

AMBULATORY BLOOD PRESSURE BIOSITUATIONAL FEEDBACK
AND SYSTOLIC BLOOD PRESSURE ESTIMATION

By

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This dissertation is dedicated to my husband and children,
Jeff, Meghan, and Matthew;
in loving memory of Eric A. Wolfe;
and to my three wonderful families,
the McGrogans, the Wolfes, and the Cittys.

Thank you to Jeff for being a wonderful friend, husband, and father.
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TABLE OF CONTENTS

	<u>page</u>
ACKNOWLEDGMENTS	iii
ABSTRACT	vi
CHAPTER	
1 INTRODUCTION	1
Definition and Scope of the Problem	1
Problem Statement	7
Purposes of the Study	8
Hypotheses	9
Definitions of Terms	9
Assumptions	11
Limitations	11
Significance of the Study	11
2 REVIEW OF LITERATURE	13
Theories of Hypertension Development	13
Systolic Hypertension	25
Issues Surrounding the Treatment of Hypertension	26
Biosituational Factors Associated with High BP	31
BP Awareness and Estimation	38
Educational Level and Health Disparities	49
Ambulatory BP Monitoring	50
Summary	52
3 PROCEDURES AND METHODS	53
Research Design	53
Population and Sample	53
Inclusion and Exclusion Criteria	55
Setting	56
Research Variables and Instruments	56
Study Protocol and Procedures	63
Methods of Statistical Analyses	68

4	RESULTS	71
	Descriptive Results	71
	Analytic Results for Hypotheses	75
	Hypotheses	78
5	DISCUSSION AND RECOMMENDATIONS	93
	Discussion of Results	93
	Conclusions	105
	Implications for Clinical Practice	106
	Recommendations for Future Research	108
 APPENDIX		
A	PRE-/POSTTRAINING SBP ESTIMATION FORM	110
B	SBP ESTIMATION STUDY TRAINING FORM	111
C	SBP ESTIMATION STUDY TRAINING FORM	113
D	HEALTH HISTORY FORM	115
	REFERENCES	117
	BIOGRAPHICAL SKETCH	126

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In an age of technological advances and medical breakthroughs, hypertension continues to be a devastating threat throughout the United States and worldwide. From 1987 to 1997, the death rate from high blood pressure increased by 13.1%. Difficulties in detection and treatment exist because hypertension is a relatively silent disease, for which patients are often asymptomatic and are feeling well. Because of the lack of observable symptoms associated with high blood pressure, patients with hypertension often have difficulty prescribing meaning to their disease threat or treatment requirements. The primary purpose of this research was to determine if subjects with hypertension can improve their awareness of their systolic blood pressure after participating in the ambulatory blood pressure and biosituational self-awareness training intervention. A repeated measure, pretest/posttest design was used for this study. Thirty-nine adult hypertensive subjects participated in the study. There were no significant differences among the group of hypertensives after training compared to before training, using a

paired samples t-test. There were, however significant differences in improvement of estimating SBP among the subgroup of college-educated hypertensives ($p = 0.04$) and between the groups who used and did not use antihypertensive medications ($p = 0.05$). Hypertensives who did not take medications showed significant improvement compared to antihypertensive medication users. This study provides support for using feedback methods to improve the ability to estimate BP in certain populations, specifically college educated hypertensives and hypertensives who are not taking antihypertension medications, and suggests that BP awareness may be improved in selected people using feedback methods.

CHAPTER 1 INTRODUCTION

This chapter introduces concepts that are under investigation including the significance of hypertension, problems with detection and treatment of high blood pressure (BP), factors associated with dismal treatment rates, potential manifestations or factors associated with high BP levels, and estimation of BP. This chapter will describe the definition and scope of the problem, the main research problem to be investigated, and the significance of the study. The definition of major terms, assumptions, and limitations will also be described.

Definition and Scope of the Problem

Hypertension is defined as systolic BP (SBP) of 140 mmHg or greater, diastolic BP (DBP) of 90 mmHg or greater, or taking antihypertensive medication. In the United States, people with hypertension comprise a rapidly growing subset of the population. Approximately 50 million Americans have high BP. High BP was the primary cause of death for 44,435 Americans in 1998 and contributed to about 210,000 deaths (American Heart Association [AHA], 2003b). Approximately 95% of people with hypertension have essential (or primary) hypertension, for which no clear cause can be identified. From 1987 to 1997, the death rate from high BP increased by 13.1% (AHA, 2000). Treatment of hypertension continues to be plagued by dismal statistics in that only 27.4% of Americans with high BP are adequately controlled on medication (AHA, 2003a).

Elevated systolic BP (SBP) specifically has been associated with increased morbidity and mortality, especially in the older population. Prospective studies have shown that there is a strong, continuous, graded, independent association between SBP and the risk of coronary heart disease, stroke, and end-stage renal disease (He & Whelton, 1999). Additionally, data from the National Health and Nutrition Examination Survey (NHANES) III found that isolated systolic hypertension was the most frequent subtype of uncontrolled hypertension, especially in subjects over 50 years of age (Franklin, Jacobs, Wong, L'Italien, & LaPuerta, 2001). The incidence and severity of complications increase with the duration and severity of hypertension (Kaplan, 1998; Lackland, 2000). Because of this, it is crucial to identify and treat high BP, and specifically high SBP, in order to reduce the risk of advanced cardiovascular disease and its associated morbidity and mortality.

Inadequate adherence to antihypertensive therapy is a major challenge and contributes to elevated BP levels in two-thirds of all patients with hypertension (JNC VI, 1997). One of the major obstacles in the diagnosis and treatment of hypertension is that it has a very insidious course, which the patient often fails or refuses to recognize because he or she may continue to "feel good." Noncompliance with antihypertensive therapy has been cited as the major cause of treatment failure (AHA, 2003c).

Noncompliance is a multi-faceted issue that results from varying behavioral, social, logistical, economic, and practical factors (Miller, Hill, Kottke, & Ockene, 1997). Failure to comply with prescribed medication regimens or other therapies can affect patients' health adversely as patients may fail to improve, worsen, or relapse. Compliance not only affects the immediate patient but the entire United States health care system and economy. Noncompliance accounts for 100 billion dollars in health care

and productivity costs in the United States. The costs of hospitalizations and practitioner visits caused by noncompliance account for 8.5 billion dollars annually (Task Force for Compliance, 1994).

Several factors have been associated with antihypertensive adherence patterns including whether or not symptoms affect daily life or work, family history of hypertension, household composition, perceived threat of complications, and perceived need and perceived effectiveness of medications (McLane, Zyzanski, & Flocke, 1995; Meyer, Leventhal, & Gutmann, 1985). Because hypertension is generally thought of as an asymptomatic disease and due to the lack of definitive symptoms associated with high BP, it can be difficult for patients to adequately prescribe meaning and importance to their disease process and treatment options (McLane et al., 1995). If high BP were associated with observable symptoms, it may be possible to improve early recognition of the disease, improve its treatment compliance and improve outcomes.

Over the past several years, more attention has been paid to preventive health care and patients have been viewed more in terms of being healthcare consumers and less as being passive participants of the healthcare process. Noncompliance in the patient with hypertension comes in the face of growing consumer empowerment among patients (Skelton, 1997). More than ever, people are trying to improve their health by participating in their care (Roter, Stashefsky-Margelit, & Rudd, 2001). In addition to pharmacologic therapy, biofeedback therapy has been used successfully to assist people in treating and preventing major health problems, such as hypertension, chronic pain, and anxiety (Fernandez & Beck, 2001; Knost, Flor, Birbaumer, & Schugens, 1999; Lal et al., 1998). Biofeedback therapy has been used successfully in both research and clinical settings to lower BP in hypertensive patients (Lal et al., 1998; Yucha et al., 2001). These

therapies would be strengthened if patients were more aware of their high BP or if they had symptoms associated with high BP that could be coupled with therapy. Healthy People 2010 identified goals to advance the prevention, detection, and treatment of hypertension, stroke, and heart disease. To increase public attention, awareness, and treatment, goal number 12-12 states that there “should be an increase in the proportion of adults who have had their BP measured within the preceding 2 years and can state whether their BP was normal or high” (Healthy People 2010, 2003).

As patients with hypertension strive to become more involved in their healthcare decisions, treatments need to be found that focus on the patient as the manager of his/her own health. High BP is a phenomenon that generally is not associated with specific symptoms or signs (AHA, 2003c). Because of this, patients with hypertension often have difficulty understanding the threat of the disease or the treatments required to manage the disease. In disorders with observable symptoms, such as diabetes mellitus, congestive heart failure or seizures, patients may be more motivated to seek and continue treatment.

Little is known about the extent to which hypertensive patients are aware of their high BP; however, several research studies and clinical experiences have shown that people can be more aware of their BP levels after different types of feedback training. If patients were aware of their high BP episodes, better-tailored treatment modalities may be developed and adherence to therapeutic treatment may be improved resulting in better patient outcomes. For example, biofeedback or relaxation therapies could be used to assist patients in lowering their BP during episodes of high BP.

While there is continued controversy over whether there are definitive symptoms associated with BP, it is generally believed that most patients with hypertension cannot accurately tell if their BP is elevated (Fahrenberg, Franck, Baas, & Jost, 1995).

However, some clinicians and researchers report that certain patients are able to detect when their BP is elevated (Barr, Pennebaker, Watson, 1988). These patients often report vague symptoms that are associated with their high BP. Symptoms such as headache, racing heart, sweaty hands, cold/warm hands, tight stomach, muscular tension, dizziness, blurred vision, lightheadedness, tension, palpitations, flushed face, and warm/cold extremities have been correlated with variations in BP (Bulpitt, Dollerly, & Carne, 1976; Pennebaker, Gonder-Frederick, Stewart, Elfman, & Skelton, 1982).

There are many hypotheses behind the development and maintenance of hypertension. These theories provide a framework for understanding how patients with high BP can be helped to recognize the subtle signs and symptoms. One hypothesis of hypertension development is the sympathetic nervous system theory of hypertension development. This hypothesis describes hypertension as a result of over-stimulation of the SNS. To substantiate SNS overactivation, several studies of patients with essential hypertension demonstrate increased levels of plasma norepinephrine and elevated norepinephrine spillover. Patients with borderline and essential hypertension have an increased sympathetic and a decreased parasympathetic drive (Rahn, Barenbrock, & Hausberg, 1999). It is hypothesized, and highly debated, whether sympathetic nervous system (SNS) activation is a trigger for high BP (defense reaction) or if SNS activation is due to a secondary phenomenon (e.g., endothelial or baroreceptor dysfunction).

Alterations and/or uncompensated increases in SNS activity in hypertensives may cause subtle physical signs and symptoms. Increased SNS activity and early hypertension are often characterized by an increased heart rate, cardiac output, and renal vascular resistance. The sympathetic nervous system elicits a “fight or flight” response when confronted with a stimulus, such as when the person is in an emergency or stressful

situation. Additionally, symptoms that are related to increased SNS activity have been reported in hypertensives and have been correlated with high BP episodes (Carels, Sherwood, & Blumenthal, 1998).

Potential manifestations of increased SNS activity include increased heart rate and stroke volume (cardiac output), increased cardiac contractility and venous return, renal retention of sodium and water, increased thirst, increased venous tone, increased angiotensin II, increased peripheral resistance, increased local vasoconstrictors/regulators (e.g., endothelin), increased blood viscosity, and decreased local vasodilators/regulators (e.g., nitric oxide). Symptoms associated with high BP may be related to overstimulation or oversensitivity of the SNS in hypertensive individuals (Esler, 2000; Kaplan, 1998; Rahn, Barenbrock, & Hausberg, 1999).

It has been reported that individuals, both normotensive and hypertensive, estimate their BP levels by using both internal sensory and external situational information (Barr et al., 1988). Estimations and beliefs about BP levels may or may not be accurate, but they are important because people act upon them. In fact, Pennebaker et al. (1982) suggest that variations in BP are correlated to different symptoms and that a person can monitor his or her BP by monitoring symptoms. Interestingly, in studies where both normotensive and hypertensive people were asked to estimate their BP levels, estimated BP was strongly associated with symptoms and moods (Baumann & Leventhal, 1985) and with feelings of physical tenseness and physical activity (Fahrenberg et al., 1995).

Several studies on whether or not people can accurately estimate their BP have been performed. The findings have been fraught with much speculation and conflicting results (Barr et al., 1988; Baumann & Leventhal, 1985; Brondolo, Rosen, Kostis, &

Schwartz, 1999; Cinciripini, Epstein, & Martin, 1979; Fahrenberg et al., 1995; Greenstadt, Shapiro, & Whitehead, 1986; Luborsky et al., 1976; Shapiro, Tursky, & Schwartz, 1970). An important variable among these studies was the addition of a feedback intervention. Among the feedback intervention-type studies, all showed an improvement in BP discrimination after feedback (Barr et al., 1988; Brondolo et al., 1999; Cinciripini et al., 1979; Greenstadt et al., 1986; Luborsky et al., 1976; Shapiro et al., 1970).

Different types of feedback have been used to assist subjects in learning to recognize symptoms, situations, and factors that are associated with their BP levels. Barr, Pennebaker, and Watson (1988) provided normotensive subjects actual biosituational factors (e.g., symptoms, moods, situations) that were related to their SBP levels. They found that 71.4% of the subjects in the biosituational feedback group had significant accuracy correlations compared with 31.3% of the subjects in the control (no feedback) group. Additionally, providing normotensive (Barr et al., 1988; Cinciripini et al., 1979; Greenstadt et al., 1986) and hypertensive subjects' (Brondolo et al., 1999; Luborsky et al., 1976;) knowledge of their actual BP levels has also been used to improve accuracy in estimating BP levels.

Problem Statement

Because of the continued prevalence and incidence of hypertension and its complications, there must be more research focused on testing detection and intervention strategies, as well as improving patient compliance (AHA, 2003c; Miller, Hill, Kottke, & Ockene, 1997). The American Heart Association Expert Panel on Compliance (Miller et al., 1997) reported that a multilevel approach featuring both behavioral and educational

strategies was needed to assist patients and providers in improving compliance (Miller, Hill, Kottke, & Ockene, 1997).

Because the majority of BP feedback intervention-type studies have been performed on normotensive, healthy volunteers, it is unknown whether adults with hypertension can accurately estimate their BP or if this awareness can be improved through BP or biosituational feedback. Specifically, it is unknown if hypertensive adults can estimate their SBP more accurately after participating in ambulatory BP feedback and biosituational self awareness training.

Purposes of the Study

The purposes of the study are as follows:

1. To determine if there are differences in the mean absolute difference (AD) among adult hypertensives after training compared to before training.
2. To determine if there are differences in the mean improvement of estimating SBP among college-educated hypertensives versus noncollege-educated hypertensives.
3. To determine if college-educated hypertensives decrease their mean AD posttraining compared to pretraining.
4. To determine if there are differences in the mean improvement of estimating SBP between hypertensives whose body mass index (BMI) is ≥ 30 and hypertensives whose BMI is < 30 .
5. To determine if there are differences in the mean improvement of estimating SBP between male hypertensives and female hypertensives.
6. To determine if there are differences in the mean improvement of estimating SBP between hypertensives who are < 48 years of age compared with those who are ≥ 48 years of age.
7. To determine if there are differences in the mean improvement of estimating SBP between hypertensives who use antihypertensive medications and those who do not use antihypertensive medications.

Hypotheses

The hypotheses investigated are listed below:

1. Adult hypertensives differ significantly in their mean improvement of estimating SBP after the ambulatory BP awareness training intervention, compared with before the training intervention.
2. College-educated hypertensives differ significantly from noncollege-educated hypertensives in their mean improvement of estimating SBP.
3. College-educated hypertensives decrease their mean AD posttraining compared to pretraining.
4. Hypertensives with a BMI < 30 differ significantly from hypertensives with a BMI \geq 30 in their mean improvement of estimating SBP.
5. Male hypertensives differ significantly in their mean improvement of estimating their SBP compared to female hypertensives.
6. Hypertensives < 48 years of age differ significantly in their improvement of estimating SBP compared to hypertensives \geq 48 years and older.
7. Hypertensives using antihypertension medication differ significantly in their mean improvement of estimating SBP compared with hypertensives not taking medications.

