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Personality, coping, and defense in the etiology of essential hypertension

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PERSONALITY, COPING, AND DEFENSE IN THE ETIOLOGY OF ESSENTIAL HYPERTENSION

by

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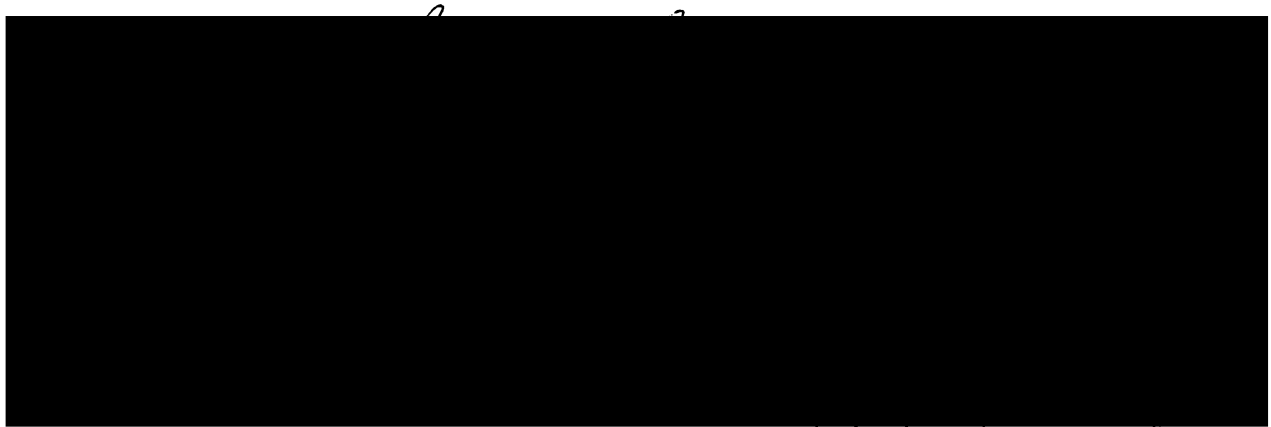

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Personality, Coping, and Defense in the Etiology of Essential Hypertension

A major objective of this study was to provide a rationale for investigating the hypothesis that personality factors are causally related to essential hypertension. This causal connection has been obscured by failure to provide a systematic notion of how such factors achieve physiological con-consequences. It is proposed that the way that a person thinks about and perceives his social-interpersonal environment is related to his health status. Stereotyped, unadaptive cognitive/perceptive processes mediating social-interpersonal stimuli can lead to a heightened sympathoadrenomedullary response which in turn may chronically raise arterial blood pressure. When used in this indiscriminate way, these mediational processes tend to cause an individual to respond to a host of social and interpersonal situations as if they were harmful or threatering to his well being. When this tendency is persistent, a heightened sympathoadrenomedullary response and subsequent chronic increase in arterial blood pressure may occur as a protective-adaptive reaction.

Subjects were 15 Black and 15 white male patients. Both groups had essential rather than secondary hypertension and were treated by clinic physicians with hydrochlarothiazide. Controls were 15 Black and 15 white male patients who, other than an occasional inability to fall asleep (for which chloral hydrate was prescribed), had no illness.

The empirical aspect of the paper focused on three areas: (1) the experimental hypothesis, (2) predicting systolic blood pressure from several personality and psychosocial variables, and (3) comparing drug treatment effect with race.

"Capacity to cope" was operationally defined by the Eysenck Personality Inventory (EPI) as moderate rather than extreme extroversion and introversion

scores. The hypothesis was partially verified in that the mean systolic pressure for a high extroversion group was significantly higher (Z_T=2.19, $p < 0.05$) than that for a moderate one. Contrary to the hypothesis, however, there was no significant difference in blood pressure between a low extroversion group and the moderate one.

Prediction of systolic blood pressure was related to a multiple regression design of the form $Y = a_1 + b_1X_1 + b_2X_2 + \text{---} b_kX_k$, where the components were social activity, extroversion/introversion, age, social stress, and number of subject's children. An $F_{5,54}$ of 4.78 ($p < 0.01$) suggested that the variance explained by this formula is significant and therefore this equation is important as a predictor of systolic blood pressure.

In the drug treatment focus, an analysis of covariance was performed in relation to race. The design for testing this idea for Black and white essential hypertensives was SYS_a (pretreatment systolic pressure) = covariate of SYS_b (posttreatment pressure), race = independent variable regressed against dependent variable, SYS_b. The data showed a covariate $F_{1,29}$ of 6.624, suggesting a significant (p 0.01) association (change) between the before treatment and after treatment blood pressures. An $F_{1,29}$ for the effect of race was 0.02 and not significant at the 0.05 level. By comparison control subjects had a covariate $F_{1,29}$ of 0.44 which was not significant at the 0.05 level.

Finally, Black and white hypertensives and Black and white normotensives were analyzed for dissimilarities in personality as measured by the Eysenck Personality Inventory. No significant differences were found. This result is inconsistent with the hypothesis that personality factors in Blacks not shared by whites are related to increased prevalence of essential hypertension in the Black community.

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INTRODUCTION

The belief that psychological factors can cause certain physical diseases is very old. In this paper, this conviction will be examined in relation to a class of disorders that collectively have come to be known as psychosomatic illnesses focusing on one such illness, essential hypertension. This disorder will be explored with the purpose of isolating some of the major psychological variables related to it.

Following this section, a theoretical model will be outlined which is intended to provide a rationale for investigating the idea that coping and defense, as dimensions of personality, are causally related to essential hypertension. An empirical model will define the experimental variables and show relations among them.

Finally, the results will be presented and discussed.

PSYCHOSOMATIC ILLNESS

This chapter begins by defining psychosomatic illnesses and briefly discussing general conceptions about them. Following this, a literature review is presented organized according to etiological theories of these disorders.

Definition and Conception

Psychosomatic illness may be generally defined as a disease thought to have an underlying psychological causation resulting in physical abnormality.

As for conception, Hambling (1965) suggested that an illness may be diagnosed as psychosomatic when (1) it shows a time correlation with situations provoking stress in the patient, and (2) it clears up or improves when the situation changes for the better or when the patient learns to adapt and lowers stress. However, the most common notion has been summarized by Lachman (1972): an illness may be diagnosed as psychosomatic when there is evidence that it is not caused by microorganisms, toxins, or other exogenous factors. This diagnosis by exclusion has been criticized by some investigators.

Psychosomatic Theories

Based on etiology, psychosomatic theories can be divided into three groups (Glock et al, 1957; Lachman, 1972) which are presented in the order of their chronological development: (1) personality theories, (2) conflict theories, and (3) physiological stress theories. The first

two groups have direct psychoanalytic roots, and the third is based on physiological concepts developed by Selye (1950, 1956, 1976). The psychoanalytic theories preceded the physiological stress models.

Mirsky (1957) argues that psychoanalytic theory has had a pervasive influence on understanding the etiology of psychosomatic disorders. In fact Mirsky and Brown (1958) have suggested that the psychosomatic approach is based almost entirely on psychoanalytic concepts. Here it is important to remember that psychoanalysis began with the study of the psychological aspects of physical symtoms (Linn, 1958). The classical monograph on hysteria by Breuer and Freud (1955) dealt mainly with the notion that certain physical symptoms can have a psychological cause.

Macleod et al (1953) proposed that the introduction of the psychoanalytic technique led to a focused interest in the manner in which "feelings, attitudes, and values influence physiological activity and are in turn influenced by it...Thus arose psychosomatic medicine."

According to Grinker (1953), psychoanalytic theory has provided two concepts about the cause of psychosomatic disorders: (1) psychosomatic disorders arise because an individual fails to modify his adaptive responses as he matures and as situational factors change, and (2) psychosomatic illnesses come about because more appropriate responses are hindered by neurotic behaviors such as phobias, obsessions, and compulsions.

The "physiological approach" emphasizes the adverse effect of stress on physiological functions. Selye's (1950) concepts have provided the foundation for this orientation. He proposed that the chronic

exposure of an organism to a highly nosious stimulus initiates a sequence of physiological events. These events were referred to as the "general adaptation syndrome". Selye proposed that this generalized, non-specific response is primarily dependent upon the arousal of the anterior pituitary gland and the subsequent activation of the adrenal cortex. Prolonged adrenal cortical activity may cause extensive physiological disorders and can result in various diseases referred to as "diseases of adaptation".

(1) Personality Theories

One of the earliest psychoanalytic formulations about the cause of psychosomatic diseases was proposed by Ferenzi (1926). He suggested that psychosomatic illnesses represent a conversion disorder. Personality theories of psychosomatic diseases began however with Dunbar in 1935. She argued that the conversion theory was too limited to explain the various psychosomatic diseases. Psychosomatic illnesses, she reported, have a high statistical correlation with certain personality types.

Following Dunbar, the psychosomatic literature was dominated by personality theory. Ruesch (1948) concluded that psychosomatic diseases occur in individuals whose personalities have not matured. A related hypothesis proposed by Spitz (1951) tied the lack of marutity to a negative mother-child relationship. Margolin (1953) wrote that psychosomatic illnesses stem from regression in the face of stress. Sainsbury (1960-1964) reported finding a significant, positive correlation between psychosomatic diseases and "introverted and neurotic" personalities.

Recently, certain investigators have attempted to expand the classification of psychosomatic diseases beyond the classical grouping suggested by Alexander (1950) and others. For example, Friedman et al (1974) found that aggressive, out-going, and hurried individuals have significantly more heart attacks than more relaxed persons. Simonton et al (1975) reported that patients with cancer are characterized by a poor self image, hopelessness, and lack of ability to develop and maintain meaningful interpersonal relationships. The authors of these two studies propose that psychosocial factors are etiologically related to these diseases.

In critical reviews Glock et al (1957), Geiger et al (1963), Davies (1970), and others have drawn attention to weaknesses in psychosomatic personality theories. Generally, the assessments of personality are too subjective. More importantly, the manner in which personality may be etiologically related to the various physical diseases is not discussed.

(2) Conflict Theories

After Dunbar, the most important contributor to psychosomatic theory was Alexander (Mendelson et al, 1956). Alexander (1950) agreed with Dunbar that psychosomatic disorders were not conversion phenomena. However, he disagreed with her emphasis on personality types. It is not a personality type that characterizes a psychosomatic disease, he wrote. Rather, each psychosomatic illness stems from a typical conflict situation. This conflict can develop in individuals with varying personalities.

Theorists influenced by Alexander continued his emphasis on conflict as a cause of psychosomatic disease. Grinker (1953) focused on conflict over fear and loss of face in wartime soldiers. Murphy (1953) concentrated on conflict in relation to responsibility and authority. Engel (1954) studied conflict in regard to dependence and independence. Baraff et al (1965) were concerned with conflict over the expression and suppression of hostility.

More recently Parker et al (1969) have emphasized what they called the increased psychosomatic death rate in persons with conflicts over desire to live or to die.

(3) Physiological Stress Theories

For convenience, physiological stress theories may be divided into (a) psychophysiological and (b) sociophysiological subgroups. Both of these emphasize the notion that stress disrupts physiological functions.

