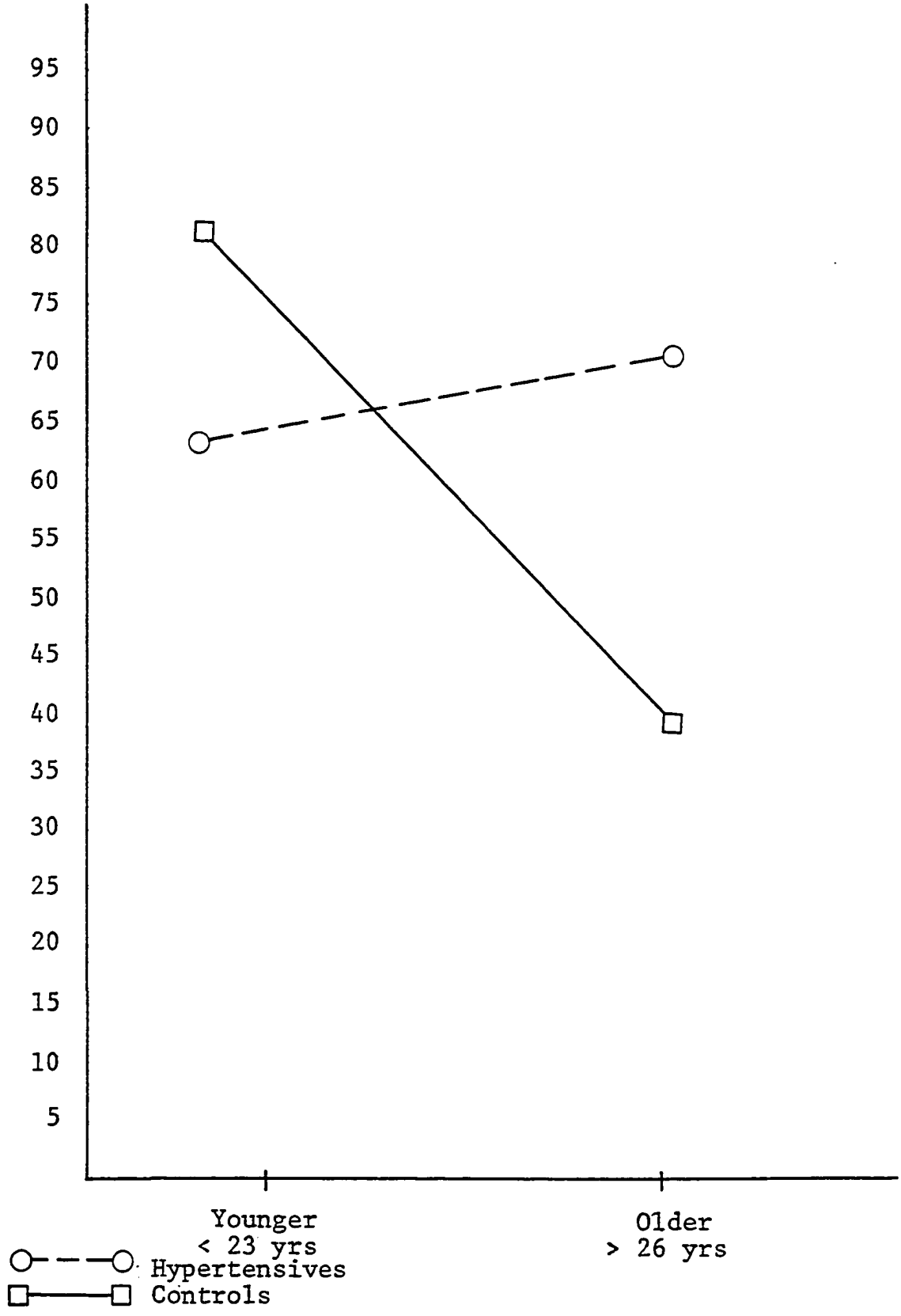

Figure 7

Comparisons of Hypertensives and Their Controls on Frequency of Feelings of Anxiety

finding here, in that the young normotensives had the highest scores. More specifically, the pattern of frequency of feelings of anxiety took this form. Older hypertensives and younger normotensives had the highest scores, which were non-significantly different from each other. Labiles had significantly lower scores than younger normotensives, but significantly higher scores than older normotensives. Thus with respect to anxiety, older normotensives reported experiencing low levels of frequency while the younger normotensives reported experiencing higher levels of frequency--the largest difference between two groups, $t = 5.09$, $p < .001$. The magnitude of this difference compared to that of the labiles could suggest that the cardiovascular systems of the labiles may be more sensitive to feelings of anxiety, since labiles experienced significantly lower levels of frequency than the younger normotensives, $t = 2.12$, $p = .04$. Thus the interaction is explained and Hypothesis III was supported.

Hypothesis IV was concerned with significantly higher levels on Frequency of Expression of Anxiety in hypertensives. No significant levels were achieved, thus no support was gained for Hypothesis IV.

Hypothesis V concerned itself with significantly higher levels on Coping Positively with Anxiety in hypertensives. Table 24 presents the results. Hypertension achieved significance as a main effect, $F = 4.47$, $p = 0.04$, indicating that hypertensives reported spending more time Coping Positively with Anxiety. Thus some support was obtained for Hypothesis V.

TABLE 24

Analysis of Variance of Positive Coping with Anxiety

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	121.015	3	40.338	1.153	0.33
Ethnicity	18.100	1	18.100	0.518	0.47
Sex	0.481	1	0.481	0.014	0.91
Weight	102.434	1	102.434	2.929	0.09
Main Effects:	203.144	2	101.572	2.904	0.06
Age	46.929	1	46.929	1.342	0.25
Hypertension	156.214	1	156.214	4.467	0.04*
2-Way Interation:	2.115	1	2.115	0.06	0.806
Age Hypertension	2.115	1	2.115	0.06	0.806

* $p < .05$

Hypothesis VI concerned itself with significantly higher levels on Duration of Feelings of Anxiety and Negative Coping in hypertensives. By inspection of Table 25, it may be seen that the main effect in general barely approached significance, $F = 4.45$, $p = 0.04$. This indicated that there was a tendency for hypertensives to spend more time experiencing feelings of anxiety and coping negatively with them. Thus Hypothesis VI was supported.

Hypothesis VII concerned itself with significantly higher levels of Frequency of Feelings and Expression of Anger in hypertensives. By inspecting Table 26, it will be seen that not only is the main effect hypertension significant, $F = 7.49$, $p = 0.007$, but the interaction between age and hypertension is also significant, $F = 4.87$, $p = 0.03$. With respect to the interaction, examination of means reveals that younger normotensives and labiles reported greater Frequencies of Feelings of Anger than older normotensives, though only the former achieved significance, $t = 2.10$, $p = 0.04$; the latter approached significance, $t = 1.87$, $p = 0.07$. However, as shown in Figure 8, the older hypertensives reported the highest level of Frequency of Feelings of Anger, though this did not prove to be significantly different from either the labiles or their controls. Thus the interaction represents relatively similar levels of Frequency of Feelings of Anger among labiles, their controls and the hypertensives in contrast to the older normotensives. Hypothesis VII received support.

TABLE 25

Analysis of Variance of Duration and Negative Coping with Anxiety

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	129.680	3	43.227	0.399	0.754
Ethnicity	70.237	1	70.237	0.649	0.423
Sex	2.494	1	2.494	0.023	0.880
Weight	56.948	1	56.948	0.526	0.470
Main Effects:	484.310	2	242.155	2.236	0.113
Age	2.078	1	2.078	0.019	0.890
Hypertension	482.232	1	482.232	4.453	0.038*
2-Way Interaction:	119.435	1	119.435	1.103	0.296
Age Hypertension	119.435	1	119.435	1.103	0.296

* $p < .05$

TABLE 26

Analysis of Variance of Frequency of Feelings and Expression of Anger

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	6475.230	3	2158.410	0.312	0.877
Ethnicity	1491.181	1	1491.181	0.215	0.644
Sex	3763.698	1	3753.698	0.544	0.463
Weight	1220.352	1	1220.352	0.176	0.675
Main Effects:	53776.168	2	26888.082	3.884	0.024*
Age	1943.445	1	1943.445	0.281	0.597
Hypertension	51832.723	1	51832.723	7.488	0.007**
2-Way Interaction:	33691.914	1	33691.914	4.867	0.03 *
Age Hypertension	33691.914	1	33691.914	4.867	0.03 *

* $p < .05$

** $p < .01$

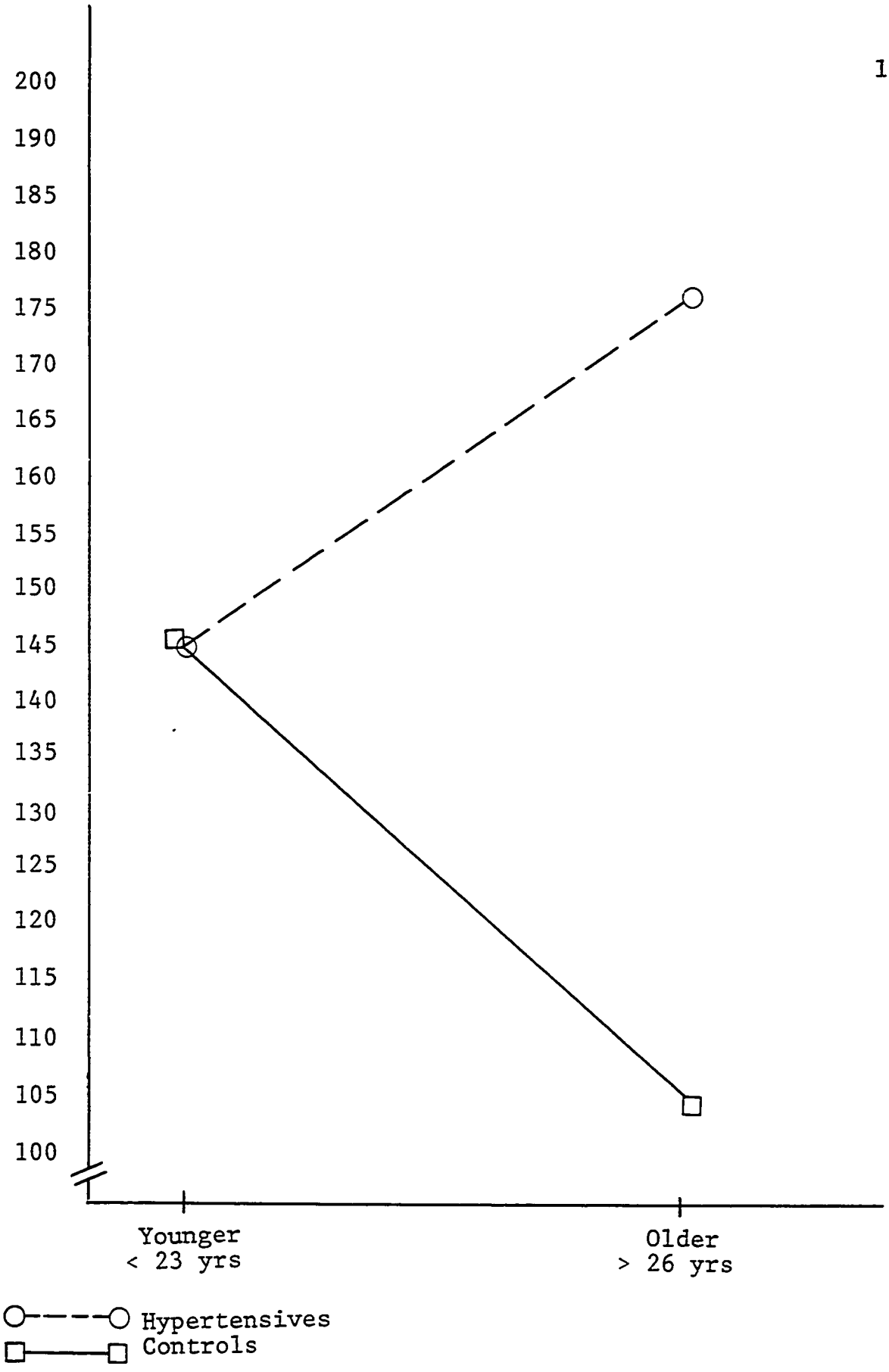

Figure 8

Comparisons of Hypertensives and Their Controls on Frequency of Feelings and Expression of Anger