Definitions of Terms

The absolute difference (AD) is defined as the absolute value of the mean difference between actual and estimated SBP. The absolute difference was calculated for mean actual SBP days 1, 2 and 3, and 4 and mean estimated SBP days 1, 2 and 3, and 4.

Actual SBP is defined as that which is measured using the ambulatory BP monitor; it is viewed as a continuous variable with parameters defined as mean, standard deviation, and variance.

Ambulatory BP feedback is defined as those BP readings from the ambulatory BP monitor that can be viewed by the patient on the unblinded LCD screen.

Ambulatory BP monitoring is defined as an automatic, noninvasive cuff-oscillometric recorder (Model 90207, SpaceLabs, Inc., Redmond, WA) which measures ambulatory BP. Subjects wear the ambulatory BP monitor cuff in a similar fashion as a standard manual sphygmomanometer. However, the ABP monitor is preprogrammed via specialized software to automatically measure BP at preset intervals throughout the day and night. The subject wears the cuff around his/her upper forearm and the main unit is strapped around the waist via a strap or belt. ABP monitoring is a reliable and naturalistic method for obtaining BP readings while subjects are in their normal environment.

Biosituational feedback is defined as feedback related to biological, situational, psychological factors that the subject has experienced. Biosituational feedback in this study is provided to the subject by providing the subject with information on their actual SBP, self-reports of their estimated SBP, and self-reports of their moods, symptoms, and activities during BP measurement.

A blinded-LCD screen is the panel on the ABP monitor that displays the time of day, but does not display the physiologic data (i.e., the patient cannot view the BP measurement).

Estimated SBP is defined as that which is estimated by each subject; it is viewed as a continuous variable.

Hypertension is defined as SBP of 140 mmHg or greater, diastolic BP of 90 mmHg or greater, and/or taking antihypertensive medication.

Hypertensive subjects are identified as hypertensive if they have BP readings greater than 140/90 on both of the two screening BP measurements or they are taking antihypertensive medications.

The mean improvement is defined as the absolute value of the mean difference of day 1 (mean actual SBP minus mean estimated SBP) minus the absolute value of the mean difference of day 4 (mean difference of actual SBP minus estimated SBP).

An unblinded-LCD screen is the panel on the ABP monitor that displays the time of day and allows the subject to view the SBP, DBP, and heart rate.

Assumptions

The following assumptions were made in this study:

1. Participants have some knowledge of their health status, including BP and ways to treat high BP.
2. Participants have some opinions about their BP patterns and factors relating to their high BP.
3. Participants have access to various sources of information about high BP.
4. Participants may have symptoms and patterns that are associated with their high BP.
5. People with high BP may have alterations in autonomic nervous system functioning that may predispose them to have symptoms during high BP episodes.
6. Symptoms, patterns, and causes of high BP vary from person to person.
7. Patients with hypertension have variability in their SBP of at least 30-50 mmHg in a 24-hour period.

Limitations

The generalizability of the results of this study is limited to adult hypertensive persons who live in the North Central Florida area. Despite this limited geographic range, the population is believed to be similar to the population of hypertensive persons in other parts of the United States.

Significance of the Study

Hypertension is a major cause of death in the U.S. and worldwide. Only about one quarter of adults with hypertension are being adequately controlled on medications

(AHA, 2003c). Because of this, there will be increased economic burden and increased morbidity and mortality associated with high BP. Hypertension is difficult to treat for a variety of reasons. One issue is that hypertension is a relatively asymptomatic disorder and patients may not even realize that their BP is elevated. Because there has been limited research inquiry into hypertensives' awareness of their BP levels, it is generally unknown whether hypertensives can improve their ability to estimate their BP. Research has indicated that SBP is an important determinant to the risk of coronary heart disease, stroke, and end-stage renal disease (He & Whelton, 1999). Because of the importance of SBP prediction and modification, this study examined the ability of adult hypertensive persons to estimate their SBP before and after a biosituational feedback training intervention. A better understanding of estimation of SBP among hypertensives will encourage researchers to study and develop new and better-tailored treatment modalities. This study also utilized ABP monitoring and a self-report diary to assist adults with hypertension to learn more about their BP patterns and associated factors. Finally, this study examined differences between different groups of hypertensives. This information will shed light on potential sub-groups that may be better or worse at estimating their SBP and may encourage a more focused inquiry into SBP estimation and biosituational feedback training. Additionally, if hypertensives can improve their ability to estimate their SBP levels, there may be improved adherence to medications, improved hypertension therapies, and improved outcomes.

CHAPTER 2 REVIEW OF LITERATURE

This chapter will present a literature review of the following areas of research: theories of hypertension development, systolic hypertension, role of sympathetic nervous system in hypertension, issues surrounding the treatment of hypertension, biosituational factors associated with high BP, BP estimation, and ambulatory BP monitoring. A summary linking these areas together to provide a research rationale for this study will conclude this chapter.

Theories of Hypertension Development

There have been several mechanisms that have been implicated in hypertension development. These mechanisms include impaired baroreceptor function, increased sympathetic nervous system activity, impaired endothelial function, and/or structural-adaptive changes in the vascular walls.

Impaired Baroreceptor Function and Baroreceptor Resetting

The baroreceptor mechanism in the central nervous system assists with the regulation and control of arterial pressure. Baroreceptors are nerve endings that lie in the walls of large arteries and are stimulated when stretched. This reflex is initiated by pressure-sensitive receptors, located in the walls of the large arteries of the neck and thoracic regions, carotid artery, and the aortic arch. The baroreceptors respond rapidly to acute drops or elevations in BP. The baroreceptor signal is transmitted, enters the medulla, and stimulates either the sympathetic nervous system (SNS) (if BP is too low)

or the parasympathetic nervous system (PNS) (if BP is too high). Stimulation of the SNS promotes the secretion of both norepinephrine (NE) and epinephrine and causes vasoconstriction in vascular smooth muscles and blood vessels and increased strength of heart contraction. Stimulation of the PNS would promote the secretion of acetylcholine and cause vasodilation of the veins and arterioles and decreased heart rate and strength of contraction. The baroreceptor mechanism is an extremely powerful and effective entity within the nervous and cardiovascular systems for short-term regulation of BP (Chapleau, Cunningham, Sullivan, Watchel, & Abboud, 1995; Harrington, Murray, & Ford, 2000; Seeley, Stephens, & Tate, 1998).

One of the problems with the baroreceptor system in the long-term regulation of arterial pressure is that the baroreceptors are continually “reset” after 1 to 2 days of prolonged pressure exposure. Consequently, they are only effective if the change in BP is acute or not prolonged. For example, if the pressure rises from the normal 100 mmHg to 170 mmHg, there would be an acute and immediate response from the baroreceptor reflex (vasodilation). The rate of impulse firing is rapid and extremely acute, and then diminishes over the course of a few seconds. The rate of impulse firing continues to decline over a period of 1 to 2 days until ultimately the rate of firing ceases, despite the fact that the arterial pressure remains at 170 mmHg. Thus, the baroreceptor has been “reset” to be accustomed to a consistently high BP level (Chapleau et al., 1995; Guyton & Hall, 1996).

It is interesting to note that studies have shown that young, mild or borderline hypertensive patients have an increase in BP variability and skeletal muscle sympathetic nerve activity, and display increased baroreceptor activity. This may be a compensatory finding that is associated with increased sympathetic nerve activity, whereby the

baroreceptors are attempting to adjust the BP toward more normal levels. In established hypertension associated with myocardial hypertrophy and decreased myocardial stretchability, baroreceptor function has been shown to decline (Chapleau et al., 1995).

The baroreceptor reflex may also not be effective in long-term regulation of BP because of the structural and functional changes that are seen in the blood vessels of patients with hypertension. Because of the anatomical location of the baroreceptor nerve endings, dysfunction of the vessel lumen/endothelium may decrease the baroreceptor pressure-sensor effectiveness (Chapleau et al., 1995; Seeley et al., 1998).

Another problem of short- and long-term baroreceptor regulation of arterial BP is that even under conditions of “normal” aging, baroreceptor function and other cardiopulmonary neural regulatory functions have been shown to be less effective with age. In animal studies, the effects of administration of acetylcholine on heart rate (i.e., bradycardia) are more pronounced in elderly normotensive subjects than in younger controls. The baroreceptor control of BP in normal subjects is reported to be comparable to that of the younger controls; however, the response to the stimulus (either high or low BP) is sluggish and slower. Thus, baroreceptor control of BP becomes impaired with the aging process, however to a lesser degree than heart rate regulation. Studies have also shown that there is impairment in the cardiogenic stretch receptors located in the cardiopulmonary region that are associated with aging (Chapleau et al., 1995; Fauvel et al., 2000; Giannattasio et al., 1994). Impaired baroreceptor function and baroreceptor re-setting may lead to uncompensated increases or decreases in BP. It remains unclear whether baroreceptor dysfunction is the cause or effect of hypertension.

Sympathetic Hyperactivity Theory

In the sympathetic hyperactivity theory of hypertension, hypertension is caused by an abnormally increased stimulation of the sympathetic nervous system. Increases in catecholamine stimulation effect BP by increasing heart rate, stroke volume, and peripheral resistance. Factors that may be associated with increased sympathetic outflow and increased total peripheral vascular resistance in essential hypertension include baroreflex re-setting; genetic composition; stress; altered renin-angiotensin-aldosterone mechanisms; alterations in circulating hormones/substances; structural-adaptive changes in vascular walls; endothelial dysfunction; endothelial derived relaxing and contracting factors; and membrane and intracellular mechanisms, including impaired adrenergic receptor numbers and types. The increase in SNS activity stimulates the release of catecholamines to effect specific target organs including the vascular smooth muscle, blood vessels, kidneys, and heart. Effects of increased SNS activity include increased heart rate and stroke volume (cardiac output), increased cardiac contractility and venous return, renal retention of sodium and water, increased thirst, increased venous tone, increased angiotensin II, increased peripheral resistance, increased local vasoconstrictors/regulators (i.e., endothelin), increased blood viscosity, and decreased local vasodilators/regulators (i.e., nitric oxide) (Lilly, 1998).

Folkow (1982) proposed a “defense-reaction” theory of increased sympathetic activity in hypertension. Folkow hypothesized that certain individuals may undergo defense reactions to conditioned stimuli on a daily basis; without the actual fight-or-flight reaction, and this would in-turn cause marked increases in sympathetic activity. If the conditioned stimuli were continually repeated, adverse structural adaptive changes of the arterioles would occur, thus leading to the further development of sustained hypertension

(Brondolo, Karlin, Alexander, Borrow, & Schwartz, 1999; Carels et al., 1998; Folkow, 2000; Wright & Angus, 1999).

In numerous studies of young patients with essential hypertension, it has been shown that there are increased levels of plasma norepinephrine and elevated norepinephrine spillover (Esler, 2000; Grassi et al., 2000; Rahn et al., 1999). In a study by Egan, Panis, Hinderliter, Schork, & Julius (1987), mildly hypertensive young humans had elevated plasma norepinephrine levels and enhanced skeletal muscle vasoconstrictor tone. These findings provide understanding of the hemodynamic profile of early human hypertension, which is characterized by increased heart rate, cardiac output, and renal vascular resistance. Increased sympathetic activity has also been shown to be a factor in elderly hypertension. In a study by Grassi et al., (2000), muscle sympathetic nerve activity was increased in 20 untreated elderly essential hypertension patients compared with age-matched controls. In addition to subjects with existing hypertension, normotensives with a family history of hypertension have higher rates of norepinephrine spillover into arterial plasma than do normotensives without a family history of hypertension. This finding may be a contributing factor and provide a link for the later development of hypertension. It was also reported that patients with accelerated essential hypertension have significantly higher levels of muscle sympathetic nerve activity than do patients with milder hypertension. There have been several proposed mechanisms for increases in muscle sympathetic nerve activity in essential hypertension. One such proposal is that increases in muscle sympathetic nerve activity may be related to increased central nervous system sympathetic outflow. Another such hypothesis is that patients with essential hypertension have impaired baroreflex sensitivity (Mark, 1996).

Increased sympathetic activity and enhanced reactivity to stress have been reported in patients with both borderline and established hypertension, and it has been suggested that they play a role in the pathogenesis of hypertension. The mechanism for these enhanced responses is unknown; however, it has been suggested that epinephrine, released from the adrenal medulla during physiological stress, is taken up into the sympathetic nerve terminal and later released as a co-transmitter with norepinephrine. The norepinephrine that has been released further stimulates norepinephrine release through its action on the presynaptic B-adrenergic receptors. In a recent study, hypertensive subjects had a 25% higher rate of whole body spillover of norepinephrine to plasma, compared to normotensive controls. Additionally, the epinephrine secretion rate was increased in hypertensives (215 +/- 209ng/min) versus normotensives (173 +/- 115 ng/min). These findings provide evidence the epinephrine may prolong and amplify the sympathetic responses at a time when circulating epinephrine concentrations are no longer elevated (Rumantir et al., 2000; Stein, Nelson, He, Wood, & Wood, 1997).

There have also been studies that demonstrate differences in SNS activity among subsets of the population. Stein, Lang, Singh, He, and Wood (2000) reported that healthy, normotensive black males (compared to age-matched white males) had markedly increased levels of vascular sensitivity to an infusion of the alpha-adrenergic vasoconstrictor substance, phenylephrine (Stein et al., 2000). This study concluded that increased sympathetically-mediated vascular tone caused by enhanced vasoconstriction and attenuated vasodilation may play a role in the pathogenesis of hypertension in blacks. It has also been reported that obese-normotensive and obese-hypertensive subjects have impaired adrenergic and baroreflex function. In a recent study, Grassi et al. (2000) reported that muscle sympathetic nerve activity is significantly

increased in lean hypertensive and overweight normotensive subjects ($p = 0.01$), compared to lean normotensive control subjects. Additionally, obese-normotensive and obese-hypertensive subjects had impaired baroreflex cardiovascular control, as measured by the infusion of vasoactive drugs (nitroprusside and phenylephrine) and the response of each substance. This study concluded that the association between obesity and hypertension triggers a sympathetic activation and an impairment in baroreflex control mechanisms (Grassi et al., 2000; Julius, Valentini, & Palatini, 2000).

Endothelial Dysfunction

The endothelium is closest to the arterial lumen, in the intimal layer, and intimate with blood flow. In the normal artery, the endothelium functions to maintain the integrity of the vessel wall by performing various metabolic and signaling functions. The endothelium functions to (a) act as a barrier and protect subendothelial space, (b) express antithrombogenic substances (heparin, thrombomodulin, plasminogen activators), (c) secrete vasoactive substances that promote vasodilation (endothelium-derived relaxing-factor and prostacyclin), and (d) inhibit smooth muscle cell migration and proliferation by secretion of heparin and endothelium-derived relaxing factor. Atherosclerotic lesions develop within the intimal layer (Lilly, 1998; Luscher, 1994).

Over the last several years, increasing attention has been paid to a substance secreted by the endothelium known as Endothelium-derived-relaxing-factor (EDRF), also known as Nitric Oxide (NO). In addition to its vasodilatory properties, NO is known to inhibit platelet aggregation and adhesion, monocyte adherence and chemotaxis, and proliferation of vascular smooth muscle cells. Endothelium-derived nitric oxide, a potent vasodilator, may be an endogenous antiatherogenic factor. In animal and human models, vasodilation caused by the release of endothelium-derived NO is diminished in

atherosclerotic vessels. In addition, hypercholesterolemia independent of observable atherosclerosis inhibits endothelium-dependent vasodilation. In addition to NO, the endothelium also produces potent vasoconstrictor substances including endothelin-1. The expression of endothelin-1 is stimulated by factors including thrombin, angiotensin-II, epinephrine, and the shear stress of blood flow (Chowdhary et al., 2000; Lilly, 1998; Luscher, 1994).

