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Blood Pressure Reactivity and Emotion Among Borderline Hypertensives, Mild Hypertensives, Diabetic Hypertensives, and Normotensives.

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Blood pressure reactivity and emotion among borderline hypertensives, mild hypertensives, diabetic hypertensives, and normotensives

Pellegrin, Alicia G., Ph.D.
The Louisiana State University and Agricultural and Mechanical Col., 1994
BLOOD PRESSURE REACTIVITY AND EMOTION AMONG BORDERLINE HYPERTENSIVES, MILD HYPERTENSIVES, DIABETIC HYPERTENSIVES, AND NORMOTENSIVES

A Dissertation

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy in The Department of Psychology

by

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Abstract

The purpose of this study was to examine the relationship between emotion and blood pressure levels and variability for 80 Caucasian subjects between the ages of 30 and 65 matched on gender. The subjects were divided into four groups: normotensives, untreated borderline essential hypertensives, treated mild essential hypertensives, and Type II diabetic hypertensives. Subjects completed five psychological measures: the Beck Depression Inventory (BDI); State-Trait Anxiety Inventory-trait anxiety (STAI-Tanx); State-Trait Anger Expression Inventory-trait anger (STAXI-Tang); the ratio of anger-in to anger-out (STAXI-IO); and the Autonomic Nervous System Response Inventory (ANSRI). Subjects then recorded their home blood pressures seven times throughout the day for a three day period with the use of portable blood pressure monitors.

Analysis showed that the four groups differed significantly in their overall scores on the five measures combined. However, further analysis found no significant differences between the groups when the measures were examined individually. Similarly, differences were found between Type II Diabetic Hypertensives and Essential Hypertensives on the five measures combined, but no differences were found when the measures were individually analyzed.
Differences were also found between home and office blood pressure readings, with home readings found to be uniformly lower, with exception of the borderline essential hypertensive group which had higher systolic readings at home. No correlations were found between the five psychological measures and blood pressure readings taken at home, or between scores on the psychological measures and the differences between home and office readings.

Further research is needed to delineate the relationship between hypertension and emotional factors.
Introduction

Epidemiologic research has shown that hypertension affects one out of every three Americans (estimated at over 60 million Blanchard, et al. 1988) and has been consistently identified as a major risk factor for heart disease, cerebrovascular disease, retinal damage, and kidney insufficiency (Castelli, 1984; Kannel, Schwartz, & McNamara, 1969; Kannel, 1976). Longitudinal studies (Rosenman, Brand, Jenkins, Friedman, Straus, & Wurm, 1975; Kannell, 1976; Veterans Administration 1970) indicate that increased risk for heart disease is from two to four times greater in hypertensive individuals than in those with normal readings.

Hypertension can be very damaging because of two primary effects. The first effect is the increased work load on the heart which causes the cardiac muscle to hypertrophy. This is the result of the very high pressure against which the left ventricle must beat causing it to increase in weight as much as two to three times. Because this increase is not accompanied by as much of an increase in coronary blood supply, relative ischemia of the left ventricle develops as hypertension becomes more severe.

The second effect is the damage to the arteries themselves caused by excessive pressure. High pressure in the arteries causes coronary sclerosis as well as sclerosis of the blood vessels throughout the body. The arteriosclerosis process causes blood clots to develop in the vessels and also
causes the vessels to weaken, leading to thrombosis or rupture.

It is estimated that only a little more than 50% of hypertensives are aware of their condition and that the number of hypertensives whose blood pressure is adequately controlled is only about 34% (Blanchard et al., 1988).


Definition

Simply stated, blood pressure (BP) is the pressure exerted by the blood on the vessel walls. Typically, blood pressure is measured at the level of the arteries and hence reflects arterial blood pressure. Blood pressure varies with
each heart beat, peaking during ventricular systole and reaching its minimum during late ventricular diastole.

Arterial pressure (AP) is the product of cardiac output (CO) and total peripheral resistance (TPR). Stroke volume, the volume of blood ejected from the ventricle with each beat, is determined by end diastolic volume and the strength of ventricular contraction. CO, in turn, the product of heart rate and stroke volume, is determined by the fluid volume and the pumping action of the heart. TPR consists of all the vascular factors which resists the flow of blood through the vascular system (Guyton, 1987). Resistance, which cannot be measured be any direct means, is calculated by dividing the pressure difference by the flow. The relationship between the various hemodynamic properties is best demonstrated in the equation \( P = CO \times TPR \). Clearly, changes in one of the properties significantly impacts the other properties.

Under the conditions of constant TPR, blood flow varies directly as blood pressure varies. However, TPR rarely remains constant, so that TPR plays a significant role in determining blood flow. The biggest single factor in determining vascular resistance to blood flow is the diameter of the vessels. More specifically, increasing vessel diameter reduces resistance to blood flow, while decreasing vessel diameter increases resistance to blood flow. This principle provides the driving force behind the regulation of local tissue blood flow. Blood flow and blood flow variability can
be caused by either increased or decreased sympathetic nervous stimulation of the peripheral blood vessels, specifically the arterioles. Inhibition of sympathetic stimulation greatly dilates the vessels and greatly increases blood flow. Conversely, very strong sympathetic stimulation can constrict the vessel so much that blood flow can sometimes be decreased to as low as zero despite high arterial pressure.

It is evident that sympathetic stimulation, with its concomitant increase in smooth muscle tone in the vascular walls increases vascular resistance, while sympathetic inhibition decreases vascular resistance. Specifically, sympathetic stimulation causes a secretion of the catecholamine norepinephrine, from the terminus of the sympathetic nerves. Norepinephrine acts directly on the "alpha" receptors of the vascular smooth muscle causing vasoconstriction, reduction in diameter, increased resistance to blood flow, and blood flow. In the absence of norepinephrine, the reverse occurs, allowing for an increase in blood flow. Under normal conditions this control mechanism allows the body to redistribute its blood flow to meet changing physiological demands such as those imposed by exercise, increasing ambient temperature, and changes in body position.

When regulation of vascular diameter becomes defective or inappropriate, cardiovascular function can ultimately be
compromised. Hypertension is a situation in which an individual has a blood pressure higher than that judged to be normal. Most frequently is results from vasoconstriction or narrowing of peripheral blood vessels, i.e. increased TPR. In order to maintain adequate blood flow, the cardiovascular system responds by raising blood pressure. This in turn increases the work load on the heart. If untreated, hypertension and the chronic elevation in work output by the heart can result in serious pathologies including cardiac, renal, and vascular damage, all of which can lead to death.

At one time it was thought that the cardiovascular sequelae of hypertension derived chiefly from the diastolic component. However, epidemiological research suggests that systolic pressure also may play an important role in increased cardiovascular mortality (Kannel, Dawber, & McGee, 1980).

The standard unit of measurement for blood pressure is mmHg (millimeters of mercury) (Hassett, 1978; Guyton, 1987). Traditionally, casual blood pressure measurements (measurements taken in a clinic or doctor's office) have been taken with the use of a mercury sphygmomanometer. The systolic blood pressure (SBP) reading is taken when the inflatable cuff first interferes with blood flow. Diastolic blood pressure (DBP) is measured when the pressure in the cuff no longer interferes with the flow of blood. This technique consistently underestimates both SBP and DBP by
about 10 mmHg and is easily affected by beat to beat differences in BP (Hassett, 1970).

There are still no uniform criterion values for the diagnosis of hypertension, often making comparisons of empirical findings difficult (Linden, 1984; Guyton 1987; The World Health Organization 1959). Most researchers recognize that deciding which values constitute hypertension is somewhat arbitrary (Linden, 1984). The most frequently cited criterion values for borderline hypertension range from 140/90 to 160/100. Borderline hypertensives may exhibit occasional readings in the normal range with no evidence of target organ damage. By contrast, sustained hypertensives do not exhibit readings in the normal range, and have readings ranging from mild (140/90) to severe (diastolic pressure greater than 105) (Julius, 1977; Obrist, Grignolo, Hastrup, Keopke, Langer, Light, McCubbin, Pollak, 1983).

Blood pressure can show marked changes with postural position, (Bevan, Honour, & Scott, 1969) time of day, (Agras, Taylor, Kraemer, Allen, & Schneider, 1980), various environmental and behavioral factors (Laughlin, Sherrard, & Fisher, 1980), daily activities (Schneider & Costiloe, 1975), and location (Harshfield, Pickering, Kleinert, Blank, & Laragh, 1982). For these reasons no firm conclusion about an individual's true blood pressure can be made from a single reading (Hassett, 1978; Tursky, 1974). Another factor making a correct diagnosis of hypertension more difficult is the
"office hypertension" phenomenon. For many years clinicians have observed that some individuals show elevated blood pressure in the physician's office, but not in other settings (Kaplan, 1986).

Adding to the difficulty of correctly diagnosing hypertension is the fact that proper equipment and measuring technique must be used. For example, it has been determined by the American Heart Association (Frohlich, Grim, Labarthe, Maxwell, Perloff & Weidman, 1993) that an individual's blood pressure is best taken in a quiet room at a comfortable temperature after the individual has rested for five minutes. Ideally the person should not have eaten or smoked for thirty minutes prior to the measurement. Additionally, correct cuff size must be of the appropriate width for the individuals' arm circumference. If the bladder is too wide, the pressure will be underestimated; if the bladder is too narrow, the pressure will be overestimated. The correct ratio of bladder to arm circumference is 0.4, which means the bladder width should be 40-50% of the upper arm circumference. Length to width ratios should be 2:1, thus insuring that if the bladder width is 40% of arm circumference, the bladder length will encircle 80% of the arm. It is also recommended that the large cuff (15 cm) be used for all adults except those with thin arms that are out of the cuff range (Frohlich, et.al; Iyriboz & Hearon, 1992).
As many as 2/3 of patients who display borderline hypertension in the clinic, may actually be false hypertensives because their blood pressure returns to normal levels when taken in the natural environment (Julius, Ellis, Pascual, Matice, Hansson, Hunyour, & Sandler, 1974; Pickering, Harshfield, Kleiner, Blank, & Laragh, 1982; Laughlin, Sherrard and Fisher, 1980; & Schneider, Egan, Johnson, Drobny, and Julius, 1986). This fact underscores the importance of utilizing ambulatory blood pressure monitoring in the natural environment in order to establish a definitive diagnosis (Pickering, Harshfield, Kleiner, Blank, & Laragh, 1982; James, Yee, Harshfield, Blank, and Pickering, 1986).

Ninety per cent of all cases of high blood pressure have no known cause and are referred to as essential hypertension (EH) (Gutmann & Benson, 1971). The remaining 10% have hypertension as the direct or indirect effect of another disease, including diabetes. These individuals are known as secondary hypertensives. Most studies in the literature combine diabetic hypertensives with other individuals whose hypertension is secondary to another disease. However, a distinction can be made since hypertension secondary to such diseases as kidney dysfunction, cerebral disease, coarctation of the aorta, or other physiological malfunctions is directly caused by these disease (Gutmann & Benson, 1971; Eyer, 1975; Linden, 1984), whereas hypertension in diabetics is only greatly influenced by the disease and not a direct result.
Almost without exception, the studies which have examined the role of psychological factors in the etiology and maintenance of hypertension have concentrated on essential hypertension, with no attention paid to the role of these factors in secondary hypertension. The reason for this is not immediately apparent, although possibly the prevailing assumption is that because secondary hypertension is due to another disease process, psychological factors do not play a role in either its development or maintenance. There is a true need for research in this area.