A major difference between the interpersonal environments of younger and older persons was hypothesized to be proximity to parents. In Figure 9 the differences in percentages, of three samples reporting mother and father as triggers and/or targets of anger is presented. One is able to observe the similarity of the two young samples in contrast to the older sample.

Hypothesis VIII concerned itself with significantly higher levels on Duration of Feelings of Anger and manner of coping with these feelings in hypertensives. Relevant data are presented in Table 27. It may be observed that only the covariate weight achieved significance, $F = 7.76$, $p = 0.006$. In explaining this, it may be helpful to observe that there is a significant negative correlation between weight and control of anger, $r = -.25$, $p = 0.01$, indicating that those who report controlling more anger tend to weigh less. However, in both Frequency of Feelings of Depression and Duration and Control of Feelings of Anger, where weight achieved significance as a covariate, and there were negative correlations with both variables, this may be further clarified by noting that for this sample, there is a very significant correlation between age and weight, $r = .38$, $p < 0.001$. Thus the significance of weight may also be pointing to a tendency for younger people in this sample to have greater Frequency of Feelings of Depression, as well as of Duration and need to Control Anger. Nevertheless, Hypothesis VIII did not obtain support.

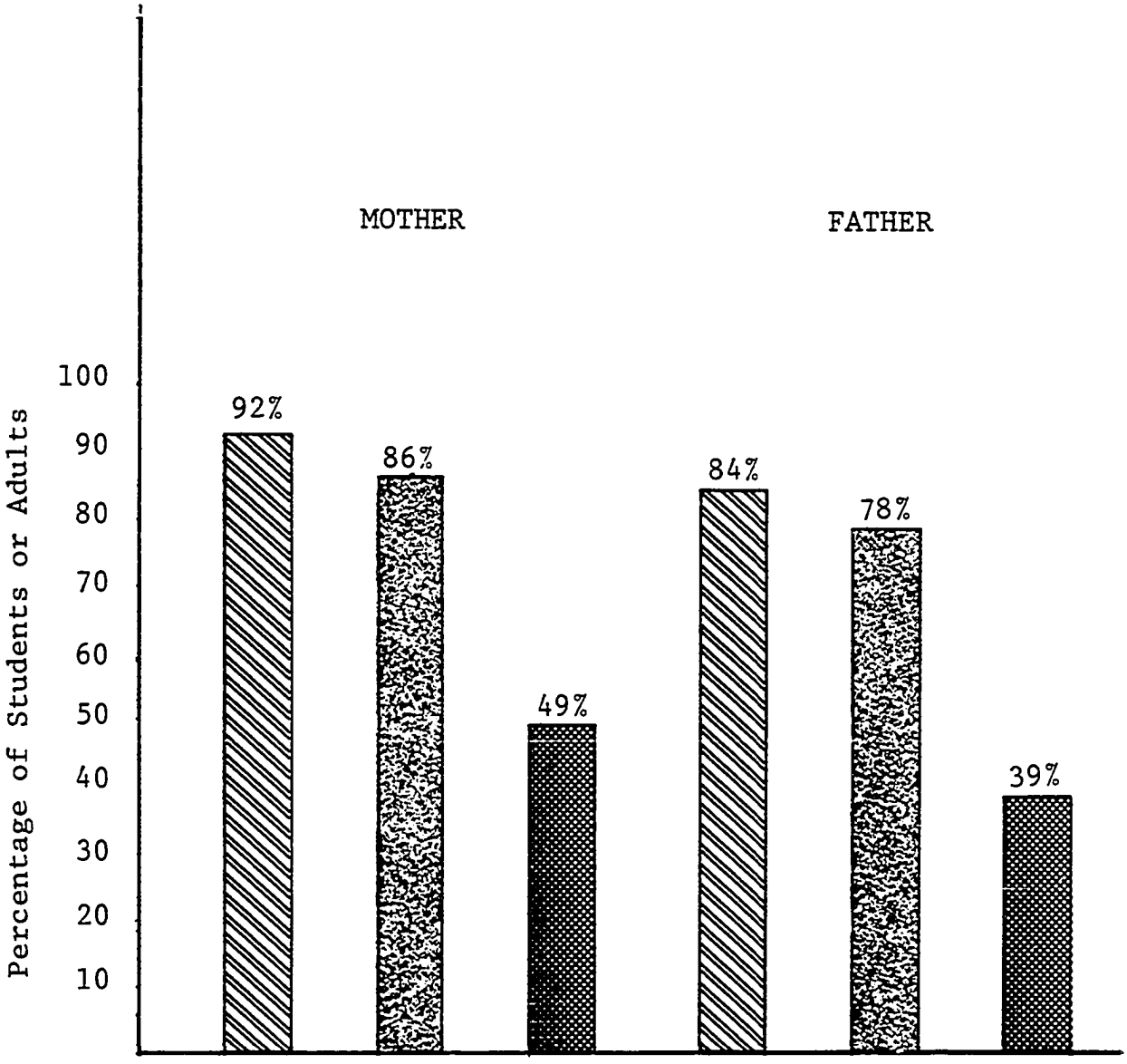

Figure 9

Differences in Triggers/Targets Between Younger and Older Samples

TABLE 27

Analysis of Variance of Duration and Coping with Feelings of Anger

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	10667.527	3	3555.842	3.038	0.03*
Ethnicity	1555.758	1	1555.758	1.329	0.25
Sex	36.016	1	36.016	0.031	0.86
Weight	9075.750	1	9075.750	7.755	0.006**
Main Effects:	1247.172	2	623.586	0.533	0.589
Age	812.770	1	812.770	0.694	0.407
Hypertension	434.402	1	434.402	0.371	0.544
2-Way Interaction:	201.258	1	201.258	0.172	0.679
Age Hypertension	201.258	1	201.258	0.172	0.679

* $p < .05$

** $p < .01$

Hypothesis IX was concerned with significantly higher numbers of negative life stressors in hypertensives. Negative life stressors cover three categories based on degree of severity. Mean ratings for each item on the PERI vary from 163--acquiring a pet--to 1036--death of a child (Dohrenwend et al., 1978). In this study, items were grouped according to three levels of severity, those under 300; those between 300 and 500; and those over 500, for negative and positive items. Results for negative stress at the least debilitating level, ratings < 300, are presented in Table 28. Covariates together almost achieve significance, and ethnicity in particular does, $F = 4.1$, $p = 0.05$. When main effects are examined, a similar pattern obtains, with age achieving significance, $F = 5.4$, $p = 0.02$. The latter may be interpreted to mean that young people report experiencing more negative stressors at the lowest level. When means comparing labiles with hypertensives on this variable are examined, significant differences obtain, $t = 2.14$, $p = 0.04$. With respect to the significance of ethnicity, it may be recalled that among the labiles, Caucasians outnumber the Blacks.

With respect to the negative stressors at the second level of severity, ratings between 300 and 500, Table 29 shows that there was no significant main effect. However, ethnicity as a covariate surfaced again, and weight achieved significance also, resulting in significant covariates. For ethnicity $F = 4.5$, $p = 0.04$ and for weight $F = 4.06$, $p = 0.05$. Not shown is a significant negative correlation between

TABLE 28

Analysis of Variance of Negative Life Stressors - Level I on the PERI

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	10.026	3	3.342	2.566	0.059
Ethnicity	5.352	1	5.352	4.108	0.045*
Sex	0.793	1	0.793	0.609	0.437
Weight	3.882	1	3.882	2.980	0.087
Main Effects:	7.135	2	3.568	2.739	0.69
Age	7.066	1	7.066	5.424	0.022*
Hypertension	0.069	1	0.069	0.053	0.818
2-Way Interaction:	0.445	1	0.445	0.342	0.560
Age Hypertension	0.445	1	0.445	0.342	0.560

* $p < .05$

TABLE 29

Analysis of Variance of Negative Life Stressors - Level II on the PERI

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	71.024	3	23.675	3.00	0.034*
Ethnicity	35.554	1	35.554	4.505	0.036*
Sex	3.385	1	3.385	0.429	0.514
Weight	32.085	1	32.085	4.065	0.046*
Main Effect:	21.915	2	10.958	1.388	0.254
Age	15.056	1	15.056	1.908	0.170
Hypertension	6.860	1	6.860	0.869	0.353
2-Way Interaction:	3.635	1	3.635	0.461	0.499
Age Hypertension	3.635	1	3.635	0.461	0.499

* $p < .05$

weight and negative stressors at level two, r = -.23, p = 0.02, indicating that persons with more of these stressors weigh less.

With respect to negative stressors at level three, ratings over 500, inspection of Table 30 shows that no significant levels obtain. However, with sex differences not surfacing throughout the entire analysis, it may be interesting to note that as a covariate here it approaches significance, F = 2.90, p = 0.09, along with hypertension F = 2.94, p = 0.09, suggesting that if you are female and hypertensive you are more likely to experience this level of stress. However results of the three tables combined do not lead to support of Hypothesis IX.

Finally Hypothesis X concerned itself with significantly lower numbers of positive life stressors in hypertensives. On inspection of Table 31 no significant main effects are observed, but the covariates achieve significance, ethnicity in particular. This indicates that Caucasians in general reported higher levels of positive stress at level one than Blacks; when means are examined they yield significant differences, t = 3.58, p = 0.001.

On positive stress, level two, no significant differences were achieved nor did anything approach significance. However, at level three, positive stress achieved significance as main effect, age, F = 4.9, p = 0.03. See Table 32.