Because of the protective nature of the endothelium, it is important that the integrity of the endothelium be intact. In response to some type of “injury” to the endothelial layer, the endothelium undergoes a continuum of changes that adversely affect the structural and functional physiology of the endothelial surface. Injured endothelium demonstrates increased permeability to large molecules and substances under the subendothelial space, reduced antithrombotic properties and increased vasoconstriction due to decreased secretion of prostacyclin and EDRF-NO, and increased smooth muscle cell migration and proliferation due to decreased secretion of EDRF-NO and platelet-derived growth factor (PDGF). Atherosclerosis is a disease of the muscular arteries (e.g., aorta, coronary and cerebral vessels) in which the intimal layer becomes “injured” and thickened by fatty deposits and fibrous tissue. Elevated levels of serum cholesterol aggravate the vessel endothelium integrity and cause changes within the vessel lumen. The earliest visible lesion of atherosclerosis is a fatty streak characterized microscopically by the subendothelial accumulation of large, lipid-laden “foam cells.” Foam cells are derived from macrophages and smooth muscle cells (SMC’s). Factors involved in monocyte migration and accumulation in the subendothelial space include increased levels of serum cholesterol, especially low-density lipoproteins (LDLs) and oxidized LDLs which encourage the presence of adhesion molecules and chemotactic

proteins. Once in the subendothelial space, the monocytes become activated macrophages and release mitogens and chemoattractants (including tumor necrosis factor, interleukins, complement fragments, PDGF, immune complexes, smooth muscle cell growth factors, and monocyte chemo-attractant proteins) that recruit additional monocytes and promote SMC growth and clot promotion. In advanced disease, a fibrous plaque of SMC origin develops in the intimal layer when there is continual accumulation of monocytes, lymphocytes, foam cells, and connective tissue. Complications occur due to weakening of the vessel wall, ulceration of the vessel wall, occlusion of vessel lumen, thrombosis and distal embolization (Chalmers, 2000; Lilly, 1998; Luscher, 1994; Schwartz, Reidy, & De Blois, 1996).

Hypertension probably is a risk factor of endothelial dysfunction, as increases in sympathetic nervous system activity have been shown to injure vascular endothelium and may increase the permeability of the vessel wall to lipoproteins and other atherogenic factors (Lilly, 1998; Toikka et al., 2000). Because endothelin-1 is stimulated by mechanisms that are affected by increased SNS activity and a majority of patients with hypertension have clinically increased SNS activity, it could be possible that these factors may influence peripheral resistance, and therefore BP. In addition, decreases in endothelium-derived vasodilating and increases in endothelium-derived constricting factors cause an increase in BP and vascular resistance and this may be a risk factor for hypertension development. In a recent study by Park, Charbonneau, and Schiffrin (2001), endothelial dilatory responses to acetylcholine infusion in the brachial artery correlates with the presence of endothelial dysfunction in human resistance arteries. In this study, endothelial-dependent dilatory responses were found to be similar in large and small arteries in hypertensive patients. This conclusion suggests that endothelial

dysfunction may have a systemic rather than a local nature in atherosclerosis and hypertension (John & Schmieder, 2000; Park et al., 2001).

Endothelial dysfunction, atherosclerosis, and/or hyperlipidemia may also precipitate alterations in the integrity of the protective endothelium and thereby increase vasoconstrictor substances, leading to hypertension. Hypothetically, if a person had early atherogenesis and/or hypercholesterolemia but no hypertension, he/she may have impaired EDRF-NO function and increased endothelin-1 stimulation and therefore may have increases in systemic BP (Lilly, 1998; Park et al., 2001).

Baroreceptor function may be modulated by factors such as prostacyclin, oxygen-free radicals, and factors released from aggregating platelets (Chapleau et al., 1995). Endothelial dysfunction and subsequent altered release of these factors contribute significantly to the decreased baroreceptor sensitivity in hypertension and atherosclerosis. Dysfunctional changes in the endothelium may impair baroreceptor function by reducing the stretch mechanisms that provide signals to the autonomic nervous system. Chapleau et al. (1995) reported that the inhibition of endogenous formation of prostacyclin and increased platelet aggregation reduced baroreceptor activity in healthy rabbits. Additionally, oxygen free-radical generation (as seen in atherosclerotic lesions and oxidized-LDL) suppressed baroreceptor activity in the normal carotid sinus (Chapleau et al., 1995).

Structural/Functional Theory

As mentioned previously, various structural and functional changes occur within the vessel wall that may encourage the development and maintenance of hypertension. Structural and adaptive changes that occur in the vessel wall and cardiovascular system include vascular and left ventricular hypertrophy, arterial stiffness, decreased vessel

compliance, and atherosclerosis of the coronary and carotid arteries. In a study comparing age-matched borderline versus normotensive subjects, increased carotid and brachial intima-media thickness was seen in the borderline hypertensive group. In addition, oxidized-LDL was increased in the borderline hypertension group compared with the control group (Toikka et al., 2000). Interestingly, a study of moderately hypercholesterolemic and hypertensive subjects reported that systolic BP and pulse pressures are associated with alterations in increased carotid-intimal thickening (Zanchetti et al., 2001).

Structural and functional changes that occur in the pathophysiological processes of atherosclerosis, SNS overactivation, and endothelial dysfunction can impair baroreflex function (Chapleau et al., 1995), impede blood flow, increase resistance of flow, increase BP, and can encourage a number of advanced adverse complications of hypertension including thrombosis formation, stroke, myocardial infarction, renal failure, retinopathy, and death. It is interesting to note, however, that human vessels can undergo massive accumulations of atherosclerotic plaque without narrowing of the lumen. This may be due to compensatory remodeling of the vessel wall and dilating to permit a normal level of blood flow. In studies of balloon-injured rabbit carotid arteries, researchers found no narrowing of the vessel lumen despite an increase in wall thickness (Schwartz et al., 1996). Increased sympathetic adrenergic activity can also increase arterial stiffness and decrease vessel compliance. Increased workload on the heart induced by hypertension and/or SNS activity causes hypertrophy of the left ventricle and decreased compliance of the ventricle to properly fill and contract blood. The level of arterial pressure exerts an important influence on the level of left ventricular muscle mass. Approximately 20% to

35% of variability in LV mass can be predicted from the level of 24-hour ambulatory BPs (Devereux, de Simone, Ganau, & Roman, 1994).

In summary, it is clear that there are many factors that are related to hypertension development and maintenance. Hypertension development and maintenance is most likely extremely individual and probably a function of a combination of the discussed mechanisms and alterations. Because of the complex nature of the vasculature, circulatory, and neurological systems, each of these theories impacts SNS activity and thereby could promote hypertension development and maintenance.

For the purposes of this study, the SNS hyperactivity theory of hypertension development will be explored as a possible link between high BP and high BP recognition. In the SNS hyperactivity theory of hypertension, high BP is caused by an abnormally increased stimulation of the SNS. The exact mechanism for increased SNS activity in hypertension is largely unknown, but has been speculated by researchers (Folkow, 1982). As mentioned previously, the increase in SNS activity stimulates the release of catecholamines to affect specific target organs including the vascular smooth muscle, blood vessels, kidneys, and heart. Stimulation of the SNS causes physiological manifestations, such as racing heart, pounding chest, increased BP, and dilated pupils.

Studies show that there are increased levels of plasma norepinephrine and elevated norepinephrine spillover in essential and borderline hypertension, seen in both younger and older hypertensives (Egan et al., 1987; Esler, 2000; Grassi et al., 2000; Rahn et al., 1999;). Because age has been shown to be a factor in increased SNS activity, it would seem plausible that adults of increased age or younger borderline hypertensives would have increased SNS output and therefore potentially more manifestations of SNS activity. Similarly, adults who are obese have been shown to have impaired adrenergic

and baroreflex function (Grassi et al., 2000). Therefore, obese adults may have physical signs or symptoms associated with BP elevation. Whether or not this activity occurs only during a high BP episode or if it occurs more consistently is unknown. It is also unknown whether obese or elderly hypertensives have an increased recognition or awareness of high BP or high levels of sympathetic activity.

Systolic Hypertension

The majority of persons with systolic hypertension are not adequately controlling their BP levels despite persuasive data from clinical trials documenting the benefit of treatment (JNC VI, 1997, p. 6). Systolic BP has been identified as a major measure in the assessment of risk in hypertensive subjects (Lackland, 1999). Observational epidemiologic studies and randomized controlled trials have demonstrated that SBP is an independent and strong predictor of risk of cardiovascular and renal disease (Franklin et al., 2001; He & Whelton, 1999). Recent data from the Systolic Hypertension in the Elderly Program (SHEP) have indicated a clear benefit of treatment with a reduction in total stroke of 36%, and a reduction of 25% and 32% in the combined end points of coronary heart disease and cardiovascular disease, respectively (Silagy & McNeil, 1992).

SBP levels have been shown to covary more with physical symptoms than either DBP or heart rate (Pennebaker et al., 1982). From a perspective of training patients to recognize high BP episodes, it has been shown that discrimination of systolic pressures occurs at a slightly faster pace than diastolic pressures (Cinciripini et al., 1979). It also may be easier for subjects to understand the estimation task as well as minimize confusion between SBP and DBP levels, thereby increasing the reliability of the SBP estimate. Because of the importance of SBP as a predictor in long-term outcomes and

the ease of conceptualization, it is valuable to solely examine the ability of hypertensive persons to estimate their SBP levels.

Issues Surrounding the Treatment of Hypertension

Overview of Treatment Statistics in High BP

A goal of therapy for patients with hypertension as defined by the JNC VI report (1997) is to reduce BP to nonhypertensive levels with minimal to no side effects. According to recent estimates from the American Heart Association (AHA), one in four U.S. adults has high BP, but because there are no symptoms, nearly one-third of these people don't even know they have it. The current goal for BP is to have BP controlled to less than 140/90 mm Hg. However, it is estimated that only 26.2% of people with high BP are on antihypertensive medications but do not have it under control. For a historical perspective, in 1972, 16% of high BP patients were controlled to less than 160/95 mm Hg, the goal at the time. A recent AHA survey indicated that the control rate for today's goal of less than 140/90 mm Hg is 29% (AHA, 2003a). Thus, it would seem that we are making progress, but we have a long way to go. The economic burden of uncontrolled hypertension is immense. For example, researchers estimated the number of cases and costs of myocardial infarction, stroke, and congestive heart failure for patients achieving BP control versus those not achieving control. For the U.S. population with hypertension, inadequate BP control was estimated to result in 39,702 cardiovascular events, 8,374 cardiovascular disease deaths, and \$964 million in direct medical expenditures. Within the medicated population with cardiovascular disease, the incremental costs of failure to attain BP goals reached approximately \$467 million. These results reflect the importance of adequate BP control, in particular, systolic BP control, in reducing cardiovascular

morbidity, mortality, and overall health care expenditures among patients with hypertension (Flack et al., 2002).

Poor adherence to antihypertensive therapy is a major therapeutic challenge contributing to the lack of adequate control in more than two-thirds of patients with hypertension (Miller et al., 1997; JNC VI, 1997). Compliance is often defined as implementation (by the patient) of the therapeutic plan that has been established (Anderson et al., 1994). Nearly three-fourths of adults with hypertension are not controlling their BP to below the recommended 140/90 mmHg (JNC VI, 1997). Noncompliance is a multi-faceted biobehavioral issue that may be related to factors such as economics, past history, perception of illness threat, effect illness has on daily activities or work, presence of symptoms associated with the illness, and perception of efficacy of therapy. Patients with chronic illnesses, especially hypertension that presents few recognizable symptoms if any, often have difficulty prescribing meaning to their illness. Therefore, these patients have problems complying with their therapeutic plans (Meyer et al., 1985; McLane et al., 1995). If patients with hypertension can learn to recognize symptoms or factors that are associated with their high BP and learn to recognize when their BP is high, their compliance with prescribed therapy and motivation to seek or continue treatment may improve.

At the same time that hypertensives are having problems with adhering to treatment regimens, people throughout the world are beginning to embrace an emerging trend called “self-managed care.” Self-managed care is a term used to describe the act of maintaining one’s own health and well-being (Strohecker, 1999). Individuals today are looking to manage their own health by becoming empowered and being vigilant healthcare consumers. Because of the recommendation by the Healthy People 2010

campaign to improve patients' awareness of their BP levels and to improve the percentage of people who know if their BP level is low, normal, or high, it is clear that it would be beneficial for patients to have increased knowledge of their BP levels and factors associated with their high BP. With this in mind, it makes sense that the major health care organizations and programs are encouraging patients to have increased awareness of BP levels and to use automatic home BP monitors to assist in the management of hypertension (AHA, 2003a; Healthy People 2010, 2003; JNC VI, 1997). Educational level has an impact on health and health outcomes, as educated people have been shown to be healthier and have more improved outcomes to treatments, whereas people of lower socioeconomic status tend to have more adverse risk factors and worse health (Winkleby, Fortmann, & Barrett, 1990). It seems reasonable that if patients were more aware of their high BP episodes and factors associated with them, they would be more motivated to seek and/or continue treatment (Meyer et al., 1985; McLane et al., 1995). Additionally, learning to recognize high BP may provide a means to teach patients to use relaxation, biofeedback, and/or pharmacologic therapies as a means of reducing elevated BP levels, thereby improving treatment outcomes.

Antihypertensive Medications

Medications, known as "antihypertensive medications," are available to treat chronic high BP. There are various types and classes of antihypertensive medications. Each type of medication works at a different site of action in the body to lower BP. Each medication has potential side effects that may occur with use of the medication. Often, antihypertensives are used alone or in conjunction with other antihypertensive medications. Because of the complex nature of hypertension, often two or more drugs or therapies are needed to control BP to a normal level. The JNC VI report on Prevention,

Detection, Evaluation and Treatment of High BP recommends that a diuretic and/or beta blocker be chosen as initial therapy for hypertension, unless there are specific contraindications or reasons to choose otherwise (JNC VI, 1997).

Diuretics are a type of medication used to treat hypertension and a variety of other illnesses that work by acting to increase urine output, thereby decreasing BP. Diuretics inhibit sodium reabsorption and affect electrolyte excretion in a particular nephron segment. Different classes of diuretics are available and they are generally classified based on their major site of action within the nephron. Depending on the diuretic class, major sites of action include the proximal tubule, thick ascending limb of the loop of Henle, early distal tubule, and late distal and early collecting tubule. Classes of diuretics include proximal tubule diuretics (Acetazolamide), loop diuretics (Furosemide), thiazide diuretics (Hydrochlorothiazide), potassium-sparing diuretics (Spironolactone), and osmotic diuretics. Diuretics are generally well tolerated and side effects are minimal; however, care should be taken to avoid electrolyte imbalances. Diuretics are frequently used alone or in combination with other antihypertensive medications for the treatment of hypertension (Smith & Reynard, 1995).

Calcium channel blockers (CCBs) are another type of medication that are used to treat hypertension. CCBs block the movement of calcium into the arteriolar smooth muscle and cardiac cells and may inhibit the mobilization of calcium within these cells. In the treatment of hypertension, CCBs act as arteriolar dilators and reduce systemic vascular resistance. CCBs are effective as monotherapy and in conjunction with other antihypertensive medications, especially beta-blockers and central sympatholytics (Smith & Reynard, 1995).

Beta blockers (BB) are also very effective in lowering BP in hypertension. BBs are competitive antagonists for norepinephrine and epinephrine receptor sites in the heart, bronchioles, and blood vessels in the skeletal muscles. The mechanism of BB action is accomplished by blocking the beta receptors in the heart, bronchioles, and blood vessels in skeletal muscle, and promoting vasodilation and decreasing BP. BBs decrease cardiac output, central sympathetic output, presynaptic beta receptor inhibition, and inhibition of renin. Different types of BBs are classified according to their site of action and selectivity of beta receptor sites. Beta-1 selective acting agents are selective for beta receptor sites in the heart. For example, two agents that are relatively cardio-selective include Metoprolol and Atenolol (Smith & Reynard, 1995).