**Borderline Essential Hypertension**

Borderline essential hypertension which is fairly common among younger individuals, is often thought to mark an early stage in the development of established high blood pressure (Julius & Schork, 1970). Borderline hypertensives have a greater chance of developing sustained essential hypertension than do normals, and the mortality and cardiovascular morbidity among subjects with borderline hypertension significantly exceeds the rates found in normotensives (Julius & Schork, 1970). The borderline phase of essential hypertension is characterized by increased cardiac output and normal peripheral resistance (Krantz, Baum, & Singer, 1983). If the disease progresses, the borderline phase gives way to a sustained phase of essential hypertension, characterized by normal cardiac output and elevated peripheral resistance. This progression appears to be positively correlated with age.
(Lund-Johanssen, 1977), and can be linked to any one or combination of the processes discussed below (Linden, 1984; Guyton, 1987; Obrist et al., 1981; Julius & Schork, 1971; Lund-Johanssen, 1977). An advantage of studying borderline hypertensives is that the structural changes found in later years among sustained hypertensives has not yet occurred.
Brief Overview of Physiological Mechanisms in Hypertension

Though the linear mechanical model (AP = CO x TPR) is generally accurate to a basic understanding of the blood pressure system, it is oversimplified since its component parts are not independent (Blanchard et al., 1988; Guyton, 1980). Arterial pressure is regulated not by any one single controlling system but by several interrelated systems which perform specific functions (Guyton, 1987). An overview of these systems and the role that they play in the etiology of hypertension follows.

Psychophysiological Response Model

The effects of stress-induced sympathetic nervous system (SNS) over-activity provide the conceptual paradigm for psychological and psychophysiological research in essential hypertension (Krantz, Baum, & Singer, 1983; Linden, 1984). As will be discussed in the next section SNS over-activity is not adequate by itself to account for chronic, sustained hypertension, however, it may be an important component in the process (Rosenman & Ward, 1988). SNS over-activity can be brought about by a variety of environmental stimuli including discriminative stimuli for objective physical danger, or the classically conditioned psychological or subjective interpretation of alarming environmental signals (Linden, 1984). These threatening environmental stimuli cause a discharge of the sympathetic nervous system and increase catecholamine production, triggering a heightened
cardiovascular response, including increased CO and TPR (DeQuattro and Miura, 1973). This response represents the circulatory component of emotional behavior, preparing the cardiovascular system for an enhanced defensive readiness (fight-flight) (Cannon, 1929; Linden, 1984). If this alarm response is maintained over time, the individual is said to experience stress (Selye, 1956) which may ultimately lead to the various pathology associated with chronically elevated blood pressure.

A number of brain areas, including the hypothalamus, are responsible for the transformation of these environmental influences into cardiovascular responses (Linden, 1984). Folkow and his co-workers (Folkow, Heymans, Neil, 1965; Folkow and Rubinstein, 1966) demonstrated that stimulation of certain areas within the hypothalamus and midbrain produces elevated arterial pressure and stimulation of the cardiac muscle. This results in complex changes in sympathetically driven cardiovascular processes including elevated heart rate, cardiac output and stroke volume, vessel constriction, and, ultimately, increased blood pressure (Linden, 1984). Prolonged elevated blood pressure can lead to long-term changes in blood vessels and arteries discussed in more detail below.

A large body of research links a variety of psychological and physical stressors induced in the laboratory to acute cardiovascular responses such as larger
blood pressure variability and slower recovery to baseline in some hypertensive individuals relative to normotensive controls (Elliot, Buell, & Dembrowki, 1982; Herd, 1983; Krantz, Baum & Singer, 1983; Steptoe, Melville & Ross, 1984; Linden, 1984; Fredridson, Danielssons, Engel, Frisk-Holberg, Strom & Sundin; Jorgensen and Houston, 1986; Weidner, Freiend, Ficarrotto, & Mendell, 1989). Research has also shown that heightened reactivity may have a familial link since normotensive offspring of hypertensive parents are more reactive than individuals without family histories of EH (Jorgensen and Houston, 1981; Rose & Chesney, 1986). However, in a review of the research on this issue, Rosenman and Ward (1988) concluded that much of the research on laboratory stressors is questionable due to methodological problems, and these authors question whether reactivity in the laboratory either predicts hypertension or accounts for differences of blood pressure variability in the natural environment.

In keeping with the autonomic mechanisms model, Obrist and his colleagues (Obrist, 1981; Obrist, Grignolo, Hastrup, Koepke, Langer, Light, McCubbin, & Pollak, 1983) suggest that behavioral challenges that evoke active coping and involve autonomic mechanisms (catecholamine release onto beta adrenergic receptors leading to elevated cardiac output and normal peripheral resistance) promote elevated resistance in the peripheral vasculature over time. These increases in peripheral resistance may occur either through structural
changes in the arterioles (hypertrophy) which cause a narrowing in these resistive vessels and elevation of the peripheral resistance, or as the result of intrinsic homeostatic processes which act to prevent an over-perfusion of body tissues (autoregulation). Thus, it is proposed that the development of an established hypertensive state is achieved largely through autoregulatory processes, which are in turn triggered by increased cardiac output which accompanies excessive cardiovascular reactions to behavioral events.

Both the structural and autoregulatory mechanisms are intrinsic effects not mediated by neurohumoral mechanisms, but involve local control of the blood flow by the arterioles. Peripheral resistance is triggered by CO elevations which flood the tissues with more blood than is necessary. To bring the flow back to appropriate levels, the arterioles begin to constrict, which raises the peripheral resistance. Reduction in blood flow dams up the blood in the upstream arteries and also reduces the venous return of blood to the heart. Thus, not only is there an increase in peripheral resistance, but also a reduction in the CO since the heart has less blood to pump.

Obrist (1981) suggests that the structural and autoregulatory mechanisms may be complementary. Autoregulation would be the most immediate effect to an elevated output. The structural changes in resistance vessels

Despite the evidence for the role of SNS over-activity in the etiology of essential hypertension, it appears that the disorder is rarely due to increases in SNS activity alone. The human blood pressure regulatory system consists of a number of checks and balances which attenuate and neutralize the effects of heightened arousal (Guyton, 1987). In the normal individual, there are several buffering mechanisms which detect short-term pressure changes and attempt to bring the system back into equilibrium (Blanchard et al., 1988). The two buffering mechanisms which have received the most attention in this respect are the renin-angiotensin and baroreceptor systems.

**Renin-Angiotensin System**

The renin-angiotensin system is a buffering mechanism which is extremely important in the regulation of blood pressure. It is largely responsible for increasing blood pressure that has fallen too much. When low pressures are detected, renin, a proteolytic enzyme is released by the kidneys. The renin, converts angiotensinogen into angiotensin I. Angiotensin converting enzyme converts Angiotensin I into Angiotensin II. Angiotensin II has a number of important
effects on the circulation related to arterial pressure control, but most importantly, it constricts vessels everywhere in the body resulting in increased systemic blood pressure. Importantly, this response chain may also be invoked through sympathetic arousal.

Since the sympathetic nervous system may modulate renin secretion by the kidney, it has been suggested that high plasma-renin activity may serve as one indicator of borderline hypertensive states that involve a more general sympathetic arousal (Manuck, Morrison, Bellack, & Polefrone, 1987). In a study of mild essential hypertensive patients differing in renin status, Esler, Julius, Zweifler, Randall, Harburg, Gardiner, and DeQuattro (1977) report that compared to normal plasma renin hypertensives, high renin hypertensives displayed higher concentrations of resting plasma norepinephrine and greater decreases in cardiac output and total peripheral resistance following pharmacologic blockade of the autonomic nervous system. The reliable differences obtained between the two groups suggests that characteristics distinguishing the high renin hypertensive condition were distinct from the consequences of the hypertension itself. Thus, the authors concluded that mild essential hypertension, when accompanied by elevated plasma renin activity, is maintained by autonomic mechanisms.
Baroreceptor Model

Linden (1984) asserts that changes in the baroreceptor system may contribute most to the etiology and maintenance of essential hypertension. This system is located in the aortic arch and the carotid sinus and consists of nerve endings which react upon deformation or strain of the blood vessels. When blood pressure increases, increased activation of the baroreceptor endings occurs. These afferent receptors then increase their rate of firing, which is detected in brain stem pressor nuclei, leading to an inhibition of vasomotor discharge via the pathway of the sympathetic nervous system. Sympathetic nervous system inhibition results in the release, or disinhibition, of parasympathetic nervous system activity which increases the activity of the cardiac vagus. The resulting vessel dilation and decrease in cardiac activity opposes the increase in blood pressure and leads to a return of normal blood pressure.

Animal research indicates that the baroreceptor possess variable adaptive set-points which increase with exposure to chronic strain (McCubbin, Green, & Page 1956; Sleight, Robinson, Brooks, & Rees 1975 as cited in Linden, 1984; Dworkin, Filewich, Miller, Craigmyle, 1979), bringing about the diminished baroreflex sensitivity often observed among hypertensives (Bristow, Honour, Pickering, Sleight, & Smythe, 1969). This decreased sensitivity may contribute to the maintenance of the disorder by permitting greater blood
pressure responses to various stimuli. However, it is often considered that the diminished baroreflex sensitivity of hypertensives is actually the result of the disorder. That is, the baroreflex habituates to a sustained high blood pressure (Ditto & France, 1990). These authors also found a strong familial influence which may affect baroreflex sensitivity.

One study suggested that an acute blood pressure increase and the ensuing baroreceptor innervation may be biologically self-reinforcing (Dworkin et al., 1979). A learning mechanism in which aversive environmental influences are perceived as less aversive when blood pressure is elevated is one plausible explanation for the long-term modification of threshold values in the baroreceptor of hypertensive animals (Linden, 1984). Support for this model is found in both animal (Dworkin et al., 1979) and human research (Linden, 1984). Dworkin and his colleagues (1979) chemically increased blood pressure in rats and observed that the animals attempted to terminate or avoid electric shock less frequently than a group of control animals, an effect no longer seen once the baroreceptor had been operatively denervated.

To test this hypothesis in humans, Linden and Feurstein (1983) compared the reporting of stressful events by untreated hypertensives, treated hypertensives, and normotensives. The untreated hypertensives reported only one
half as many stressful events as the other groups. While treated hypertensives and the controls showed expected normal values on a scale for depression, the untreated hypertensives obtained depression scores which were significantly lower than the expected normal values. In addition, they displayed elevated scores on the Social Desirability Scale, a measure of psychological defensiveness, (Crowne & Marlowe, 1966) and indicated no awareness of ever feeling depressed during the study. The authors hypothesized lowered perception of stress and "repressive-defensive cognitive styles" in some hypertensives, whereby elevated physiological responses during stress are accompanied by low self-report of trait anxiety, suggesting low arousal.

This pattern of responding among hypertensives finds support in a study conducted by Sapira, Scheib, and Moriarty (1971). In this study, a group of hypertensives and a group of normotensives were shown two movies depicting two types of doctor-patient interaction; one film displayed a physician who was unpleasant and disinterested while the second physician presented a relaxed, personal, and warm style. During viewing, blood pressure and pulse rate responses in the hypertensive group were small but significantly greater than those in the normal group. In an interview following the film, hypertensives interviewed by the "bad doctor" whom they had just seen displayed a significantly greater pressor response than did the normotensives or the hypertensives who
were interviewed by a neutral physician. The most striking finding was that the hypertensive group tended to deny seeing any differences between the doctors depicted in the two movies while the normal group had no such difficulty. The authors assert that these findings may be suggestive of a defect in perception which enables the hypertensive to screen out potentially noxious stimuli as a defense against cardiovascular hyper-reactivity. While certainly intriguing, this hypothesis requires further investigation.