When means are compared, there is a significant difference between young normotensives and older normotensives,

TABLE 30

Analysis of Variance of Negative Life Stressors - Level III on the PERI

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	5.360	3	1.787	1.079	0.362
Ethnicity	0.324	1	0.324	0.196	0.659
Sex	4.809	1	4.809	2.904	0.091
Weight	0.227	1	0.227	0.137	0.712
Main Effects:	6.289	2	3.144	1.899	0.155
Age	1.415	1	1.415	0.854	0.358
Hypertension	4.874	1	4.874	2.943	0.089
2-Way Interaction:	0.410	1	0.410	0.247	0.620
Age Hypertension	0.410	1	0.410	0.247	0.620

TABLE 31

Analysis of Variance of Positive Life Stressors - Level I on the PERI

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	82.426	3	27.475	3.797	0.013*
Ethnicity	61.095	1	61.095	8.443	0.005**
Sex	0.037	1	0.037	0.005	0.943
Weight	21.294	1	21.294	2.943	0.089
Main Effects:	12.420	2	6.210	0.858	0.427
Age	5.758	1	5.758	0.796	0.374
Hypertension	6.661	1	6.661	0.921	0.340
2-Way Interaction:	0.382	1	0.382	0.053	0.819
Age Hypertension	0.382	1	0.382	0.053	0.819

* $p < .05$

** $p < .01$

TABLE 32

Analysis of Variance of Positive Life Stressors - Level III on the PERI

N = 108 Ss

Source of Variation	Sum of Squares	df	Mean Square	F	Signif. of F
Covariates:	3.173	3	1.058	1.50	0.219
Ethnicity	0.046	1	0.046	0.065	0.800
Sex	0.959	1	0.959	1.360	0.246
Weight	2.169	1	2.169	3.077	0.082
Main Effects:	3.463	2	1.732	2.456	0.091
Age	3.457	1	3.457	4.904	0.029*
Hypertension	0.006	1	0.006	0.008	0.928
2-Way Interaction:	0.003	1	0.003	0.005	0.946
Age Hypertension	0.003	1	0.003	0.005	0.946

* $p < .05$

$t = 2.81$, $p = 0.007$, but differences between labiles and hypertensives do not even approach significance. Older normotensives then seem to be experiencing the most gratification from their lives. However, when data from the three levels of positive stress are combined, Hypothesis X did not obtain support.

With respect to variables monitored, no significant correlations obtained between smoking and hypertension, or parents with hypertension and hypertension. In the latter case, this is likely to be largely a function of poor self-report and can therefore be questioned.

While most hypertensive subjects were on a diuretic and a sympathetic blockade, generally a thiazide and propanolol, the sample was not large enough, nor were amounts of medication clearly enough indicated, to make this information the basis of classification.

Correlations of the SESI and the longer established inventory, the PERI, were computed. Although these inventories have different content domains, they should complement each other. Thus one would hypothesize that there should be significant positive correlations between most of the scales of the SESI and the PERI. In Table 33 correlations between the SESI and the three negative scales of the PERI are presented. There are no significant correlations between negative items on the PERI and the Anxiety scales on the SESI. However, there are significant correlations between levels one and three of negative stress on the PERI and frequency of

TABLE 33

Correlations of the SESI and Negative Stressors on the PERI

DEPRESSION	r	p
Frequency of feelings/NSTRSS I	.21	0.03
/NSTRSS II	.17	0.07 NS
/NSTRSS III	.20	0.04
Duration/NSTRSS I	.19	0.05
/NSTRSS II	.21	0.03
/NSTRSS III	.18	0.06 NS
ANGER		
Frequency of feelings/NSTRSS II	.20	0.04
/NSTRSS III	.35	0.000
Duration/NSTRSS III	.22	0.02
Coping		
Withholding/NSTRSS III	.29	0.002
Controlling/NSTRSS III	.20	0.03

feelings of depression ($p = 0.03$, $p = 0.04$, respectively). The first two levels of negative stress on the PERI are correlated with duration of feelings of depression ($p = 0.05$, $p = 0.03$, respectively). Level three approached significance, $p = 0.06$.

When correlations between the Anger scale on the SESI and levels of negative stress on the PERI are examined, significant correlations occur only with the two higher levels of the PERI. Frequency of feelings of anger correlate with level two of negative stress, $p = 0.04$, and correlate more highly with level of negative stress, $p = 0.000$. Duration of feelings of anger also correlate significantly with level three of negative stress, $p = 0.02$. This level of negative stress also correlates with withholding anger, $p = 0.002$ and with controlling anger, $p = 0.03$.

We next consider correlations between positive stressors on the PERI and subjective distress scales on the SESI. Perhaps the most striking observation is the validation of the social readjustment theory which holds that despite the positive designation, events involving favorable changes are nonetheless stressful. There are positive correlations between the three major scales of the SESI and positive stress at levels one and two of the PERI. See Table 34.

Depression correlates significantly with level two of positive stress, with frequency of depression, $p = 0.003$; with duration, $p = 0.01$ and with negative coping, $p = 0.02$.

TABLE 34

Correlations of the SESI and Positive Stressors on the PERI

DEPRESSION	r	p
Frequency of feelings/PSTRSS II	.28	0.003
Duration/PSTRSS II	.24	0.01
Negative Coping/PSTRSS II	.22	0.02
ANXIETY FEELINGS		
Expression/PSTRSS II	.25	0.009
Coping negatively/PSTRSS II	.27	0.004
Duration/PSTRSS II	.19	0.05
ANGER		
Duration/PSTRSS I	.30	0.001
/PSTRSS II	.38	0.000
Coping		
Withholding/PSTRSS I	.39	0.000
/PSTRSS II	.43	0.000
Controlling/PSTRSS I	.22	0.02
/PSTRSS II	.22	0.02

Anxiety did correlate significantly with positive stressors on the PERI. At level two it is associated with expression of anxiety, $p = 0.009$; with negative coping with anxiety, $p = 0.004$ and with duration of anxiety, $p = 0.05$.

Finally, neither levels one or two of positive stress correlate with either frequency of feelings of anger or frequency of expression of anger. They do correlate significantly with duration of feelings of anger, $p = 0.001$, $p = 0.000$ respectively; with withholding anger each correlates $p = 0.000$ for each level; and with control there is a smaller correlation, for both, $p = 0.02$.

In understanding the above correlations it may be helpful to recall two important characteristics of the sample studied: (i) It is predominantly female, and (ii) a significant proportion of the sample is young. In general one notes that a significant relationship between the SESI and the PERI obtained, one that has served to further clarify the nature of each instrument's content domain. However, in terms of future research, it would be worthwhile to observe if similar relationships occurred in a predominantly male sample.

CHAPTER VI: DISCUSSION

Discussion will focus on four aspects of this research and their implications for future work. They are: (i) the new inventory, the SESI, (ii) the characteristics of the samples studied, (iii) the patterns of the physiologic data, and (iv) implications of the role of subjective distress with respect to the problem of causality of essential hypertension.

At the most fundamental level it must be observed that the Subjective Emotional Stress Inventory proved to be an instrument adequate to the task of discriminating frequency, duration and manner of coping with subjective distress in two samples, but perhaps most importantly in finding differences between the hypertensive and normotensive samples. In succeeding where others have failed (Ostfeld, 1967), it may be a critical factor in a breakthrough in this area of research.

Indeed given the presence of the two stress pathways uncovered by Cannon and Selye and the ease and frequency with which they can be activated, and also as presented in the review of the literature, their relatively direct relationship to blood pressure and therefore hypertension, it is not surprising that some differences obtained, between hypertensives and normotensives. This points to the importance of specific and well-focused psychological scales. It is also

important to note that finding significant differences at the group level is a mere beginning. Neither group, hypertensive nor normotensive are likely to be homogeneous types. First, there is the traditional problem of overlap: this needs examination and explanation. Why are there some subjects in the hypertensive group who reported relatively low levels of subjective distress and some in the normotensive group who reported relatively high levels of subjective stress? At least three possibilities need to be explored here: (i) To what extent is this simply false or incorrect reporting in both groups? (ii) To what extent, in the case of normotensives, is this ineffective use of defense mechanisms but a particularly resistant circulatory system? (iii) To what extent in the case of hypertensives is this effective use of defense mechanisms but a particularly vulnerable circulatory system?

Second, among the hypertensives who report high levels of subjective distress, to what extent is this ineffective use of defense mechanisms contributing directly to higher blood pressure? To what extent can these defense mechanisms be identified and changed so that they indeed lower blood pressure?

Also worthy of note is the high levels of subjective distress and life stress identified in the lives of the younger people in the samples. One is reminded of the perils of youth, a matter that developmental psychologists, such as Erik Erikson (19), have talked about. Also one observes the relatively consistent relationship between life stress and subjective distress; a follow-up study might consider the

extent to which these high levels of subjective distress continue in adult life, first as a function of continued high levels of life stress but second, and perhaps more important, simply as a function of psychological habit, in the absence of high levels of life stress.

Closely related to the experience of subjective distress is its perceived source. As may be recalled, the literature reviewed suggested that hypertensives felt forced to live in a relatively uncomfortable interpersonal environment; often having hostile feelings toward persons on whom they are dependent, resulting in high levels of repressed anger. The SESI was capable of identifying this profile quite clearly in younger persons. In particular, they reported more depression than older persons and labiles reported having the greatest frequencies of prolonged depression. In addition, both groups also reported relatively high levels of frequency of feelings of anger. Their anger was less than of older hypertensives but more than that of older normotensives. Also, although this did not achieve significance, the younger groups reported experiencing longer periods of feeling anger and needing to withhold and control these feelings. In fact, it is worthwhile to note that in the research sample, frequency of feelings of depression and duration of anger were significantly correlated, $r = .45$, $p < .001$. Feelings of depression and withholding anger are even more highly correlated, $r = .52$, $p < .001$.

The profiles of the older hypertensives on the SESI are only slightly different from that of the labiles; they had the highest anger scores and lower depression scores without either achieving significance. Anxiety scores were very similar. Thus while older hypertensives may have a little more freedom to express themselves, the high levels of subjective distress that obtain for both groups, as a function of their interpersonal environments, are by far the more significant observation.

It would seem that the ability of the SESI to identify subjective distress as it is perceived to be related to one's interpersonal environment is a unique and important quality that warrants attention and expansion. In most studies on stress, the stressors examined are often crises such as illness requiring hospitalization of the individual or a close relative. Less attention is generally paid to the subtle but powerful role of an individual's continuing interpersonal environment. Nonetheless, this research suggests that it may be a critical source of stress. In addition it differs from other more episodic sources of stress. First, while continuing difficult interpersonal relationships may be acknowledged to be stressful by individuals, particularly where the individual is dependent or perceives himself to be dependent, the resulting subjective distress tends to be regarded as inevitable. Individuals may tend to adopt the "learned helplessness" attitude. Thus in contrast to acute stressors such as physical illness, individuals are less likely to seek

help in alleviating this form of chronic stress. Second, even for individuals who are willing to seek aid, often appropriate types of assistance are not available, and when available, direction is not always clear. Thus stress associated with interpersonal environments is far more subtle than that associated with illness or major loss; however, it would seem that it may still be a major source of subjective distress.