Another type of antihypertensive medication is the angiotensin-converting enzyme inhibitors (AI). AIs are generally well tolerated and the most common adverse effect is chronic cough. The mechanism of action of AIs is on the Renin-Angiotensin-Aldosterone System (RAAS). Briefly, the RAAS is a key player in the regulation of human BP. Renin is an enzyme that is found in the kidney and responds to a drop in BP, stimulation of the SNS, or decreased extracellular sodium concentration. Renin is the catalyst for the conversion of angiotensin I to potent, vasoconstricting, angiotensin II. Angiotensin I is converted to angiotensin II by an enzyme found in the lung, angiotensin-converting enzyme. The system assists the body in maintaining BP. In hypertension, where there may be abnormally high levels of SNS activity or abnormal renin activity, AIs work to disrupt the conversion of angiotensin I to angiotensin II (Porth, 1998). Types of AIs include Captopril, Enalapril, Fosinopril, Rimipril, Quinapril, and Benzepiril (Smith & Reynard, 1995).

Biosituational Factors Associated with High BP

Alterations and/or uncompensated increases in SNS activity in hypertensives may cause physical signs and symptoms. As described, increased SNS activity and hypertension are often characterized by an increased heart rate, cardiac output, and renal vascular resistance. These effects increase BP, flush the skin, increase fatigue, increase heart rate, and cause a “pounding or racing” heart (Seeley et al., 1998).

The SNS also promotes numerous metabolic effects throughout the body. These effects include: enhanced metabolic rate of body cells, increases in blood glucose levels, mobilization of fats to be used as fuels, and increased mental alertness via stimulation of the reticular activating system (RAS) of the brain stem. Additionally, increased SNS activity may promote smooth muscle cell growth and increase the likelihood of atherosclerotic lesions and the development and/or acceleration of hypertension (Grassi et al., 2000).

A number of studies suggest that both normotensives and hypertensives have symptoms associated with fluctuations in their BP levels (Dimenas et al., 1989; Pennebaker et al., 1982). In a study by Pennebaker et al. (1982), young, normotensive subjects were evaluated to see if symptoms correlated with fluctuations in BP. Within subject analysis found that 77% of the subjects had at least one significant symptom-SBP correlation. Interestingly, the within-subject correlation varied from subject to subject, indicating that different people perceive different symptoms during fluctuations in BP. Despite the individual variations, however, symptoms of heavy breathing, pounding heart, and fast pulse tended to be high for the majority of subjects. In contrast, another study reported that hypertensive subjects experienced more emotional distress and

cardiac and respiratory symptoms (i.e., sweating, flushing, dry mouth, coughing, dizziness, and dyspnea) (Dimenas et al., 1989).

BP Variability

BP is labile and normally fluctuates in response to both behavioral and biosituational factors. These include activity level, posture, emotional state, communication pattern, bodily function, and internal or external environment. People with hypertension display significantly greater 24-hour variations in mean arterial pressure than do normotensives (Mancia, Di Rienzo, & Parati, 1993). In our laboratory, for example, the range of SBP of 10 hypertensive subjects varied from a minimum range of 19 mmHg to a maximum of 56 mmHg. BP variability is influenced by both biosituational and behavioral factors, presumably through central modulation of autonomic drive to the heart and sympathetic blood vessels. This may be due to greater pressor responses to emotional and other behavioral stimuli due to an increased central emotional reactivity in essential hypertensives (Esler, 2000).

Factors such as dietary intake, gender, ethnicity, alcohol/caffeine intake, stressors, seasonal variations, circadian fluctuations, cocaine and similar drug use, tobacco use, or others may effect BP fluctuations (Campbell, McKay, Chockalingam, & Fodor, 1994; Gellman et al., 1990). Brondolo et al. (1999) noted similar findings when they investigated the effects of workday communication patterns on physiologic parameters. It was found that naturally occurring interpersonal interactions were associated with increases in SBP and heart rate.

Headache

Several studies have assessed whether or not the symptom “headache” was related to BP levels. Kruszewski, Bieniaszewski, Neubause, and Krupa-Wojciechowski

(2000) reported that although 30% of stage 1 and 2 hypertensive subjects (N = 150) experienced headache during 24-hour ABPM, headache was not associated with BP elevations, mean BP levels were not significantly higher than those during headache-free periods, BP means 1 hour before and 1 hour after the headache were not significantly different, and in the majority of hypertensives, the maximal BP values were recorded outside the headache periods. Dimenas et al. (1989) similarly reported that hypertensive subjects did not complain of headaches, as compared to other studies which show that headache is more frequent in patients with hypertension (Bulpitt, Dollery, & Carne, 1976). Headache has been speculated to be related to increased pressure and stretching of the vessels of the dura at the base of the brain (Seeley et al., 1998).

Mood/Communication Pattern

Mood has been reported to be associated with BP. Positive mood accounted for 6% of the within subject variance for systolic and diastolic BPs (Gellman et al., 1990). Negative mood accounted for 8% of the within subject variance for systolic and diastolic BPs. The BPs were generally higher during the positive and negative mood states and were lowest during a neutral mood state. Mood was classified into three categories: (a) neutral mood (i.e., content); (b) negative mood (i.e., tense, annoyed, upset, angry); and (c) positive mood (happy and smiling). In previous studies, it was reported that primarily negative mood was associated with increases in BP (Brondolo, Karlin, Alexander, Borrow, & Schwartz, 1999; James, Yee, Harshfield, Blank, & Pickering, 1986).

Communication patterns have also been associated with increases in BP. Brondolo et al., (1999) reported that interacting with the public, supervisor, or coworker within the prior 15 minutes of BP measurement had a stimulatory effect on BP and cardiovascular reactivity in normotensives and hypertensives. Elevated BP responses to

positive or negative mood or communication patterns may elicit a cardiovascular response, similar to the defense reaction hypothesis proposed by Folkow (1982).

Anger, Hostility, Stress, and Anxiety

Durel, Carver, Spitzer, Llabre, Weintraub, and Saab (1989) examined BP levels and dispositional anger and hostility in 135 African Americans and Caucasian male and female normotensives and unmedicated mild to moderate hypertensives. Using ABPM, this study revealed that cognitive anger and state-trait anxiety were strongly associated with higher SBP and DBP levels at work. In this study, women showed significant positive relationships between hostility, anger, and anxiety and elevated BP at work. Male subjects showed no association between anger measures and ABPM levels. Shapiro, Goldstein, and Jamner (1996) examined the association between cynical hostility, anger, defensiveness, and anxiety on BP in African American and Caucasian college students. This study reported that high-hostile African American subjects had higher SBP during the day and at night compared to high or low hostility Caucasian subjects. African American subjects who scored high on both anxiety and defensiveness had higher waking DBP. These studies suggest that there is an association between anger and hostility and higher BP levels. Additionally, these studies suggest that gender, ethnicity, type of self-report instrument, activity, and other personality traits may influence the association (Carels et al., 1998). Factors such as anger and stress have been shown to effect the “fight or flight” response, thereby increasing catecholamine release and subsequent SNS effects (Seeley et al., 1998).

Environment

A stressful home environment can cause elevations in BP similar to those seen in the work environment (Blumenthal, Towner, Thyrum, & Seigel, 1995; Carels et al.,

1998). Blumenthal et al. (1995) reported that married women had significantly higher BP levels than unmarried women, but married and unmarried men had similar pressures. In a study by Schnall et al. (1992), 262 employed males were studied and it was found that social support did not affect BP independently, but the association of job strain with DBP was stronger for the subjects who had low levels of social support.

Mild hypertensive subjects have also been shown to have greater home versus work differences in BP, as compared to normotensives (Durel et al., 1989; Gellman et al., 1990). Additionally, Durel et al. (1989) found that there was a significant correlation among Caucasian and African American women between work related hostility and anger and BP. This finding may be related to increased or augmented SNS activity in response to stressors seen in patients with hypertension.

Work characteristics, such as perceived psychological job demands and decision latitude, may contribute to work-related stress. Job strain is defined as “a combination of high psychological demands together with low decision latitude.” At least 12 studies have examined job strain and ABPM in a naturalistic environment (Carels et al., 1998). Theorell, Perski, Akerstedt, Sigala, Ahlberg-Hulten, and Svensson (1988) examined 73 normotensive men and women in six different occupations and found increased SBP during work hours among those reporting high job strain, relative to those reporting low job strain. Other studies examined hypertensive and normotensive subjects and discovered that job strain was related to increased SBP and DBP at work, home, and during sleep (Schnall, Schwartz, Landsbergis, Warren, & Pickering, 1992; Vrijkotte, van Dooren, & de Geus, 2000). Elevations of BP at home, work, or stressful job environments may be related to activation of the SNS and the “fight or flight” response (Brondolo et al., 1999).

Posture

Various postural positions effect BP levels. For example, in a study performed on 87 normotensive and 44 hypertensive subjects, the effects of posture on BP were examined. It was found that 33% to 47% of the within-subject variance in SBP and DBP could be explained by changes in posture. As subjects in this study went from lying down to sitting to standing, their BP systematically increased (Gellman et al., 1990). The baroreflex mechanism is a possible physiological mechanism for changes seen through the effects of posture. This reflex is initiated by pressure-sensitive receptors, located in the walls of the large arteries of the neck and thoracic regions, carotid artery, and the aortic arch. The baroreceptors respond rapidly to acute drops or elevations in BP. Upon standing, gravitational forces push blood downward and blood flow rapidly decreases from the head and neck regions. Baroreflex stretch receptors sense changes in pressure/stretch and react, causing a rapid increase in action potentials toward the cardiorespiratory center in the medulla to increase pressure.

Physical Activity

BP levels are profoundly influenced by physical activity levels. Acute physical activity and/or exercise increase BP levels (Carels et al., 1998). Over an extended period of habitual exercise, subjects have improved their cardiorespiratory endurance and eventually lower resting BP and control hypertension (Jessup, Lowenthal, Pollock, & Turner, 1998). Physical activity acutely raises BP due to the increased aerobic activity, which increases oxygen demand, blood flow, cardiac output, and BP (Seeley et al., 1998).

Lifestyle Factors: Smoking, Caffeine, and Sodium Intake

Laboratory studies suggest that smoking a cigarette results in an immediate and marked increase in BP. In addition, studies have shown that ABP is higher throughout the day in smokers compared to nonsmokers (Groppelli, Giorgi, Omboni, Parati, & Mancina, 1992), particularly for those smokers who have consumed caffeinated beverages (Narkiewicz et al., 1995). Smokers also tend to have much more BP variability than do nonsmokers. Caffeine increases BP levels and potentiates cardiovascular and neuroendocrine effects of stress in both habitual and light consumers (Lane, Adcock, Williams, & Kuhn, 1990). Hypertensive subjects, in contrast to normotensives, displayed significant increases in SBP and DBP after consumption of coffee. This is due to the vasoconstrictive properties of the drug caffeine (Hartely et al., 2000; Rakic, Burke, & Beilin, 1999). A review of literature on sodium intake and BP reported that higher intake of sodium is associated with higher BP levels. This response may be due to the physiological water-conserving effects of sodium, thereby increasing blood volume and BP (Chobanian & Hill, 2000).

Type A Personality

The Type A individual is characterized by feelings of time urgency, impatience, hostility, aggressiveness, and competitiveness. The Type A personality has been associated with increased risk of coronary heart disease. Type A individuals exhibit higher cardiovascular responses in the natural environment, but only under certain circumstances (i.e., stressful situation, job strain). Type A individuals have higher heart rates and BP levels and greater BP variability than Type B individuals (Carel et al., 1998; Stepoe, 2000). This response is most likely related to Type A individuals having increased reactivity of the SNS and therefore continual “defense reactions.”

In summary, BP is affected by numerous biological, situational, and behavioral factors. Research studies have shown relationships between these factors and BP variability (Brondolo et al., 1999; Carels et al., 1988; Durel et al., 1989; Gellman et al., 1990; James et al., 1986; Lane et al., 1990; Theorell et al., 1988). Despite the growing research literature on relationships between biosituational or behavioral factors and higher BP levels, it is widely held that high BP is a relatively asymptomatic event.

BP Awareness and Estimation

Discrimination of physiological processes has been of interest to researchers for some time. Laboratory procedures have been developed to assess a subject's accuracy of physiological parameters. Discrimination of heart rate, BP, skeletal muscle tension, and blood glucose (Barr et al., 1988; Greenstadt et al., 1986) has been reported.

Discrimination of BP by hypertensive patients is of interest to researchers and clinicians because hypertension is considered a relatively "silent" disease in which immediate sensory consequences are not available to the individual. The development of procedures that facilitate detection of BP changes may be useful in the management of hypertension. According to Cinciripini, Epstein, and Martin (1979), techniques used to facilitate BP discrimination should utilize procedures that are easily applied in the natural environment and not too disruptive to the patient's lifestyle.

In the clinical setting, patients with high BP often report that they can identify when their BP is higher than normal. Often these patients are correct in their awareness and it has led them to receive treatment based on their physiological measurements after subjective reporting. Patients often provide clues to their high BP through such statements as, "I just don't feel right," "I feel pulsing or throbbing in my head," or "I feel hot and tense." While it seems clear that some people are better at sensing high BP,

the question remains as to why some people are able to do this while others are not. One potential hypothesis is that patients with high BP have a higher SNS output and are aware of symptoms relating to this physiologic phenomenon. While there is no direct link between BP estimation and SNS activity, there are studies that show elevated SNS neurotransmitters in patients with high BP (Rahn et al., 1999).

Individuals, both normotensive and hypertensive, may estimate their BP levels by using both internal sensory and external situational information (Barr et al., 1988). Estimations and beliefs about BP levels may or may not be accurate, but they are important because people act upon them. In fact, Pennebaker et al. (1982) suggest that variations in BP are correlated with different symptoms and that a person can monitor his or her BP by monitoring symptoms. In studies where both normotensive and hypertensive people were asked to estimate their BP levels, estimated BP was strongly associated with symptoms and moods (Baumann & Leventhal, 1985) and with feelings of physical tenseness and physical activity (Fahrenberg et al., 1995).

Several studies tested whether or not people can accurately estimate their BP. The findings have been fraught with much speculation and conflicting results (Barr et al., 1988; Baumann & Leventhal, 1985; Brondolo et al., 1999; Cinciripini et al., 1979; Fahrenberg et al., 1995; Greenstadt et al., 1986; Luborsky et al., 1976). An important variable among these studies was the addition of a feedback intervention.

Clinical Relevance of BP Awareness and Estimation

The question of what is a good level of accuracy in estimating BP has not necessarily been answered with a definitive number. However, several studies examine BP and coronary event outcomes. For example, studies assessing the effects of BP reduction and outcomes found significant associations between relatively small

reductions in usual BP (5, 7.5, and 10 mmHg) and 34%, 46%, and 56% less stroke and at least 21%, 29%, 37% less coronary heart disease (MacMahon et al., 1990). Therefore, even incremental changes or awareness in BP may be a good outcome of BP estimation research. Additionally, several studies have found that awareness of BP level is a predictor of health outcomes in patients with hypertension (Asai et al., 2001; Hyman & Pavlik, 2001). Therefore, it is clinically important for patients to be more aware of their health status and BP.

BP Estimation Without a Feedback Intervention

Only two studies address the question “Can people estimate their BP without any type of feedback or training intervention?” Table 2-1 describes the sample descriptions, methods, and findings of each study. In both studies, subjects were generally and statistically inaccurate in estimating their BP correctly. Interestingly, perceived BP was associated with symptoms and moods, rather than with actual BP in a majority of subjects. Although some participants were better estimators than others, no differences among subject characteristics were found (Baumann & Leventhal, 1985; Fahrenberg et al., 1995).

Fahrenberg and his colleagues (1995) assessed whether subjects’ estimation of BP was related to various self-assessments (feeling tense, physical activity, feeling nervous) or actual BP or heart rate. This research inquiry involved 51 hypertensive (defined by WHO criteria) male subjects, ages 22 to 60 years and a second group of 30 volunteer hypotensive or normotensive student subjects ages 20 to 28 years. The hypertensive group was enrolled in a rehabilitation center and was simultaneously receiving exercise therapy, health education, group therapy, and relaxation training. The hypertensive group participated in 3 days of psychological and physiologic monitoring.