The Renal-Body Fluid System

Guyton (1987) claims that the baroreceptor system cannot play a role in the establishment of sustained hypertension since the rate of baroreceptor firing returns to normal levels in one or two days regardless of the continuing abnormal blood pressure levels. He asserts that long-term regulation of blood pressure is chiefly achieved through the renal-body fluid-pressure control system. While the other mechanisms previously discussed have been shown to be relatively short-acting regulators of blood pressure, the kidneys are designed in part to regulate blood pressure over the long term through a process of balancing fluid/electrolytes until blood pressure is normalized (Blanchard et al., 1988).

According to Guyton, (1987) the system works as follows: A rise in blood pressure directly causes the kidneys to purge increasing amounts of fluid and electrolytes
resulting in a decreased extracellular fluid and blood volume. This process may involve atrial peptide as well as local mechanisms. The decreased blood volume decreases heart pumping which further helps return arterial pressure to normal. According to Guyton (1987), sustained hypertension can only be caused by a change in the renal output curve or by different levels of water and salt intake. It should be noted that the latter is regulated via CNS mechanisms which are responsive to long and short-term stressors. Behavioral stressors can exert substantial influences on renal functioning. For instance, it has been observed that shock avoidance induces significant sodium and fluid retention in dogs (Kranz & Manuck, 1984).

If greater than normal pressure is required to cause the kidneys to excrete salt and water, then the long-term mechanisms for raising the pressure will become progressively more active until the pressure rises to the high level that is required to make the kidneys excrete normally. Increased pressure can come about either through salt and water retention, activation of vascular constrictor mechanisms, or both. Therefore, Guyton (1987) classifies hypertension into two different types: 1) volume loading hypertension, and 2) vasoconstrictor hypertension.

Volume-loading hypertension occurs when excess extracellular fluid volume accumulates in the body even though all other circulation functions are normal, and can be
divided into two stages. The first stage is the result of increased fluid volumes and increased cardiac output (characteristic of borderline hypertension). The second stage is characterized by high blood pressure and high total peripheral resistance but return of the cardiac output to near normal (characteristic of sustained hypertension). This second stage of increased total peripheral resistance is considered to be secondary to the hypertension rather than its cause.

Volume-loading occurs in two different ways. In the first, the kidney mass decreases to only about 30% of normal. At this time, the intake of salt and water increases sixfold. The acute effect is that extracellular fluid volume increases as does blood volume, and cardiac output is approximately 20-40 per cent above normal. At the same time, arterial pressure begins to rise, but at first not nearly as much as the fluid volumes and the CO. After these early acute changes in the circulatory system occur, more prolonged secondary changes take place during the next several weeks. Perhaps most important is the progressive increase in total peripheral resistance and the simultaneous decrease in CO to near normal.

A second type of volume-loading hypertension is due to excess aldosterone or other steroids in the body. Aldosterone increases the rate at which salt and water are reabsorbed by the tubules of the kidney. This reduces the loss of these
substances in the urine but causes an increase in the extracellular fluid volume, resulting in mild to moderate hypertension. If there is a simultaneous increase in salt intake, an even greater increase in blood pressure occurs. If the condition continues for prolonged periods, it appears that the excess aldosterone causes pathological changes in the kidneys which makes them retain still more salt and water, resulting in severe hypertension. Vasoconstrictor hypertension is caused by continuous infusion of a vasoconstrictor agent into the circulation or by excessive secretion of a vasoconstrictor by one of the endocrine glands. The vasoconstrictors which are especially prone to cause hypertension are angiotensin II, norepinephrine, and epinephrine. These agents cause marked increase in total peripheral resistance by constricting the arterioles. Conversely, there is a drop in cardiac output due to the intense arteriolar constriction.

The hypertension caused by angiotensin-induced vasoconstriction is, like volume-loading hypertension, a high resistance type of hypertension. That is, the angiotensin II constricts the arterioles which results in increased total peripheral resistance. Thus, even in the vasoconstrictor type of hypertension, it is the effect of the vasoconstrictor on kidney output of salt and water that determines the arterial pressure level at which the hypertension stabilizes. In order to sustain the increased pressure, the kidney output curve is
abnormally reset higher and higher in response to various changes in the system that drive up blood pressure (diet, weight, and stress), and chronic hypertension can result. It is often stated, for instance, that increased amounts of sodium in the diet can result in increased blood pressure through affecting renal function and fluid retention.

Once again, it is important to note the importance of neural/sympathetic activation in this process. Two of the vasoconstrictors which may ultimately act to cause a shift in the kidney output curve are the catecholamines epinephrine and norepinephrine, products of sympathetic nervous system activity. An example of how sympathetic activation can act through the renal pathway to change blood pressure is provided by Light, Koepke, Obrist and Willis (1983). These authors discovered that, in individuals who had been pre-loaded with fluid, psychological stress resulted in a hypertensive effect of increasing fluid retention (restricting kidney output). They also found that physical stress had the opposite effect of lowering blood pressure due to enhancing kidney functions.

Physical Effects of Hypertension

Hypertension can be damaging due to an increased work load on the heart as well as damage to the arteries themselves by the excessive pressure. The cardiac muscle hypertrophies when its work load increases. In hypertension, the very high pressure against which the left ventricle beats
causes it to increase greatly in weight. However, this increase is not accompanied by an adequate increase in coronary blood supply. Therefore, relative ischemia of the left ventricle develops as the hypertension becomes more severe. This can become serious enough that the person develops angina pectoris (Guyton, 1987).

High pressure in the arteries can cause coronary atherosclerosis, a symptomless condition characterized by narrowing and deterioration of the arteries and vessels nourishing the heart (Krantz and Manuck, 1984). In addition to coronary artery sclerosis, prolonged hypertension can also result in sclerosis of blood vessels throughout the body. The arteriosclerotic process causes clots to develop in the vessels, and it also weakens blood vessels. They may eventually rupture and bleed, causing damage to surrounding organs such as the brain (stroke). Arteriosclerosis also narrows arterial lumen which decreases blood flow, permitting clots to form and block the coronary artery. This sequence of events can lead to coronary ischemia and myocardial necrosis and ultimately, to myocardial infarcts. Two important types of damage resulting from hypertension are cerebral hemorrhage and hemorrhage of renal vessels inside the kidney, further exacerbating the hypertension (Guyton, 1987).

Summary

In summary, a review of the literature suggests that essential hypertension is most likely produced by an
interaction of physiological, environmental, psychological and genetic factors. When these variables interact, normal blood pressure regulation mechanisms can be defeated in susceptible individuals, not just over the short run, but for the long term as well, to produce chronic essential hypertension. Although the exact process(es) are not known, four models have been presented each of which explain the psychophysiological mechanism(s) involved in the progression from acute to essential hypertension. Each of the models can be influenced by environmental stressors and autonomic nervous system activity. The next section will examine ways in which the psychological factors' effect on the autonomic nervous system may result in a stereotypic response among some individuals which may ultimately lead to hypertension.
Psychological Components of Hypertension

Much of the work exploring the relationship between physiological response and emotion is the outgrowth of the James-Lange hypothesis of emotion. That hypothesis posited that the perception of emotion was based on the central nervous system receiving a complex set of peripheral physiological responses. The theory assumed that individuals could make distinctions among the idiosyncratic bodily reactions accompanying divergent emotional reactions.

While this theory is still generally controversial, it has given birth to three well-accepted principles of psychophysiology: stimulus-response (S-R) specificity, individual response (I-R) specificity, and individual response (I-R) stereotypy. S-R specificity occurs when certain classes of stimuli bring forth certain patterns of autonomic nervous system (ANS) response across individuals, i.e., the stimulus evokes a specific response regardless of the subject (Cannon, 1929; Lacey and Lacey, 1970). I-R specificity occurs when an individual tends to respond more with one particular ANS innervated response than others, i.e., the individual's response is specific to the stimulus (Malmo & Shagass, 1949; Wenger, Clemens, Coleman, Cullen, and Engel, 1961). The principal of (I-R) stereotypy takes I-R specificity one step further and describes the tendency of some individuals to respond to a variety of stressors with the same particular ANS response pattern, i.e., regardless of the stressor, one
particular ANS response pattern will be more activated than any others (Lacey, 1950; Engel & Bickford, 1961; Wenger et al., 1961; Wilson, Albright, Steiner & Andreassi, 1991). Some individuals have little variation across stimuli (rigid reactors), while others vary greatly across stimuli (random reactors) (Sternbach, 1966).

An early example of stimulus response specificity is found in the work on cardiac function by John and Beatrice Lacey (Lacey, 1950; Lacey and Lacey, 1970; Lacey and Lacey, 1958). They demonstrated that, for most people, a task such as solving a mental arithmetic problem led to a classical arousal reaction in which both heart rate and skin conductance increased. When the same individuals listened to a series of tones their heart rate decreased while their skin conductivity increased. Based upon this, they concluded that attention to internal events (environmental rejection) led to increased heart rate, while attention to external events (environmental intake) led to a decrease in heart rate.

Not only can differences be found between responses to stimuli and between individuals, but it also appears that these differences are stable over time. In a study examining the temporal stability of psychophysiological responses, Arena, Goldberg, Saul and Hobbs (1989) measured electromyographic (EMG) activity, hand surface temperature (HST), and heart rate (HR) response to two different stimuli. Based upon a statistical determination, 42% of their subjects
responded within a single system regardless of stressors (individual stereotypy) and 20% responded to stressors differentially (stimulus-response specificity). Similarly, Manuck and Garland (1980) and Allen, Sherwood, Obrist, Crowell, and Grange (1987) found stable individual differences in cardiovascular reactivity to laboratory stressors over extended periods of time. Obrist and colleagues have reported numerous studies which support the hypothesis of individual-response specificity of cardiovascular functioning (Obrist, 1981; Light & Obrist, 1983; Light, 1985; Sherwood, Allen, Obrist, & Langer, 1986; Sherwood, Dolan, & Light, 1990). The general finding of these studies is that the cardiovascular responses exhibited by an individual during psychological stress may be determined by the interaction of physiological predisposition, psychological makeup, and the type of stress situation, specifically, whether or not the individual has control over the outcome of events (active versus passive coping). It is important, therefore, to consider these factors when examining the role of stress in the etiology of hypertension.

Contrasting a group of normotensives with hypertensives, Engel and Bickford (1966) measured nine physiologic response functions (including SBP, DBP, breathing rate, heart rate, and heart rate variability) to five stimuli. The authors' findings indicate that hypertensives are generally over-reactive to environmental stressors in blood pressure, and
that they tend to respond with more consistent individual response patterns than do normotensives.

These findings have been supported in a number of subsequent studies. In particular, Fredrikson, Danielssons, Engel, Frisk-Holmberg, Strom, and Sundin (1985) determined that hypertensive patients are more consistent in their physiologic responses to stressors than are normotensives. The hypertensive group had their maximum high response in blood pressure, whereas the maximum response of the normals was variable. Specifically, the HT group showed the most reactivity in systolic pressure, with diastolic pressure next. Heart rate and skin conductance were the least reactive systems with respiration, hand blood flow and finger temperature falling between. These authors also found that beta-adrenergic receptor antagonists (beta-blockers) reduced levels of cardiovascular activity and attenuated reactivity, but did not affect the amount of I-R specificity. Similar results were obtained in other studies (Hodapp, Weyer & Becker, 1975; Fredrikson, Dimberg, Frisk-Holmberg & Strom, 1982).

A large number of laboratory studies report greater BP reactivity among hypertensives in response to various stimuli when compared to normotensives (Brod, 1970; Elliot, Buell, & Dembrowki, 1982; Herd, 1983; Krantz, Baum & Singer, 1983; Steptoe, Melville & Ross, 1984; Linden, 1984; Fredridson, Danielssons, Engel, Frisk-Holberg, Strom & Sundin, 1985;
Jorgensen and Houston, 1986; Weidner, Freiend, Ficarrotto, & Mendell, 1989; Fredrikson, Dimberg, & Frisk-Holmberg, 1980). While this appears to be a robust finding, it is not without its critics. Some researchers question the methodological soundness of these laboratory studies (Julius & Schork, 1971; Rosenman & Ward, 1988), and maintain that hypertensives do not show greater blood pressure variability than do normals.