With respect to the SESI, despite its current ability to discriminate various levels of subjective distress, further work needs to be done on standardization of the inventory and the establishment of norms for various populations. Also, more work is needed on the level of intensity variable and on feelings versus expression: if these, when given a better chance to discriminate, do not (for example, when used as an interview schedule, as opposed to a self-administered inventory), it would indicate that they be deleted and the inventory take a shorter form.

Also, it is important to look at the physiologic aspects of this research and their implications. The most striking event in this research is the physiologic sensitivity of the labiles to questions about members of their interpersonal environment. It should be repeated that the experimental conditions were in no manner stressful, any stress was a function of past associations with persons referred to on the inventory. This points to Cannon's notion

of the role of classical conditioning as a mediator of prolonged subjective distress. Thus at the physiologic level, despite its conservative strategy, the research design turned out to be quite powerful.

Also noteworthy is the pattern of physiologic data. The significant differences between hypertensives and labiles and their controls have been noted. Also, the sometimes not significant differences in heart rate. However, although physiologic data have been presented on the hypertensives, the concern was more with verification of the physiologic status of the labiles. But since it is open to inspection, one should note the high readings despite the fact that most hypertensives were on medication. True some had "not taken their medication" on that day, but the data point to the great dependence on the medication and also its limited efficacy.

Heart rate was recorded because it was interesting to see whether heart rate and blood pressure would rise together or whether one would rise and not the other. When Tables 18 and 20 are compared, some interesting observations may be made: in general hypertensives and labiles have higher heart rates than their controls. Also, on the face of it, hypertensives and labiles have similar heart rates. But when standard deviations for the labiles are examined, one notes that they are large: (i) in comparison to their controls and (ii) in comparison to the hypertensive. From the review of

the literature, we know that there is generally some increase in cardiac activity to psychological stress, but since many of the hypertensives were on propanolol, a beta-adrenergic blockade that suppresses heart rate, labiles who were not on medication could be anticipated to have higher heart rates. Indeed, some labiles did have a spell of tachycardia on the first reading, some heart rates were well over 100 beats per minute.

Finally, it should be noted that although it was not possible to monitor or control for medication, this is a variable that should be of great concern to psychologists working in this area. Medication introduces its own source of variance which interacts with the dependent variable in varying ways. Among the most frequently used oral medications for hypertension by trade names are Aldomet, Apresoline and Dralzine, Inderal, Isemelin, Minipress and Catapress. Hypertensive medications, as pointed out earlier in this study, affect various parts of the nervous system and in this manner generate their various anti-hypertensive effects and side effects. Thus for example, with respect to moods, Reserpoid will aggravate depressions and Eutonyl will increase anxiety-tension states and/or insomnia. With respect to particular conditions, Isemelin can lead to postural dizziness or diarrhea and Apresoline tends to aggravate tension and/or migraine headaches and/or palpitations and heart consciousness. Thus, if possible, monitoring medications and their

side effects could lead to the identification of an important source of variance. Perhaps it should be pointed out that with respect to medication, there are two distinct factors: (i) direct variance coming from the side effects of the drugs and (ii) indirect variance caused by patients' feelings about taking or not taking their medication.

It is noteworthy, however, that in this study, young labiles, who were not on medication, had very similar profiles on all three measures of subjective distress, anxiety, depression and anger, as older diagnosed hypertensives, who were on medication. Additionally, frequency of feelings of anxiety were highest in the young normotensives. This could suggest that in this sample, the source of most of the variance was subjective distress associated with oneself or one's interpersonal environment, rather than subjective distress caused by medication or concern over condition.

Finally the implications of the role of subjective distress with respect to the problem of causality of essential hypertension needs to be addressed. This research has provided some correlational evidence supporting the possibility of a causal link between subjective emotional distress and essential hypertension. If continued support for this line of research is assumed, what could this mean? What are the implications for psychosomatic research? What are the implications for treatment? What are the implications for public health?

With respect to psychosomatic research, a question of clarification arises in reference to this study. Does the constellation of emotions, anxiety, depression and anger, when experienced too frequently, lead only to disturbances of the cardiovascular system? Although this study limited its focus to a single disturbance of the cardiovascular system, it does not imply that the cardiovascular system is the only system that is subject to the ravages of stress. On the contrary, there is an awareness that any of the bodily systems that are subject to frequent use--for example the digestive and respiratory systems--and/or suffer from any genetic weaknesses, are particularly vulnerable to the strain of subjective distress. Thus with respect to future research it would be worthwhile, once the SESI has been further refined, to test its capability on populations suffering from peptic ulcer disease and bronchial asthma.

Also in need of clarification are the implications of this study for treatment of essential hypertension. As before, if a causal link is assumed, does this mean that there should be differences in treatment plans, with more focus on controlling subjective distress? Actually, for the fixed hypertensive whose elevation of blood pressure is of several years duration, it is important to note that the current medical strategy of combining a diuretic with either a sympathetic blockade or a vasodilator, should continue. This is critical because treatment has to address itself not only to the cause of the problem, but also to the symptoms, especially if

they generate secondary problems, as well as the structural damage done by the disorder.

In the case of essential hypertension, success in controlling, though not curing, the disease comes from the ability of current medications to compensate for structural maladaptations of the cardiovascular system, such as narrowed arteries or increased cardiac activity. Without the aid of these medications, subjective emotional distress coupled with these maladaptations of the cardiovascular system, would cause blood pressure to rise to life threatening levels.

However, it should be made clear that treatment based on a causal link between subjective emotional distress and essential hypertension is already being used at one level. Sympathetic blockades and vasodilators, as described in the section on 'Therapeutic Drugs,' do block sympathetic nervous system activity at various sites.

What still remains to be addressed is the source of the sympathetic overactivity. Presumably medication blocks activity that has already been triggered. Thus it would seem that reducing the frequency of subjective emotional distress could be still more effective in controlling sympathetic overactivity. The literature on cognitive control, in particular the studies focused on relaxation (Benson, 1975) and the biofeedback studies directed towards lowering blood pressure have had limited success (Schwartz, Shapiro & Tursky, 1971; Patel, 1973, 1975; Goldman, Kleinman et al., 1975; Surwit, Shapiro & Good, 1978). At least two factors are of critical

relevance here: (i) the personal discipline, time and effort that those techniques require in order to have any observable effect, and (ii) their very limited ability, if any, to compensate for structural damage of the cardiovascular system. These two factors combine to limit the effectiveness of these techniques. Thus the recommendation would be that they be used in conjunction with prescribed medication to avoid the need for increases in medication over time and possibly to reduce dosage to some comfortable minimum within a given time. It should also be mentioned that relaxation exercises and various strategies of cognitive control are most effective as preventive measures or in mild cases of essential hypertension. It is recommended that these techniques be taught to younger persons and those at risk with respect to essential hypertension as one measure of reducing the incidence of this disorder.

Finally, the implications of the role of subjective distress as a causal factor in essential hypertension for public health will be addressed. Moriyama (1964), Chief of the Office of Health Statistics Analysis, pointed out that in the period 1939-1960 the crude death rate dropped from 17.2 to 9.5 per 1,000 population. However, the figures also indicate that during 1950-1960 the trend levelled off. Moriyama expressed concern about this stationary trend in 1950-1960:

> The failure to experience a decline in mortality during this period is unexpected in view of the intensified attack on medical problems in the post

> war years. . . If the levelling off of the death rate has resulted from failure to prevent deaths that are preventable, this is of public health significance.

It is also noteworthy that this quantitative pattern in mortality is paralleled by an important qualitative difference since 1900. In general, there has been a significant reduction in acute infectious diseases causing death, and a gradual rise in mortality due to chronic disorders (Stewart, 1963). Despite intensified research, heart disease, cancer and stroke continue overwhelmingly to be the leading causes of death in the United States. And as mentioned in the introductory chapter, hypertension is the leading complication in heart disease and strokes. Thus reduction in prevalence and severity of essential hypertension is important in reducing the leading causes of death.

A consistent finding that may be related to frequent experience of anxiety, depression and anger is the higher levels of essential hypertension in Black Americans, with higher rates of related morbidity and mortality. Here sociocultural factors play a critical role that help define the severity and number of life stressors. Thus Harburg et al. (1973) were able to show that the prevalence of high blood pressure is greater in poor Black Americans than in middle-class Black Americans. Poor Black Americans live in more crowded conditions, have higher rates of divorce, and move more frequently than middle class Blacks and experience deep resentment with respect to police brutality, but do not feel that they can express these resentments (Harburg, 1973).

As Weiner (1979) comments,

> The correlation between socioeconomic conditions and status and blood pressure levels in Blacks support the hypothesis that sociocultural factors play an etiological role in essential hypertension and hypertensive disease. The data partly elucidate how social and socioeconomic factors may elicit unexpressible or unexpressed anger and resentment in hypertensive patients. In this case the anger is a response to real danger, external prejudice and violence. (p. 24)

It would seem, then, that in terms of public health the prevalence of essential hypertension in almost epidemic proportions reflects at least in part the attempts of current populations to cope with overwhelming stressors that impact on their lives. Thus public health policy may have to include type and number of life stressors that may play a major role in the etiology of current chronic and often fatal disorders, and plan intervention based on these indicators.

CHAPTER VII: CONCLUSIONS

Hypotheses based on each of the subscales of the SESI were tested. Support was obtained for hypotheses based on at least one subscale from each of the three major scales, Depression, Anxiety and Anger. Of the eight null hypotheses associated with the SESI, only three were not rejected. This indicated that hypertensives in this sample tended to experience all three types of subjective distress more frequently than normotensives. At a more specific level, data analysis revealed that in the case of Depression, although there were no significant differences in how frequently hypertensives experienced feelings of depression, they reported that their experience of these feelings were of longer duration and caused them to engage more frequently in positive coping strategies, such as listening to particular kinds of music. With respect o Anxiety, hypertensives reported experiencing feelings of anxiety more frequently, as well as of longer duration. In addition, they indicated that they spent more time engaged in coping with these feelings in a positive manner, e.g. reaching out to a friend and talking about the tension until they felt better. They also indicated spending more time in negative coping, e.g. taking a drink or two when feeling tense or nervous. Finally, with respect to Anger, hypertensives reported having more frequent feelings of anger, as well as expressing anger

more frequently than normotensives. This anger was triggered and/or targeted within family and/or work environments.