The first 2 days were consecutive and the 3rd day was approximately 14 days after the first days. The normotensive/ hypotensive group participated using a SpaceLabs 90207 ambulatory BP monitor (SpaceLabs, Inc, Redmond, WA). Personality assessments and self-evaluations of physical symptoms were also collected. A programmable pocket computer (Casio PB 1000) was used by both groups to estimate their SBP (in mmHg) and record self-report items.

Table 2-1. Research studies: BP estimation without feedback

Authors	Sample description	Methods	Findings
Fahrenberg, Franck, Baas, & Jost (1995)	51 hypertensive males & 30 normotensive male and female students.	BP measured every 30 minutes about 25 times; concurrent diary of estimated BP, physical activity, & subjective states.	Estimated BP & actual SBP were poorly correlated; Self-ratings tense & activity were significantly related to estimated BP.
Baumann & Leventhal (1985)	20 hypertensive & 24 normotensive male & female subjects.	BP measured 2 times per day for 10 days. BP estimated categorically (same, higher, or lower than usual) & assessed moods/ symptoms.	Estimated BP & actual SBP were poorly correlated; 6 out of 41 subjects had significant correlations between SBP & estimated BP; Estimated BP related to symptoms.

Within-subject correlations revealed that estimated BP was not related to actual BP. More extended experience in BP estimation tasks did not enhance the correlation coefficients in hypertensive patients (day 2, $r = 0.32$ and day 3, $r = 0.27$). Estimated SBP was related to self-reports of symptoms and activity. Stepwise regression indicated that self-ratings of tenseness and heart rate predicted estimated SBP in hypertensive patients; however, actual SBP was not related to estimated SBP in any of the regression models.

It is unclear whether subjects had variability in their estimations or actual BP and how often their BP was higher than 140 mmHg (normal BP cut-off point) (JNC VI, 1997). This issue is important because there may be no physiological cues for the patient to refer to if there are not any higher than normal readings. Differences among the hypertensive and student groups may have occurred because of differing settings (i.e., rehabilitation center versus naturalistic environment) and treatments (i.e., rehabilitation environment versus no additional training). Although the findings present insight into awareness of BP, it is premature to generalize these findings to cohorts of either hypertensive or normotensive subjects because of the presence of potential confounding variables and differences among groups (e.g., geographic, treatment, instrument).

Baumann and Leventhal (1985) performed a similar study that assessed three main research questions: (a) whether moods or symptoms are associated with BP in the work setting, (b) whether people are accurate in assessing their BP levels, and (c) whether there are dispositional factors that are associated with people's ability to predict elevated BP. They used a convenience sample and included a heterogeneous group of 44 insurance company employees (20 subjects with hypertension, 24 subjects without hypertension). The subjects' actual BP levels were measured two times per day (in the morning and in the afternoon) for 10 days. The actual BPs were measured using a mercury column Baumanometer and a single tube stethoscope. There were six experimenters who were trained as screener technicians by the Wisconsin Heart Association. Collected data included the following:

- Actual BP.
- Estimated BP level (i.e., categorical variable-higher than usual, same as usual, lower than usual).

- Moods/symptoms (i.e., 10-item mood list and a 12-item symptom list regarding how the subject felt within the last hour).
- Personality measures (i.e., self-esteem scale, private-body consciousness scale).
- BP estimation confidence rating (i.e., 1 = guess, 2 = confident, 3 = very confident).
- Initial interview questionnaire and poststudy questionnaire (i.e., questions pertaining to whether subjects can tell if BP is up or down).

Baumann and Leventhal (1985) found that only 6 out of 41 (15%) correlations of actual to predicted BP were statistically significant ($p = 0.01$) with an accuracy correlation “ r ” of greater than 0.14. It was not clear how the researchers computed the numerical estimated BP levels, as estimated BP in this study was a categorical variable (i.e., higher, same, or lower). The results also show that BP predictions and symptoms were correlated more strongly (56% at $p = 0.05$) than actual BP to predicted BP (15% at $p = 0.05$). Interestingly, subjects claimed to be fairly confident in the BP predictions, with a mean confidence rating of 2.38 out of a possible 3.

In summary, both studies found that people are generally inaccurate in estimating their BP. Additionally, both studies reported that people estimate their BP higher when they are experiencing symptoms that they associate with high BP. These studies provide a glimpse into the question of whether people are aware of their BP.

BP Estimation With a Feedback Intervention

Other studies have been undertaken to answer the questions “Are people accurate in judging their SBP?” and “Does feedback improve estimation?” Table 2-2 describes studies that examined BP estimation and provided subjects some type of feedback intervention.

Table 2-2. Research studies: BP estimation with feedback

Author	Sample	Design/methods	Findings
Luborsky, McClintock, & Bortnichak (1976)	21 male & female subjects, 9 of those taking antihypertension medications	Five sessions of feedback/no feedback, 2 assigned comparison groups.	Feedback group improved after feedback from ± 11.5 mmHg to ± 7.4 mmHg. Accuracy was maintained over 5 phases.
Cinciriprini, Epstein, & Martin (1979)	18 normotensive subjects	BP measured twice daily for 20 days/4 weeks. 2 randomly assigned groups received either feedback or no-feedback.	Feedback group accuracy improved & effects were maintained through conditions.
Greenstadt, Shapiro, & Whitehead (1986)	72 normotensive subjects	Experiment #1: 4 sessions (1 pre, 1 post, 2 feedback training). Experiment #2: 2 sessions (1 feedback, 1 no feedback). Experiment #3: single session of feedback of DBP	Feedback improved estimation of SBP. Initial feedback did not improve subsequent accuracy of BP estimations. Feedback improved estimation of DBP.
Barr, Pennebaker, Watson (1988)	64 normotensive male & female subjects	Experimental design with random assignment to 4 treatment groups (no feedback, internal feedback, external feedback, or biosituational feedback). Estimated SBP & reported symptoms.	43.8% of subjects had significant correlations between actual & estimated SBP after the feedback compared with 26.6% before feedback training.
Brondolo, Rosen, Kostis, & Schwartz (1999)	54 mildly-hypertensive males	Estimated BP & recorded moods/symptoms an average of 7.5 times. Subjects provided prior BP feedback.	Significant within-person association of actual to predicted BP.

Luborsky et al. (1976) performed a study on 21 subjects (16 normotensives, 5 stage-1 hypertensives) to assess the ability of people to estimate their SBP after being given feedback of daily BP information. In this study, mean raw error (absolute value) scores for numerical SBP were compared between baseline and feedback groups. Feedback, in the form of providing the subject their mean previous SBP readings, improved the estimation of SBP by 5 mmHg. The authors concluded that the key to becoming more accurate in estimating SBP is learning your individual range of BP levels.

Cinciripini and colleagues (1979) studied 18 normotensive student volunteers to assess the effects of providing BP feedback on the ability to discriminate systolic and diastolic BP. The subjects were randomly assigned to two groups, one that provided feedback and one that provided feedback after an extended baseline period. Group 1 consisted of feedback (i.e., the mean of two BP readings for that session) that was provided to the subjects for five days in a multiple baseline fashion. The procedure began with an initial screening for BP variability, 5 days of baseline (no feedback), 5 days of feedback, and 10 days postbaseline (no feedback). Group 2 subjects had an extended baseline period followed by a feedback condition. The subjects were asked to estimate their systolic and diastolic BP levels twice a day for 20 consecutive days prior to measuring them using a mercury sphygmomanometer. This study evaluated the difference between estimated and actual BP using the absolute deviation in mmHg between the estimate and actual mean daily BP. Those in group 1 improved their ability to estimate their actual BP after the sequential implementation of feedback. The mean SBP daily deviation score at baseline for group 1 was 9.6 mmHg and after feedback it declined to 5.9 mmHg. This improvement continued after the feedback sessions and was

maintained during the no feedback, postbaseline period. Those in group 2 showed no statistically significant improvement during the extended baseline period; however, their accuracy level improved after the addition of feedback during the last week of training. These subjects improved from a mean SBP daily deviation score of 9.0 mmHg to a score of 3.6 mmHg after feedback was provided (Cinciripini et al., 1979).

Greenstadt and colleagues (1986) performed an experimental study on 72 healthy normotensive volunteers to assess the benefit of discrimination training on the ability of normotensive subjects to detect changing levels of their own BP. Overall, this study concluded that normotensive subjects have relatively no awareness of small BP variations, but that feedback in the form of “knowledge of results” improves BP discrimination.

Barr et al. (1988) studied 64 normotensive subjects for 3 sessions (3 months apart) to assess the effects of internal and environmental feedback on SBP estimation. This study was unique in that it utilized biosituational feedback methods. Biosituational feedback involves providing feedback to the patient regarding internal (e.g., actual BP, symptoms, moods) and external (e.g., environment, posture, diet) factors that occur during the measurement of BP (Barr et al., 1988). In the feedback phase of the study, subjects were randomly assigned to one of four groups: no feedback, symptoms/mood feedback, situational/activity feedback, or biosituational feedback (a combination of the previous two feedback types). Approximately 71.4% of the subjects in the biosituational feedback group had significant accuracy correlations, compared with 31.3% in the symptoms/moods group, 44.4% in the situational group, and 31.3% in the control (no feedback) group.

Brondolo et al. (1999) provided 54 mildly-hypertensive subjects with their SBP range after a baseline period. This study found significant within-subject associations of actual to predicted SBP ($p = 0.002$) and DBP ($p = 0.02$) in 54 mildly hypertensive male subjects. The authors also took into consideration factors that may influence judgments about BP estimation including home BP monitoring and use of medications. The findings indicate that, given some information about their previous BP, subjects display a limited but reliable relationship between their actual and estimated SBP.

In summary, five studies that have provided feedback to people to improve their ability to estimate their BP have shown an improvement in BP discrimination after feedback. Different types of feedback have been used to assist subjects in learning to recognize symptoms, situations, and factors that are associated with their BP levels. Providing normotensive (Barr et al., 1988; Cinciripini et al., 1979; Greenstadt et al., 1986) and hypertensive subjects (Brondolo et al., 1999; Luborsky et al., 1976) knowledge of their actual BP levels has been somewhat successful in improving the accuracy of BP estimation.

Discussion of BP Estimation Studies

Among the feedback intervention-type studies, all showed an improvement in BP discrimination after feedback (Barr et al., 1988; Brondolo et al., 1999; Cinciripini et al., 1979; Greenstadt et al., 1986; Luborsky et al., 1976). In contrast, both studies that did not provide feedback failed to show associations between actual and estimated BP levels (Baumann & Leventhal, 1985; Fahrenberg et al., 1995). However, both studies found relationships between estimated BP and self-reports of physical symptoms and subjective state. Limitations for generalizability include the use of normotensive, young-student, or convenience samples; the amount and frequency of the feedback interventions and/or BP

estimations; and the lack of application to real-life situations and circumstances of American hypertensive patients. If patients can be trained to recognize when their BP is elevated, they may be candidates for some further intervention (e.g., biofeedback training) to help control their BP. However, more research is needed to conclusively state that patients with hypertension are either accurate or inaccurate in estimating their SBP levels.

This review of research provides support for using feedback methods to improve the ability to estimate BP and suggests that BP awareness may be improved in some people using feedback methods. The limited number of studies studying hypertensive persons with high BP suggests that more research is needed to further assess the effects of BP awareness feedback training among this group. Research is needed to evaluate clinical outcomes of BP awareness training, such as BP control, patient motivation and compliance, cost-effectiveness, and morbidity and mortality.

Over the past several years, changes have occurred in health care that have made patients more than mere passive participants of their healthcare. Patients are much more willing and able to learn more about their health and well-being than previous generations (Strohecker, 1999). Teaching people about their BP and BP patterns is an effective way to improve health of patients and empower people with hypertension to have more control over their own life and health (Healthy People 2010, 2003). This review of literature suggests that BP feedback interventions may be an effective means to teach people how to learn more about their BP patterns and when their BP is elevated. While this research is promising, more inquiry is needed to decide if training patients with hypertension can improve their awareness of their high BP episodes and if this training will ultimately improve healthcare outcomes.

Educational Level and Health Disparities

Major disparities exist among population groups, with a disproportionate burden of death and disability from cardiovascular disease in minority and lower socioeconomic populations. Health disparities are defined as differences in the incidence, prevalence, mortality, and burden of diseases and other adverse health conditions that exist among specific population groups in the United States. Several research studies have reported that higher educated people tend to be healthier and have improved outcomes to treatments, whereas people of lower socioeconomic status tend to have more adverse risk factors and poorer health (Mylykangas, Pekkanen, Haukkala, Vahtera, & Salomaa, 1995; Winkleby et al., 1990; Winkleby, Jatulis, Frank, & Fortmann, 1992). For example, data from the NHANES III study showed that there were highly significant differences in BP, body mass index (BMI), and physical inactivity for both African- and Mexican-American women compared to white women when educational level and ethnicity were adjusted for (Winkleby, Kremer, Ahn, & Varady, 1998). Disparities also exist in the prevalence of risk factors for cardiovascular disease. Lower educated persons and racial and ethnic minorities have higher rates of hypertension, BMI, physical inactivity, and non-HDL cholesterol, tend to develop hypertension at an earlier age, and are less likely to undergo treatment to control their high BP (NIH Online, 2003). In a study by Goldman and Smith (2002) differences in treatment adherence by education level are examined in patients with HIV and diabetes. It was found that patients with higher socioeconomic status and higher educational levels had improved treatment adherence and outcomes. In this study, the more-educated patients were more likely to adhere to therapy and have better self-reported general health. The less-educated patients were more likely to switch treatments, which led to worsening general health. The authors assert that the large

differences in health outcomes exist, not solely because of poor access to care or poor health behaviors, but because of differences in educational level (Goldman & Smith, 2002).

Ambulatory BP Monitoring

Ambulatory BP monitoring (ABPM) is a naturalistic BP measurement technique that has been evolving over the past 30 years. It is a method that allows a clinician, patient or researcher to monitor multiple BP readings over a 24- to 48-hour period. These devices can measure BP over time and introduce minimal intrusion into the person's daily routine. ABPM is used clinically to assess and diagnose types of hypertension, evaluate pharmacologic and/or nonpharmacologic therapies, and monitor resistant and/or borderline hypertension.

Ambulatory BP monitoring has now become an established research tool in clinical trials. The use of ABPM decreases threats to external validity and the potential "white coat" effect of observers on physiological and psychological responses. Oscillometric monitors measure SBP, mean arterial pressure (MAP), and heart rate (HR), from which DBP, pulse pressure (PP), and average 24-hour BP, diurnal changes, BP Load (percentage of systolic and diastolic readings greater than 140 and 90 mmHg during the day and greater than 120 and 90 mmHg during the night), and BP variability (the standard deviation of the average 24-hour daytime and nighttime measures) can be calculated. Ambulatory BP measurements correlate with the extent of target organ damage or cardiovascular risk. For example, Verdecchia (2000) reported that ambulatory SBP, DBP, and PP were independently and directly associated with cardiovascular risk.

While the use of the ABPM is minimally intrusive to the person, it may pose comfort issues such as annoyance from the beeping sound, weight of the ABPM device,

and bulkiness of the device. Over the past several years, improvements have been made to the devices to make them more “user friendly” and comfortable for subjects to wear for longer periods of time. Adherence has been shown to be enhanced following empathetic discussion and demonstration of the device. The safety of ABPM techniques have been established and complications are rare (NHBPEP-ABPM, 1992).