(a) Psychophysiological stress theories. In this subgroup, stress is viewed as stemming primarily from interpersonal interaction. As stated, physiological stress theories are based on Selye's (1950) protective-adaptive concepts. One of the earliest and most important contributors in this group is Wolff (1950, 1968). He initiated a reaction to the line of thought developed by Dunbar and Alexander. According to Wolff (1950), neither of these authors paid enough attention to physiological and psychophysiological factors that may cause psychosomatic diseases. He proposed that the body defends itself from stress by a complex protective-adaptive reaction. This reaction is intended only for short-term, emergency responses to stress. When stress is prolonged, malfunctions

in the protective-adaptive system can occur. Sustained malfunctioning in this system may result in widespread organ and tissue damage.

A number of psychosomatic investigators have been influenced by Wolff's ideas. Deutsch (1955) proposed that psychosomatic diseases develop because of chronic disturbances of physiological activity at stressful periods. Szasz (1957) regarded a psychosomatic illness as a regression to infantile modes of physiological functioning. This is characterized by a relatively greater physiological reactivity to stimuli.

Contrary to Selye's and Wolff's emphasis on the adrenal cortex, Baker et al (1968) suggested that prolonged activation of any biological mechanism causes psychosomatic disease.

(b) Sociophysiological stress theories. Authors such as Wardwell et al (1968), Paasikvi et al (1971), Reeder et al (1973), and Levi (1974) are representative of this group. The main idea they advance is that an explanation of psychosomatic disease must be sought in the stressful context of the larger society rather than exclusively in interpersonal interaction. Social isolation, alienation, and life crisis are examples of these wider sources of stress. According to Levi and others, these social stresses may upset normal physiological processes and cause a number of physical illnesses. These authors suggest that this "sociophysiological" concept explains the accelerated increase in chronic (i.e. psychosomatic) as opposed to acute diseases that have occurred over the last two decades. Reeder et al and other investigators in this group contend that this disease shift necessitates the wider causal view.

Holmes et al (1967) have developed a questionnaire which measures social stress (life events). They proposed that the higher the

number of these stresses that a person experiences, the more likely he will develop a physical illness in the near future.

Rahe et al (1972) have consistently found a high positive correlation between the number of life stresses and physical illness. They reported that when their subjects' yearly "life change unit" (LCU) value was from 0 to 150, the majority of them remained "in good health" in the succeeding year. When subjects' yearly LCU value ranged between 150 and 300, an illness during the following year occurred in approximately half of them. For the relatively few subjects who registered over 300 LCUs per year, an illness occurred during the following year in 70 per cent of them. They also reported that illnesses which were associated with over 300 LCUs per year tended to be multiple.

Rycroft (1965), Caplan (1974), Cobb (1976), and others have emphasized the role of alienation and lack of a network of supporting social contacts as sources of stress that may be etiologically related to physical diseases. Rycroft suggested that the effect of a person's social network or social support system on the onset and development of his illness is generally a function of at least three factors: (1) the nature of the social, legal, and biological ties existing among the persons involved in the social network, (2) physical and psychological proximity of the persons, and (3) each person's perception of his role in the social network.

In a comparison between supported and unsupported males undergoing the stress of prolonged unemployment, Cobb (1974) reported that the unsupported subjects had higher cholesterol levels than did the

supported ones. The Framingham (1968) study found that elevated cholesterol is one of the chief risk factors associated with heart attack, stroke, and hardening of the arteries.

ESSENTIAL HYPERTENSION

Of the psychosomatic illnesses, essential hypertension is the most prevalent and has the greatest mortality consequences (Build and Blood Pressure Study, 1959). The 1960-62 National Health Survey indicated that about 15 per cent of the U.S. population suffers from definite hypertension (systolic pressure of 160 mm Hg or higher or diastolic pressure of 95 mm Hg or higher). A similar proportion has borderline hypertension (systolic pressure of 140 to 159 mm Hg or diastolic pressure of 90 to 94 mm Hg).

With 1976 population figures, it is estimated that almost 60 million Americans have or potentially have blood pressures in the ranges that are associated with increased mortality. The Build and Blood Pressure Study concluded that at any age, the higher the blood pressure above an optimal level of 88 to 97 systolic and 48 to 67 diastolic, the greater the chance of a cardiovascular related death.

Abnormally high blood pressure may have a known or an unknown organic cause or set of causes. When a specific causal factor or set of such factors can be identified (e.g. a diseased kidney), the hypertension is said to be "secondary". This type of hypertension accounts for about ten per cent of all hypertensive cases. In the remaining 90 per cent, a specific organic cause or set of such causes cannot be found. Here the hypertension is said to be "primary" or "essential". Pickering (1968) defined essential hypertension as "high blood pressure without evident cause."

Normal and Abnormal Blood Pressure Levels

Efforts to determine the point at which blood pressure becomes abnormal have led to some controversy and a number of conceptual approaches. According to Geiger et al (1963), current efforts to establish normal and abnormal levels of blood pressure are based on three approaches: (1) a statistical model of the normal curve of pressures (i.e. the usual distribution of blood pressures in a population), (2) empirical examination of mortality statistics, and (3) establishing arbitrary operational criteria for the purpose of standardization in epidemiological research and clinical practice.

Pickering (1961, 1968), Platt (1963), and others have been associated with the first approach. Platt suggested that blood pressure has a bimodal population distribution. That is, it is divided into normal and abnormal groups which differ qualitatively. In opposition to this view, Pickering argued that blood pressure is unimodally distributed. Therefore, there is no qualitative division of blood pressure into hypertension and normotension. Blood pressure and mortality are quantitatively related; the higher the pressure the worse the prognosis.

The second approach has been largely established by the Build and Blood Pressure Study (1959). This model has been developed in connection with the large U.S. life insurance studies. The main idea is that "normal" blood pressure is that which is associated with least mortality from disease of the circulatory system.

The third paradigm is identified with the World Health Organization (1962). Investigators there analyzed data from a number of independent studies. It was found that, when combined with specific ages

and sex, blood pressures above a certain point (160/95) are associated with a significantly greater risk of hypertensive mortality. These pressures are considered abnormal.

Increased Morbidity and Mortality Associated with Hypertension

In the past decade, understanding of the relation between hypertension and cardiovascular disease processes has been considerably increased. This is reflected by the widespread view that the higher the blood pressure (above an optimal level) the more severe the illness consequences. In general it appears that high blood pressure is a factor in the acceleration of atherosclerosis (Framingham, 1968), in hemorrhagic stroke (Pickering, 1968), in renal failure (Schalekamp et al, 1975), and in heart failure (Cohn, 1975).

AGE, SEX, AND RACE AS INDICES OF RISK OF ESSENTIAL HYPERTENSION

Age

The prevalence of essential hypertension increases with advancing age. Life insurance data from the Build and Blood Pressure Study indicate that men 50 to 59 years of age with a "moderate blood pressure of 150/100 mm Hg" have twice the mortality of males the same age but whose blood pressure is normal. The same moderate pressures in the 40 to 49 year age group are related to a mortality increase of 3.6 times the "standard risk". At ages 30 to 39, these pressures are associated with a mortality five times greater than that for normotensives of the same age. Thus, when appearing relatively early in life, even a moderate elevation of blood pressure is correlated with a greatly increased risk of premature death.

Sex

According to the Build and Blood Pressure Study, the prevalence of essential hypertension up to age 50 is greater for men than for women. After this age the situation reverses. Comstock (1957) proposed that sex differences related to prevalence suggest either that (a) counter-active mechanisms (associated with endocrine function or child bearing) are at work in the female under 50, or (b) that the causal or primary mechanisms producing blood pressure elevation differ in nature, effect, or magnitude by sex.

Even with the greater prevalence in later years, women have a lower overall mortality rate. At a given hypertensive level women relative to men have a better prognosis (Freis, 1976). Mathisen et al (1959) and others reported that mortality was approximately twice

as high in men as in women. In an earlier study, Comstock wrote that male/female mortality differences were related to socioeconomic status. The female hypertensive rates were higher than the male's in the lower socioeconomic group. This was reversed for the higher status subjects. Both Blacks and whites demonstrated this pattern. He concluded that etiology is rarely a function of a single factor. Causation tends to be multiple, though a given variable may have a primary role in one situation and a secondary one in another.

Race

Most investigations support the suggestion that essential hypertension in Blacks is more prevalent and severe than in whites. With respect to prevalence, the National Health Survey (1966) found a blood pressure of 160/95 mm Hg or higher in 27 per cent of the adult Black population as compared to 14 per cent of white adults. Since the survey was a representative sampling of the national population, it has been concluded that the prevalence of essential hypertension in the United States is almost twice as high in Blacks as in whites. The survey indicated that this excess prevalence in Blacks was approximately the same in every age group.

Blacks also have a higher death rate from hypertensive heart disease. Vital statistics for the year 1967 show that this rate is 66 per 100,000 for Black men compared to 16 per 100,000 for white men. This represents a mortality difference of approximately 4 to 1. Before age 50, this death rate is about six to seven times higher in Blacks than in whites.

Several hypotheses have been advanced to explain the higher hypertensive morbidity and mortality rates among Blacks. The major

ones are: (1) the physical exertion hypothesis which argues that Blacks are more likely than whites to be engaged in manual labor occupations, and that greater physical exertion leads to higher mortality from hypertension (Lennard et al, 1957), (2) the psychological stress hypothesis which contends that Blacks are severely frustrated and stressed by racial discrimination (Stamler et al, 1960, 1962), (3) the genetic hypothesis which purports that Blacks differ genetically from whites in ways that predispose Blacks to higher hypertensive rates (Nichaman et al, 1962; Scheckloth et al, 1962; McDonough et al, 1964), (4) the associated disorder hypothesis which is that Blacks are more prone to develop diseases (e.g. pyelonephritis) which cause blood pressure to rise (Stamler et al, 1960), (5) the diet hypothesis which holds that Blacks have dietary habits which increase their susceptibility to hypertension (Burch et al, 1957; Wadsworth, 1960), and (6) the medical care hypothesis which states that Blacks receive poorer or less frequent medical care than do whites (Howard, 1965).

A number of criticisms of these hypotheses has been raised. For example, the empirical findings for some (e.g. hypothesis 1) are mixed (Stamler et al, 1962). In other cases (e.g. hypothesis 3), the assumptions are difficult to test. Generally, the authors do not discuss psychological factors which may underlie their investigations. It is possible that discrepancies in medical care alone cannot adequately explain different hypertensive rates. Perhaps a person's thoughts, feelings, and attitudes about the medical profession influence his decision to seek medical attention. In addition to this decision, there may be psychological factors which affect how carefully the physician's orders are followed.

Perhaps the best conclusion is that more work needs to be done in this area before it can be better understood.

PSYCHOLOGICAL THEORIES OF ESSENTIAL HYPERTENSION

The general hypothesis that psychological factors have a causal role in the etiology of essential hypertension has been under increasingly sophisticated investigation. During the evolution of this hypothesis, studies have ranged from relatively undifferentiated notions of emotional stress to more elaborate conceptions of a hypertensive personality.