The apparent controversy may, in fact, be due to confusion between the definitions of variability and reactivity. Julius and Schork (1971) define blood pressure variability as "wide spontaneous fluctuations of the blood pressure in a person as compared to the naturally occurring changes in a group of normal subjects", while vascular reactivity is thought of as "the change in blood pressure in response to a defined stimulus" (Julius and Schork, 1971, p.319). Thus, variability is the extent to which an individual's blood pressure fluctuates spontaneously compared to normals; reactivity is the extent to which an individual's blood pressure fluctuates due to a known external or internal stimulus.

A number of authors disagree with the long-held assumption of increased spontaneous pressure oscillation or variability among hypertensives, particularly among borderline hypertensives, (Julius & Schork, 1971; Harshfield, et. al, 1985; Kannel et al., 1980; Rosenman and Ward, 1988). However, as will be discussed in greater detail below, these
authors fail to examine variability in the natural environment, and do not take emotional factors into consideration. A growing body of research supports the finding of increased blood pressure fluctuations in the natural environment when borderlines are compared to normotensives, especially when emotional factors are considered (reactivity) (Schwartz, Weinberger, & Singer, 1981; Linden and Feurstein, 1983 as cited in Linden, 1984).

Much of the research on cardiovascular reactivity can be explained by individual response specificity and stereotypy in high stress situations, possibly due to genetic influences (Jorgensen and Houston, 1981). The large body of research cited previously suggests that hypertensive individuals react to emotion in a stereotypic manner which may have implications for the development of cardiovascular diseases such as hypertension. Specifically, I-R stereotypy can lead to short-term as well as more lasting changes in blood pressure through the various mechanisms discussed above. Following is an overview of the literature pertaining to emotional and psychological factors which appear to correlate with hypertension.

**Psychological Factors Associated with Elevated Blood Pressure**

Emotional behavior is a basic construct that can be seen as linking psychological factors to pathophysiologic processes (Diamond, 1984). Plutchik (1980) used the term emotional behavior to refer to a complex sequence of
reactions that include cognitive evaluations, subjective changes or perceptions, physiological changes (autonomic arousal), and action tendencies having both expressive as well as instrumental qualities.

The earliest investigators of hypertension did not clearly formulate the role of emotion in the development of hypertension. Rather, they concentrated on the search for an elusive personality type among hypertensives. Moschowitz (1919) and later investigators, proposed the existence of a "hypertensive personality", and described such characteristics as a "high strung" quick tempered character (Ayman, 1933): ambition, extroversion, intelligence, and expressiveness (Palmer, 1950); and devotion, perfectionism, avoidance of dependence (Tucker, 1959). Additional concepts linked to hypertension were submissiveness, inhibition, and neurotic symptomatology (Binger, Ackerman & Cohn, 1949; Dunbar, 1943; Hambling, 1951; Rennie, 1939).

Foremost among these early theories about hypertension was Alexander's (1939) notion of a "central conflict" in hypertensives between passive dependent feelings and strong hostile impulses. This hostility was accompanied by anxiety incompletely repressed and incapable of appropriate overt expression. Based on anecdotal evidence, Alexander contended that chronic inhibition of hostile tendencies led to permanent histological changes with an ensuing elevation of blood pressure. Unfortunately, the personality literature
failed to provide a description of the psychophysiological mechanisms accounting for the pathogenic interaction of personality traits and the development of hypertension, and there are other problems as well. For example, Diamond (1982) maintains that the formulations of the "hypertensive personality" were poorly integrated, and the specificity of the personality pattern to the hypertensive population questionable. He also states that the studies are not generalizable since the samples contain an over-representation of white, middle class neurotics. In addition, most of the work was biased by the researcher's knowledge of the diagnosis of the patients involved, violating standards of objectivity. Finally, because personality variables were assessed concurrently with blood pressure levels, the direction of causality is unclear. However, Alexander's notion of suppressed hostility in hypertensives has received some empirical support and is being studied still today, though the exact nature of the relationship between emotion and hypertension is still debated.

**Anger and Hostility**

Anger has been described as a primary emotion arising when an organism is blocked in the attainment of a goal or fulfillment of a need (Diamond, 1982; Izard, 1977; Novaco, 1975). As with any emotion, the occurrence of anger will likely depend on the organism's appraisal of events and assignment of meaning to those events (Arnold, 1960).
Systematic study of the physiology of anger began with Cannon (1929) who described the "fight or flight" response mediated by adrenaline, involving increases in blood pressure and heart rate, skeletal muscle vasodilation, visceral vasoconstriction, and biochemical changes associated with mobilization of energy. Schacter (1957) found that hypertensives manifested larger systolic BP increases than normotensives to pain and anger. As Mason (1971) points out, elevations in both epinephrine and norepinephrine levels may occur as a result of psychological stimuli, though selective responses of one hormone or the other may occur in a non-unitary fashion. In a still classic study, Albert Ax (1953) found differential responses to fear and anger which suggested that the cardiovascular correlates of fear were similar to an epinephrine response, while the anger pattern appeared similar to combined epinephrine-norepinephrine reactions.

Among the first researchers to examine the physiological correlates of anger expression were Funkenstein, King, and Drolette (1954) who hypothesized that the physiology of anger is mediated by the direction of anger expression (inward toward self or outward toward others.) The anger-in and anxiety groups were similar, with systolic BP and HR increases and predominantly epinephrine secretion while the response associated with anger-out predominantly involved norepinephrine secretion and large diastolic BP increase,
with little heart rate change and elevated peripheral resistance. These early studies helped to establish the possibility that epinephrine and norepinephrine might be selectively related to different emotional states (Mason, 1972).

The connection between anger and hostility is a close one, and researchers often use the terms interchangeably, making distinctions between the two constructs difficult, if not impossible, to ascertain. Buss (1961) described hostility as an attitude involving an implicit evaluative verbal response. Plutchik (1980) thought of hostility as a combination of anger and disgust. He associated it with indignation, contempt and resentment. Saul (1976) defined hostility as a "motivating force, a conscious or unconscious impulse, tendency, intent or reaction...usually accompanied by the feeling or emotion of anger" (p7). Diamond (1982) agrees with Saul's definition and asserts that all hostility contains an element of destructiveness.

Spielberger, Jacobs, Russell, and Crane (1983) have examined the research literature on anger and hostility and have proposed that the concept of anger usually refers to a simple emotional state that consists of feelings that vary in intensity from mild irritation to fury and rage. While hostility usually involves angry feelings, these authors define it as complex set of attitudes which motivate
aggression directed toward destroying objects or injuring others.

One major research program focused on anger expression in the black population, hypothesizing that suppressed hostility may be implicated in the greater prevalence of essential hypertension among black Americans (Harberg, Erfurt, Hauenstein, Chape, Schull, & Schork 1973). The authors observed that persons residing in high stress areas who used "anger in/guilt" coping styles, which they labeled "suppressed hostility", had significantly higher diastolic blood pressure and a greater incidence of hypertension than men with "anger out/no guilt" coping styles, labeled "expressed hostility." This finding of increased diastolic BP pressure among the anger-in group is inconsistent with the findings of Funkenstein et al. (1954) discussed above.

Harburg and colleagues also found that black males living in high stress areas had higher blood pressure than any other group. These high stress males also reported more "anger in" and guilt when they responded to the hypothetical situations involving verbal abuse by others. A similar pattern of results was found for white males residing in high and low stress areas, i.e., higher blood pressure levels were associated with "anger in" and guilt.

Mattson (1975 as cited in Diamond, 1982) compared black hypertensives, diabetic-hypertensives, diabetics, and medical controls with the Hostility and Direction of Hostility
Questionnaire (HDHQ; Caine, Foulds, & Hope, 1967). Consistent with the findings of Harberg and colleagues (1973) his most salient finding was that a tendency toward overt anger expression was related to lower blood pressure among hypertensives. Harburg, Blakelock, and Roeper (1979) maintained that styles of coping with anger provocation (anger-in v. anger-out) vary with social class and that such differences may be associated with blood pressure levels.

A reanalysis of the data from the studies by Harburg and colleagues (Gentry, Chesney, Gary, Hall, & Harburg, 1982) reconfirmed the relation between anger-in, or low levels of anger expression as an habitual coping pattern (trait v state), to be related to elevated blood pressure, both systolic and diastolic. They relate an individual's trait anger-coping style to hypertensive disease rather than to the specific situation and interpreted these findings as providing strong support Alexander's suppressed hostility hypothesis.

Dimsdale, Pierce, Schoenfeld, Brown, Zusman, and Graham (1986) found that systolic blood pressure was significantly related to suppressed anger, and that normotensives were twice as likely as hypertensives to exhibit no suppressed anger. However, suppressed anger was found to be unrelated to diastolic blood pressure.

Esler, Julius, Zweifler, Randall, Harburg, Gardiner, and DeQuattro (1977) maintain that suppressed hostility can be
found only in a specific subgroup of hypertensives. They found that only those hypertensives with high renin levels displayed a tendency to suppress hostility on a number of psychological measures. The high renin essential hypertensives were controlled, guilt prone and submissive with high levels of unexpressed anger. The hypertensives with low renin production did not differ psychologically from the normal controls.

Schneider, Egan, Johnson, Drobney, and Julius (1986) examined two groups of borderline hypertensives: one which maintained high blood pressure outside of the clinic as measured by ambulatory monitors, the other whose average BP returned to normal at home. These authors found that the high home BP group reported greater intensity and greater suppression of anger than the low-home group. Mills, Schneider, and Dimsdale (1988) found that anger expression, particularly anger expressed outwardly is related to lower heart rate and catecholamine responses to mental stress. Based upon their findings, they suggest that the ability to express anger outwardly may be related to reduced blood pressure and reduced incidence of heart disease as well as reduced reactivity. Since reactivity is a potential risk factor for high blood pressure, the authors postulate that expression of anger outwardly along with its possible association with lower reactivity, is a mediating mechanism
for the maintenance of lower blood pressure and reduced cardiovascular disease.

In a laboratory experiment, Goldstein, Edelberg, Meier, & Davis (1988) studied the relationship between the experience of anger, the expression of anger in the individual, the expression of anger in the subjects’ family of origin, and resting blood pressure. The subjects’ blood pressure was measured only once in a laboratory setting. The normotensives showed a significant relationship between anger and blood pressure levels, but the hypertensives did not.

A number of studies regarding the role of hostility have shown a consistent relationship between high hostility and cardiovascular reactivity. However, there is a question as to whether hostility alone is sufficient to produce increased reactivity, whether it must have some mediating factor, and if so, what the mediator might be. Sallis, Johnson, Treverrow, Kaplan, and Hovell (1987) found no relationship between hostility as measured by either the MMPI or the Cook Medley Hostility Scale (Cook & Medley, 1954) and blood pressure variability in response to laboratory stressors.

Weidner, Friend, Picarrotto, and Mendell (1989) examined the relationship between Cook Medley Hostility Scale scores and blood pressure and heart rate reactivity in male and female normotensives. Both men and women scoring high on hostility (Ho) had greater blood pressure responses to a stressful lab test of unsolvable anagrams. Those subjects
scoring high on hostility also reported more anger in response to the task as compared to low Ho subjects. The authors questioned whether anger as opposed to suspiciousness contributed significantly to the variation in blood pressure reactivity. To test this, the relationship between blood pressure readings obtained during the task (stress scores) and anger ratings was evaluated by computing the partial correlation (controlling for baseline) between the stress value and the anger score. The results indicated that anger alone was not related to either SBP or DBP reactivity. These results are consistent with findings by Smith and Houston (1987) who found that Ho was related to increased state anger, but not to increased physiological reactivity. Together, these findings suggest that arousal of mistrust and suspicion rather than anger per se may be responsible for the greater blood pressure reactivity found among high Ho subjects. Unfortunately, these authors failed to assess whether or not the task elicited suspiciousness or mistrust among the high hostility subjects who responded to the task with greater reactivity, significantly weakening their conclusions.