A typical, fully-automatic ABPM device is battery-driven and consists of an arm cuff that can be programmed to inflate automatically throughout a 24- to 48-hour period. BP is determined in the arm by detection of (a) Korotkoff sounds by one or two piezoelectric microphones under the cuff (auscultatory method) and (b) oscillations transmitted from the brachial artery to the cuff (oscillometric method). The Spacelabs 90207 ABPM device (Spacelabs, Inc, Redmond, WA) measures BP using the oscillometric technique. Auscultatory and oscillometric techniques have not been rigorously compared to each other to see if one is more preferable for ambulatory BP monitoring. However, the auscultatory technique is more sensitive to environmental and distracting noises, such as automobiles and large machinery. Oscillometric techniques detect systolic and mean BP and use algorithms to calculate diastolic BP. This may be a weakness as these algorithms are not appropriate for all subjects. Additionally, oscillometric methods are affected by muscle artifacts and tremors generated beneath the cuff. To avoid invalid or erroneous readings, the device should be calibrated properly and the cuff should be fit to the subject prior to use. In short, ABPM is a mature and clinically appropriate method for obtaining multiple, naturalistic ambulatory BP readings over a period of 24- to 48-hours (NHBPEP-ABPM, 1992).

Summary

In summary, hypertension, specifically high SBP, continues to be a major predictor of morbidity and mortality of people in the United States and worldwide. As many as 50 million American people are estimated to have hypertension (AHA, 2003a). Isolated systolic hypertension is prevalent among the elderly and people greater than 50 years of age (Franklin et al., 2001). Current diagnostic and treatment modalities have been wrought with difficulties due to a variety of physiologic, psychological, socio-economic, and practical factors. Current research suggests that the sympathetic nervous system plays a major role in the development and/or maintenance of hypertension (Rumantir et al., 2000). Activation of the SNS leads to a documented psychophysiological “fight or flight” response and associated manifestations. It is unknown whether high BP is associated with symptoms; however the majority of current knowledge suggests that it is an asymptomatic phenomenon. Despite the overwhelming support that hypertension is an asymptomatic disease, studies using BP and biosituational feedback have shown that people can be trained to become more aware of their BP levels. It is unknown whether the combination of ambulatory BP methods and biosituational self-awareness training improves subjects’ ability to recognize when their BP is elevated. Due to the recent surge of knowledge regarding the sympathetic nervous system’s connection with hypertension, it seems likely that some people, if not all people, could be trained to become more aware of the increased SNS activity.

CHAPTER 3 PROCEDURES AND METHODS

The purpose of this research was to determine if subjects with hypertension could improve their estimation of their SBP after an ambulatory BP feedback and biosituational self-awareness training intervention.

Research Design

A prospective cohort, repeated measures, pretest/posttest design was employed for this study. A repeated measures design allows subjects to serve as their own control and within-subject differences to be analyzed. The design, analysis groupings, and data measured are graphically displayed in Table 3-1.

Population and Sample

The population under investigation was adult hypertensive persons, aged 21 to 65, in the northern Florida area. Subject recruitment was done through both flier advertising and BP screening. The investigator offered BP screening over the course of a 12-month period at various locations. Before BP was measured, potential subjects were told that they would be offered the opportunity to take part in a research study if they qualified. BP was measured twice 2 minutes apart after the subject sat quietly for 3 to 5 minutes. If the BP measurements differed by more than 5 mmHg, an additional BP measurement was taken. The initial BP screening was obtained by averaging the two BP readings that agreed within 5 mmHg. Subjects who met the inclusion and not the exclusion criteria were asked to participate in the study. Every attempt to include diverse participants

Table 3-1. Description of design, analysis groupings, and data measured

Group	Hypothesis (H)	Pretraining	Training	Posttraining
Adult hypertensives (total sample)	H 1	ASBP, ESBP, mean AD	ASBP, ESBP	ASBP, ESBP, mean AD
Adult hypertensives college educated	H 2, H 3	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Adult hypertensives noncollege- educated	H 2, H 3	ASBP, ESBP, mean AD	ASBP, ESBP	ASBP, ESBP, mean AD
Adult hypertensives BMI < 30	H 4	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Adult hypertensives BMI ≥ 30	H 4	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Adult hypertensives male	H 5	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Adult hypertensives female	H 5	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Hypertensives < 48 years of age	H 6	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Hypertensives ≥ 48 years of age	H 6	ASBP, ESBP, MI	ASBP, ESBP	ASBP, ESBP, MI
Antihypertensive medication nonusers	H 7	ASBP, ESBP	ASBP, ESBP	ASBP, ESBP
Antihypertensive medication Users	H 7	ASBP, ESBP	ASBP, ESBP	ASBP, ESBP

Note: ASBP represents actual SBP, ESBP represents estimated SBP, mean AD represents mean absolute difference, and MI represents mean improvement.

(i.e., gender, race, socioeconomic, age, and ethnicity) was made. To determine the sample size, it was estimated that subjects could improve their estimation of SBP by decreasing the difference by half. For example, if the mean difference between the actual and estimated SBP was 10 mmHg on day 1, this difference would drop to 5 mmHg. Assuming that the deviation of the difference was 4.0 mmHg, setting an alpha of 0.05, and using a 2-tailed test, 8 subjects would be required to achieve at least 80% power. Recognizing that subjects may not be able to improve their estimation this much with only a 2-day training period, a second determination of sample size was completed based on an improvement of 2 mmHg, the smallest effect that would be important to detect.

Again, if on day 1 the mean difference between the actual and estimated SBP was 10 mmHg and the mean difference on day 4 was 8 mmHg, this would constitute an improvement of 2 mmHg. Assuming a standard deviation of the difference to be 4.0 mmHg, setting an alpha of 0.05, and using a 2-tailed test, 34 subjects would be required to achieve at least 80% power. Recognizing that these are estimates and there are no data suggesting the appropriate effect size to use, 42 subjects were recruited for study to allow for attrition and incomplete data.

To ensure that subjects would have adequate variability to be able to detect differences, we randomly selected ambulatory BP data from 10 hypertensive subjects in Dr. Yucha's research study. For these 10 subjects, the average daytime range in SBP was 33.2 mmHg, ranging from a minimum of 19 to a maximum of 56 mmHg. Therefore, we felt confident that subjects would have adequate variability in their BP to detect differences (unpublished BP variability data, 2001).

Inclusion and Exclusion Criteria

The specific inclusion criteria were as follows:

- men or women 21 to 65 years diagnosed with hypertension or taking antihypertensive medications, living in the North Florida area.
- ability to come to the research office at least four times.
- ability to speak and understand English.
- able to verbally communicate with intact memory.
- ability to read English at an eighth grade level or greater.

Subjects who could respond to requests for participation were considered to have adequate communication skills and memory ability. Subjects were excluded from the

study if their history demonstrated significant cardiovascular, renal, or psychiatric diseases. There was no exclusion of subjects based on gender or race.

Setting

The setting for this study was a county located in Northern Florida. Initially, the subjects were screened in the laboratory or field setting. The pretraining, training, and posttraining sessions occurred in the subjects' natural environment during daytime hours while the subjects were awake.

Research Variables and Instruments

Demographic Data Sheet

The demographic data sheet included information regarding age, gender, race, marital status, how long with diagnosis of hypertension, height, weight, body mass index (BMI), and education.

Health History Form

The health history form included yes/no type questions regarding the presence or absence of health conditions including high BP, diabetes, heart and cerebral disease, psychiatric disorders, and other chronic diseases. Additionally, questions regarding past or present smoking, alcohol use, high cholesterol, exercise level, medications, and family cardiovascular health history were included.

Ambulatory BP Monitor

Naturalistic ambulatory monitoring of BP in human subjects was preferred in this study because it enhances the generalizability of the findings to outside of the laboratory setting and it does not interfere with most of the subjects' usual daily activities. The SpaceLabs ABPM (Model 90207, SpaceLabs, Inc., Redmond, WA), an automatic noninvasive oscillometric recorder, was used to collect SBP data. This monitor measures

BP by detection of oscillations transmitted from the brachial artery to the cuff. The monitor was equipped with four different size adult cuffs. A SpaceLabs (Model 9029, Redmond, WA) Data Interface Unit was used for data retrieval and report generation. The ABPM can be programmed to display the BP readings on its' LCD screen immediately after measurement (i.e., unblinded) or not to display the BP readings (i.e., blinded). This feature worked well for this study, as different time periods required "blinding" or "unblinding" of the LCD screen. The reliability of the SpaceLabs ABPM device has been studied extensively over the last few years. Correlation coefficients between two sets of readings have ranged between 0.72 and 0.93 for SBP, indicating that the reliability is acceptable (Pickering et al., 1994). Pickering et al. suggest that at least five or six readings would give an adequate representation of the average pressure in a particular setting such as work or home. In addition, a sampling frequency of one reading every 30 to 60 minutes has been suggested to adequately describe average SBP levels in different settings. This instrument has a high level of accuracy and clinical performance and meets Association for the Advancement of Medical Instrumentation guidelines and the guidelines of the British Hypertension Society (O'Brien, Atkins, & Staessen, 1995). Artifactual readings were eliminated using the Casadei procedure, a standard editing criteria (Winnicki, Canali, Mormino, & Palatini, 1997). Similar to other editing criteria, the Casadei procedure eliminates measurements that fall outside 50 to 240 mmHg for SBP, 40 to 140 mmHg for DBP, 40 to 125 beats per minute for heart rate, and 20 to 100 mmHg for pulse pressure.

Rigorous calibration of the monitor was made prior to ABP monitoring. A calibration procedure comprised of three calibration readings taken simultaneously with a

mercury column sphygmomanometer and the ABP monitor, by means of a “T-connector” between the two instruments. Readings for both SBP and DBP agreed within 5 mmHg of one another on all three attempts.

For the purpose of this study, the ABPM was initialized to measure BP every 30 minutes. Actual ambulatory BP measurements were recorded as numerical continuous response variables. After measurement of ambulatory BP, BP data were downloaded using SpaceLabs Data Management Software (Redmond, WA) and the data.

Actual BP

Actual BP was a continuous variable that was measured using a SpaceLabs Ambulatory BP Monitor (Model 90207, SpaceLabs Inc., Redmond, WA).

Estimated Systolic BP

Estimated SBP was a continuous numerical variable that was estimated by the participant in mmHg and was based on the guideline provided to the participant. Participants were also invited to circle the range of SBP that they thought their SBP was in at the time of cuff inflation and BP measurement. This was done to improve the conceptualization of the participant to estimating his/her own SBP. If range information was the only method of estimating for the subject, the average of the range was computed and entered as the subjects’ estimation of SBP.

Absolute Difference

The absolute difference (AD) is defined as the absolute value of the mean difference. The absolute difference was calculated for mean actual SBP day 1, mean estimated SBP day 1, mean actual SBP day 4, and mean estimated SBP day 4.

Mean Improvement

The mean improvement is defined as the absolute value of the mean difference of day 1 (mean actual SBP minus mean estimated SBP) minus the absolute value of the mean difference of day 4 (mean difference of actual SBP minus estimated SBP).

Pre- and Posttraining SBP Estimation Form

The pre- and posttraining SBP Estimation Form provides subjects with a guideline for SBP estimation that is based on the classification defined by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP (JNC VI, 1997). This tool categorizes SBP based on the JNC VI (1997) classification and provides categorical descriptions of each range of SBP category. This serves to help subjects conceptualize their SBP, so that they can estimate their SBP level. Subjects are instructed to write an estimate of what they think their SBP level is at the start of cuff inflation. Subjects were instructed that they may circle the range of where they think their SBP falls, if this was more understandable for the subject.

SBP Estimation Training Form and Self-Awareness Checklist

The training form is a form that is used during days 2 and 3. One form was used for each BP measurement/estimation. The form consists of an area for the subject to write the time of BP measurement, estimated SBP level (subject estimates), and actual SBP level (from the monitor). The Self-Awareness Checklist is a yes/no checklist. It is made up of 38 mood, symptoms, and situation items. This checklist has been adapted from research on physical symptoms and factors relating to BP (Barr et al., 1988; Brondolo et al., 1999; Gellman et al., 1990).

Demographic Variables

Demographic characteristics of subjects were examined by nine indicators: gender, race, education, marital status, age, height, weight, body mass index, and how long with diagnosis of hypertension.

Gender. Gender was a categorical variable coded as male or female.

Race. Race was a categorical variable coded as White, Black, Hispanic, Asian, and other.

Education. Education was categorized into seven groups according to the number of years of formal education which the participants completed: less than 7 years, junior high school (grades 7-9), some high school (grades 10-11), high school graduate, some college or technical school, college graduate, and graduate school (master's degree or beyond). For data analysis purposes, education was further compressed into two variables: H.S./technical school and college educated.

Marital status. Marital status was coded into one of four categories reflecting the status of married, widowed, divorced/separated, or never married.

Age. Age was recorded as actual years and was coded into five categories reflecting years of age: 21-30, 31-40, 41-50, 51-60, 61-65. For data analysis purposes, age was further compressed into two categories: ≥ 48 years of age and < 48 years of age.

Weight. Weight was a continuous numerical variable that was recorded in kilograms (kg).

Height. Height was a continuous numerical variable that was recorded in centimeters (cm).

Body Mass Index (BMI). BMI was calculated as the ratio of the weight in kg to the square of the height in meters.

Length of time since diagnosis of hypertension. Length of time since diagnosis of hypertension was categorized as follows: less than 5 years, 5-10 years, 11-20 years, 21 years or more.

Health Status Variables

Four indicators were utilized to identify the health status, family cardiovascular health history, and the use of prescribed and nonprescribed medicines. These variables included (a) existence of health problems, (b) number and type(s) of medications used daily, (c) family cardiovascular health history, and (d) lifestyle factors. Below is a description of these variables.

Health problems. The participant was asked to identify his or her health problems from a list of different illnesses. The answer was coded zero when the problem did not exist and one if the problem existed.

Use and type of medications. The use of all types of medications was a categorical variable that was coded zero if there were no medications used and one if the participant used medications on a daily basis. If the answer was yes, the participant was asked to name all prescribed and nonprescribed medications that are used daily. For data analysis purposes, the medication variable was further described to account for differences among types of medications and antihypertensive medications. A variable coded as “htntype” was created and was coded as 0 if they were taking no hypertensive medications, 1 if they were using ace inhibitors, 2 if they were using calcium channel blockers, 3 if they were using beta blockers, 4 if they were using diuretics, 5 if they were using other antihypertensives, and 6 if they were using 2 or more antihypertensive medications.

Family cardiovascular health history. Each participant was asked to identify illnesses that his or her blood relatives have had or currently have. Family cardiovascular health history was coded into five categorical variables including heart attack, high BP, stroke, diabetes, and high cholesterol. The answer was coded zero if there was no family history of the disease. The variable was coded 1 if there was a blood relative with one of the identified illnesses, 2 if there were 2 identified illnesses, 3 if there were 3 illnesses chosen, 4 if there were 4 chosen, and 5 if there were 5 illnesses chosen.

Lifestyle factors. Lifestyle factors were considered questions relating to alcohol use, caffeine use, exercise level, and cholesterol elevation. The responses were coded zero if the respondents chose no and one if the respondents chose yes.

Table 3-2. Instruments used and variables measured during the study periods

Instrument	Variables
ABP Monitor	Actual SBP
Pre-/Posttraining Form	Estimated SBP
Training Form/Self-Awareness Checklist	Estimated SBP Actual SBP Biosituational factors
Demographic Data Sheet	Gender Race Education Marital status Height Weight BMI Time with hypertension Veteran status Age Date of Birth
Health History Form	Personal history of cardiovascular, renal, liver, thyroid diseases, diabetes mellitus, caffeine, alcohol and tobacco use, exercise, medication usage, and family history.

Classification of Adult BP

The criteria for classifying BP as defined by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP (JNC VI, 1997) was used to assist subjects to estimate their actual BP. Table 3-3 shows the classification for adult BP as defined by the JNC VI (1997).

Table 3-3. Adult BP classification

Category	Systolic BP (mmHg)		Diastolic BP (mmHg)
Optimal	Less than 120	and	Less than 80
Normal	Less than 130	and	Less than 85
High-Normal	130-139	or	85-89
Hypertension			
Stage 1	140-159	or	90-99
Stage 2	160-179	or	100-109
Stage 3	180 or greater	or	Greater than 110

Study Protocol and Procedures

This research consisted of three phases: (a) initial interview and pretraining measurement and estimation of BP (one day period), (b) ABPM and biosituational self-awareness training (2-day period) and (c) posttraining measurement and estimation of BP (1-day period).