Theories of the psychological causation of essential hypertension can be placed in three broad groups (Glock et al, 1957). In the order of their chronological development, the groups are: (1) Theories of hypertensive personality, (2) Theories of intra-psychic conflict, and (3) Theories of physiological stress.

(1) Theories of hypertensive personality

A number of researchers have suggested that essential hypertension is causally related to certain personality characteristics. Ayman (1933) proposed that essential hypertensives tend to be highstrung and quick tempered. They are inclined to be unusually sensitive and hurt by small matters. In youth and often in later life, they have a tendency to blush easily and to be readily embarrassed. When dealing with events of life, they are unusually serious and worry about trivialities.

Dunbar (1935, 1939) wrote that patients with essential hypertension are shy in social relationships, fearful of not making the grade, and apt to be self-centered. In their childhood they are ambivalent towards their fathers and hostile and fearful of their mothers.

Tucker (1949) suggested that hypertensives are conscientious,

strongly devoted to duty, scrupulous, perfectionistic, and meticulous. They have high ideals and are never satisfied with their work. In addition, they are highly sensitive to criticism and prone to be crushed by failure.

Gressel et al (1949) offered the view that patients with essential hypertension have a tendency to "Obsessive-compulsive behavior and subnormal assertiveness." Patterns of anxiety and hysteria are also prevalent. Weiss et al (1952) also reported finding compulsiveness in essential hypertensives.

Palmer (1950) described the hypertensive personality as ambitious, energetic, and liable to mood alterations that are not related to life situations. In contrast to Dunbar (1939), Palmer reported that patients with essential hypertension are extroverted and habitually talkative. However, Sainsbury (1960) found lower levels of "extroversion" in essential hypertensives than in controls. Davies (1971) concluded that there is no consistent relationship between blood pressure and extroversion.

In the hypertensive personality literature, the most frequently mentioned traits are hostility, anger, anxiety, and nervousness. Saslow et al (1950) emphasized that their patients with essential hypertension "recurrently and frequently inhibited impulses to overt action." They suggested that these repressed impulses are manifested as outward hostility and raised blood pressure. Grace et al (1952) reported that anxiety is chronically present in essential hypertensives because they feel that they must be constantly prepared to meet all possible threats. Benedict (1956) also commented on observing anxiety in her patients with essential hypertension.

More recent hypertensive personality studies shifted from an emphasis on traits such as anger and hostility to a concern with the causative role of neuroses. In a population at large sample, Robinson (1962) found "relatively high levels of neuroticism" in subjects previously diagnosed as essential hypertensives. Sainsbury (1964), using the Eysenck Personality Inventory (EPI), also found higher neuroticism in the hypertensives than in the normotensives.

Kidson (1971, 1973) conducted a series of neurosis/essential hypertensive studies. In the 1973 study, he compared three groups: (1) persons whose blood pressures were at hypertensive levels (these subjects were unaware of this condition, as they had no previous diagnosis of hypertension), (2) diagnosed essential hypertensives, and (3) normotensives. Each subject in the second group was aware of the hypertensive disorder and of having been professionally diagnosed. In comparison to the other groups, the subjects in group two had higher levels of neuroticism and insecurity. Kidson (1973) proposed that these disorders were related to the subject's knowledge of the diagnosis.

In agreement with Tucker, Gressel et al, and Ayman, Kidson (1970) wrote that relative to normotensives, essential hypertensives are angry, sensitive, and anxious. While not all studies in this group agreed on every specific discovery, one may be impressed with the large number of findings of a significant, positive correlations between personality and essential hypertension.

Hypertension and Personality Change

The issue of whether personality factors cause essential hypertension or whether essential hypertension produces certain personality

traits was addressed by Harris et al (1953). If certain personality traits cause essential hypertension, they reasoned, then these traits should appear before the disease. Subjects were pre-hypertensive (i.e. borderline hypertension) and normotensive college women. They reported that relative to the controls, the pre-hypertensives were high-strung and anxious. In a follow-up study, Sokolow et al (1961) confirmed Harris et al's basic results.

These studies suggest that personality traits such as anxiety and nervousness develop early in life and may cause essential hypertension in later years.

Negative Results

Three studies reported negative findings of personality differences between essential hypertensives and normotensives. The most recent of these was by Ostfeld et al (1959, 1960), and the earlier one was by Stornment (1951). The 1959 study, using the MMPI and the Rorschach, compared three groups: (1) essential hypertensives, (2) renal hypertensives, and (3) controls drawn from surgery, general medical clinics, and gynecology. The authors reported finding no significant personality differences between the hypertensive and control groups. In commenting on these findings, Pickering (1968) suggested that the MMPI is intended to identify serious rather than mild mental disorders. Another problem with this study is that it is difficult to compare it with most of the other investigations because the Ostfeld et al sample was predominantly female; most personality/essential hypertension studies have used a male dominated sample.

In the 1960 study, Ostfeld et al separated their subjects into four categories: labile essential hypertensives, labile normotensives,

stable essential hypertensives, and stable normotensives. They found that the labile hypertensives were similar to the labile normotensives. Both stable groups differed significantly from the two labile ones. They concluded that differences in personality are found not between hypertensives and normotensives but between labile and stable blood pressure groups.

In this study an important methodological issue arose. They did not control for the interaction of the mean and the variability of blood pressure. A number of investigators (Geiger et al, 1963) have observed that mean blood pressure level is affected by lability (i.e. variability). Therefore, if variability is studied without controlling for the mean, certain personality factors may correlate with lability, mean blood pressure, or both. In this event interpretation may be difficult. Geiger et al suggested that, in such studies, one of the two variables should be controlled.

In Stornment's study, there were five groups: 25 subjects with essential hypertension, 25 with rheumatic heart disease, 19 with atherosclerosis, 8 with coronary occlusion, and 13 controls "with infections and other non-cardiac diseases".

Data were collected with the Guilford and Guilford-Martin personality tests. The author reported that the groups did not significantly differ.

Stornment's design incorporated a number of features which made it superior to many previous ones. For example, she used groups to control for the possible effects of serious illness on personality. There was a problem in getting subjects to complete the experiment. She reported that

a few patients were reluctant to cooperate because they feared that the questionnaires might have a negative effect on their medical treatment. Others were said to have been adverse or unavailable for personal reasons.

Critique of personality/hypertensive studies

Discussions under this heading will focus on theoretical integration, conceptual issues, and methodological problems.

A major criticism of the studies reviewed in this group is that the research instruments are not related to a larger theory. As a result, the causal relationship between the behavior measured and the illness cannot readily be explained. Ayman, for example, reports that essential hypertensives are high-strung and quick tempered. He does not discuss why these rather than other traits appear together. Also it would be valuable to know what these particular traits have in common theoretically and how they operate in the etiological process.

Investigators do not explain the conceptions and assumptions which lead them to emphasize or observe one aspect of personality rather than another.

Most authors do not clarify whether they are suggesting that essential hypertensives uniquely possess traits such as hostility and aggression or whether they merely have these traits to a greater degree than do other persons. Some writers (Dunbar, 1939) propose that it is the total pattern of particular traits in interaction that is unique to essential hypertensives. However, this approach is very general.

An important methodological problem is that control groups are either not used or they do not help clarify data analysis.

(2) Theories of intra-psychic conflict

Investigators in this group proposed that essential hypertension is etiologically related to the inability of certain individuals to effectively resolve conflicts. Alexander (1939), a pioneer in this area, argued that the specific hypertensive conflict is between needs for dependency and aggression. This results in inhibition and repression which are eventually expressed in chronically elevated blood pressure. Saul (1939) and others suggested that essential hypertensives experience conflict in relation to the desire to express hostility and reluctance to do so because of fear of the consequences. Hambling (1952) indicated that patients with essential hypertension have conflict over dependence and independence in their everyday lives. Moses et al (1956) proposed that essential hypertensives have conflicting needs for expressing and suppressing anger. Binger (1951) and Saslow et al (1950) had previously alluded to the ability of anger to raise blood pressure.

More recently Brown et al (1976) and Reiss et al (1967) reported that patients with essential hypertension experience conflicts in family interactions. Hafner (1974) noted a relationship between patients with essential hypertension and "aggressive-defensive" responses to interpersonal conflicts.

Critique of conflict/hypertensive studies

Relative to the personality group, the conflict studies have more theoretical integration. This is in part related to the fact that the hypotheses in this latter group are derived from psychoanalysis, "a relatively solid psychological theory" (Glock et al, 1957).

In comparison to the personality and stress hypotheses, the conflict hypotheses are more dependent upon a specific conflict-disease syndrome. For example, conflict over dependent and independent feelings toward one's parents is thought almost always to result in essential hypertension. The extent to which this particular conflict also characterizes other diseases is not explored by authors in this group. Also there is often little attempt by individual investigators to integrate their findings with the broader psychoanalytic theory.

In conflict theory, anxiety has a key role where a certain amount of anxiety in relation to incompletely repressed feelings is always present (Freud, 1946). Given the prevalence of anxiety in the general population and in a host of illnesses, it seems that conflict theorists must be specific about how anxiety is related to essential hypertension. They must also identify the particular aspect of anxiety that is purported to raise blood pressure.

Writers in this group relative to those in the personality group have studied noticeably smaller samples. In addition, the subjects are most often patients with essential hypertension who are not compared with controls. As a consequence, few meet desired standards of statistical significance.

(3) Theories of physiological stress

Stress in this group refers most generally to a state in which a stimulus places a demand upon a person's adjustive mechanisms. These mechanisms serve to restore the individual to psychophysiological equilibrium.

Two major assumptions underlying the essential hypertension-stress position are: (a) that an individual reacts to many different kinds of stress with a characteristic physiological arousal, and (b) that this arousal is causally related to essential hypertension.

Wolff (1950), a major figure in this area, suggested that "protective reaction responses" are consequences of psychological stress. He and other theorists in this group contend that essential hypertension results from a prolonged protective-reactive response to psychological stress. The notion is that this reaction is meant biologically only as a short-term, emergency adjustment to increased stress. Consequently, when stress is chronic, protective-reactive mechanisms may break down. In such a case, the acutely increased blood pressure may become chronically raised. Authors such as Margolin (1953), Grinker (1953), and Wolf et al (1968) emphasized this notion in interpersonal situations.

Reiser et al (1968) formulated their stress-essential hypertension hypothesis in relation to life stress. Saslow et al (1968) focused on "negative emotions" as sources of stress, and Ackerman (1968) concentrated on lack of character development.

Critique of physiological stress/hypertensive studies

A major strength in this group is that the authors attempt to be specific about possible mechanisms in essential hypertension. In addition direct causal links between stress and this disease are discussed. Generally, investigators in this group maximize physiological concepts and minimize psychological ones.

Most of the authors do not specify the conditions under which an individual will experience stress or indicate when essential hypertension will be the psychophysiological consequence.

Methodological shortcomings center around the use of patients with essential hypertension without controls.