Jorgensen and Houston (1988) found a positive correlation between hostility and cardiovascular reactivity. Diastolic blood pressure reactivity was associated with not overtly expressing hostility. In a study designed to examine cardiovascular reactivity (DBP, SBP, MAP, and heart rate) in
high and low hostile men during increased interpersonal conflict elicited by a standard laboratory task, Suarez and Williams (1989) found that men who scored high on hostility showed exaggerated cardiovascular arousal when harassment caused them to experience anger and irritation. These authors suggested that the mediating variable between hostility and blood pressure reactivity is anger.

Perhaps the best known attempt to link anger and/or hostility to cardiovascular disease is the Type A Behavior Pattern (TABP) put forth by Friedman and Rosenman. They found that many hypertensives exhibited a characteristic behavior pattern, which they labelled "Pattern A" or "Type A," and summarized as "excessive and competitive drive and an enhanced sense of time urgency" (Friedman, Byers, and Rosenman, 1960, p. 758).

As originally envisioned by these authors, Type A was a global concept. Some researchers soon argued, however, that within the category of Type A were several subgroups, a question still undecided among TABP researchers. Some later studies on the specific components of TABP found consistent links to cardiovascular disease only with the hostility component (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985). Linden's review of the literature (1987) suggests that potential for hostility and anger—in as typical emotional responses to strain appear to be the most powerful predictor components of TABP. It should be noted that the
construct anger-in is not an element of the original definition of TABP. Linden suggests that whether or not potential for hostility and anger-in overlap is an area open to further investigation.

Friedman and Booth-Kewley (1988) used meta-analysis to dispute the notion that hostility and anger-in are the main predictor components in TABP. They found anxiety, hostility, and depression all to be highly correlated with coronary heart disease.

Anxiety

In examining the long-suspected relationship between hypertension and level of anxiety, conflicting results have been obtained. One possible reason may be the use of different non-standardized measures of anxiety, including the use of a combined anxiety-agitation rating, (Heine, Sainsbury, & Chynoweth, 1969), a measure of test-anxiety among college students (Harburg, Julius, McGinn, McLeod, & Hoobler, 1964), and clinical observation in the physicians office (Friedman & Bennett, 1977).

In one study, Whitehead, Blackwell, DeSilva, and Robinson (1977) instructed hypertensives to take their blood pressure 4 times daily for 7 weeks and to rate their anxiety and anger on analog scales. Anxiety was more highly correlated to elevations in both diastolic and systolic blood pressure than was anger.
Banahan, Sharpe, Baker, Liao, & Smith (1978) found a weak relationship between trait anxiety and hypertension, but a strong one between state anxiety and hypertension. McGrady and Higgins (1989) studied 24 unmedicated patients previously diagnosed as having borderline hypertension. Over a six week baseline measurement period, 15 decreased clinic mean arterial pressure (MAP) by at least 5 mmHg. This "unstable" group had significantly higher state and trait anxiety scores, heart rates, and DBP than did the nine patients in the "stable" group. These findings add further support for and possible identification of those individuals who may exhibit "office hypertension." Foster and Bell (1983) studied 30 medically diagnosed hypertensives (16 men and 14 women) and their normotensive spouses to confirm a suspected relationship between hypertension and trait anxiety. However, no significant differences were found between the normotensive and hypertensive groups on either state or trait anxiety. In addition, the anxiety scores of both the normotensive and hypertensive groups fell within the normal range for general and medical patients. Although this study used a standardized measure of anxiety, it did not control for other variables such as concurrent treatment by a physician with antihypertensive medication.

Van der Ploeg, van Buuren, and van Brummelen (1985) used the Dutch State-Trait Anger Scale, and the Dutch State-Trait Anxiety Inventory to investigate the role of anger and
anxiety in patients with essential hypertension. For the total male/female group, a significant difference was found between hypertensives and the control group for state anxiety. In addition, male hypertensives scored significantly higher in state anxiety than did the male controls, though females did not. No significant difference was found for either the total group or the male or female subgroups for trait anxiety or for either of the anger scales. This study also did not control for the effects of medication.

Schneider et al. (1986) found no greater anxiety, as measured by Spielberger's State-Trait Anxiety Inventory, among a group of borderline hypertensives who maintained high blood pressure outside the clinic than among borderlines whose pressure returned to normal at home. However, there was no normotensive control group against which to compare relative levels of anxiety for both groups of borderlines.

McGrady and Higgins (1990) conducted a study to determine which factors correlated with decreases in BP readings taken at home versus those taken in the physician's office. Of the small number of patients studied (n=24) 15 decreased their mean arterial pressure by at least 5 mmHg during a six week period of home measurement while 9 did not. The 9 patients with BP that did not change had lower state and trait anxiety scores as measured by the State-Trait Anxiety Inventory, lower diastolic BP, and lower heart rates compared to the group whose BP decreased. These findings
suggest that under certain conditions, a relationship between anxiety and elevated BP may exist for a segment of the hypertensive population.

**Depression**

Depression has received surprisingly little attention in the psychosomatic study of hypertension (Coelho, Hughes, Fonseca, & Bond, 1988). Several studies report higher rates of depressive symptoms among hypertensives (Kidson, 1973; Thaler, Freidman, Harshfield, & Pickering, 1985; Bulpitt, Hoffbrand, & Dollery, 1976; Coelho, Hughes, Fonseca, & Bond, 1988) while others show no such relationship (Boutelle, Epstein, & Ruddy, 1987; Santonastaso, Canton, Ambrosio, & Zamboni, 1984; Wheatley, Balter, Levine, Lipman, Bauer, & Bonato, 1975; Heine, Sainsbury, and Chynoweth, 1969; Friedman and Bennett, 1977). These mixed findings may be accounted for in part by the fact that some studies assessed depression by subscales of larger inventories rather than employing specific inventories designed to measure depression only.

Fuller (1988) found that 30-37% of hypertensives suffer from depression which is significantly higher than the 2-9% prevalence of depression in the general population. Which causal factors are significant is unclear. Current etiological hypotheses may be summarized as: 1) a common physiological factor underlies both depression and hypertension (Freidman & Bennett, 1977); 2) depression is a side-effect of some anti-hypertensive medications (Bant,
1974; Avorn, Everitt, & Weiss, 1986; Paykel, Fleminger, & Watson, 1982); 3) depression is secondary to experiencing a chronic illness such as hypertension (Bant, 1974; Paykel et. al., 1982); 4) depression results from treatment that lowers the blood pressure in such a way as to cause cerebral insufficiency in the elderly (Paykel, et. al., 1982); 5) the association between hypertension and depression is coincidental (Hyapaya & Ananth, 1980).

To summarize the findings from the literature on blood pressure and emotion, it appears that the suppression of anger and hostility may play a role in increased blood pressure among some individuals. The role of suspiciousness and mistrust has also been postulated as a possible mediating variable between increased anger and increased blood pressure, but further work is needed to draw any firm conclusions.

The anxiety literature is less clear, with some studies finding a relationship between hypertension and anxiety and others finding none. Few studies have attempted to differentiate between the role of anxiety in the pathogenesis of the disorder and its role as a possible consequence of being diagnosed as hypertensive. The depression literature is also equivocal, with some authors supporting the finding of increased depression among hypertensives while others do not. As with anxiety and perhaps anger, it is possible that the depression seen in many hypertensives may be secondary to
being diagnosed with a chronic illness (MacDonald, Sackett, Haynes, & Taylor, 1984). One of the few studies to address this issue was done by Mourn, Naess, Sorensen, Tambs, and Holmen (1990). These authors found that while changes in psychological well-being were not significantly related to labelling or BP status, there was a deterioration in psychological well-being among patients who had been subjected to one or more negative life events (stressors other than labelling) in the preceding 12 months. More research is needed to test this possibility.

Emotion and BP Reactivity

As was briefly discussed above, whether or not hypertensives exhibit more spontaneous blood pressure variability than normotensives has been controversial. A number of studies report negative findings (Julius and Schork, 1971; Harshfield, Pickering, Kleinert, Denby, Kleiner, Kaplan, Tucher, and Laragh, 1982; Pickering, Harshfield, Kleiner, Blank, & Laragh; Rosenman & Ward, 1988), while others find support for greater variability in hypertensives (Peiss, 1967; Linden & Feurstein, 1983 as cited in Linden, 1984). A central issue has been whether or not borderline hypertensives exhibit larger blood pressure variability and/or slower recovery to baseline values than do normotensives following exposure to a stressor in the natural environment, i.e. whether or not borderlines are more reactive than normotensives and whether their reactions are
more persistent. A number of studies have examined this issue, but many have not included normal control groups for comparison purposes.

Sokolow, Werdegar, Perloff, Cowan and Brenestuhl (1970) utilized an ambulatory sphygmomanometer and found that blood pressure fluctuated wildly among hypertensives, with an average range of 60 mmHg systolic and 32 mmHg diastolic. The highest blood pressure values correlated significantly with feelings of anxiety and time pressure. Hostility and depression did not correlate with observed blood pressure increases. Contentment and the subjective sense of well-being was associated with low blood pressure values.

Yee, Harshfield, Blank, and Pickering (1986) utilized hour ambulatory blood pressure monitoring and found position (sitting or standing), situation (work, home, elsewhere), and emotional state (happiness, anger, anxiety) to be significant sources of variation in blood pressure, with emotional state being the most significant. On average, anger and anxiety were associated with increased pressure more than happiness. Anxiety had a greater relation away from home than at home, but this was not the case for either happiness or anger. They also found a relationship between variability and intensity of emotion. An increase in happiness was found to be associated with a decrease in systolic pressure; an increase in anxiety intensity was found to be associated with an increase in diastolic pressure.
Reported anger intensity was not associated with either systolic or diastolic pressure. Finally, the greater the daily pressure variability of the borderline hypertensive, the greater the increase in pressure associated with emotional response.

Schneider, et al. (1986) compared borderline hypertensives who maintained high blood pressure outside of the clinic with a group which had normal blood pressure at home. Both groups were monitored with a 24 hour ambulatory device and given the State-Trait Personality Inventory (Spielberger, Jacobs, Barker, Russell, Crane, & Worden (1984) and the Anger Expression Scale (Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1984). They reported greater intensity of anger and more anger suppression among the high BP at home group. The groups did not differ in anxiety, nor was variability significantly different between the groups.

Linden and Feurstein (1983) compared reactivity among hypertensives to normotensives. They found that individuals with mild untreated hypertension (mean 150/90 mmHg) responded to a relatively minor stressor with average changes of 20 mmHg systolic and 10 mmHg diastolic whereas subjects with normal resting values displayed blood pressure changes of about half that magnitude. This study did not employ ambulatory devices, but relied instead upon multiple readings taken in the laboratory.
Crowther, Stephens, Koss, and Bolen (1987) examined the relative impact of five behavioral factors on the 24 hour blood pressure variability of normotensives, borderline hypertensives, and sustained essential hypertensives. 24 hour readings were taken with an automatic preprogrammed monitor. Activity, posture, location, and social involvement accounted for more variability among the normotensives than for either of the other groups. They summarize their results as showing that normotensive individuals may be more sensitive to behavioral factors than either borderline or sustained hypertensives. However, the authors also note that "tension" accounted for a larger percentage of the variability in the borderline hypertensive group, but failed to provide a clear definition of the term.