Subject Recruitment

After the appropriate institutional review and approval, subjects were recruited from northern Florida. The investigator recruited participants using fliers and advertisements that were posted near the University of Florida, the Veterans Administration clinics, hospitals, and various public areas in the north Florida area. Recruitment fliers were also sent to female veterans with hypertension in the northern Florida area. Attempts to include diverse participants (i.e., gender, age, and race) were

made. Those subjects who met the inclusion criteria and who did not meet the exclusion criteria were asked to participate in the study.

Initial Screening Procedures

The initial screening occurred in either the laboratory or field setting. The procedure for measuring BP was in accordance with JNC VI recommendations, using the ABPM. To assure that the ABPM readings are valid, calibration of the equipment was performed. The investigator calibrated the ABPM using simultaneous determinations of BP by auscultation and a mercury sphygmomanometer (using T connector) and agreement of at least 3 sequential readings to within 5 mmHg systolic and diastolic was found (NHBPEP-ABPM, 1992).

The BP measurement began after approximately 3 to 5 minutes of quiet rest, sitting in a chair. The subject was seated in a chair with his/her back supported and nondominant arm bared and supported at the heart level. The appropriate cuff size was determined to ensure accurate measurement. The bladder within the cuff encircled approximately 80 % of the subject's arm in accordance with JNC VI recommendations (JNC VI, 1997). The investigator provided the subject with an initialized, programmed and fitted ambulatory BP monitor. The investigator performed two BP measurements approximately two minutes apart, in accordance with recommendations of the JNC VI (1997, p. 12). If these two measures were more than 5 mmHg apart, a third measure was taken. The average of the measurements were provided to the patient as their "average BP."

Ambulatory BP Monitoring Protocol

The subject was instructed to refrain from excessive physical exertion and water activities while wearing the BP monitor. The subject was instructed to keep a regular

sleep and wake pattern and to avoid any unusual physical exertion or excessive stress during the study period. The subject was instructed that he/she could remove the monitor for short time periods if these activities were unavoidable. Then, the subject was given the opportunity to use and become familiar with the ABPM.

Ambulatory BP was measured on an ordinary work, home, or school day for each subject. To ensure that the subjects were experiencing “usual” symptoms or situations, subjects were asked prior to beginning each study day how they were feeling on that day and if they were feeling “well” or “usual.” If the subject was not feeling as he/or she normally feels (e.g., has a cold/flu or other anomaly), the session was postponed until the following day or a more “usual” day.

To ensure subject safety, subjects were instructed to sit, rest, and call their primary health care provider in the event that their BP was greater than 180 mmHg systolic or 110 mmHg diastolic over two consecutive periods. Because of the nature of the ABPM device, subjects were instructed that they could push the “start” button on the monitor and measure their BP in more frequent intervals than were programmed. In addition, subjects were instructed to call their health care provider or seek emergency care if they experienced any other serious discomforts other than the minimal discomforts associated with the use of the ambulatory BP monitor. Subjects were instructed that symptoms such as chest pain, shortness of breath, or numbness or tingling of face, legs, or arms should be reported immediately to their healthcare provider.

Subjects were fitted with an ABP monitor and familiarized with its use. The monitor was programmed to measure BP every 30 minutes over a 6-hour period. Subjects were instructed that they could wear the monitor during their usual awake hours, generally between the hours of 6 am and 10 pm. Each subject was fitted with a proper-

sized BP cuff, fitted according to JNC VI recommendations (JNC VI, 1997). To ensure that the cuff was not too tight, the investigator inserted a finger between the bladder of the cuff and the subjects' arm. Subjects were provided an ABPM tote bag or hip strap to assist in carrying the ABP monitor.

The monitor emits a series of 5 beeps prior to measurement of BP and cuff inflation. The subject was instructed to listen for these sounds and to hang their arm freely at their side during cuff inflation. They were also instructed to keep the bladder of the cuff at or near the level of their heart, to avoid measurement errors. At cuff inflation, subjects were instructed to estimate their BP. On days 2 and 3, subjects were also instructed to document their actual SBP as well as their moods, symptoms, and activities during the BP measurement.

Day 1

After the initial screening for inclusion/exclusion criteria, a convenient meeting date and time to start the study was arranged. Informed consent was obtained and a copy of the informed consent and contact information for the investigator and dissertation chairperson was provided to each participant. Subjects were informed that participation in this study will not change the way they are treated for high BP. The subject was instructed to continue doing exactly what his/her doctor has prescribed. Each participant was advised of his or her rights as a research participant and the right to decline without penalty. The investigator arranged a time and place for the initial interview, either at the research office or the subject's home. The investigator instructed the subject about the study procedures and that data would be collected over a 4-day period. The participants were notified that there was monetary compensation of \$10.00 per day for each day that was completed.

After informed consent was obtained, the subject was asked to answer questions related to demographics, health status, family history, and medication usage. The entire interview took approximately 15-30 minutes per subject. After completion of the initial interview, data were entered into a data spreadsheet for analysis.

When the subject was comfortable with the ABPM operation and function, the subject started Day 1 data collection period and took the ABPM home, work and/or to their “natural” environment. The participant was instructed to estimate numerically their SBP using guidelines from the JNC VI (1997). The LCD screen on the ABPM was “blinded” (i.e., no BP readings were displayed). The subject was instructed to return to the clinic the following day with the ABPM, or arrangements for a field visit were made.

Days 2 and 3

On the second study day, day 1 data were downloaded and edited using the SpaceLabs (Model 9029, Redmond, WA) Data Interface Unit. Data were entered into a data spreadsheet for analysis. The ABPM was initialized and reprogrammed to display the BP readings on the LCD screen. The subject was provided information on potential biosituational factors that may affect BP. The subject was given the opportunity to use and become familiar with the Training Form/Self-Awareness Checklist. The subject was instructed to fill out the Self-Awareness Checklist at each BP reading. The subject was instructed that SBP, DBP, and HR are visible in the LCD screen after each BP measurement. Next, the subject was asked to look at and write down his or her actual SBP level as it appears on the LCD screen after each reading. Subjects were instructed to estimate their SBP in a similar fashion as in day 1, when the cuff began to inflate.

When the subject was confident with using the Self-Awareness checklist and Training Form, he or she was instructed to wear the ABPM for two consecutive days in

the “normal” environment and perform the instructed tasks every 30 minutes for six hours each day.

Day 4

On the fourth study day, the subject and investigator met again. The data were downloaded onto a spreadsheet for analysis. The ABPM was initialized and re-programmed not to display the BP readings. Similar to day 1, the LCD screen was “blinded” to the subject (i.e., the BP reading were not be visible to the subject). The subject was instructed to think about the biosituational factors that occurred during their SBP measurements and when SBP was high. Subjects were given an opportunity to assess the biosituational self-awareness factors that were related to high SBPs (according to individual responses).

Subjects were instructed to wear the ABPM monitor for 6 hours and estimate their SBP, making decisions based on their biosituational self-awareness factors and BP readings, during the previous 2 days.

At the conclusion of the study, subjects were instructed to return the ambulatory BP monitor and all forms to the investigator. Subjects were thanked for their participation and were given a printed analysis of the 4-day ABPM readings. Each subject received \$10.00 compensation for each day they completed. The subjects received a total of \$40.00 monetary compensation for participation in this study. Study procedures are outlined in Table 3-4.

Methods of Statistical Analyses

Data were analyzed using SPSS (SPSS, Inc., Chicago, Illinois). Descriptive statistics were computed to obtain the summary measures for the data addressing the research hypotheses. Estimated SBP data was obtained from the pre-/posttraining SBP

Estimation Form. Actual SBP measurement data was obtained from the data recorded using the ABP monitor and the report generated by the SpaceLabs (SpaceLabs, Inc., Redmond, WA) Data Interface Software. These data were entered into data files for analysis using Microsoft Excel (Microsoft Inc.) software and SPSS (SPSS, Inc., Chicago, Illinois) statistical software. Descriptive statistics were computed to identify the demographic characteristics of the participants, number and types of medications used, health problems, and family health history. Study variables (estimated and actual SBP, absolute difference (AD) of the mean scores of day 1 and 4, and mean improvement) were summarized and graphed across time. For data analysis purposes, day 2 and 3 were combined and a total mean score for actual SBP, estimated SBP, and absolute difference were calculated for the two days. Analysis concerning the relationships between actual and estimated SBP were performed using paired-samples t-tests. For Hypothesis 1 and 3, paired-samples t-tests were used to compare the mean improvement between day 1 and day 4 within groups. For Hypotheses 2 and 4 through 7, independent samples t-tests were used to compare the means between groups of subjects. See Table 3-1 for a description of the design, analysis groupings, and data measured in this study.

Table 3-4. Procedures for SBP estimation study

Phase 1: Initial Interview and Pretraining (Day 1)

In laboratory/Field Setting:

- Screen for Inclusion/Exclusion criteria
- Informed Consent Process
- Calibrate & initialize ABPM (BP readings not shown) and determine cuff size
- Obtain demographic, health status, health and family health history, and medication usage data
- Provide basic information about SBP
- Obtain “average” baseline BP and provide information to subject
- Introduction to Pre-/Posttraining Estimation Form and ABPM
- Allow subject to practice using SBP estimation form and ABPM

In natural setting:

- Subject estimates SBP (LCD blinded) for 6 hours at start of each cuff inflation

Phase 2: Training (Days 2 & 3)

In laboratory:

- Initialize ABPM (BP readings shown)
- Provide information on SBP Estimation and BSMA factors
- Demonstrate ABPM and Training Form/Self-Awareness Checklist
- Allow subject to practice using ABPM, BP estimation, and Self-Awareness Checklist

In natural setting:

- Subject estimates SBP for 6 hours at start of cuff inflation
- Complete Training Form (SBP estimation, Self-Awareness Checklist, & actual SBP)

Phase 3: Posttraining (Day 4)

In laboratory:

- Initialize ABPM (BP readings not shown)
- Instruct patients to think about biosituational self-awareness factors & SBP feedback while estimating their SBP

In natural setting:

- Estimate SBP (LCD blinded) for 6 hours at start of cuff inflation

CHAPTER 4 RESULTS

The primary purpose of this study was to determine if hypertensive persons could learn to estimate their SBP using a BP feedback and biosituational self-awareness training intervention. This was determined by comparing the accuracy of the SBP estimation before and after training. The secondary purpose of this research was to compare the differences in the mean improvement of actual to estimated SBP between different groups of hypertensives within the sample. These groups include college-educated (CE) hypertensives compared to non-CE (NCE) hypertensives, hypertensives with a body mass index (BMI) < 30 compared to hypertensives with a BMI ≥ 30, male hypertensives compared to female hypertensives, hypertensives less than 48 years of age compared to hypertensives ≥ 48 years of age, and hypertensive medication (HM) users compared to hypertensive medication (HM) nonusers.

This chapter will first present descriptive statistics, including means, standard deviations, and frequency distributions for each variable. The hypotheses posed in Chapter 1 will be addressed using paired samples t-tests and independent samples t-tests. For all results, data will be expressed as means ± standard deviations and/or percentages.

Descriptive Results

Sample Characteristics

Over 60 potential subjects were screened for inclusion in this study. However, only 42 subjects met the final inclusion criteria. Of these 42 subjects, 3 subjects were

excluded from the analysis for different reasons. One male subject was excluded after completion of day 1 because his BP on day 1 was low. This subject had a mean SBP on day one of 101 mmHg and a minimum BP of 74/52. The subject reported symptoms of “not feeling well” and was being treated for chronic hypothyroidism. It was recommended that the subject seek care from his healthcare provider and withdraw from the study. A female subject was excluded from the study after day 1 because her BP levels were excessively high. Her mean SBP level was 192 mmHg and her maximum BP was 201/116. She was advised to obtain immediate medical care. She contacted her physician, obtained medical treatment, and was released from the study. A third subject withdrew from the study after day 1 because of difficulties that she had in performing the protocol activities during her normal work/home day.

A total of 39 subjects completed the study protocol. Of the total, 15 subjects were male and 24 subjects were female. The male group ranged from 26 to 65 years with a mean age of 45.1 years. The female group ranged from 21 to 65 years, with a mean age of 50.4 years.

Subject demographics expressed in numbers and percentages were gender, race, age, marital status, family history of hypertension, veteran status, time with diagnosis, education level, hypertension medication type, overall medication type, and habits of cigarette smoking, alcohol use, caffeine use, and exercise. Table 4-1 shows the subject demographics for the total hypertensive sample (N = 39), NCE subjects (N = 15), and CE subjects (N = 24). Table 4-2 compares the lifestyle variables for the total hypertensive sample, NCE subjects, and CE subjects. Table 4-3 compares the health status data of the total sample of hypertensives, NCE subjects, and CE subjects.

Table 4-1. Comparison of demographic data for the total sample, college-educated subjects, and noncollege-educated subjects

	Total sample (N = 39)	Noncollege- educated subjects (N = 24)	College-educated subjects (N = 15)
	N (%)	N (%)	N (%)
Gender			
Male	15 (38.5)	9 (37.5)	6 (40)
Female	24 (61.5)	15 (62.5)	9 (60)
Race			
Caucasian	32 (82.1)	18 (75.0)	14 (93.3)
African American	7 (17.9)	6 (25.0)	1 (6.7)
Age			
30 and under	4 (10.3)	1 (4.2)	3 (20.5)
31-40 years	5 (12.8)	2 (8.3)	3 (20.5)
41-50 years	12 (30.8)	6 (25.0)	5 (33.3)
51-60 years	13 (33.3)	12 (50.0)	2 (13.3)
61-65 years	5 (12.8)	3 (12.5)	2 (13.3)
Marital Status			
Married	27 (69.2)	16 (66.7)	11 (73.3)
Never married	3 (7.7)	2 (8.3)	1 (6.7)
Widowed	2 (5.1)	2 (8.3)	0 (0.0)
Divorced	7 (17.9)	4 (16.7)	3 (20.0)
Education Level			
HS graduate	8 (20.5)	8 (33.3)	0 (0.0)
Some college/tech.	16 (41.0)	16 (66.7)	0 (0.0)
College graduate	6 (15.4)	0 (0.0)	6 (40.0)
Graduate school	9 (23.1)	0 (0.0)	9 (60.0)
VA/Veteran Affiliate			
No	22 (56.4)	18 (75.0)	4 (26.7)
Yes	17 (43.6)	6 (25.0)	11 (73.3)

Table 4-2. Comparison of lifestyle data for the total sample, college-educated subjects, and noncollege-educated hypertensive subjects

	Total sample (N = 39)	Noncollege- educated subjects (N = 24)	College-educated subjects (N = 15)
	N (%)	N (%)	N (%)
Current Tobacco Use			
No	30 (76.9)	18 (75.0)	12 (80.0)
Yes	9 (23.1)	6 (25.0)	3 (20.0)
Regular Alcohol Use			
No	18 (46.2)	13 (54.2)	5 (33.3)
Yes	21 (53.8)	11 (45.8)	10 (66.7)
Regular Caffeine Use			
No	5 (12.8)	5 (20.8)	0 (0.0)
Yes	34 (87.2)	19 (79.2)	15 (100.0)
Regular Exercise			
No	15 (38.5)	10 (41.7)	5 (33.3)
Yes	24 (61.5)	14 (58.3)	10 (66.7)

Table 4-3. Comparison of health status data for the total sample, college-educated hypertensive subjects, and noncollege-educated hypertensive subjects.