A Comprehensive Approach to the Etiology of Essential Hypertension

Investigators such as Geiger et al (1963) and Gunn et al (1972) have called for an integration of the various physical and psychological factors thought to interact in the etiology of essential hypertension. It does not appear that this has been fully accomplished. The personality and conflict theories, for example, do not discuss the role of physiological processes and mechanisms that stress theorists suggest are causally involved in the hypertensive disorder. Physiological stress theorists on the other hand give inadequate attention to personality and conflict factors which may underlie essential hypertension. The present model (outlined in the following section) is designed to be broader than the ones discussed. This is accomplished by integrating the psychological and psychophysiological components of the paradigms reviewed.

Possible Mechanisms in Essential Hypertension

A frequent criticism in the literature is that investigators do not describe the mechanisms of the personality/hypertensive interaction. In response to this, some brief statements about such mechanisms

are presented here. According to Pickering (1968) and others, there are three basic theories of the mechanisms that cause essential hypertension: neural, humoral, and neurohumoral. Neural theories propose that chronically elevated blood pressure results primarily from alterations in neural regulatory mechanisms, including increased sympathetic vasoconstriction. Humoral theorists suggest that increased blood pressure is mainly a function of vascular and blood volume changes resulting largely from renal and adrenal hormonal factors. Neurohumoral (interaction) theories indicate that neural and humoral elements collaborate in the production of essential hypertension.

Investigators who are interested in a psychological or psychophysiological causal model also propose an interaction theory. The notion is that chronically elevated blood pressure results from an interaction of higher cortical activity, psychological variables such as stress, neural, and endocrine factors.

THEORETICAL MODEL

This section presents a graphic illustration of a stress/psychosomatic illness (EH) model and explains its theoretical and functional components. In addition, it discusses how such a model attempts to solve the problems raised in the literature review.

The present model draws heavily from the stress paradigms of Selye (1950, 1976) and Henry et al (1974), from the coping theories of Janis (1958) and Lazarus (1966), and from the neobehavioral models of Miller et al (1960) and Pribram (1967). By incorporating ideas from these paradigms, the present model is thought to be more comprehensive than the ones reviewed earlier. The model may be diagrammed as follows:

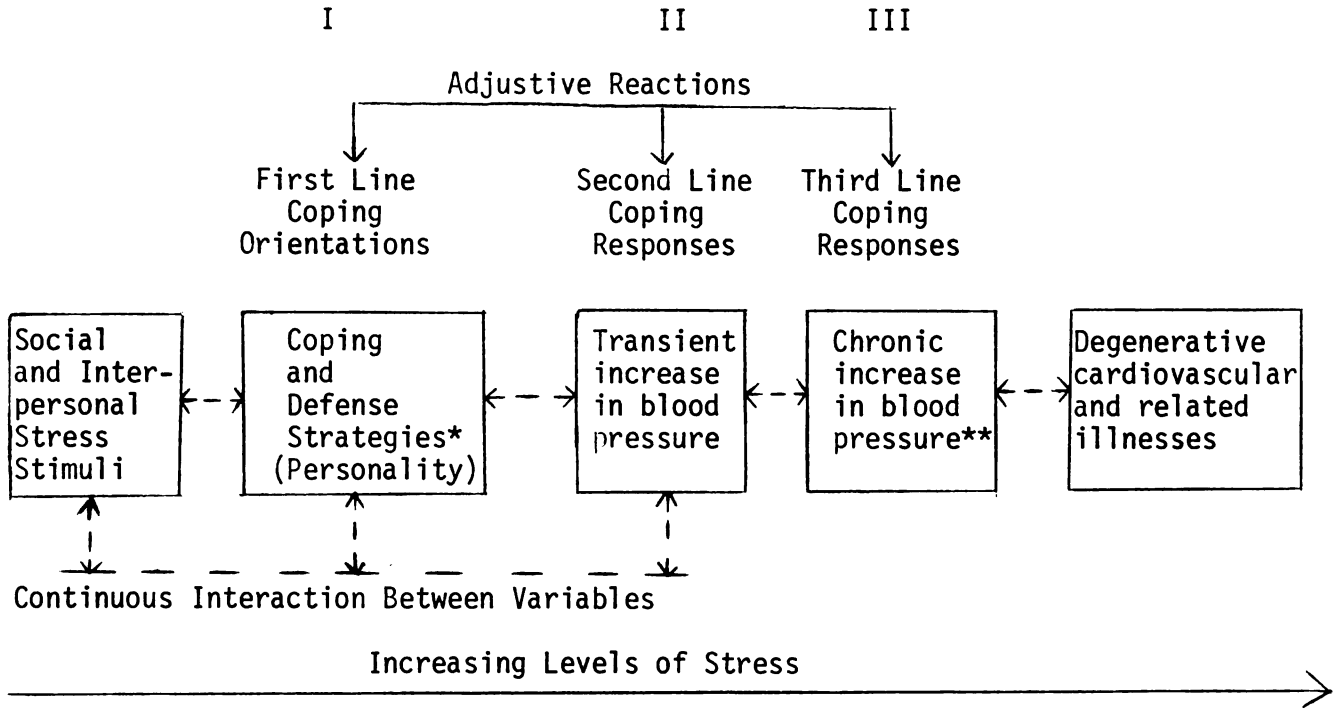

Figure 1

*Extroversion and introversion
**Essential Hypertension

Definitions and Conceptions

Social and interpersonal stimuli environment. Social stimuli result from gatherings such as ball games where no words are directly exchanged between those present. Interpersonal stimuli by contrast refer to a complex of verbal and non-verbal transactions between or among individuals. Stress theorists (e.g. Appley et al, 1967) have suggested that these two types of stimuli are the most pervasive and potent sources of stress in modern urban life.

First line coping and defense (personality) strategies. The basis idea here is that when social or interpersonal stimuli either threaten to or actually impinge upon an individual, his preconceived coping or defensive strategy is set into motion. This strategy at its basic level is designed to mediate these stimuli so that stress does not increase and cause blood pressure to rise. Since this coping concept is crucial to the present model, it was decided to discuss it more extensively than the other components in the paradigm.

In the model here, coping and defense focus on cognitive-perceptive processes rather than on overt behavior. This is related to Lazarus' (1966) suggestion that an analysis of coping and defensive theories may profitably center on the cognitive-perceptive processes that are presumes, at some level, to underlie all behavior.

Coping (including defense) may be broadly defined as a strategy for satisfying an individual's adjustive demands. It is proposed that

coping is based on an approach/avoidance directional dichotomy. In this paper, this dichotomy specifically applies to the directional way that an individual cognitively and perceptively handles stimulus input. An approach orientation describes a person who habitually copes by seeking out stimuli. An individual with an avoidance inclination characteristically copes by screening out or avoiding stimuli.

What are the major factors that determine the selection of one directional orientation over the other? In an unpublished paper, Kagan (1973) proposed an interesting possibility. She wrote that Rotter's (1966) "locus of control" is the central concept in coping theories related to physical illness. Rotter (1966) explained that the locus of control concept refers to a person's generalized belief about the effect that his behavior has on situational events and outcomes. If a person believes that his behavior is capable of influencing these outcomes, he is said to have an internal locus of control. An "internalized" person is disposed to seek an active intervention into those aspects of his situational environment which he feels affects his well being. This person intervenes so as to favorably influence events and outcomes. This is an approach orientation. An "externalized" person, by contrast, presumes that events are not effected by his actions. Instead he assumes that situational outcomes are determined by forces beyond his control. By not attempting to influence events, this person protects himself from failure and disappointment. This is an avoidance strategy.

The locus of control distinction emphasized here is not between active and passive behaviors. The concern is with a cognitive-perceptive set oriented towards approach versus avoidance. Though they differ in particulars, both directional propensities have the same objective. Namely, to cope with one's environment as effectively and efficiently as possible.

In short, it seems that the locus of control notion provides a rationale for understanding an approach versus an avoidance coping preference.

Coping Models

Various coping models extant in the literature can be understood in terms of the "locus of control". Four examples will serve to illustrate this: (1) participatory versus preparatory processes, (2) leveling versus sharpening, (3) coping versus defense, and (4) extroversion versus introversion.

(1) Participatory versus preparatory processes

This model proposes that an individual has an innate tendency to seek a "neurophysiological equilibrium" between stimulus input and output (Pribram, 1967). Participatory and preparatory coping processes are designed to accomplish and maintain this equilibrium.

Participatory orientations facilitate stimulus input. This inclination is designed to provide an individual with flexibility "through a more complex neural organization". It leads him to attempt

to adjust his internal systems to the arousal consequences of increased stimulus input. This process involves "an engagement, an involvement, or commitment to environmental events...that stem beyond the individual." This is a towards or approach coping style.

Preparatory processes, by contrast, screen out stimuli and correspond to an avoidance set. According to Pribram, the clinical and experimental literature is replete with examples of these processes. Concepts such as repression, suppression, and perceptual defense can be interpreted as preparatory processes, for they are forms of defensive "gating out."

(2) Sharpening versus leveling

Generally, sharpening refers to a process of emphasizing stimulus input, of accentuating or sharpening its distinguishing characteristics. Sharpening corresponds to an approach coping orientation.

The leveling concept identifies a tendency to screen out or overlook stimulus input and to minimize or level out its discriminating characteristics. Leveling is associated with an avoidance inclination.

(3) Coping versus defense

At the cognitive level, coping (i.e. mature, adaptive ego processes) is indicated by a preference for such ego functions as intellectualization and sublimation. Copers have an approach preference.

Defense (i.e. immature, unadaptive ego processes) suggest a propensity for repressive and suppressive ego processes. Defense is seen as avoidance.

(4) Extroversion versus introversion

Like participatory, sharpening, and coping orientations, extroversion* is an approach strategy. It refers to an orientation to think about and perceive social stimuli in certain ways. The extrovert's basic propensity is to screen in (rather than screen out) social stimuli. This inclination may behaviorally manifest itself as a tendency to seek out other people and to avoid being alone.

The extrovert's coping preference, according to Eysenck (1967), is related to his being underconditioned. Interpersonal relationships can be complex. In order to successfully negotiate them, an individual must have acquired certain social skills. These are usually learned during the process of socialization. Because of underconditioned social skills, the extrovert is ill-prepared to deal with the complexities of cause-effect interpersonal interactions.

The extreme extrovert appears coarse and overpowering in interpersonal relations. Lack of sensitivity or understanding of social intricacies prevents his improving his negative social impact. These factors expose him to reactionary hostility and other forms of stress-producing interactions. Extreme extroversion can therefore be seen as representing a decreased capacity to cope with interpersonal and social stimuli.

Introversion, as with preparatory, leveling, and defensive orientations, is an avoidance strategy. It refers to a conditioned way

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*It may be noted that Eysenck (1967) has said that his extroversion and introversion concepts are similar to but not identical with those developed by Jung.

of thinking about and perceiving social and interpersonal stimuli. Generally, these stimuli are screened out (rather than in). With an extreme introvert, this process is facilitated by obsessive-compulsive ruminations about facts, figures, and other non-social phenomena. By cluttering his mind in this way, he prevents social thoughts from intruding.