In conclusion, findings from studies examining BP reactivity relative to emotion are mixed; A number of studies support heightened reactivity in response to emotion among hypertensives, but many of these lack comparison groups. Most of these studies do not compare hypertensives with normotensives, nor do they compare borderline subgroups with subgroups of sustained hypertensives. None of the studies to date examine the issue of emotional reactivity among secondary hypertensives. Further, the findings regarding patterns of reactivity accompanying the core emotions are also mixed: Some authors report greater reactivity in response to anger, while others find greater
increases in BP associated with anxiety. The intriguing issue of what emotional factors may contribute to decreased blood pressure taken at home versus in the clinic has just begun to be examined. Much more work is needed in this area to be able to arrive at any firm conclusions.

Summary

Much of the research dealing with hypertension suffers from major methodological flaws such as the use of inadequate methods to diagnose hypertension, (e.g. the reliance on casual office readings), the use of non-validated measures of emotion and personality, lack of control or contrast groups, and the frequent inclusion of patients who are being treated with anti-hypertensive medications at the time of blood pressure assessments.

Borderline hypertension is an important risk factor for the development of sustained essential hypertension. A significant percentage of individuals with borderline hypertension do not develop established hypertension or related complications. This may be due to the fact that as many as two-thirds of those individuals diagnosed as borderline hypertensives have diagnoses based on casual readings and may not exhibit higher than normal readings outside the clinical setting. This is a tentative finding which requires further research and examination with the use of portable blood pressure monitors.
If these findings are indeed replicated, there are several important ramifications. First, better identification of true borderline hypertensives may lead to an increased understanding of the psychological and physiological factors involved in the progression from borderline to sustained essential hypertension. Second, misdiagnosing a patient as borderline hypertensive may result in heightened psychological distress which might then affect blood pressure readings. For instance, it appears that while some hypertensives exhibit a greater degree of anxiety and neuroticism, it is unclear whether this is a causal factor in the pathogenesis of the disease or if it is a consequence of being so diagnosed. Besides these negative psychological implications, incorrect diagnosis also exposes the individual to the potentially harmful effects of unnecessary medication and iatrogenic illnesses.

A sorely overlooked group in the research on hypertension is those who suffer from hypertension either directly from another disease, or whose hypertension is greatly influenced by another disease, such as diabetes mellitus. Many studies go to great lengths to exclude secondary or diabetic hypertensives from any study examining psychological factors, and virtually no attention has been paid to what psychological and physiological differences between essential and secondary or diabetic hypertensives might actually exist. It appears that there is the assumption
that blood pressure of these individuals are somehow immune to the psychological factors which appear to affect essential hypertensives.

A review of the literature failed to produce a single study which examined differences in reactivity between these groups to test this long-standing and well-entrenched implicit assumption. Is it possible that, like borderline hypertension, secondary and diabetic hypertension is also overdiagnosed as a result of one casual reading? Because there have been no well-controlled, systematic studies using ambulatory or portable BP monitoring, we do not know whether or not borderline hypertensives exhibit greater blood pressure variability in response to emotional situations (reactivity) in the natural environment than do secondary or diabetic hypertensives. It would be worthwhile to examine the contribution of psychological and emotional factors for this neglected group of hypertensives.
Hypotheses

The purpose of this study is to examine the relationship between emotion and cardiovascular reactivity for four groups of subjects: untreated borderline essential hypertensives, treated mild essential hypertensives, Type II diabetic hypertensives who are treated with the same medications as the mild essential hypertensives, and normotensives. Among the hypertensive subjects, only patients medicated with calcium channel blockers or ACE inhibitors will be included since these do not inhibit emotional responsivity (Dimsdale, et al, 1992). The study will examine five specific hypotheses. Following are the hypotheses and a rationale for the inclusion of each in this study.

1. Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ on trait measures of anxiety, anger, depression, and self-report of autonomic nervous system activity during emotions. Though there have been a number of studies examining the relationship between emotion and hypertension, none have included all four of these subgroups.

2. Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ in the degree of correlation between trait measures of anxiety, anger, depression, and self-report of autonomic nervous system
activity and blood pressure level and variability as determined by repeated BP measurements at home. The few studies examining BP reactivity in the natural environment have resulted in equivocal findings. There is a need for further examination of BP reactivity in general and its relationship with these three subgroups in particular.

3. Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ in the difference between casual office blood pressure readings vs. repeated blood pressure readings at home. The "white coat" phenomenon is generally accepted for borderline hypertensives. It has not been examined for mild essential hypertensives or diabetic hypertensives.

4. Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ in the degree of correlation of trait measures of anxiety, anger, depression, and self-report of autonomic nervous system activity with the difference between casual blood pressure readings and BP readings taken at home. This hypothesis attempts to identify those emotional components which contribute to "white coat" hypertension among those hypertensives exhibiting this phenomenon.

5. Two-thirds of those individuals diagnosed as essential borderline hypertensive, mild essential
hypertensive or diabetic hypertensive based on casual readings will record blood pressure readings in the normal range when the readings are taken at home. "White coat" hypertension is a generally accepted phenomenon among borderline hypertensives. However, to date, no study has examined whether or not it exists among mild essential hypertensives or diabetic hypertensives.

Subjects

80 subjects were chosen from a clinical outpatient population; 20 untreated borderline essential hypertensives, 20 treated mild essential hypertensives, and 20 treated diabetic hypertensives, and 20 untreated normotensives. Medication for the treated subjects was either angiotensin converting enzyme (ACE) inhibitors or calcium channel blockers since these drugs do not inhibit emotional responsivity. While it would have been optimal to include only unmedicated subjects in this study, it would have been impossible to obtain the necessary number of subjects if medicated patients were excluded because virtually all mild and diabetic hypertensives are medicated. Individuals on beta blockers were excluded from the study since it has been shown that this treatment does inhibit emotional responses (Dimsdale et al, 1992).

The age of the subjects was restricted to 30-60 years old. In addition, all subjects were caucasian and matched on sex. Restricting the study to caucasians was due to the
findings of Harburg and colleagues (Harburg et al., 1964; Harburg et al., 1973; Harburg et al., 1979) who found differing BP levels, patterns of reactivity, and response to anger among racial groups. According to Harburg et al. (1973) African American blacks have higher blood pressure levels, as well as higher morbidity and mortality from hypertension and other cardiovascular diseases than do American caucasians. Limiting the study to one racial group eliminated potential confounds due to these differences.

**Instrumentation**

With the advent of portable blood pressure monitoring devices, it is now possible to take blood pressure readings in the natural environment, and as often as desired. Calibration studies comparing several different portable devices to readings taken casually show these to be as accurate and more valid than casual readings (Harshfield, Pickering, Blank, Lindhal, Stroud, & Laragh, 1983; Weber, Drayer, & Chard, 1983; Ward & Hansen, 1983).

Psychological measures consisted of Spielberger’s State-Trait Anxiety Inventory (STAI) (Spielberger, 1983), a measure of trait and state anxiety, Spielberger’s Anger Expression Scale (AX), a trait measure which provides a ratio score of anger-in to anger-out (Spielberger, 1987), the Beck Depression Inventory (BDI), a self-report measure of depression (Beck, 1967), and the Autonomic Nervous System Response Inventory (ANSRI), a self-report instrument designed to assess patterns
of ANS response to emotional stimuli (Waters et al., 1984). All measures have been found to possess adequate psychometric properties.

Test-retest reliability (stability) data for the STAI A-Trait scale range from .73 to .86 while those for the A-State scale tend to be much lower (range of .45 to .60) as would be expected for a measure designed to be influenced by situational factors. Both scales have a high degree of internal consistency. (Spielberger, 1983). In addition, the STAI has been found to be a valid test of state and trait anxiety as indicated by concurrent validity studies, with correlations between the STAI and other measures ranging from .53 to .85. Spielberger’s Anger Expression Scale has also demonstrated both adequate reliability (coefficients of alpha range from .73 to .84 for internal consistency) and adequate convergent and divergent validity.

Beck, Weissman, Lester, and Trexler (1974) have demonstrated high levels of internal-consistency reliability. Concurrent validity was assessed by comparing BDI scores with global clinical assessments. Beck and Beamesderfer (1974) reported that in nine studies carried out in the US and Europe, correlations in all cases were between .61 and .73. This finding was more recently corroborated by Hamilton (1982). A number of factor-analytic studies have identified physiological and negative self-image factors (Rabkin & Klein, 1987).
The ANSRI has also shown to be a reliable instrument as discussed in Waters et al., (1984). Test-retest reliabilities were found to be statistically significant and reasonably high for both males and females. In addition, the coefficients alpha were also significant and very high, with most falling in the .90 range.

**Procedure**

Subjects were assigned to the appropriate groups based on their physicians' diagnoses. After subjects signed a standard consent form, they were given the psychological measures, and instructed in the use of the portable blood pressure monitor. The unit was then be attached to the patient, and calibration readings taken simultaneously by trained assistants with a mercury column sphygmomanometer and stethoscope and the portable home BP recorder. Five consecutive readings with two procedures agreeing to within 5mmHg for both systolic and diastolic pressure were obtained before calibration was considered adequate (Harshfield et. al., 1988).

Non-office readings were taken after an individual's morning awakening routine, at mid-morning, mid-day (immediately before lunch), mid-afternoon, evening before dinner, night-time between dinner and bedtime, and night-time at bed time. Subjects were instructed to take their blood pressure in the right arm while sitting and to follow the proper procedures described above to insure that accurate
readings were taken. A subject's mean blood pressure level was expressed as the overall average across all readings. Blood pressure variability was expressed in terms of the individual's standard deviation from the mean.

**Statistical Method**

Hypothesis 1 was tested using MANOVA. The variables examined were anxiety, anger, depression, and ANS activity. The rationale for using this statistical method is that the use of independent univariate tests leads to an inflated Type I error rate. In addition, univariate tests ignore important information such as the correlations among the variables, whereas the multivariate test incorporates the correlations into the test statistic. Finally, it was thought possible that the groups might not significantly differ on any of the variables individually, but that jointly, the set of variables might reliably differentiate the groups. In this case, the multivariate test would be more powerful (Stephens, 1986). The alpha level was set at .01.

Hypotheses 2 and 4 were both tested with z tests of correlation. This test of equality of correlations was appropriate since these hypotheses was concerned with the differences in correlations among the three groups (Steele & Torrey, 1984). Hypothesis 2 compared correlations of trait measures of anxiety, anger, depression, and self-report of ANS activity with both level and variability of blood pressure taken at home for all groups. Hypothesis 4 compared
correlations of the various trait measures mentioned above with the difference between casual blood pressure readings and BP readings taken at home for the three hypertensive groups. P values were set at .01.

Hypothesis 3 was tested using ANOVA to assess the differences among the three groups between causal blood pressure readings versus at-home BP readings, with P values set at .01. Hypothesis 5 was tested using a simple binomial test where p = .75. This hypothesis was concerned with replicating previous findings that as many as 2/3 of clinic hypertensives are actually normotensives when tested away from the physician's office.
Results

To test Hypothesis I (Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ on trait measures of anxiety, anger, depression, and self-report of autonomic nervous system activity during emotions), MANOVA was carried out to compare the four groups across the five psychological indices of interest: Beck Depression Inventory (BDI), State-Trait Anxiety Inventory-trait anxiety (STAI-Tanx), State-Trait Anger Expression Inventory-trait anger (STAXI-Tang), the STAXI ratio of anger-in to anger-out (STAXI-IO), and the Autonomic Nervous System Response Inventory (ANSRI). Using Wilks' criterion the exact F was 2.19 (p.<.0075), indicating significant differences among the four groups on the five psychological measures. Results are given in Table 1.