With respect to life stressors, there were no significant differences between hypertensives and normotensives when compared on the PERI, on both positive and negative stressors at each of three levels of severity. What emerged, nonetheless, was first an age variable, young persons in this sample had higher levels of negative stress at level one and older normotensives had highest levels of positive stress at the third level. And, second, an ethnicity variable, Caucasians reported higher levels of positive stress than blacks. However, it may be important to note that despite the absence of significant differences, there are significant correlations between the PERI and the SESI.

At a more general level, it is felt that this investigation accomplished three of its main aims. First, there has been a comprehensive and integrative review of the medical literature related to the problem under study. This in turn provided a foundation for pursuing empirical work on the relationship between subjective distress and essential hypertension. Second there was construction of a new inventory for assessing important negative emotions complete with detailed justification and partial validation. And third, a research design was implemented that was sensitive enough to identify labiles and then discriminate differences between labiles and hypertensives and their controls in these important areas of emotion.

It is hoped that these findings add to our progress in understanding and assessing the psychodynamic and social aspects of hypertension--and eventually may find some useful practical application.

APPENDIX I

INSTRUCTIONS FOR TAKING THE SESI

MIND SET:

- Focus should be upon WHO and WHAT causes you most emotional stress, that is to feel angry, anxious or depressed.

- If these target persons or situations are not identified in the questionnaire, substitute the person and/or the situation by writing them in, in one of the four item blocks that are "Not Applicable" for you. For example, neighbors are not indentified on the questionnaire.

- Also please note that the examples given (e.g.) with each question are only examples. That is, they need only be similar to situations in your life, not identical.

- If there are any questions, please feel free to ask.

RESPONSE ALTERNATIVES:

Responses to questions are:

Several times a Day (std)

Every Day (evd)

Every other Day (eod)

Once a Week (onw)

Once a Month (onm)

Two or Three times a Year (tty)

Never (n)

Not Applicable (na)

If you have feelings or experiences which do not occur precisely at these frequencies, mark the frequency that is closest to it. For example, if something happens three times a week, mark "eod," since this would be the closest. Again questions are encouraged.

ANXIETY SCALE

HOW OFTEN DO YOU GET UPSET -

1. Over your health (e.g. worrying about getting sick?)
2. Over the thought of your death (e.g. worrying about what would happen to your children?)
3. About not having enough money to buy things (e.g. clothes, car, stereo, etc.?)
4. About not knowing where enough money will come from to pay, e.g. rent or buy food?

HOW OFTEN DO YOU GET UPSET ABOUT YOUR CHILDREN -

5. (e.g. worrying about them getting sick?)
6. (e.g. fearing that they may catch or have some awful disease?)
7. (e.g. worrying about the company they keep?)
8. (e.g. fearing that they may be taking drugs?)

HOW OFTEN DO YOU GET UPSET ABOUT -

9. Your spouse/lover (e.g. he/she cheating on you?)
10. Your spouse/lover (e.g. he/she left you or may leave you?)
11. Not having a job or a good job?
12. Losing your job?

HOW OFTEN DO YOU GET TENSE AND UPSET -

13. When relating to your supervisor?
14. About completing a task at work on time?
15. About how to do a task at work?
16. Because you just don't get much satisfaction from your work?

HOW OFTEN DO YOU GET-

17. Nervous & tense because you have to compete with your co-workers a great deal?

18. Upset at not having completed H.S. education?

19. Upset about not being able to get more education (e.g. a degree?)

20. Tense & nervous because you are not getting enough sex (e.g. partner often absent or tired?)

21. Tense & nervous because you are not putting enough into your sex life (e.g. not making time for it, not being a full participant?)

HOW OFTEN -

22. Are you bothered by constipation?

23. Do you have diarrhea?

24. Do you have frightening dreams?

25. Do you have a horrible feeling that something awful is going to happen to you?

26. Are you caught up in sudden attacks of fear or panic (e.g. your hands tremble & you feel weak?)

HOW OFTEN DO YOU -

27. Become so nervous you develop chest pains, or chest feels tight?

28. Become so nervous you get indigestion?

29. Break out in hives when something important is about to happen (e.g. exam, ask for a raise?)

30. Get headaches?

31. Suffer from insomnia (e.g. not being able to sleep two nights in a row?)

32. Fear being mugged or robbed?

ANXIETY SCALE (page 3)

HOW OFTEN DO YOU -

33. Get tense & upset because you feel you just can't cope with your problems at home?

34. Become so tense & nervous, you feel faint and dizzy?

35. Become so tense you bite your nails or pull out your hair?

36. Take a drink or two when you feel tense or nervous?

HOW OFTEN DO YOU -

37. Do some exercise when you feel tense or nervous (e.g deep-breathing, running in place, etc.?)

38. Drink in order to forget a particular problem?

39. Find that listening to certain kinds of music calms you when you feel tense or upset?

40. Find that you take Valium, Lithium or similar pills in order to calm down?

HOW OFTEN DO YOU FIND THAT YOU -

41. Can reach out to a friend, talk about your tension, and calm down?

42. Take Excedrin, Sominex or similar non-prescription pills to fall asleep?

43. Can take hold of yourself when nervous by repeating a sentence over & over to yourself or imagining a pleasant scene?

HOW OFTEN DO YOU FEEL NERVOUS & TENSE -

44. For about an hour or less?

45. All morning or all afternoon?

46. For about a day or two?

47. For three days or more?

ANXIETY SCALE

ANSWER SHEET

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
1.	stD	evD	eoD	onW	onM	ttY	N
2.	stD	evD	eoD	onW	onM	ttY	N
3.	stD	evD	eoD	onW	onM	ttY	N
4.	stD	evD	eoD	onW	onM	ttY	N
5.	stD	evD	eoD	onW	onM	ttY	N
6.	stD	evD	eoD	onW	onM	ttY	N
7.	stD	evD	eoD	onW	onM	ttY	N
8.	stD	evD	eoD	onW	onM	ttY	N
9.	stD	evD	eoD	onW	onM	ttY	N
10.	stD	evD	eoD	onW	onM	ttY	N
11.	stD	evD	eoD	onW	onM	ttY	N
12.	stD	evD	eoD	onW	onM	ttY	N
13.	stD	evD	eoD	onW	onM	ttY	N
14.	stD	evD	eoD	onW	onM	ttY	N
15.	stD	evD	eoD	onW	onM	ttY	N
16.	stD	evD	eoD	onW	onM	ttY	N
17.	stD	evD	eoD	onW	onM	ttY	N
18.	stD	evD	eoD	onW	onM	ttY	N
19.	stD	evD	eoD	onW	onM	ttY	N
20.	stD	evD	eoD	onW	onM	ttY	N

ANXIETY SCALE ANSWER SHEET (page 2)

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
21.	stD	evD	eoD	onW	onM	ttY	N
22.	stD	evD	eoD	onW	onM	ttY	N
23.	stD	evD	eoD	onW	onM	ttY	N
24.	stD	evD	eoD	onW	onM	ttY	N
25.	stD	evD	eoD	onW	onM	ttY	N
26.	stD	evD	eoD	onW	onM	ttY	N
27.	stD	evD	eoD	onW	onM	ttY	N
28.	stD	evD	eoD	onW	onM	ttY	N
29.	stD	evD	eoD	onW	onM	ttY	N
30.	stD	evD	eoD	onW	onM	ttY	N
31.	stD	evD	eoD	onW	onM	ttY	N
32.	stD	evD	eoD	onW	onM	ttY	N
33.	stD	evD	eoD	onW	onM	ttY	N
34.	stD	evD	eoD	onW	onM	ttY	N
35.	stD	evD	eoD	onW	onM	ttY	N
36.	stD	evD	eoD	onW	onM	ttY	N
37.	stD	evD	eoD	onW	onM	ttY	N
38.	stD	evD	eoD	onW	onM	ttY	N
39.	stD	evD	eoD	onW	onM	ttY	N
40.	stD	evD	eoD	onW	onM	ttY	N
41.	stD	evD	eoD	onW	onM	ttY	N
42.	stD	evD	eoD	onW	onM	ttY	N

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
43.	stD	evD	eoD	onW	onM	ttY	N
44.	stD	evD	eoD	onW	onM	ttY	N
45.	stD	evD	eoD	onW	onM	ttY	N
46.	stD	evD	eoD	onW	onM	ttY	N
47.	stD	evD	eoD	onW	onM	ttY	N

DEPRESSION SCALE

HOW OFTEN DO YOU FIND YOU FEEL SO UNHAPPY, YOU -

1. Are over-sleeping (e.g. getting up too late for work or appointments?
2. Are skipping a day at work or school?
3. Skip meals?
4. Fail to eat and lose weight (e.g. 5 lbs. or more?)

HOW OFTEN DO YOU FIND YOU FEEL SO UNHAPPY, YOU -

5. Overeat (e.g. eating until you feel uncomfortable?)
6. Overeat and gain weight (e.g. 5 lbs. or more?)
7. Do not care how you look?
8. Feel that you look ugly or repulsive?

HOW OFTEN DO YOU FIND YOU FEEL SO UNHAPPY, YOU ARE -

9. Unable to do little things at work (e.g. keep track of where you left things?)
10. Not working as well as you used to (e.g. you do less work?)
11. Not setting social goals for yourself (e.g. attending new social functions?)
12. Not socializing as you used to (e.g. you go less to discos, concerts, church, etc.?)

HOW OFTEN DO YOU FIND YOU ARE SO UNHAPPY, YOU -

13. Are not setting financial goals (e.g. budgeting for things you want or need?)
14. Are spending more money than you can afford on entertainment (e.g. drinks, eating out, theatre, etc.?)
15. Cry easily?
16. Are just unable to control tears running down your cheeks?