According to Eysenck (1967), introverts are overconditioned. They have a low acquired capacity to screen out stimuli, and they over-respond to social and interpersonal situations. Because of this, they attempt to avoid or control these situations. However, this approach is often impractical and stressful. In addition, by avoiding social contact, the extreme introvert forfeits the possibility of influencing outcomes that affect his welfare.

It is emphasized that social and interpersonal stimuli are stressful for both extroverts and introverts. The former are stressed by these stimuli because their lack of sensitivity exposes them to social backlash and the latter because they have too little experience with them.

Rationale for use of Extroversion/Introversion in Present Model

In the present model, extroversion and introversion are viewed as coping strategies, possibly the most important. This view represents a departure from customary usage in the literature, where extroversion and introversion are simply considered personality variables. Borrowing from the "locus of control" concept as described earlier, extroversion and introversion are recognized as representing ways of thinking about and perceiving social and interpersonal stimuli.

The particular way that a person thinks about and perceives stimuli is, of itself, a way of coping with them. In other words, extroverted and introverted orientations are considered coping processes because they influence the way that a person responds to certain types of stimuli.

Of all the coping theories available, extroversion/introversion is most specifically concerned with cognitive-perceptive factors mediating social and interpersonal stimuli. The other theories are more general, focusing on a variety of psychological, physical, and environmental stimuli. Extroversion/introversion, therefore, is considered the more appropriate model for testing hypotheses relating social and interpersonal stimuli to stress and illness.

One further concept deserves mention because it has been associated with measures of extroversion and introversion. This is the concept of neuroticism (N) (emotionality) defined by Eysenck (1967) as a tendency to be easily and unduly aroused. In this paper, this instability is construed as stress.

With respect to extroversion/introversion and N, the position in this paper is that the former is more crucial to coping and defensive processes. This is because the present model focuses on the influence of extroverted/introverted behavioral tendencies on coping and defense.

Theoretical Causation

It was pointed out in the review that different investigators assembled various traits purported to be causally related to essential hypertension. No rationale was given, for example, as to why anxiety should bear more of a relationship to essential hypertension than hostility. It did not appear that "hypertensive personality" traits

were selected on the basis of a guiding theoretical construct. A causal explanation requires a rationale as to why a particular personality variable is etiologically related to essential hypertension. It is suggested that coping strategies provide a logical place to look for such a variable. These strategies help a person adapt to his environment. If they fail, illness may result. In this way, an aspect of personality, namely coping, may be shown to be related to disease. While other personality variables can be associated with illness, coping may provide the causal explanation.

The causal connection between coping and essential hypertension may be stated as follows: When extroverted/introverted coping strategies effectively monitor social and interpersonal stimuli, an individual is protected from stress that may chronically raise his blood pressure. When these coping strategies are ineffective, there is no protection from stress and blood pressure may become elevated.

Possible Psychological/Essential Hypertension Mechanisms

As stated, a comment on the question of possible mechanisms involved in the interaction of psychological factors and essential hypertension seems appropriate. The psychophysiological model discussed earlier addressed this question. It states that essential hypertension stems from the interaction of neural, humoral, cerebral, and psychological mechanisms. As for the psychological mechanisms, stress is the most important variable and results from social and interpersonal stimulus overload.

Second line transient increase in blood pressure. This second line "coping response" occurs when the first line "coping orientation" fails to protect against increased stress. It denotes a condition where blood pressure fluctuates between elevation and normalcy. Note that the model makes a distinction between coping orientations and coping responses. The former refers to extroverted and introverted inclinations to cognize and perceive stimuli and emphasizes a psychological orientation. The latter concerns transient and chronic increases in blood pressure and is viewed as a physiological response.

Third line chronic increase in blood pressure (essential hypertension). This coping response results when the first and second line processes fail to reduce stress. First and second line failures usually exist for a number of years before this third line response becomes well established. This level represents a chronically elevated blood pressure and is generally more serious than the second line response.

Degenerative cardiovascular and related reactions. Cardiovascular and related pathologies stem from prolonged third line adjustive processes.

Adjustive reactions. This refers to the different levels of psychological (extroversion and introversion) and physiological (elevation of blood pressure) coping processes involved in reacting to social and interpersonal stress stimuli.

The increasing levels of stress line indicates that stress progresses from low to high levels. The progression occurs as various coping levels fail to contain stress.

Stress. This notion is defined as a state of neural/humoral disequilibrium, which may result from (a) a discrepancy between stimulus input and adjustive accommodation, or (b) the absence of adequate or needed stimuli. The former occurs more frequently than the latter.

A certain level of stress is always present. Increased degree and duration of its elevation is what is causally important for essential hypertension.

The broken lines among the model's variables suggest that the interactions are not absolute and equal.

In summary, this section has proposed a theoretical model which is intended to provide a rationale for investigating the causal connections between personality and essential hypertension. It was suggested that causation is theoretically related to those aspects of personality which operate to help an individual adapt to his environment. These are coping orientations. Specific coping strategies, extroversion and introversion, were proposed and a theoretical justification for their use was discussed.

THE EMPIRICAL MODEL

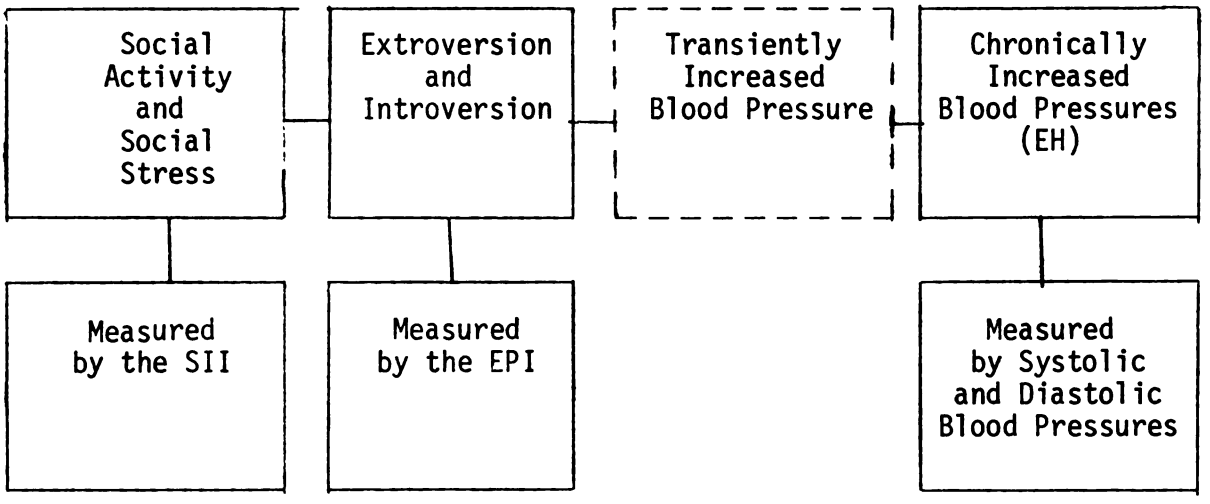

Figure 2

Operational Definitions and Conceptions

Social activity and social stress. Social activity (SA) was defined as a person's actual level of social contact as measured by the Social Interaction Inventory (SII) (Please refer to Appendix C for key to abbreviations). Social stress (SS) was also measured by the SII. The items were related to a person's feelings about his social activity.

Extroversion and introversion. The EPI provides only one score, E, for its extroversion/introversion dimension. This score is often interpreted as indicating degrees of extroversion. As E increases (0 to 24), so does the level of extroversion. An introverted score may be derived from the E value. It is arrived at by subtracting the given E from 24. For example, an E of 5 translates into an introverted score of 19 (24 - 5 = 19). A simpler approach is that an E score of 5 may be interpreted as low extroversion or high introversion. In this study, E, EXT, and extroversion are used interchangeably.

As stated, the experimental hypothesis is that as the capacity to cope decreases, blood pressure increases. A decreased coping capacity is reflected by an extreme (high or low) E score. An extremely high E score was defined as one at or above the 92nd percentile and an extremely low one at the 12th percentile or lower. These figures were provided by Eysenck's (1963) normative results. Because of the brevity of the scale, the distribution of percentile scores was not continuous.

Transiently increased blood pressure. This notion was included here so that the empirical and theoretical models could be easily compared.

Chronically increased blood pressure (Essential Hypertension). The duration of an elevated blood pressure was inferred from medical histories. Blood pressure was measured by the indirect method, using pressure cuff and mercury manometer. Both systolic and diastolic pressures were obtained.

Systolic pressure (SYS) was defined as the pressure reading at the time the first sound appeared, when the blood resumed its flow through the artery following occlusion. The point at which sound was no longer heard from the artery was defined as the diastolic pressure (disappearance of sound).

Essential hypertension. Based on criteria set by the World Health Organization (1962) and previous research studies, essential hypertension in this study was operationally defined as a blood pressure of 145/95 mm Hg or above. Normal pressure was defined as 120/80 mm Hg or below.

Demographic variables. In order to provide a comprehensive model of the factors that affect blood pressure, the following demographic variables were added to the personality factors previously discussed: age, number of children (i.e. child), marital status (MAR), and race. Age was included because it is known to be associated with blood pressure. Marriage and parenthood were involved because they concern important interpersonal interaction and race because of its psychosocial consequences.

Age and number of subject's children were recorded as continuous variables and marital status (i.e., married or not) and race as dichotomous (0 and 1) ones. It may be noted that the discrete entities are of no consequence in this design, since an assumption is that the dependent variable has a normal distribution for the fixed set of independent factors.

Procedure

Essential hypertensive and normotensive subjects formed the basic groups. An objective of this study was to create a model of personality and psychosocial factors that predict level of blood pressure. The details of this aspect of the study are described in a later section. Another goal was to assess a component of the "differential treatment" explanation of higher rates of hypertension for Blacks relative to whites. This involved selecting Black and white patients with hypertension, who were using the same antihypertensive medication for approximately the same length of time, and whose hypertension was essential rather than secondary. The preclusion of secondary causes of hypertension was accomplished in consultation with clinic physicians who, on the basis of reviewing test and other information in patients' charts, confirmed the secondary exclusion.

Since the hypertensive subjects were taking medication, it was considered desirable to select a control group that was also using drugs. It was also intended that the drugs used by the two groups should be comparable in effects (or non-effects) on behaviors measured in this study. The drugs used, hydrochlarothiazide for experimentals and chloral hydrate (a medium strength, 250 mg. sleeping medication prescribed at bedtime) for controls, are considered by physicians to have minimal effects on the behaviors of concern in this investigation. In each group, individuals had been receiving their respective drugs for approximately six weeks prior to this study.

Other than an occasional inability to fall asleep, control subjects had no identifiable illnesses. All were normotensive, approximately the same age and weight as the hypertensives and had the same number of Blacks and whites as the latter.

All subjects were Black and white males between the ages of 20 and 45, had at least a high school education, and were employed in blue and white collar jobs. Females were not used because of the need to control for sex.