Further analysis was carried out to assess the contribution of each of the five psychological measures to these findings. One-way ANOVAs found no significant differences between the four groups on the five psychological measures when the measures were examined individually. A canonical analysis was also performed (Huberty and Morris, 1989). With the exception of the ANSRI, the psychological measures each contributed to the finding of overall significance, with no one measure contributing substantially more than the others. Results are given in Table 2.
### TABLE 1

**MANOVA ANALYSIS OF GROUPS BY PSYCHOLOGICAL MEASURES**

<table>
<thead>
<tr>
<th>Comparison</th>
<th>F</th>
<th>P=</th>
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<tbody>
<tr>
<td>All Groups</td>
<td>2.1920</td>
<td>.0075*</td>
</tr>
<tr>
<td>Normals &amp; BEH</td>
<td>1.9435</td>
<td>.0962</td>
</tr>
<tr>
<td>Normals &amp; EH</td>
<td>0.9653</td>
<td>.4441</td>
</tr>
<tr>
<td>Normals &amp; DH</td>
<td>2.4974</td>
<td>.0374</td>
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<tr>
<td>BEH &amp; EH</td>
<td>2.2789</td>
<td>.0545</td>
</tr>
<tr>
<td>BEH &amp; DH</td>
<td>1.6772</td>
<td>.1496</td>
</tr>
<tr>
<td>EH &amp; DH</td>
<td>4.1411</td>
<td>.0021</td>
</tr>
</tbody>
</table>

* p < .01

BEH = Borderline Essential Hypertensives  
EH = Essential Hypertensives  
DH = Diabetic Hypertensives
**TABLE 2**

ALL GROUPS CANONICAL ANALYSIS

<table>
<thead>
<tr>
<th></th>
<th>Canonical 1</th>
<th>Canonical 2</th>
<th>Canonical 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canonical Correlations</td>
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<td>.302</td>
<td>.187</td>
</tr>
<tr>
<td>p=</td>
<td>.0075*</td>
<td>.21</td>
<td>.40</td>
</tr>
<tr>
<td>Canonical Coefficients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TANX</td>
<td>.53</td>
<td>.25</td>
<td>.41</td>
</tr>
<tr>
<td>TANG</td>
<td>.68</td>
<td>.15</td>
<td>-.19</td>
</tr>
<tr>
<td>BDI</td>
<td>-.87</td>
<td>.08</td>
<td>.58</td>
</tr>
<tr>
<td>ANSRI</td>
<td>-.09</td>
<td>.78</td>
<td>.19</td>
</tr>
<tr>
<td>ANGER IN/OUT</td>
<td>.65</td>
<td>-.45</td>
<td>.38</td>
</tr>
</tbody>
</table>

*p < .01

TANX = Trait Anxiety  
TANG = Trait Anger  
BDI = Beck Depression Inventory  
ANSRI = Autonomic Nervous System Response Inventory
Another MANOVA found significant differences between Type II Diabetic Hypertensives and Essential Hypertensives (EH) \( (F=4.1411, \ p=.0021) \) on the five tests (Table 1). Again, further analysis was carried out to assess the contribution of each of the five psychological measures to these findings. One-way ANOVAs found no differences between the DH and EH on any of the five measures. Canonical analysis revealed that, once again, with the exception of the ANSRI, the psychological measures each contributed to the finding of significance between EH and DH. The range of the other four coefficients was small, again indicating that no one measure made a substantially greater contribution than the others. Results are given in Table 3. Table 4 shows the means and standard deviations for each of the four groups on each of the five measures.

ANOVA was used to test Hypothesis 3 (Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ in the difference between casual office blood pressure readings vs. repeated blood pressure readings at home). ANOVA results showed that there were no significant differences across the groups in the difference between casual office diastolic blood pressure readings and home diastolic blood pressure readings \( (F=3.61, \ p=.02) \). Average home diastolic readings were significantly lower than average casual office readings for all groups. Table 5 gives
TABLE 3
PAIR-WISE CANONICAL ANALYSIS

<table>
<thead>
<tr>
<th></th>
<th>Normals &amp; BH</th>
<th>Normals &amp; EH</th>
<th>Normals &amp; DH</th>
<th>BH &amp; EH</th>
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<td></td>
<td></td>
<td></td>
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<td>.367</td>
<td>.353</td>
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<tr>
<td>Coefficients</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TANX</td>
<td>.56</td>
<td>.14</td>
<td>.65</td>
<td>.43</td>
</tr>
<tr>
<td>TANG</td>
<td>.48</td>
<td>-.40</td>
<td>.58</td>
<td>.78</td>
</tr>
<tr>
<td>BDI</td>
<td>-.35</td>
<td>.88</td>
<td>-.61</td>
<td>-.88</td>
</tr>
<tr>
<td>ANSRI</td>
<td>.62</td>
<td>.58</td>
<td>.02</td>
<td>.21</td>
</tr>
<tr>
<td>ANGER IN/OUT</td>
<td>.05</td>
<td>-.31</td>
<td>.73</td>
<td>.24</td>
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</table>

<table>
<thead>
<tr>
<th></th>
<th>BH &amp; DH</th>
<th>EH &amp; DH</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
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<td>Correlation</td>
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</tr>
<tr>
<td>p</td>
<td>.15</td>
<td>.0021*</td>
</tr>
<tr>
<td>Canonical</td>
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<td></td>
</tr>
<tr>
<td>Coefficient</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TANX</td>
<td>.18</td>
<td>.44</td>
</tr>
<tr>
<td>TANG</td>
<td>.18</td>
<td>.64</td>
</tr>
<tr>
<td>BDI</td>
<td>-.35</td>
<td>-.89</td>
</tr>
<tr>
<td>ANSRI</td>
<td>-.63</td>
<td>-.26</td>
</tr>
<tr>
<td>ANGER IN/OUT</td>
<td>.81</td>
<td>.71</td>
</tr>
</tbody>
</table>

*p < .01

BEH = Borderline Essential Hypertensives
EH = Essential Hypertensives
DH = Diabetic Hypertensives
TANX = Trait Anxiety
TANG = Trait Anger
BDI = Beck Depression Inventory
ANSRI = Autonomic Nervous System Response Inventory
### TABLE 4

**MEANS AND STANDARD DEVIATIONS FOR PSYCHOLOGICAL MEASURES**

<table>
<thead>
<tr>
<th>GROUP</th>
<th>TANX MEAN</th>
<th>TANX SD</th>
<th>TANG MEAN</th>
<th>TANG SD</th>
<th>BDI MEAN</th>
<th>BDI SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normals</td>
<td>37.56</td>
<td>7.47</td>
<td>18.60</td>
<td>4.49</td>
<td>9.07</td>
<td>7.28</td>
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<tr>
<td>BEH</td>
<td>33.35</td>
<td>7.35</td>
<td>16.00</td>
<td>2.20</td>
<td>9.35</td>
<td>5.97</td>
</tr>
<tr>
<td>EH</td>
<td>35.33</td>
<td>11.86</td>
<td>18.71</td>
<td>4.51</td>
<td>5.96</td>
<td>5.06</td>
</tr>
<tr>
<td>DH</td>
<td>31.76</td>
<td>7.82</td>
<td>16.62</td>
<td>4.89</td>
<td>9.23</td>
<td>6.73</td>
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</table>

<table>
<thead>
<tr>
<th>GROUP</th>
<th>ANSRI MEAN</th>
<th>ANSRI SD</th>
<th>ANGER IN/OUT MEAN</th>
<th>ANGER IN/OUT SD</th>
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</thead>
<tbody>
<tr>
<td>Normals</td>
<td>130.83</td>
<td>38.29</td>
<td>1.29</td>
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</tr>
<tr>
<td>BEH</td>
<td>112.48</td>
<td>11.71</td>
<td>1.35</td>
<td>0.37</td>
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<tr>
<td>EH</td>
<td>122.37</td>
<td>22.03</td>
<td>1.29</td>
<td>0.52</td>
</tr>
<tr>
<td>DH</td>
<td>128.81</td>
<td>26.23</td>
<td>1.06</td>
<td>0.30</td>
</tr>
</tbody>
</table>

**BEH = Borderline Essential Hypertensives**  
**EH = Essential Hypertensives**  
**DH = Diabetic Hypertensives**  

**TANX = Trait Anxiety**  
**TANG = Trait Anger**  
**BDI = Beck Depression Inventory**  
**ANSRI = Autonomic Nervous System Response Inventory**
the means and standard deviations for each of the four groups.

In further analysis pertaining to Hypothesis 3, ANOVA showed that there were significant differences across the groups in the difference between casual office systolic blood pressure readings and home systolic blood pressure readings ($F=7.14$, $p=.0002$). A post-hoc analysis using Tukey's Studentized Range (HSD) showed significant differences between EH (13.11 mmhg) and BH (-1.85 mmhg) and between DH (12.83) and BH (-1.85 mmhg). Table 5 gives the means and standard deviations for each of the four groups. Average home systolic readings were significantly lower than the average office readings for all groups except BH.

In examining "white coat" hypertension, as set forth in Hypothesis 5, (Two-thirds of those individuals diagnosed as essential borderline hypertensive, mild essential hypertensive or diabetic hypertensive based on casual office readings will record blood pressure readings in the normal range when the readings are taken at home), office and home BP readings for the three hypertensive groups produced the following results: of the 6 BH with abnormal office SBP, 3 had normal home SBP, and of the 5 BH with abnormal office DBP, 4 had normal home DBP; of the 11 EH with abnormal office SBP, 8 had normal home SBP, and of the 16 EH with abnormal office DBP, 15 had normal home DBP; of the 13 DH with abnormal office SBP, 9 had normal home SBP, and of the 10 DH
### TABLE 5
DIFFERENCES BETWEEN OFFICE AND HOME BP READINGS

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<tr>
<th></th>
<th>MEAN</th>
<th>SD</th>
<th>p</th>
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<tr>
<td>OFF-SBP</td>
<td>120.96</td>
<td>10.57</td>
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<tr>
<td>HO-SBP</td>
<td>120.76</td>
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<td></td>
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<tr>
<td>DIFF</td>
<td>0.20</td>
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<td>0.93</td>
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<tr>
<td>OFF-DBP</td>
<td>80.46</td>
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<tr>
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<td>76.03</td>
<td>5.60</td>
<td></td>
</tr>
<tr>
<td>DIFF</td>
<td>4.43</td>
<td>7.20</td>
<td>0.01</td>
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</table>

<table>
<thead>
<tr>
<th>GROUP</th>
<th>OFF-SBP</th>
<th>SD</th>
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</tr>
</thead>
<tbody>
<tr>
<td>BEH</td>
<td>133.87</td>
<td>12.87</td>
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<tr>
<td>HO-SBP</td>
<td>135.72</td>
<td>16.25</td>
<td></td>
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<tr>
<td>DIFF</td>
<td>-1.85</td>
<td>16.33</td>
<td>0.62</td>
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<tr>
<td>OFF-DBP</td>
<td>88.20</td>
<td>8.43</td>
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<td>HO-DBP</td>
<td>83.79</td>
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</tr>
<tr>
<td>DIFF</td>
<td>4.41</td>
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<table>
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<tr>
<td>EH</td>
<td>140.86</td>
<td>10.51</td>
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<td>11.75</td>
<td></td>
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<td>12.28</td>
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<th>SD</th>
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<tbody>
<tr>
<td>DH</td>
<td>145.19</td>
<td>10.91</td>
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<tr>
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<td>16.13</td>
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<td>HO-DBP</td>
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<tr>
<td>DIFF</td>
<td>13.58</td>
<td>12.35</td>
<td>0.0001*</td>
</tr>
</tbody>
</table>

* P < .01

BEH = Borderline Essential Hypertensives
EH = Essential Hypertensives
DH = Diabetic Hypertensives

OFF-SBP = Average Office Systolic Blood Pressure
HO-SBP = Average Home Systolic Blood Pressure
OFF-DBP = Average Office Diastolic Blood Pressure
HO-DBP = Average Home Diastolic Blood Pressure
DIFF = Difference Between Office and Home Blood Pressure
with abnormal office DBP, all 10 had normal home DBP. Two-thirds or more of the members of each of the three groups recorded blood pressure readings in the normal range when the readings were taken at home in support of Hypothesis 5.