DEPRESSION SCALE (page 2)

HOW OFTEN DO YOU FIND -

17. You are so unhappy that you lose interest in sex (e.g. your partner has to coax you?)

18. You are so unhappy you don't engage in sex for several weeks?

19. Yourself feeling so unhappy that you blame yourself for everything that goes wrong?

20. Yourself feeling very unhappy and noticing all the ways in which you could be harmed?

HOW OFTEN DO YOU FIND YOURSELF FEELING QUITE UNHAPPY -

21. And feeling that you are being punished?

22. And you feel you deserve to be punished?

23. And hating yourself?

24. And thinking of ways you could harm yourself?

HOW OFTEN DO YOU FEEL -

25. So unhappy it seems as if no one cares about you?

26. So unhappy you think people would be better off without you?

27. Very unhappy but you are afraid to discuss your feelings with anyone?

28. Quite unhappy but you can't find anyone to talk over your feelings with?

HOW OFTEN DO YOU FEEL SO UNHAPPY IT SEEMS LIKE -

29. There is nothing to look forward to?

30. Life is hopeless and things will just get worse?

31. You have accomplished very little?

32. You are just one big failure?

DEPRESSION SCALE (page 3)

HOW OFTEN DO -

33. You feel so unhappy you can't sleep through the night?

34. You feel so unhappy, that you get up tired after not sleeping well for several nights in a row?

HOW OFTEN DO YOUR FEELINGS OF UNHAPPINESS LAST -

35. An hour or less?

36. All morning or all afternoon?

37. A day or two?

38. Three days or more?

HOW OFTEN DO YOU FEEL -

39. Renewed or strengthened after a period of unhappiness?

HOW OFTEN DO YOU FIND, AFTER A PERIOD OF UNHAPPINESS -

40. You feel exhausted?

41. You emerge with clearer ideas?

42. You feel ashamed or guilty?

43. You feel as if a burden has been lifted from your shoulders?

HOW OFTEN DO YOU TAKE -

44. Elavil or Tofranil when you feel very unhappy?

45. An upper when you feel very unhappy?

DEPRESSION SCALE (page 4)

HOW OFTEN DO YOU FIND THAT AFTER A PERIOD OF UNHAPPINESS -

46. You feel more dependent on a particular person (e.g. spouse/lover/friend?)

47. You seem able to understand others more easily?

HOW OFTEN DO YOU FIND THAT DURING OR AFTER A PERIOD OF UNHAPPINESS -

48. You feel anxious or afraid?

49. You feel angry or disgusted with yourself?

HOW OFTEN DO YOU FIND THAT -

50. Certain music helps you feel better when you are unhappy?

51. After a period of unhappiness you feel that something good is going to happen to you?

DEPRESSION SCALE

ANSWER SHEET

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
1.	stD	evD	eoD	onW	onM	ttY	N
2.	stD	evD	eoD	onW	onM	ttY	N
3.	stD	evD	eoD	onW	onM	ttY	N
4.	stD	evD	eoD	onW	onM	ttY	N
5.	stD	evD	eoD	onW	onM	ttY	N
6.	stD	evD	eoD	onW	onM	ttY	N
7.	stD	evD	eoD	onW	onM	ttY	N
8.	stD	evD	eoD	onW	onM	ttY	N
9.	stD	evD	eoD	onW	onM	ttY	N
10.	stD	evD	eoD	onW	onM	ttY	N
11.	stD	evD	eoD	onW	onM	ttY	N
12.	stD	evD	eoD	onW	onM	ttY	N
13.	stD	evD	eoD	onW	onM	ttY	N
14.	stD	evD	eoD	onW	onM	ttY	N
15.	stD	evD	eoD	onW	onM	ttY	N
16.	stD	evD	eoD	onW	onM	ttY	N
17.	stD	evD	eoD	onW	onM	ttY	N
18.	stD	evD	eoD	onW	onM	ttY	N
19.	stD	evD	eoD	onW	onM	ttY	N
20.	stD	evD	eoD	onW	onM	ttY	N

DEPRESSION SCALE ANSWER SHEET (page 2)

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
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25.	stD	evD	eoD	onW	onM	ttY	N
26.	stD	evD	eoD	onW	onM	ttY	N
27.	stD	evD	eoD	onW	onM	ttY	N
28.	stD	evD	eoD	onW	onM	ttY	N
29.	stD	evD	eoD	onW	onM	ttY	N
30.	stD	evD	eoD	onW	onM	ttY	N
31.	stD	evD	eoD	onW	onM	ttY	N
32.	stD	evD	eoD	onW	onM	ttY	N
33.	stD	evD	eoD	onW	onM	ttY	N
34.	stD	evD	eoD	onW	onM	ttY	N
35.	stD	evD	eoD	onW	onM	ttY	N
36.	stD	evD	eoD	onW	onM	ttY	N
37.	stD	evD	eoD	onW	onM	ttY	N
38.	stD	evD	eoD	onW	onM	ttY	N
39.	stD	evD	eoD	onW	onM	ttY	N
40.	stD	evD	eoD	onW	onM	ttY	N
41.	stD	evD	eoD	onW	onM	ttY	N
42.	stD	evD	eoD	onW	onM	ttY	N

DEPRESSION SCALE ANSWER SHEET (page 3)

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
43.	stD	evD	eoD	onW	onM	ttY	N
44.	stD	evD	eoD	onW	onM	ttY	N
45.	stD	evD	eoD	onW	onM	ttY	N
46.	stD	evD	eoD	onW	onM	ttY	N
47.	stD	evD	eoD	onW	onM	ttY	N
48.	stD	evD	eoD	onW	onM	ttY	N
49.	stD	evD	eoD	onW	onM	ttY	N
50.	stD	evD	eoD	onW	onM	ttY	N
51.	stD	evD	eoD	onW	onM	ttY	N

ANGER SCALE

HOW OFTEN -

1. Does your father make you feel angry - (e.g. by not giving you something you think you should have?)

2. Do you express anger to your father (e.g. having an argument over something about which you disagree?)

3. Does your father make you feel very angry (e.g. so angry you feel as if you are going to explode inside?)

4. Do you get very angry with your father (e.g. have a quarrel, yelling or shouting & leave hating each other?)

HOW OFTEN -

5. Does your mother make you feel angry (e.g irritating you by not helping you to do something?)

6. Do you express anger to your mother (e.g. have an argument over something you disagree about?)

7. Does your mother make you very angry (e.g. so angry you feel as if you are about to explode inside?)

8. Do you get real angry with your mother (e.g. quarrel with her, shouting and leave hating each other?)

HOW OFTEN -

9. Does your spouse/lover make you feel angry (e.g. irritating you by not living up to your expections?)

10. Do you express anger to your spouse/lover (e.g. have an argument over something he/she may have done?)

11. Do you feel very angry with your spouse/lover (e.g. so angry you feel as if you could hurt him or her?)

12. Do you get very angry with your spouse/lover (e.g. so angry that you deprive them or hit them?)

HOW OFTEN -

13. Does your sister(s) make you feel angry (e.g. irritating you by having things or talents you don't have?)

14. Do you express anger to your sister(s) (e.g. having heated arguments over matters upon which you disagree?)

15. Does your sister(s) make you feel very angry (e.g. they become so jealous and mean that you wish they were dead?)

16. Do you get very angry with your sister(s) (e.g. quarreling and shouting and end hating each other?)

HOW OFTEN -

17. Does your brother(s) make you feel a bit angry (e.g. irritating you by displaying things or talents you don't have?)

18. Do you express anger to your brother(s) (e.g. argue over something you disagree about?)

19. Does your brother(s) make you feel very angry (e.g. becoming so jealous that you wish they were dead?)

20. Do you get very angry with your brother(s) (e.g. quarreling & shouting and leave hating each other?)

HOW OFTEN -

21. Does your girl(s) make you feel a bit angry (e.g. doing things they should not do?)

22. Do you express your anger to your girl(s) (e.g. shouting at her not to do something?)

23. Does your girl(s) make you feel very angry (e.g. competing with you for your husband's attention?)

24. Do you get very angry with your girl(s) (e.g. so angry you find yourself wishing you never had them?)

HOW OFTEN -

25. Does your boy(s) make you feel a bit angry (e.g. doing something he should not do?)

26. Do you express your anger to your boy(s) (e.g. shouting at him not to do something?)

27. Does your boy(s) make you feel very angry (e.g. forcing you to go without things in order to provide for him?)

28. Do you get very angry with your boy(s) (e.g. so angry you wish you had never given birth to them?)

HOW OFTEN -

29. Do you feel a bit angry with your supervisor (e.g. annoyed that you have to do something over again?)

30. Do you express anger to your supervisor (e.g. argue about how something ought to be done?)

31. Do you feel very angry with your supervisor (e.g. very frustrated that he favors someone over you?)

32. Do you get very angry with your supervisor (e.g. argue heatedly & end up hating each other?)

HOW OFTEN -

33. Do your male co-workers make you feel a bit angry (e.g. by not completing a particular task?)

34. Do you express your anger to them (e.g. by pointing out to them that you are doing their job?)

35. Do your male co-workers make you feel very angry (e.g. by being mean or jealous?)

36. Do you get very angry with your male co-workers (e.g. argue heatedly & end up disliking each other?)

ANGER SCALE (page 4)

HOW OFTEN -

37. Do your female co-workers make you feel a bit angry (e.g. by not completing a particular task?)

38. Do you express your anger to them (e.g. pointing out to them that you are doing their job?)

39. Do your female co-workers make you feel very angry (e.g. by being mean or jealous?)

40. Do you get very angry with your female co-workers (e.g. argue heatedly and end up disliking each other?)

HOW OFTEN -

41. Do you feel a bit angry with yourself (e.g. annoyed that you did something badly again? (goofed again?))

42. Do you express anger at yourself (e.g. muttering or swearing under your breath?)

43. Do you feel very angry with yourself (e.g. hating yourself for being so stupid?)

44. Do you get very angry with yourself (e.g. banging on the table, shouting and slamming doors?)

HOW OFTEN -

45. Do you find yourself angry without knowing why (e.g. feeling hostile towards people around you?)

46. Do you express anger without knowing why (e.g. flaring up at someone who didn't really bother you?)

47. Do you feel very angry without knowing why (e.g. as if you are going to explode inside?)

48. Do you get very angry without knowing why (e.g. shouting at kids for no reason at all?)

ANGER SCALE (page 5)

HOW OFTEN -

49. Does your anger last for about 40 minutes or less?

50. Does your anger last about an hour or two?

51. Does your anger last three to four hours/ all morning or afternoon?

52. Does your anger last all day?

53. Does your anger last a day or two?

HOW OFTEN -

54. Do you hold back your anger because you are afraid of what someone could do to you?

55. Do you hold back your anger because you think people will have a poor impression of you?

56. Do you hold back your anger because you think it is wrong to express it?

57. Do you become angry but find it difficult to express your anger?

58. Do you feel that if you express your anger you will lose the affection of someone you love?

HOW OFTEN DO YOU -

59. Get a headache when you get angry?

60. Get a backache when you get angry?

61. Develop chest pains when you are angry?

62. Feel very tired or drained when you are angry?

ANGER SCALE (page 6)

HOW OFTEN DO -

63. You feel helplessly caught up in a fit of anger?

64. Your hands tremble with rage when you are angry?

65. You break things when you are angry?

66. You hit someone when you are angry?

HOW OFTEN DO YOU -

67. Discuss your anger at someone with a third person?

68. Express your anger by writing?

69. Feel that expressing your anger increases others' appreciation of you?

70. Feel refreshed after expressing your anger?

HOW OFTEN DO YOU -

71. Find people changing in the manner you suggested when you were angry?

72. Find yourself changing in the manner you realized you should when you were angry?

73. Find that you feel quite friendly towards the person who made you angry, once you've expressed yourself?

74. Jog or exercise in order to rid yourself of anger?

ANGER SCALE (page 7)