Two statistical models were employed, multiple regression and analysis of covariance. The former was used in connection with the theoretical paradigm (figure 1) which suggests that the most important behavioral factors causally associated with blood pressure are personality variables related to coping with social and interpersonal stress. This model was also employed in analyzing the experimental hypothesis that as the capacity to cope decreases, blood pressure increases. Because coping is defined as a moderate rather than a high and a low extroverted and introverted score, a "U" shaped (curvilinear rather than a linear) relationship is predicted. A multiple regression design was used because it is best suited to handle a study that has several independent variables.

As for the analysis of covariance, the test for affects of treatment by race required removing extraneous variation from the dependent variable, posttreatment pressure. In the ANOVA program (SPSS), regression procedures accomplish this removal, and a conventional analysis of variance is subsequently performed on the "corrected" scores.

Description of clinic. All subjects were out-patients at the John Hale Medical Center, a group practice in San Francisco whose staff includes family physicians, internists for continuing primary care, and medical students serving clerkships. A number of the staff physicians are members of the clinical faculty of the University of California, San Francisco, School of Medicine.

Subject selection. Subjects were selected from the active case file and in most instances had been scheduled for clinic visits in November, 1976. On the average, each patient had two pretreatment pressures recorded in his chart which had been taken over several weeks. These were averaged for each subject and provided his before (chart) treatment pressure.

There were 60 subjects, 30 with essential hypertension and 30 normotensives. These were subdivided into four groups of fifteen based on race and pressure: Black hypertensives, Black normotensives, white hypertensives, and white normotensives. For each group, the first 30 charts of patients who met the selection criteria were chosen. Then 15 charts were randomly taken from the 30 selected. Each of the 15 designated patients was contacted by letter and asked to participate in this study. Patients who did not respond to the letter were telephoned or sent a follow-up letter. The response success was about 85 percent. This resulted in having to add two or three patients from the initial pool of thirty. Each subject received ten dollars ($10.00) for participation in the study.

Method

For posttreatment pressures, hypertensive and normotensive patients were randomly scheduled for clinic visits by Judi Dale, R.N., the assisting nurse. Following a brief on site orientation by Ms. Dale and the principal investigator, each subject was led to an examination room and asked to lie supine on an examination table. The subject was requested to lie quietly in this position for fifteen minutes. At the end of this period, the investigator took the first blood pressure (systolic and diastolic). Two additional pressures were recorded at five minute intervals. These three pressures were then averaged. This was done to compensate for possible fluctuations caused by the procedure itself.

Pressures were measured by an indirect method. The brachial artery in a subject's right arm was occluded with a 13 cm pressure cuff which was connected to a mercury manometer. Following the measurements, each subject was escorted to an adjacent room and asked to complete two questionnaires.

Since blood pressure varies during the day, all subjects were seen between 10 a.m. and 2 p.m.

Instruments

Two scales were used. The first, Eysenck Personality Inventory (EPI), measures two orthogonal (independent) dimensions of personality, extroversion/introversion and neuroticism, N (stress). It is the most widely used self-report research instrument for measuring extroversion

and introversion. An additional measuring instrument, Social Interaction Inventory (SII), was devised for this study. It is a two part scale measuring self-report of (1) social and interpersonal interactions, i.e. social activity (SA), and (2) feelings of satisfaction and dissatisfaction about perceived level of SA, i.e. social stress (SS).

Whereas the EPI provides self-report of feelings and attitudes about social and interpersonal interactions on an hypothetical level, the new scale was intended to measure self-report of <u>actual</u> degree of such interaction.

RESULTS AND DISCUSSION

Since multiple variable analysis has many facets, it seems necessary to explain its use in this study. The main purpose of the regression design was to provide empirical support for the basic conceptual model shown in figure 1. This involved an inferential use of multiple regression, where the soundness of the basic model can be judged by analyzing population parameters estimated for the variables stemming from the model. Regression coefficients (b) and multiple R will be discussed in this connection.

Another interest was in getting some idea of how well the independent variables that operationally define the conceptual model predict the dependent variable. This focus required examining r and r^2.

Concerning the main objective, the variables which were generated by the basic model (EXT, SA, NEU, SS) will be discussed prior to those added (age, marriage, children) for reasons previously cited. For convenience of presentation, blood pressure dependent variables will be limited to the systolic.

Of the four model variables (table 1), $F_{1,51}$ was significant ($F = 4.02$, $p < 0.05$) for two, SA and EXT. If the variables had been included in the regression model in an optional hierarchical manner, SS would also have been significant. As it stands, with an $F_{1,51}$ of 3.6, SS was almost significant. This result indicates that SA and EXT are significantly associated with essential hypertension since in the majority of cases systolic pressure and these components rose together. Also, the correlation for these two measures did not exceed 0.164.

The two significant and one nearly significant variables provide good empirical support for the soundness of the theoretical model. Additional support for the model is provided in subsequent discussion.

REGRESSION RESULTS RELEVANT TO THE THEORETICAL MODEL

Variable	B	Beta	Std Error B	F
SA	7.702	0.267	3.682	4.605
EXT	3.050	0.298	1.238	6.062
AGE	1.605	0.206	1.029	2.432
SS	6.793	0.230	3.577	3.607
CHILD	9.767	0.165	7.098	1.893
MAR	4.432	0.046	11.623	0.145
NEU	0.269	0.026	1.468	0.034
(CONSTANT)	11.827			

TABLE 1

None of the added factors were significant at the 0.05 level, though one of these factors, MAR, was not measured precisely in that only a "yes" or "no" answer was permitted which did not consider other living situations.

Before discussing the prediction results, some of the statistics which summarize this data (table 2) may be mentioned. The R square change (RSQ change) shows the numerical contribution of each variable to variance explained.

CUMULATIVE RESULTS OF VARIABLES IN THE PREDICTION EQUATION

Variable	Multiple R	R Square	RSQ Change	Simple R	B	Beta
SA	0.35590	0.12666	0.12666	0.35590	7.90202	0.26731
EXT	0.43627	0.19033	0.06366	0.30141	3.05010	0.29893
AGE	0.48953	0.23964	0.04931	0.20292	1.60550	0.20602
SS	0.52974	0.28062	0.04098	0.22485	6.79321	0.23043
CHILD	0.55398	0.30689	0.02627	0.17269	9.76770	0.16545
MAR	0.55623	0.39339	0.00250	0.03806	4.43273	0.04639
NEU	0.55688	0.31012	0.00045	0.01886	0.26904	0.02628
(CONSTANT)	11.82732					

TABLE 2

The non-partial correlation of each independent variable with the dependent one is shown by the simple R. r^2 and multiple R (multiple correlation coefficient) reveal progressive reduction of the variance as each variable was added to the regression equation. These two statistics have a similiar meaning. The square of multiple R is equal to percentage of explained variance for all of the independent variables in interaction. Stated differently, multiple R square = r^2 = percent variance explained. Finally, the variables in table 2 have been positioned by step-wise regression procedures which list variables in the order of decreasing contribution to variance explained.

Concentrating on multiple R, when squared (0.5568^2), it equals 0.310 and may be used to indicate the percentage of variation explained by a best fitting equation of the form $Y = a + b_1X_1 + b_2X_2 + ---b_kX_k$. Substituting from table 2, the present regression equation is

SYS = 11.8 + .26 SA + .29 EXT + .20 AGE + .23 SS + .16 CHILD + .04 MAR + .02 NEU, where 11.8 is the constant term (a) and .26 et cetera are beta coefficients which show the relative effect of each variable on the dependent variable. However, this effect is not the same as contribution to variance explained, which determined the sequential arrangement of the terms. Multiple R indicates that the correlation coefficient of all the variables in the equation is 0.5568. Multiple R^2 simply states that taken together these variables explain 0.310 percent of the variance associated with the dependent variable. It does not take into consideration other important factors such as the standard error, the combination of the variables, and each variable's contribution to total reduction of variance. In the present case, when the standard error of the equation is considered, MAR and race relative to variables before them increased rather than decreased the error term. The negligible numerical contribution of MAR and race to r^2 is another reason for limiting the present equation to the first five variables cited in the previous equation. In consideration of just these factors, the best prediction equation is SYS = 11.8 + .26 SA + .29 EXT + .20 AGE + .23 SS + .16 CHILD, where five rather than seven variables are involved. Table 3 shows the multiple R for this equation (0.5539) and indicates that it can be tested for level of significance. Significance in one instance is related to rejecting the null hypothesis, H_o:R=0, which is equivalent to the null hypothesis that all k regression coefficients are equal to zero in the population, i.e., $H_o:B_1=B_2=\ldots=B_k=0$.

ANALYSIS OF VARIANCE TEST FOR SIGNIFICANCE OF REGRESSION EQUATION

	Analysis of Variance		
Multiple R = 0.5539 r Square = 0.3068 Adjusted r square = 0.2427 Standard error = 37.0798	Sum of squares of regression= 3287.88, of residual=7424.44 Degrees of freedom of regression=5, of residual=54	Mean square of regression=6574.77, of residual=1374.91	F= 4.781

TABLE 3

The alternative hypothesis, stated in terms of population regression coefficients, is $H_1: B_i \neq 0$ for one or more i. If the null hypothesis is rejected, it may be concluded that one or more of the population regression coefficients has an absolute value greater than zero. For the five term equation, table 3 shows an $F_{5,54}$ of 4.78 which with 5 and 54 degrees of freedom (DF) is significant at the 0.01 level. The null hypothesis is therefore rejected, suggesting that one or more of the independent variables has received a value that closely approximates the true value in the population at large. Consequently that variable(s) has an appreciable effect on the dependent variable. It has been shown that SA and EXT are two such factors. In addition, table 3's significant F may be interpreted as indicating that the values estimated for the independent variables are close to the actual values in the population. Hence, the basic model is in good accord with empirical evidence.

While not independently significant, a variable may be combined with another to make an important impact on the dependent variable. The

cumulative multiple R provides an indication of this. As table 2 shows, multiple R for the first two factors is only 0.43627 but is increased to 0.55398 when the three lesser variables are added.

The percentage of variance explained by a straight line regression is indicated by r and r^2. (Given a multiple regression of Y on several Xs, the regression line is actually a plane in three-dimensional space.) r indicates the degree to which variation (or change) in one variable is related to variation in another. When the linear regression line is a good fit to the data, r will be close to + 1.0 or - 1.0. While high r may indicate a strong linear relationship between two variables, r can be 0 and yet there may be a perfect curvilinear relationship as shown in figure 3. This observation results from the fact that r, being a measure of the goodness of fit of the least-squares straight line, denotes a linear rather than a curvilinear relationship. Usually this limitation is of little consequence since a linear regression line is satisfactory or even preferred in most behavioral studies. However, linearity in the present case does not suffice since curvilinearity is specified by the hypothesis.