Hypotheses 2 (Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ in the degree of correlation between trait measures of anxiety, anger, depression, and self-report of autonomic nervous system activity and blood pressure level and variability as determined by repeated BP measurements at home) was not supported, nor was Hypotheses 4 (Individuals diagnosed as borderline essential hypertensive, mild essential hypertensive, diabetic hypertensive, and normotensive will differ in the degree of correlation of trait measures of anxiety, anger, depression, and self-report of autonomic nervous system activity with the difference between casual office blood pressure readings and BP readings taken at home). No differences were found among the four groups in the degree of correlation of the five psychological measures with either blood pressure level or variability (Hypothesis 2), nor were there differences among the groups in the degree of correlation of the five psychological measures with the difference between casual office blood pressure readings and readings taken at home (Hypothesis 4).
Discussion

Results of this study were mixed. Of the five hypotheses set forth, three were partially supported. Regarding Hypothesis 1, differences were found among the four groups with respect to their overall scores on the five psychological measures. Further analysis showed an overall difference on the five psychological measures between the diabetic hypertensives and the essential hypertensives, although no significant differences were found for any of the specific psychological measures across any of the groups, including DH and EH. Canonical analysis revealed that the ANSRI made no significant contribution to these findings, and that no one of the other four measures made a significantly greater contribution than did the others.

The fact that there were differences among the groups on the five measures of emotion appears to support the belief that there is a relation between emotion and hypertension. However, the present findings make it difficult to ascertain the specific nature of the relation. The finding that DH differed from EH is not surprising in that it was speculated that the Type II Diabetics would differ from other subjects emotionally, if only due to the fact of their serious, lifestyle changing, and sometimes life threatening underlying illness.

The lack of differences found between the Normals and the three hypertensive groups (BH, EH, and DH) is somewhat
more difficult to explain. Given the existing data on the increased emotional reactivity of Borderline Hypertensives versus Normals, and because the literature is replete with positive findings of a role for emotion in the development of hypertension, it was thought that such differences would be found between Normals and BH and EH respectively when emotion was examined; but this was not the case.

The argument could be made, based on this study and others that have not shown a clear link between emotion and hypertension, that the causes of hypertension must be purely due to genetic or non-psychological environmental influences on blood pressure regulation. However, it remains possible that the role of emotion in the development of hypertension is more subtle in nature. Indeed, the results of this study indicate no one emotion may contribute significantly to the development of hypertension. Perhaps there is an additive effect with each emotion playing a relatively small and difficult to determine role, becoming significant only when all are considered together, or that the effect of psychological factors differ for each individual. It is also possible that the psychological effects cannot be clearly shown in a cross-sectional study such as this one, but can only be seen through longitudinal studies.

There is, in fact, evidence that this is just the case. The possibility that emotion, specifically anxiety, may play a role in the development of hypertension, at least in some
individuals, is supported by the findings of recently published results from the longitudinal Framingham Study (Markovitz, Mathew, Kannel, Cobb, & D'Agostino, 1993). The study followed a group of 1123 initially normotensive individuals for 20 years and looked at a number of emotions including anxiety, trait anger, and anger-in to predict which individuals developed hypertension. The authors concluded that high levels of anxiety in middle-aged men (45-60) was second only to baseline systolic blood pressure as a predictor of future hypertension. No other psychological factor was a significant predictor of future hypertension, nor was anxiety a predictor for women or younger men.

Hypothesis 3 was partially confirmed. There was a difference in the difference of office v. home readings across the groups for systolic blood pressure, but not for diastolic pressure. Further, the finding of overall differences in the difference between home and office systolic readings is suspect when closely analyzed. Post-hoc pair-wise analyses determined that the only differences to emerge between groups were that BH differed from EH and DH, and that this was due to the fact that the average home systolic reading was actually higher than the average office systolic reading for BH, while it was lower for EH and DH. The former was an unexpected finding, contrary to the "white coat" phenomenon, and there is no apparent explanation in the
present data as to why that phenomenon occurred only for BH, and then only for BH's systolic blood pressure.

Hypothesis 5 was confirmed and the "white coat" phenomenon was found for Borderline Hypertensives' diastolic blood pressure as well as for Essential Hypertensives' and Diabetic Hypertensives' systolic and diastolic blood pressure. Previous studies have found that as many as 2/3 of individuals diagnosed as BH may actually exhibit blood pressure in the normal range when readings are taken at home. An examination of the home and office readings taken in the present study confirm those observations but also found that the borderline essential group had readings for both home and office in the normal range. Specifically, 10 of the 20 borderlines had normal office SBP and DBP, while only two of 20 had both abnormal SPB and DBP. In the present study, BH evidenced lower home readings only for DBP.

The fact that most BH had normal office DBP raises the question of why the diagnosis of borderline essential hypertension was made. There are several possible explanations. Given the prevalence of hypertension in our society and the extreme adverse consequences which can result from this disease, physicians may be medically conservative by making liberal diagnoses of borderline essential hypertension. Morbidity data clearly show that individuals who carry the diagnosis of borderline hypertension are at greater risk for heart disease than those individuals who do
not receive the diagnosis. Another reason for the apparent normal office and home readings among the borderlines could be that the diagnoses may have been based on other information available to the physician, such as family history, information which was not obtained for the purposes of this study. Further, the availability of no-cost non-invasive treatment such as dietary changes and exercise may predispose the physician to make a diagnosis of borderline essential hypertension even for patients who may show few measurable signs of the disease.

In examining home BP readings versus office BP readings for the essential and diabetic hypertensives the phenomenon was clearly in evidence, as well over 2/3 of both EH and DH who had office blood pressure readings in the abnormal range had normal blood pressure readings when measured at home. This is the first study to provide evidence that "white coat" hypertension may occur not only in individuals diagnosed as Borderline Hypertensives, but in individuals diagnosed as Essential and Diabetic Hypertensives as well. These findings may be of some significance. If replicated, it is possible that attending physicians could have not only BH, but also EH and DH perform home blood pressure readings to determine if they show the same high readings outside of the office. If they do not, it is possible that anti-hypertensive medication might be adjusted accordingly. These findings may also hold significance for future research examining the relationship
between psychological stress and hypertension. The results of this study indicate that the setting in which blood pressure readings are taken (home v. office) has a great influence on the readings. It is important to determine whether lower home blood pressure readings following a particular intervention, such as relaxation training, are the result of the intervention, or are the result of the setting in which the readings were taken.

The concepts of stimulus response specificity and classical conditioning may provide a framework for explaining the white coat phenomenon. As discussed earlier, studies of psychophysiological responses to emotion-provoking stimuli (Ax, 1953; Lacey and Lacey, 1959) have suggested that at least some emotions can be distinguished physiologically on the basis of cardiovascular and other physiological response patterns. This work led to the formulation of the concept of stimulus-response specificity, that certain classes of stimuli bring forth certain patterns of autonomic nervous system (ANS) response across individuals. In other words, a particular stimulus tends to evoke a specific response pattern across individuals.

The experience of having one's blood pressure taken in the doctor's office is a stimulus that may elevate blood pressure levels via anxiety or frustration/anger responses to past readings, to anticipated readings themselves, or to their implications. Thus it is possible that the physician's
office, BP equipment, and even his or her white coat may serve as conditioned stimuli (CS) which elicit the affective response of anxiety or frustration/anger part of which may be increased BP.

Hypotheses 2 and 4 postulated that correlations would be found between the five psychological measures and blood pressure level and variability measurements taken at home, and that there would be correlations between each of the five psychological measures and the differences between home and office readings. Positive findings were not obtained for either of these hypotheses, nor were any correlations found between blood pressure readings and the five psychological measures. This attempt to establish a more precise relationship between emotion as measured at one point in time, and multiple blood pressure readings taken during the same time span did not produce statistically significant results. However, the possibility remains that emotion does play a role in the development of hypertension, but the relationship is a subtle one that develops over a considerably longer period of time than can be measured in a cross-sectional study such as this one.

Hypertension is an exceedingly complex illness. Biological data support the role of genetics, weight, diet, and a myriad of other factors as important etiological variables. Simply because this and other studies have not yet provided a definitive role for the contribution of
psychological factors in the etiology of hypertension does not mean that a meaningful role does not exist, as some have maintained. As concluded in a recent editorial in the Journal of the American Medical Association: (Pickering, 1993):

That the existing data supporting these views [the role of psychological factors in the development of hypertension] are conflicting should come as no surprise, but it should also be stressed that such confusion in no way invalidates the concept. The role of salt intake in the development of hypertension continues to be debated just as hotly, yet the quantification of dietary salt is trivial in comparison with the problems of measuring personality. (P.2494).

One of the major difficulties inherent in cross sectional research relating personality to hypertension is the problem of separating cause and effect. It is difficult to determine whether an association between a psychological variable such as anxiety and a life threatening chronic disease such as hypertension is causal or is a psychological consequence of the disease. Another problem in conducting this type of research is that while measuring personality characteristics by questionnaire removes observer bias and allows for better reliability, it has a disadvantage in that it relies exclusively on self-report, allowing subjects to portray themselves as they would choose to be perceived.

Additional longitudinal research, of the type done in the Framingham study, should be designed to determine which emotions may play a role for which individuals in the subsequent development of hypertension. It is possible, if not probable, that anxiety may play a role for some hypertensives while anger may play a role for others and both
may be a factor for still others. Certainly both emotions generate considerable cardiovascular activation. Behavioral therapies, such as relaxation training, biofeedback, and other interventions have not been useful for all hypertensive patients (Jacob, Chesney, Williams & Ding, 1991), but better results may be obtained by selecting anxious hypertensives for this type of treatment. Similarly, should anger appear to be a relevant factor in the development of hypertension in some individuals, anger management strategies may prove more beneficial.
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Vita

Alicia Gail Pellegrin was born September 5, 1958 in Baton Rouge, Louisiana. She attended public schools in that city and graduated from high school in 1976. She attended Louisiana State University and received a B.A. in English in May, 1981. After working as a salesperson for several years, she returned to school and received an M.S. degree in Counseling Psychology from the University of Southern Mississippi in August, 1987. She then entered private practice as a psychometrician under the supervision of Donald Hoppe, Psy.D. in Baton Rouge, where she has since worked, and began studying for her qualifying exams for the doctoral program at Louisiana State University. She entered that program in January, 1989. Ms. Pellegrin served a year of pre-doctoral internship at the Veterans Medical Center in Houston, Texas beginning in August 1991. She has taught freshman and sophomore psychology courses at Southeastern Louisiana University in Hammond, Louisiana and in the Harris County Community College system. She is the author or co-author of three articles and two presentations at state professional conventions. Ms. Pellegrin is married to Robert Pellegrin and is the mother of a fourteen year old daughter, Karlye Hoyt, and the step-mother of a nineteen year old son, Michael Pellegrin.
DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Alicia Pellegrin

Major Field: Psychology

Title of Dissertation: Blood Pressure Reactivity and Emotion Among Borderline Hypertensives, Mild Hypertensives, Diabetic Hypertensives, and Normotensives

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

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