HOW OFTEN DO YOU -

75. Do discotheque dancing in order to rid yourself of anger?

76. Clean up house/office in order to rid yourself of anger?

77. Jog or take similar exercise?

78. Go discotheque dancing?

HOW OFTEN DO YOU -

79. Punch pillows or a bag when you are angry?

80. Eat something in order to calm yourself when you are angry?

81. Have a drink in order to calm yourself when you are angry?

82. Take Excedrin or aspirin in order to calm yourself when you are angry?

83. Take Valium or a prescribed tranquilizer to calm yourself when angry?

ANGER SCALE

ANSWER SHEET

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
1.	stD	evD	eoD	onW	onM	ttY	N
2.	stD	evD	eoD	onW	onM	ttY	N
3.	stD	evD	eoD	onW	onM	ttY	N
4.	stD	evD	eoD	onW	onM	ttY	N
5.	stD	evD	eoD	onW	onM	ttY	N
6.	stD	evD	eoD	onW	onM	ttY	N
7.	stD	evD	eoD	onW	onM	ttY	N
8.	stD	evD	eoD	onW	onM	ttY	N
9.	stD	evD	eoD	onW	onM	ttY	N
10.	stD	evD	eoD	onW	onM	ttY	N
11.	stD	evD	eoD	onW	onM	ttY	N
12.	stD	evD	eoD	onW	onM	ttY	N
13.	stD	evD	eoD	onW	onM	ttY	N
14.	stD	evD	eoD	onW	onM	ttY	N
15.	stD	evD	eoD	onW	onM	ttY	N
16.	stD	evD	eoD	onW	onM	ttY	N
17.	stD	evD	eoD	onW	onM	ttY	N
18.	stD	evD	eoD	onW	onM	ttY	N
19.	stD	evD	eoD	onW	onM	ttY	N
20.	stD	evD	eoD	onW	onM	ttY	N

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
21.	stD	evD	eoD	onW	onM	ttY	N
22.	stD	evD	eoD	onW	onM	ttY	N
23.	stD	evD	eoD	onW	onM	ttY	N
24.	stD	evD	eoD	onW	onM	ttY	N
25.	stD	evD	eoD	onW	onM	ttY	N
26.	stD	evD	eoD	onW	onM	ttY	N
27.	stD	evD	eoD	onW	onM	ttY	N
28.	stD	evD	eoD	onW	onM	ttY	N
29.	stD	evD	eoD	onW	onM	ttY	N
30.	stD	evD	eoD	onW	onM	ttY	N
31.	stD	evD	eoD	onW	onM	ttY	N
32.	stD	evD	eoD	onW	onM	ttY	N
33.	stD	evD	eoD	onW	onM	ttY	N
34.	stD	evD	eoD	onW	onM	ttY	N
35.	stD	evD	eoD	onW	onM	ttY	N
36.	stD	evD	eoD	onW	onM	ttY	N
37.	stD	evD	eoD	onW	onM	ttY	N
38.	stD	evD	eoD	onW	onM	ttY	N
39.	stD	evD	eoD	onW	onM	ttY	N
40.	stD	evD	eoD	onW	onM	ttY	N
41.	stD	evD	eoD	onW	onM	ttY	N
42.	stD	evD	eoD	onW	onM	ttY	N

ANGER SCALE ANSWER SHEET (page 3)

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
43.	stD	evD	eoD	onW	onM	ttY	N
44.	stD	evD	eoD	onW	onM	ttY	N
45.	stD	evD	eoD	onW	onM	ttY	N
46.	stD	evD	eoD	onW	onM	ttY	N
47.	stD	evD	eoD	onW	onM	ttY	N
48.	stD	evD	eoD	onW	onM	ttY	N
49.	stD	evD	eoD	onW	onM	ttY	N
50.	stD	evD	eoD	onW	onM	ttY	N
51.	stD	evD	eoD	onW	onM	ttY	N
52.	stD	evD	eoD	onW	onM	ttY	N
53.	stD	evD	eoD	onW	onM	ttY	N
54.	stD	evD	eoD	onW	onM	ttY	N
55.	stD	evD	eoD	onW	onM	ttY	N
56.	stD	evD	eoD	onW	onM	ttY	N
57.	stD	evD	eoD	onW	onM	ttY	N
58.	stD	evD	eoD	onW	onM	ttY	N
59.	stD	evD	eoD	onW	onM	ttY	N
60.	stD	evD	eoD	onW	onM	ttY	N
61.	stD	evD	eoD	onW	onM	ttY	N
62.	stD	evD	eoD	onW	onM	ttY	N
63.	stD	evD	eoD	onW	onM	ttY	N
64.	stD	evD	eoD	onW	onM	ttY	N

ANGER SCALE ANSWER SHEET (page 4)

	several times a day	every day	every other day	once a week	once a month	two or three times a year	never or not applicable
65.	stD	evD	eoD	onW	onM	ttY	N
66.	stD	evD	eoD	onW	onM	ttY	N
67.	stD	evD	eoD	onW	onM	ttY	N
68.	stD	evD	eoD	onW	onM	ttY	N
69.	stD	evD	eoD	onW	onM	ttY	N
70.	stD	evD	eoD	onW	onM	ttY	N
71.	stD	evD	eoD	onW	onM	ttY	N
72.	stD	evD	eoD	onW	onM	ttY	N
73.	stD	evD	eoD	onW	onM	ttY	N
74.	stD	evD	eoD	onW	onM	ttY	N
75.	stD	evD	eoD	onW	onM	ttY	N
76.	stD	evD	eoD	onW	onM	ttY	N
77.	stD	evD	eoD	onW	onM	ttY	N
78.	stD	evD	eoD	onW	onM	ttY	N
79.	stD	evD	eoD	onW	onM	ttY	N
80.	stD	evD	eoD	onW	onM	ttY	N
81.	stD	evD	eoD	onW	onM	ttY	N
82.	stD	evD	eoD	onW	onM	ttY	N
83.	stD	evD	eoD	onW	onM	ttY	N

Instructions for Scoring

Numerical Value of Response Alternatives:	stD	evD	eoD	onW	onM	ttY	N/NA
	6	5	4	3	2	1	0

Scale Scores: Anxiety Scale

Feelings 1: Add #s 1, 3, 5, 7, 9, 12, 14, 16, 21, 21, 32.

Feelings 2: Add #s 2, 4, 6, 8, 10, 11, 13, 15, 17, 20, 33.

Expression: Add #s 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 34, 35.

Coping - : Add #s 36, 38, 40, 42.

Coping + : Add #s 37, 39, 41, 43.

Duration: Add #s 44, 45, 46, 47.

Scale Scores: Depression Scale

Feelings 1: Add #s 1, 3, 5, 7, 9, 11, 13, 15, 17, 19, 21, 23, 25, 28, 29, 31, 33.

Feelings 2: Add #s 2, 4, 6, 8, 10, 12, 14, 16, 18, 20, 22, 24, 26, 27, 30, 32, 34.

Duration: Add #s 35, 36, 37, 38.

Coping - : Add #s 40, 42, 44, 45, 48, 49.

Coping + : Add #s 39, 41, 42, 46, 47, 50, 51.

Instructions for Scoring

Numerical Value of Response Alternatives:	stD	evD	eoD	onW	onM	ttY	N/NA
	6	5	4	3	2	1	0

Scale Scores: **Anger Scale**

Feelings 1: Add #s 1, 5, 9, 13, 17, 21, 25, 29, 33, 37, 41, 45.

Expression 1: Add #s 2, 6, 10, 14, 18, 22, 26, 30, 34, 38, 42, 46.

Feelings 2: Add #s 3, 7, 11, 15, 19, 23, 27, 31, 35, 39, 43, 47.

Expression 2: Add #s 4, 8, 12, 16, 20, 24, 28, 32, 36, 40, 44, 48.

Duration: Add #s 49, 50, 51, 52, 53.

Withholding: Add #s 54, 55, 56, 57, 58.

Suffering: Add #s 59, 60, 61, 62.

Physical: Add #s 63, 64, 65, 66.

Controlling: Add #s 67, 68, 69, 70, 71, 72, 73.

Doing: Add #s 74, 75, 76, 77, 78, 79.

Unresolved: Add #s 80, 81, 82, 83.

APPENDIX I

The Subjective Emotional Stress Inventory (SESI) as presented here represents current efforts to construct an inventory that measures frequency and levels of anxiety, depression and anger in normal persons. With the steady rise in stress-related disorders, there is a need for an inventory that focuses on these emotions as they find expression in the lives of millions of people who pursue an active lifestyle.

The SESI is currently available in a single format with limited normative data for college students and normotensives and hypertensives. While the format incorporates several new features described in the text, more work needs to be done in several areas: For example, although satisfactory reliability coefficients and validity scores obtained, item analysis is needed to identify more precisely which items are making major contributions to each scale as well as those that are not. Decisions to shorten or lengthen scales will be made on this basis.

In addition, normative data on larger numbers of college students as well as normotensives and hypertensives need to be established. Also normative data on other populations suffering from psychosomatic diseases, such as ulcers and migraine headaches, are important.

While the item format will remain the same, future revisions will include construction of new items that are peculiar to various populations and age groups, and more

careful attention will be paid to how well items discriminate the intensity levels. Additionally, once satisfactory discriminations have been made, scoring procedures may be revised to reject the presence of the intensity variable.

Thus while the SESI has proved to be useful in its present form, further work refining the inventory for future use is planned.

APPENDIX II

INTERCORRELATION MATRIX: SESI

BASED ON COLLEGE STUDENTS

N = 101

	DEFEELO	DEFEELT	DURA	COPNP	COPNN	AXFEELO	AXFEELT	AXEXPR	COPP	COPN	DUR	AGFEELO	AGFEELT	AGEXPRO	AGEXPRT	DORA	WITHOLD	CONTROL
DEFEELO	=																	
DEFEELT	95✓	=																
DURA	71✓	69✓	=															
COPNP	42	43	41	=														
COPNN	70✓	69✓	56✓	40	=													
AXFEELO	21	24	08	06	07	=												
AXFEELT	19	19	08	00	04	91✓	=											
AXEXPR	63✓	62✓	53✓	20	44	18	16	=										
COPP	16	18	20	44	06	03	-00	15	=									
COPN	47	46	41	20	47	-07	-11	50✓	10	=								
DUR	50✓	52✓	60✓	28	38	12	09	58✓	27	30	=							
AGFEELO	12	11	09	00	-02	52✓	61✓	08	-05	-04	06	=						
AGFEELT	04	04	01	-06	-05	50✓	62✓	00	-08	-12	-00	96✓	=					
AGEXPRO	02	01	02	-04	-08	47	58✓	-01	-08	-11	-02	97✓	96✓	=				
AGEXPRT	00	01	-01	-08	-06	50✓	61✓	01	-11	-10	-03	94✓	98✓	95✓	=			
DORA	43	41	47	21	37	10	06	48	16	36	40	16	06	07	03	=		
WITHOLD	48	44	36	11	42	10	09	55✓	07	37	40	14	09	05	04	52✓	=	
CONTRL	25	22	22	33	22	06	-05	16	27	22	06	05	-01	-02	-06	49	27	=