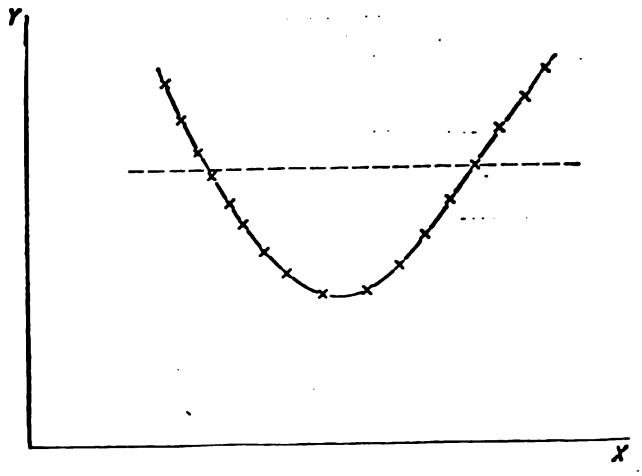

Figure 3. Scattergram of perfect nonlinear relationship for which r = 0. (Reproduced from H.M. Blalock, Social Statistics; McGraw-Hill Book Company, New York, 1972)

The r^2 statistic is considered a measure of the proportion of the total variation in one variable explained by the other. (It ranges from a minimum of 0 to a maximum of 1.0). In some ways r^2 is a better measure of the strength of association and therefore of prediction than is r. This is in part true because, according to Blalock (1972), first while there is no simple and direct interpretation for r, r^2 may be explained as the equivalent of "explained sum of squares", $\sum (Y-\bar{Y})^2$. Second, since r is usually larger than r^2 (table 4), the importance of r as an indicator of strength of association can be misleading. Also r can be unduly influenced by extreme scores. In table 4, $1-r^2$ represents the proportion of unexplained variation corresponding to a given r and r^2, and the quantity $\sqrt{1-r^2}$ represents the square root of the proportion of the total sum of squares that is unexplained by the independent variable. As the table shows, an r^2 of .25 is equivalent to an r of .50. The objective of this discussion is to provide a context in which to gauge the explanatory or predictive strength of the r^2 for the five equation terms. This r^2 of .306

r	r^2	$1-r^2$	$\sqrt{1-r^2}$
.90	.81	.19	.44
.80	.64	.36	.60
.70	.49	.51	.71
.60	.36	.64	.80
.50	.25	.75	.87
.40	.16	.84	.92
.30	.09	.91	.95
.20	.04	.96	.98
.10	.01	.99	.995

Table 4 Numerical relationships among r, r^2, $1-r^2$, and $1-r^2$ (Reproduced from H.M. Blalock, Social Statistics; Mcgraw-Hill Book Company, New York, 1972)

should not be thought of as a .306r, since the former explains as much variance as a .553r. The significance of this r^2 can be judged by comparing it with multiple R. Remembering that the square root of r^2 = multiple R, and since the latter was significant at the 0.01 level (table 3), it may be estimated that the $.306r^2$ has considerable predictive strength.

Shifting the discussion to the present experimental hypothesis, the prediction is that persons with high blood pressure will be found

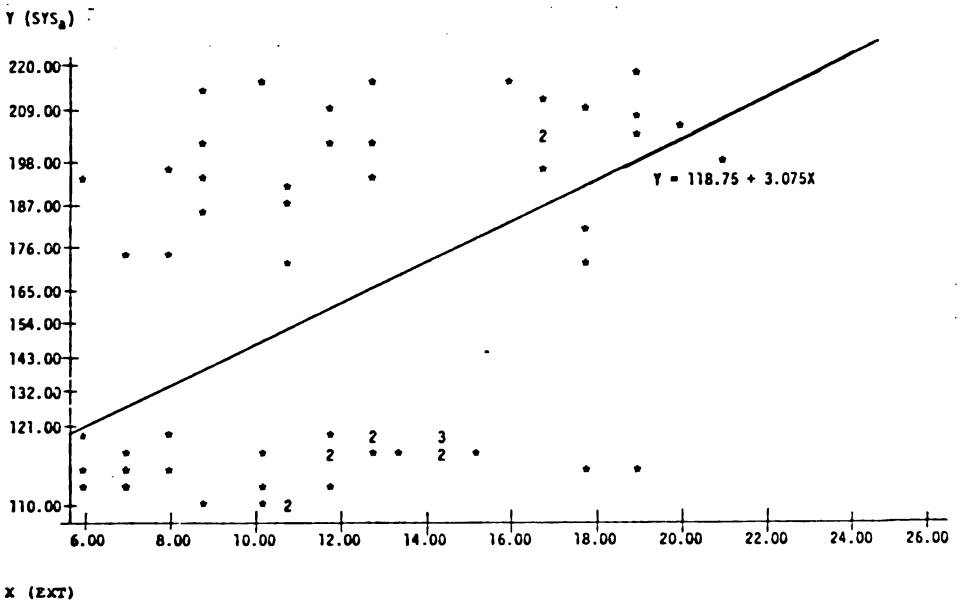

Figure 4 Scattergram of EXT and systolic blood pressure with regression line.

in extremely low E groups to a significantly greater extent than in a moderate one. Stated more succinctly, SYS = (f)E which suggests that

systolic pressure is a predictive function of E. (The ways that the experimental hypothesis have been stated are equivalent.) This predictor concerns one independent variable and can be graphed (figure 4) as E (Y/X) = a + bX, where the notation E (Y/X) emphasizes concern with the expected value of Y which depends on X, a = the intercept, the value of Y at the point where the regression line crosses the Y axis, b = the slope of the line, denoting changes in Y per one unit change in X, and X is the predictor (independent) variable.

The slope (b) of the data in figure 4, according to the principle of lease squares, is equal to r (s_y/s_x) where r = Pearson correlation coefficient of systolic pressure (Y) and E (X), s_y = the standard deviation of Y, and s_x = the standard deviation of X. The intercept (a) is equal to $\bar{Y} - b\bar{X}$. These and other values related to figure 4 are present in table 5.

SUMMARY DATA FOR SYS_a/E REGRESSION

N = 60		r = .301
Mean	$\bar{X}$ = 12.5	$\bar{Y}$ = 157.3
Standard deviation	S_x = 4.17	S_y = 42.6

TABLE 5

As stated, b = r (s_y/s_x) = 3.075 [.301 (42.6/4.17) = .301 x 10.21 = 3.075] and a = $\bar{Y} - b\bar{X}$ = 118.7 (157.3 - 3.075 x 12.5 = 118.8). Hence, the regression equation for the scattergram in figure 4 is Y' = 118.8 + 3.075X. As the figure suggests, the hypothesis was only partially confirmed in that the SYS_a/E relationship was much stronger for

the high E scores than for the low ones. This implies that, as expected, systolic pressures for the high E scores were significantly higher than for the moderate ones, and that contrary to prediction, systolic pressures for the low E scores were not significantly higher than those for the moderate ones.

Another, perhaps more graphic way of analyzing the data in relation to the hypothesis is to look at the distribution of pressures corresponding to E scores grouped into high, moderate, and low categories based on Eysenck's (1963) normative data. Table 6 shows that the high group consisted of the four highest E scores, 21-18, the middle groups of the moderate scores, 17-10, and the low group of the four lowest scores, 9-6. As was suggested by figure 4, the mean systolic pressure for the high E group was significantly higher ($Z_T = 2.19$, $p < 0.05$) than that for the moderate one. For the low and moderate E groups, mean systolic pressures did not significantly differ.

The prediction failure for the subjects at the low extreme may be partially explained by observing that the hypothesis predicts blood pressure for persons with extreme extroverted and extreme introverted profiles. As pointed out in the empirical section, the EPI actually assesses degrees of extroversion rather than measuring extroversion and introversion in a bipolar fashion. The EPI suggests that persons with low extroversion scores are equivalent to or no different from persons with high introversion values. This assumption may not always be warranted, and the EPI's introvert, being a type of extrovert, may be unique among introverts. A possible consequence of this is that the EPT did not allow an adequate test of the present hypothesis as it relates to introverts in general. Even so, the EPI was the most suitable instrument available.

HIGH, MODERATE, AND LOW GROUPS BASED ON E SCORES

High Group (21 - 18)				Moderate Group (17 - 10)				Low Group (9 - 6)			
E	SYS	Age	Race	E	SYS	Age	Race	E	SYS	Age	Race
18	173	41	B	17	212	40	B	9	201	35	W
18	179	21	W	17	203	27	B	9	214	39	B
18	115	36	W	17	204	32	W	9	184	25	W
18	210	37	B	17	197	25	W	9	195	45	W
19	115	24	B	16	217	40	B	9	112	30	W
19	218	36	B	16	118	36	B	8	196	28	W
19	208	33	W	15	119	34	B	8	175	31	W
19	204	36	B	15	118	30	B	8	116	27	B
20	205	25	B	15	117	28	W	8	119	34	B
21	198	22	W	15	120	34	W	7	116	36	W
				15	120	29	W	7	175	34	B
				14	118	25	W	7	114	29	B
				13	216	34	B	7	118	36	W
				13	202	38	W	6	195	27	B
				13	117	31	B	6	114	34	B
				13	119	31	W	6	120	35	B
				13	119	37	B	6	115	29	W
				13	194	40	W				
				12	117	33	B				
				12	113	39	W				
				12	209	40	B				
				12	120	36	B				
				12	118	32	W				
				12	201	29	W				
				11	172	39	B				
				11	110	38	B				
				11	192	36	W				
				11	188	29	W				
				11	110	26	W				
				10	113	28	W				
				10	110	32	W				
				10	117	24	B				
				10	216	41	B				

Summary Table

n = 10	n = 33	n=17
Race	Race	Race
6 B	16 B	8 B
4 W	17 W	9 W
$\bar{X}$ SYS = 182.5	$\bar{X}$ SYS = 152.6	$\bar{X}$ SYS = 151.8
$\bar{X}$ Age = 31.1	$\bar{X}$ Age = 33.1	$\bar{X}$ Age = 32.5

TABLE 6 N = 60

On a positive note, table 6 shows that all E groups had approximately the same number of Black and white subjects. This situation is encouraging since some of the most widely used instruments such as the MMPI and the Holmes et al (1967) Life Events Scale have been criticized as yielding racially differentiated scores. It may also be noted that the mean ages of the groups in table 6 do not significantly differ so that this variable does not explain the findings. This point is made in connection with the observation that in the population at large, blood pressure and age tend to rise concomitantly.

Finally, an analysis of covariance was done on the racial subgroups. A variation of the differential drug treatment hypothesis suggests that the greater prevalence of essential hypertension among Blacks relative to whites results from poorer medical compliance in the former race compared to the latter. The design for testing this idea was SYS_a (pretreatment systolic pressure) = covariate of SYS_b (posttreatment pressure), race = independent variable regressed against "corrected" dependent variable, SYS_b. Control subjects provided a comparison group. The two questions relevant to the hypothesis are (1) is there a significant association between the pre- and posttreatments, and (2) is race significantly related to treatment effect. Referring to the first question, table 7 shows a covariate (SYS_a) $F_{1,29}$ of 6.624, suggesting a significant (p 0.01) association (change) between the before treatment (SYS_a) and after treatment (SYS_b) blood pressures. For the second question, the table shows that the $F_{1,29}$ for the effect of race is 0.02 and not significant at the 0.05 level. These findings are also evident in that of the total sum of squares variation explained (1487.7), SYS_a accounted for 1483.4 units and race for only 4.48. Moreover, the $F_{2,27}$

for the total sum of squares explained (r^2) was significant (F = 3.32, $p < 0.05$) so that the variance explained is significant.