N.B. Decimals have been omitted
✓Correlations ≥ 50

APPENDIX III

INTERCORRELATION MATRIX: SESI

BASED ON HYPERTENSIVES AND NORMOTENSIVES

N = 108

	DEFEELO	DEFEELT	DURA	COPNP	COPNN	AXFEELO	AXFEELT	AXEXPR	COPP	COPN	DUR	AGFEELO	AGFEELT	AGEXPRO	AGEXPRT	DORA	WITHOLD	CNTRL
DEFEELO	=																	
DEFEELT	94✓	=																
DURA	38	37	=															
COPNP	24	19	63✓	=														
COPNN	58✓	59✓	65✓	58✓	=													
AXFEELO	27	32	10	-02	13	=												
AXFEELT	17	22	05	-05	05	91✓	=											
AXEXPR	52✓	61✓	19	01	37	50✓	42×	=										
COPP	36	35	28	27	24	28	16	26	=									
COPN	59✓	67✓	32	17	43×	43×	32	71✓	43×	=								
DUR	41×	41×	73✓	53✓	67✓	07	03	28	22	30	=							
AGFEELO	29	31	13	-08	11	52✓	58✓	30	05	24	10	=						
AGFEELT	35	39	18	-07	19	52✓	57✓	36	05	26	15	95✓	=					
AGEXPRO	29	32	16	-06	14	47×	53✓	28	03	23	09	97✓	94✓	=				
AGEXPRT	36	42×	20	-06	23	50✓	55✓	39	06	33	16	92✓	98✓	93✓	=			
DORA	43×	45×	24	15	28	25	22	37	01	33	34	36	42×	37	42×	=		
WITHOLD	48×	52✓	23	07	24	32	27	44×	13	41×	19	39	42×	41×	42×	71✓	=	
CNTRL	24	27	11	21	09	20	15	23	16	25	13	24	23	28	26	63✓	74✓	=

N.B. Decimals have been omitted
✓ Correlations ≥ 50
× Correlations 40-49

APPENDIX IV

THE PSYCHIATRIC EPIDEMIOLOGY RESEARCH INTERVIEW (PERI) LIFE EVENTS SCALE

SCH: 1. Started school or a training program after not going to school for a long time
2. Changed school or training programs ____
3. Graduated from school or training program ____
4. Had problems in school or in training program ____
5. Failed school, training program ____
6. Did not graduate from school or training program ____

WRK: 7. Started work for the first time ____
8. Returned to work after not working for a long time ____
9. Changed jobs for a better one ____
10. Changed jobs for a worse one ____
11. Changed jobs for one that was not better and no worse than the last one ____
12. Had trouble with a boss ____
13. Demoted at work ____
14. Found out that was not going to be promoted at work ____
15. Conditions at work got worse other than demotion or trouble with boss ____
16. Promoted ____
17. Had significant success at work ____
18. Conditions at work improved not counting promotion or other personal successes ____
19. Laid off ____
20. Fired ____
21. Started a business or profession ____
22. Expanded a business or profession ____
23. Took on a greatly increased work load ____
24. Suffered a business loss or failure ____
25. Sharply reduced work load ____
26. Retired ____
27. Stopped working, not retirement, for an extended period ____

L&M: 28. Became engaged ____
29. Engagement was broken ____
30. Married ____
31. Started a love affair ____
32. Relations with spouse changed for the worse, without separation or divorce ____
33. Married couple separated ____
34. Divorce ____
35. Relations with spouse changed for the better ____
36. Married couple got together again after separation ____
37. Marital infidelity ____
38. Trouble with in-laws ____
39. Spouse died ____

CHL: 40. Became pregnant ____
41. Birth of a first child ____
42. Birth of a second or later child ____
43. Abortion ____

CHL: 44. Miscarriage or stillbirth ____
45. Found out that cannot have children ____
46. Child died ____
47. Adopted a child ____
48. Started menopause ____
FMY: 49. New person moved into the household ____
50. Person moved out of the household ____
51. Someone stayed on in the household after he was expected to leave ____
FAM: 52. Serious family argument other than with spouse ____
53. A change in frequency of family get-togethers ____
54. Family member other than spouse dies ____
RES: 55. Moved to better residence or neighborhood ____
56. Moved to worse residence or neighborhood ____
57. Moved to residence or neighborhood no better or no worse than the last one ____
58. Unable to move after expecting to be able to move ____
59. Built a home or had one built ____
60. Remodeled a home ____
61. Lost a home through fire, flood or other disaster ____
62. Assaulted ____
C&L: 63. Robbed ____
64. Accident in which there are no injuries ____
65. Involved in a law suit ____
66. Accused of something for which a person could be sent to jail ____
67. Los drivers license ____
68. Arrested ____
69. Went to jail ____
70. Got involved in a court case ____
71. Convicted of a crime ____
72. Acquitted of a crime ____
73. Released from jail ____
74. Didn't get out of jail when expected ____
FIN: 75. Took out a mortgage ____
76. Started buying a car, furniture or other large purchase on the installment plan ____
77. Foreclosure of a mortgage or loan ____
78. Repossession of a car, furniture or other large items bought on the installment plan ____
79. Took a cut in wage or salary without a demotion ____
80. Suffered a financial loss or loss of property not related to work ____
81. Went on welfare ____
82. Went off welfare ____
83. Got substantial increase in wage or salary without a promotion ____
84. Did not get an <u>expected</u> wage or salary increase ____
85. Had a financial improvement not related to work ____
S.A. 86. Increased church or synagogue, club, neighborhood, or other organizational activities ____
87. Took a vacation ____

S.A.:	88. Was not able to take a planned vacation	____
	89. Took up a new hobby, sport, craft or recreational activity	____
	90. Dropped a hobby, sport, craft or recreational activity	____
	91. Acquired a pet	____
	92. Pet died	____
	93. Made new friends	____
	94. Broke up with a friend	____
	95. Close friend died	____
MIS:	96. Entered the Armed Services	____
	97. Left the Armed Services	____
	98. Took a trip other than a vacation	____
HLT:	99. Physical health improved	____
	100. Physical illness	____
	101. Injury	____
	102. Unable to get treatment for an illness or injury	____

APPENDIX V

INFORMED CONSENT

We are inviting your cooperation in this study. Your blood pressure will be taken before, during and after the interview. We will be asking you how often you experience certain feelings, attitudes and behaviors. Answering these questions will result in no injury to your health. We are also asking that you respond as accurately as you can to these questions, because we are trying to learn more about people's inner feelings, in order to better serve them. We are requesting persons connected with the St. Vincent's General Medical Clinic to answer these questions and everyone will be asked the same questions. The interview will last approximately one hour.

We would like to say that refusal to answer the questionnaire will not in any way affect your medical treatment or any other service provided by the hospital. Cooperation on your part, however, we hope will result in a stimulating and meaningful experience.

"I agree to respond to the questions asked. I understand that all information will be kept confidential and that it will be used for scientific purposes only. My cooperation will not expose me to any psychological risks.

"I also hereby release St. Vincent's Hospital and Medical Center and the Graduate Center of the City University of New York, parent and affiliated organizations, officers, directors, employees and students from any and all liability, including any matter or thing committed or omitted which may arise during the interview or from the data derived therefrom."

SIGNED _____

DATE _____

PRINT NAME _____

WITNESSED _____

DATE _____

NAME: _____ Male [ ] Female [ ]

ADDRESS: _____ Zip _____

AGE: 20-25 [ ] 26-30 [ ] 31-35 [ ] 36-40 [ ] 41-45 [ ]
46-50 [ ] 51-60 [ ] over 60 [ ]

ETHNIC AFFILIATION: White [ ] Black [ ] Hispanic [ ] Other [ ]

ARE YOU EMPLOYED: Yes [ ] No [ ]

CHECK YOUR HIGHEST BLOOD PRESSURE READING: 120/80 [ ] 130/85 [ ] 140/90 [ ]
145/92 [ ] 150/95 [ ] 160/98 [ ]
165/100 [ ] 170/105 [ ] 180/105 [ ]
185/110 [ ] 190/110 [ ]

DO YOU SMOKE 10 OR MORE CIGARETTES A DAY: Yes [ ] No [ ]

HOW LONG HAVE YOU BEEN SMOKING: 1 year or less [ ] 2-5 years [ ]
6-8 years [ ] 9-12 years [ ]
13-15 years [ ] Over 16 years [ ]

HOW MUCH DO YOU WEIGH: Less than, or 100 lbs [ ]
101-110 lbs [ ] 111-120 lbs [ ]
121-130 lbs [ ] 131-140 lbs [ ]
141-150 lbs [ ] 151-160 lbs [ ]
161-170 lbs [ ] 171-180 lbs [ ]
181-190 lbs [ ] 191-200 lbs [ ]
201-210 lbs [ ] Over or 211 lbs [ ]

ABOUT HOW TALL ARE YOU: Less than, or 4'4" [ ] 4'5"-5' [ ]
5'1"-5'3" [ ] 5'4"-5'6" [ ]
5'7"-5'9" [ ] 5'10"-6' [ ] Over 6' [ ]

ARE YOU ON A LOW SALT DIET: Yes [ ] No [ ]

HOW LONG HAVE YOU HAD HYPERTENSION: Less than 4 mths [ ] 5-9 mths [ ]

10-12 mths [ ] 1-2 yrs [ ] 3-4 yrs [ ]

5-6 yrs [ ] 7-8 yrs [ ] 9-10 yrs [ ]

11-12 yrs [ ] 13-15 yrs [ ] 16-20 yrs [ ]

21-25 yrs [ ] 26-30 yrs [ ] Over 31 yrs [ ]

DO YOU HAVE DIABETES: Yes [ ] No [ ]

DO YOU HAVE, OR HAVE YOU HAD KIDNEY PROBLEMS: Yes [ ] No [ ]

HAVE YOU EVER HAD A HEART ATTACK: Yes [ ] No [ ]

DO YOU HAVE ANGINA PAINS: Yes [ ] No [ ]

HAVE YOU EVER HAD A STROKE: Yes [ ] No [ ]

	MOTHER	FATHER
HAVE EITHER OF YOUR PARENTS EVER HAD HYPERTENSION:	Yes [ ] No [ ]	Yes [ ] No [ ]
HAVE EITHER OF YOUR PARENTS EVER HAD A HEART ATTACK:	Yes [ ] No [ ]	Yes [ ] No [ ]
HAVE EITHER OF YOUR PARENTS EVER HAD A STROKE:	Yes [ ] No [ ]	Yes [ ] No [ ]

DATA SHEET

Physiological Measures

NAME: _____

Identification Number: _____

Date _____ Time _____

Temperature _____ °F Barometric reading _____

1st Blood pressure: Systolic RA _____ Diastolic RA _____
LA _____ LA _____

Heart rate _____

2nd Blood pressure: Systolic RA _____ Diastolic RA _____
LA _____ LA _____

Heart rate _____

3rd Blood pressure: Systolic RA _____ Diastolic RA _____
LA _____ LA _____

Heart rate _____

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