ANALYSIS OF COVARIANCE WHERE TREATMENT EFFECT IS REGRESSED WITH RACE FOR EXPERIMENTAL SUBJECTS

Source of Variation	Sum of Squares	DF	Mean Square	F	Significance of F
Covariates	1483.228	1	1483.228	6.624	0.015
SYSA	1483.228	1	1483.228	6.624	0.015
Main Effects	4.485	1	4.485	0.020	0.999
Race	4.485	1	4.485	0.020	0.999
Explained	1487.715	2	743.857	3.322	0.050
Residual	6045.738	27	223.916		
Total	7533.453	29	259.774		

TABLE 7

Results did show that the mean pretreatment systolic blood pressure for Black hypertensives was 202.6 mm Hg compared to 194.2 mm Hg for whites; this difference was not significant at the 0.05 level ($Z_T = 1.82$).

By comparison, table 8 shows that control subjects had a covariate $F_{1,29}$ of 0.44 which was not significant at the 0.05 level. This suggests that as predicted, chloral hydrate has no significant effect on the behaviors measured in this study. Though not significant, race was a bigger factor in this group relative to the hypertensive one. This is shown both in the sum of squares (364.0 for race and 124.9 for SYS_a) and $Fs_{2,27}$ (1.288 for race and 0.442 for SYS_a).

The four subgroups were analyzed for differences, and no significant ones were found.

ANALYSIS OF COVARIANCE WHERE TREATMENT EFFECT IS REGRESSED WITH RACE FOR CONTROL SUBJECTS

Source of Variation	Sum of Squares	DF	Mean Square	F	Significance of F
Covariates	124.958	1	124.958	0.442	0.999
SYSA	124.958	1	124.958	0.442	0.999
Main Effects	364.077	1	364.077	1.288	0.266
Race	364.077	1	364.077	1.288	0.266
Explained	489.035	2	244.518	0.865	0.999
Residual	7632.411	27	282.683		
Total	8121.477	29	280.051		

TABLE 8

In conclusion, while the data could have been stronger and more definitive, the larger objective of providing a degree of empirical evidence for the theoretical model appears to have been satisfactorily accomplished.

The regression model was shown to identify some of the psychological and psychosocial factors that affect blood pressure variability.

As expected, E was a key variable in the prediction equation and in the experimental hypothesis. In the former case, E made an important contribution to the variance explained, and in the latter, it verified the hypothesis for the high group. However, since E's relation to blood pressure was linear rather than curvilinear as predicted, the hypothesis

was not confirmed for the low group. A possible explanation for the outcome of the low group involved the suggestion that introversion was not measured as the equivalent of extroversion so that E was actually a scaled extroversion.

An unique feature of this study was that it made extensive comparisons between Black and white essential hypertensives and Black and white normotensives. No significant personality differences as measured by the Eysenck Personality Inventory were found. This result is inconsistent with the hypothesis that personality factors in Blacks not shared by whites are somehow related to increased prevalence of essential hypertension in the Black community.

Similarly, the analysis of covariance finding (table 7) of no significant Black/white difference in systolic blood pressure between pre- and posttreatments is also contrary to the proposition that a higher incidence of essential hypertension in Blacks relative to whites is related to the Black's lack of compliance with a hypertensive treatment regimen.

Finally, it is felt that the results can be generalized to Black and white males who are in the middle to lower socioeconomic stratum.

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Appendix A

SOCIAL INTERACTION INVENTORY

DIRECTIONS. Please note that each question has two parts. For example, question 1 is divided into 1A and 1B. Please check one answer for each part of the question. It is realized that not all questions will fit your situation exactly. In this case please check the answer that is most nearly true for you.

Please check one for each question.

1A. How many times a week on the average do you visit friends or relatives or have them visit you?
_____ less than once a week.
_____ more than once a week.

1B. Are you satisfied with the above situation?
_____ No.
_____ Yes.

2A. When you go out, do you
_____ most always
_____ hardly ever
go to places where there are lots of people?

2B. Are you satisfied with the above situation?
_____ Yes.
_____ No.

3A. How much of your leisure time or free time do you spend alone?
_____ almost all of it.
_____ hardly any of if.

3B. Are you satisfied with the above situation?
_____ No.
_____ Yes.

4A. How many times a week on the average do you go to parties or other social functions?
_____ more than once a week.
_____ less than once a week.

4B. Are you satisfied with the above situation?
_____ Yes.
_____ No.

5A. At work, do you
_____ hardly ever
_____ often
have to meet and talk to other people?

5B. Are you satisfied with the above situation?
_____ Yes.
_____ No.

6A. At work, do you usually eat your lunch
_____ by yourself?
_____ with 1 or more other people?

6B. Are you satisfied with the above situation?
_____ No.
_____ Yes.

7A. At work, how many other people do you work with?
_____ none.
_____ 1 or more.

7B. Are you satisfied with the above situation?
_____ Yes.
_____ No.

8A. At work, do you
_____ often
_____ hardly ever
have to meet and talk with your supervisor or foreman?

8B. Are you satisfied with the above situation?
_____ No.
_____ Yes.

Appendix B

RAW DATA

CASE-N	EXT	NEU	SA	SS	RACE	AGE	MAR	CHILD	SYSA	SYSB
1	17.	14.	3.	4.	1.	40.	1.	2.	212.	160.
2	9.	5.	5.	3.	1.	39.	0.	0.	214.	155.
3	13.	6.	5.	5.	1.	34.	0.	0.	216.	163.
4	7.	10.	2.	3.	1.	34.	0.	0.	175.	115.
5	20.	16.	6.	3.	1.	25.	0.	0.	205.	160.
6	19.	9.	4.	4.	1.	36.	0.	0.	218.	152.
7	18.	8.	2.	1.	1.	41.	0.	1.	173.	158.
8	17.	16.	3.	3.	1.	27.	0.	2.	203.	130.
9	12.	8.	2.	3.	1.	40.	1.	0.	209.	118.
10	6.	15.	0.	5.	1.	27.	1.	0.	195.	155.
11	19.	3.	3.	3.	1.	36.	1.	0.	204.	161.
12	16.	12.	4.	4.	1.	40.	0.	0.	217.	158.
13	10.	8.	5.	1.	1.	41.	0.	3.	216.	155.
14	11.	5.	3.	3.	1.	39.	0.	2.	172.	156.
15	18.	11.	2.	6.	1.	37.	1.	0.	210.	139.
16	13.	12.	6.	7.	0.	38.	1.	1.	202.	154.
17	8.	15.	4.	3.	0.	28.	0.	0.	196.	145.
18	21.	23.	2.	5.	0.	22.	0.	0.	198.	159.
19	17.	18.	3.	6.	0.	32.	0.	0.	204.	157.
20	19.	18.	5.	2.	0.	33.	0.	0.	208.	160.
21	17.	16.	4.	1.	0.	25.	1.	0.	197.	154.
22	9.	11.	2.	3.	0.	25.	0.	1.	184.	150.
23	9.	6.	2.	3.	0.	45.	0.	0.	195.	148.
24	12.	8.	3.	3.	0.	29.	0.	2.	201	156.
25	11.	9.	3.	5.	0.	36.	0.	2.	192.	120.
26	9.	18.	4.	6.	0.	35.	0.	0.	201.	155.
27	8.	16.	1.	3.	0.	31.	0.	0.	175.	115.
28	11.	17.	3.	2.	0.	29.	1.	1.	188.	118.
29	18.	15.	5.	3.	0.	21.	0.	0.	179.	120.
30	13.	18.	4.	3.	0.	40.	0.	0.	194.	148.

CASE-N	EXT	NEU	SA	SS	RACE	AGE	MAR	CHILD	SYSA	SYSB
31	15.	13.	1.	1.	1.	34.	0.	0.	119.	120.
32	19.	9.	3.	1.	1.	24.	0.	0.	115.	114.
33	11.	8.	1.	4.	1.	38.	0.	0.	110.	152.
34	12.	10.	4.	4.	1.	36.	0.	0.	120.	108.
35	12.	16.	3.	3.	1.	33.	1.	1.	117.	148.
36	13.	10.	2.	3.	1.	31.	0.	0.	117.	110.
37	15.	9.	0.	1.	1.	30.	0.	1.	118.	116.
38	6.	13.	4.	5.	1.	35.	0.	0.	120.	110.
39	13.	16.	2.	1.	1.	37.	1.	0.	119.	115.
40	6.	10.	3.	4.	1.	34.	0.	0.	114.	113.
41	7.	15.	3.	3.	1.	29.	0.	0.	114.	112.
42	8.	14.	5.	2.	1.	34.	0.	0.	119.	104.
43	8.	8.	0.	3.	1.	27.	1.	0.	116.	108.
44	16.	12.	1.	3.	1.	36.	1.	0.	118.	110.
45	10.	11.	5.	5.	1.	24.	0.	1.	117.	110.
46	14.	11.	2.	3.	0.	25.	1.	1.	118.	134.
47	12.	12.	2.	2.	0.	32.	0.	0.	118.	108.
48	12.	11.	4.	4.	0.	39.	0.	0.	113.	148.
49	13.	14.	4.	3.	0.	31.	0.	0.	119.	110.
50	11.	17.	3.	6.	0.	26.	0.	0.	110.	134.
51	15.	11.	3.	2.	0.	28.	0.	1.	117.	120.
52	15.	15.	2.	1.	0.	34.	1.	1.	120.	152.
53	9.	14.	1.	3.	0.	30.	0.	1.	112.	100.
54	7.	5.	1.	4.	0.	36.	0.	0.	116.	151.
55	6.	18.	4.	3.	0.	29.	1.	0.	115.	108.
56	7.	10.	2.	4.	0.	36.	0.	0.	118.	108.
57	18.	10.	2.	3.	0.	36.	0.	0.	115.	145.
58	15.	14.	3.	3.	0.	29.	1.	1.	120.	132.
59	10.	12.	2.	4.	0.	32.	0.	1.	110.	105.
60	10.	19.	3.	1.	0.	28.	0.	0.	113.	110.

1 - 15 - Black hypertensives
16 - 30 - white hypertensives
31 - 45 - Black normotensive
46 - 60 - white normotensive

Appendix C

Table 9

ABBREVIATIONS USED IN TEXT

B	Black Subject
BHG	Black hypertensive group
BNG	Black normotensive group
CHILD	Number of children in family
DIA	Diastolic blood pressure
E/EXT	Extroversion
MAR	Marital status
N/NEU	Neuroticism
SA	Social activity
SII	Social Interaction inventory
SPSS	Statistical package for the behavioral sciences
SYS_a	Before treatment or chart systolic blood pressure
SYS_b	After treatment or experimental systolic blood pressure
SS	Social stress
SYS	Systolic blood pressure
W	White subject
WHG	White hypertensive group
WNG	White normotensive group