	Total sample (N = 39) N (%)	Noncollege- educated subjects (N = 24) N (%)	College-educated subjects (N = 15) N (%)
Time with diagnosis of hypertension			
Less than 5 years	27 (69.2)	14 (58.4)	13 (86.7)
5-10 years	6 (15.4)	6 (25.0)	0 (00.0)
11-20 years	4 (10.3)	2 (8.3)	2 (13.3)
21 or more years	2 (5.1)	2 (8.3)	0 (0.0)
Family history of cardiovascular disease			
No family history	2 (5.1)	0 (0.0)	2 (13.3)
1 FH item selected	4 (10.3)	2 (8.4)	2 (13.3)
2 FH item selected	9 (23.1)	6 (25.0)	3 (20.0)
3 FH item selected	6 (15.4)	3 (12.5)	3 (20.0)
4 FH item selected	10 (25.6)	5 (20.8)	5 (33.4)
5 FH item selected	8 (20.5)	8 (33.3)*	0 (0.0)*
Hypertension medication type			
Not taking HTN meds	16 (41.0)	9 (37.5)	7 (46.7)
Ace inhibitor only	8 (20.5)	4 (16.7)	4 (26.7)
Calcium channel blocker only	3 (7.7)	3 (12.5)	0 (0.0)
Beta blocker only	2 (5.1)	0 (0.0)	2 (13.3)
Diuretic Only	2 (5.1)	2 (8.3)	0 (0.0)
Other HTN med only	1 (2.6)	1 (4.2)	0 (0.0)
2 or more HTN meds	7 (18.0)	5 (20.8)	2 (13.3)
Overall medication type			
Not taking medications	6 (15.4)	3 (12.5)	3 (20.0)
Taking HTN meds only	10 (25.6)	4 (16.7)	6 (40.0)
Taking other type of meds only	10 (25.6)	6 (25.0)	4 (26.7)
Taking other med and HTN med	13 (33.4)	11 (45.8)*	2 (13.3)*

* indicates $p \leq 0.05$ by Mann-Whitney-U Nonparametric Tests.

Clinical Characteristics

The clinical characteristics of the subjects, including age, weight, height, BMI, actual and estimated SBP day 1, actual and estimated SBP day 2 and 3, actual and estimated SBP day 4, mean absolute differences of actual SBP (ASBP) minus estimated SBP (ESBP) for each day, and number of observations are expressed using means and standard deviations and are presented in Table 4-4. The mean scores for each study day and across all study days are summarized in Table 4-4. The mean ASBP measurements

were similar among the total sample on days 1 and the average of days 2 and 3; 137.0 ± 11.0 mmHg and 136.1 ± 15.3 mmHg, respectively. On day 4, the mean ASBP was slightly lower at 136.1 ± 12.1 mmHg, but this reduction was not statistically significant. Among the 39 subjects, there were 485 BP measurements/estimations on day 1, 880 BP measurements/estimations on day 2 and 3, and 482 BP measurements/estimations on day 4. In total, there were 1847 BP measurements/estimations among the 39 subjects for the total 4-day study period.

Table 4-4. Clinical characteristics for the total sample (N = 39)

Total Sample (N = 39)				
	Initial screening	Day 1	Day 2 & 3	Day 4
Age (yrs.)	48.4 ± 11.5			
Weight (lbs.)	194.1 ± 46.3			
Height (in.)	66.7 ± 4.6			
BMI (kg/m^2)	30.5 ± 5.4			
Actual SBP (mmHg)		137.0 ± 11.0	137.1 ± 9.27	136.1 ± 12.1
Mean Actual SBP Range (mmHg)		51	36	52
Min ASBP (mmHg)		94	91	99
Max ASBP (mmHg)		176	192	174
Estimated SBP (mmHg)		137.3 ± 8.6	136.0 ± 5.49	136.1 ± 11.8
Absolute difference ASBP-ESBP (mmHg)		10.1 ± 3.5	7.5 ± 5.70	9.3 ± 3.2
Number of ASBP-ESBP observations		12.4 ± 2.3	20.9 ± 6.9	12.3 ± 1.7

Analytic Results for Hypotheses

Procedure for Calculating Mean Scores

As described previously, the study protocol involved 4 days of BP measurement, at a frequency of every 30 minutes for 6-hours each day. Therefore, theoretically each subject should have 12 observations or BP measurements/estimations per day. However, on Day 1, the number of BP observations for each subject ranged from 9 to 20 observations, with a mean number of observations at 12.4. Likewise, on day 4 the number of BP observations for each subject ranged from 9 to 15 observations with a mean of 12.3 observations. This variation occurred for a number of reasons. First, some

subjects correctly had their BP taken 12 times but the BP measurement was deleted due to an error. The error may have been caused by improper inflation/position of the cuff, too much activity of the arm or body during cuff inflation, and/or the BP reading was higher than the previous BP reading and the cuff did not inflate to an adequate level to obtain the reading. Secondly, a few subjects did not follow instructions completely and performed either too few (9 to 11) readings or too many (13 to 20) readings. The majority of subjects performed the tasks as directed and performed 12 readings. Data were included if there were at least 9 observations per day. All 39 subjects had at least 9 observations per day. To assure that the mean score reflected the variation in number of observations per day, each individual subject's scores were analyzed separately to compute a mean actual SBP, mean estimated SBP, and a mean absolute difference between actual and estimated SBP for each subject.

Absolute difference. The absolute difference (AD) is defined as the absolute value of the mean difference. Without using absolute difference, overestimates and underestimates would average to a smaller mean difference. The absolute difference has been calculated for day 1 (mean actual SBP minus estimated SBP day 1), day 2 and 3 (mean actual SBP minus estimated SBP day 2 and 3 combined), and day 4 (mean actual SBP minus estimated SBP day 4).

Mean improvement. The mean improvement is defined as the absolute value of the mean difference of day 1 (mean actual SBP minus mean estimated SBP) minus the absolute value of the mean difference of day 4 (mean difference of actual SBP minus estimated SBP). The mean improvement represents a measure of improvement in SBP estimation between day 1 and day 4.

The Paired Sample t-Test

The paired sample t-test was used to compare the means of two scores from related samples. The assumptions of a paired t-test are that the variables are at interval or ratio levels and that they should be normally distributed. Figure 4-1 depicts the improvement scores of the entire sample of adult hypertensives. It illustrates a relatively normal distribution of improvement scores. Because of the robustness of a t-test, it is appropriate to use a paired t-test for these data.

The Independent Samples t-Test

The independent samples t-test compares the means of two independent groups. The assumptions of this test are that the two groups are independent of each other, the dependent variable must be measured on an interval or ratio level, and the scores should be normally distributed. All assumptions have been met for this test (refer to Figure 4-1) for distribution of mean improvement scores.

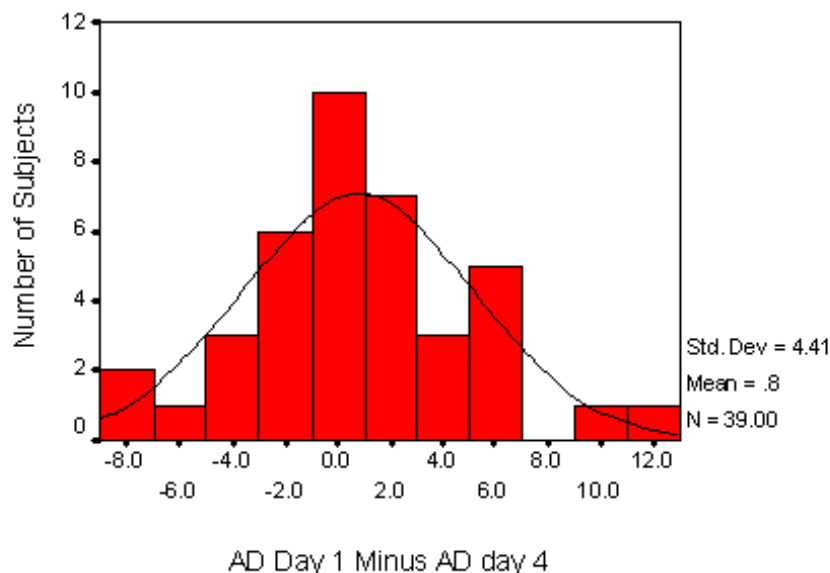


Figure 4-1. Distribution of mean improvement scores for total sample (N = 39). Mean and standard deviation of the improvement in SBP estimation for the entire sample of hypertensive persons is provided.

The mean actual SBP and estimated SBP across all study days was 136.5 and 136.1 mmHg, respectively. The mean difference across all study days was + 0.43 mmHg. This finding suggests that subjects were extremely accurate in estimating their SBP; however, the mean difference does not take into account the variability of SBP measurement/estimations and the over- and underestimators of SBP. The absolute value of the difference between estimated and actual SBP for each subject was calculated and averaged and was found to be ± 8.6 mmHg. Therefore, subjects were actually estimating on average within ± 8.6 mmHg of their actual SBP level across all study days. The AD was calculated and used in this study to take into consideration that there would be over- and underestimations of SBP and to gain the true picture of SBP elimination. This method has been used by Luborsky et al. (1976) in a similar BP estimation study.

Hypotheses

Hypothesis 1. Adult hypertensives differ significantly in their mean AD after the ambulatory BP awareness training intervention, compared with before the training intervention.

For hypothesis 1, the mean absolute difference of day 1 was compared to the mean absolute difference of day 4, using a paired-samples t-test. The mean absolute difference on day 1 (pretraining) was 10.1 ± 3.5 mmHg and the mean absolute difference on day 4 (posttraining) was 9.29 ± 3.2 mmHg. The hypertensive subjects improved their mean scores after the training, however the improvement was not statistically significant ($t = 1.094$, $df = 38$, $p = 0.281$). Of the 39 subjects, 18 subjects showed no improvement and 21 subjects (54%) showed improvement in estimating their SBP after the training. See Figure 4-1 for a graphical display of the AD of day 1 minus the AD of day 4 (mean improvement) of the total hypertensive sample. As Figure 4-1 shows, the mean improvement was 0.8 ± 4.4 mmHg for the total sample of hypertensive subjects ($N = 39$).

Hypothesis 2. College-educated hypertensives differ significantly from noncollege-educated hypertensives in their mean improvement of SBP estimation.

For hypothesis 2, CE hypertensives were compared to NCE hypertensives to assess differences in their mean improvement scores for day 1 and day 4. The CE subgroup was composed of 15 subjects (6 males, 9 females), mean age 43.2 ± 13.7 , mean BMI = 29.02, and mean improvement 2.0 ± 4.1 mmHg. The NCE subgroup was composed of 24 subjects (9 males, 15 females), mean improvement 0.04 ± 4.5 mmHg, mean age 51.6 years, mean BMI 31.4. The CE and NCE groups had similar marital status and frequencies of reported tobacco, caffeine, and alcohol use. Compared to CE subjects, NCE subjects were older ($p = 0.05$), more frequently taking medications for hypertension ($p = 0.05$), and had more family cardiovascular disease history ($p = 0.05$). In addition, trends in NCE subjects included a longer personal history of hypertension, less alcohol use, and had a greater African American racial percentage; however, these were not statistically significant compared with CE subjects. The mean actual SBP for the CE group was significantly lower compared to the NCE group on day 1; 132.8 mmHg and 139.6 mmHg respectively ($p = 0.05$). Similarly, the mean SBP on day 4 was lower for the CE group, however not significantly ($p = 0.108$). Refer to Tables 4-1, 4-2, 4-3, and 4-5 for demographic, health status, lifestyle factors, and clinical data for these groups.

To test hypothesis 2, an independent samples t-test was used. The difference between the two groups was not statistically significant ($t = -1.333$, $df = 37$, $p = 0.19$). The mean improvement scores of NCE hypertensives (mean improvement 0.04 ± 4.5 mmHg) were not significantly different than the mean improvement of CE hypertensives (mean improvement = 2.0 ± 4.1 mmHg). Because the analysis performed in hypothesis 2

is comparing the two independent groups improvement using an independent samples t-test, it is unclear if the college-educated group alone improved significantly between day 1 and day 4. Therefore, a paired-samples t-test was performed to assess the change in mean AD from day 1 to day 4 among the CE subjects.

Hypothesis 3. College-educated hypertensives decrease their mean AD post-training compared to pretraining.

A paired samples t-test was calculated to compare the pretraining mean AD to the posttraining mean AD among CE hypertensives. The mean AD on day 1 was 9.74 ± 3.4 mmHg and the mean AD on day 4 was 7.78 ± 2.0 mmHg. A significant decrease in mean AD from pretraining to posttraining was found using a one-tailed test ($t = 1.86$, $df = 14$, $p = 0.04$). This supports the hypothesis that CE hypertensives improve their accuracy significantly after training. Refer to Tables 4-1, 4-2, and 4-3 for a description of the demographic, health status, and lifestyle factors data of the CE subjects. Refer to Table 4-5 for a description of the clinical characteristics of the group of CE hypertensives, expressed using means and standard deviations.

An independent samples t-test was calculated to compare the mean improvement scores of female CE hypertensives compared to mean improvement scores of male CE hypertensives. The mean improvement scores for female CE hypertensives ($N = 9$) was 3.31 ± 4.76 mmHg. The mean improvement for male CE hypertensives was -0.093 ± 1.17 mmHg. This difference between groups approached, but did not reach statistical significance ($p = 0.069$). As shown in Figure 4-2, male CE hypertensives actually worsened their ability to estimate their SBP after the training, while the female CE hypertensives improved at near statistically significant levels ($p = 0.069$).

Table 4-5. Clinical characteristics of college-educated hypertensives (N = 15).

	College-educated hypertensives (N = 15)			Noncollege-educated hypertensives (N = 24)		
	Initial screening	Day 1	Day 4	Initial screening	Day 1	Day 4
Age (yrs.)	*43.2 ± 13.7			*51.6 ± 8.8		
Weight (lbs.)	191.0 ± 47.9			196.0 ± 46.2		
Height (in.)	67.7 ± 4.2			66.1 ± 4.8		
BMI (kg/m ²)	29.0 ± 5.0			31.4 ± 5.6		
Actual SBP (mmHg)		*132.8 ± 10.3	132.1 ± 11.3		*139.6 ± 10.8	138.5 ± 12.1
Estimated SBP (mmHg)		135.9 ± 7.8	132.7 ± 10.6		138.1 ± 9.2	138.2 ± 12.2
Absolute difference ASBP-ESBP (mmHg)		9.7 ± 3.4	7.8 ± 2.0*		10.3 ± 3.7	10.2 ± 3.5
Number of ASBP- ESBP Observations		12.8 ± 2.9	13.1 ± 1.6		12.2 ± 1.7	11.8 ± 1.6

Values are expressed as means ± standard deviations.

* $p \leq 0.05$ versus pretest scores by paired t-test within groups or independent samples t-tests between groups.

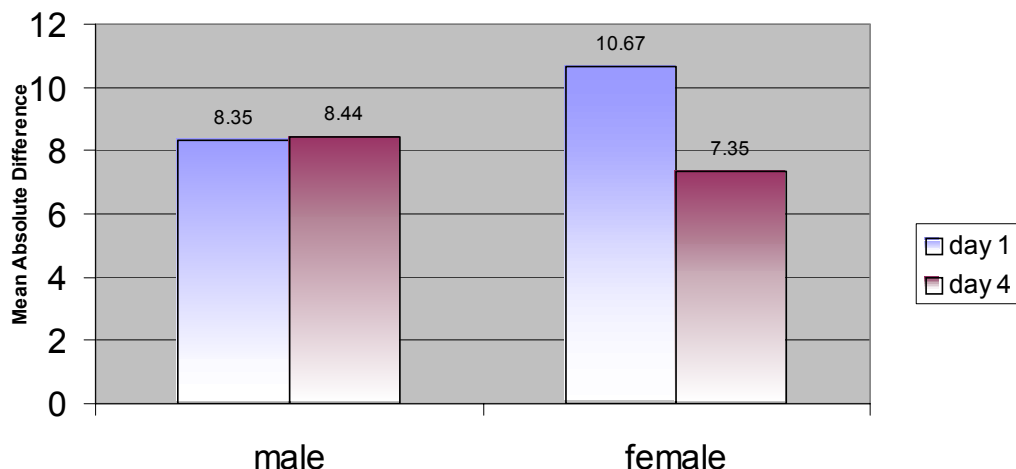


Figure 4-2. Gender effects on estimation of SBP among college-educated hypertensives (N = 15).

Hypothesis 4. Hypertensives with a BMI <30 differ significantly than hypertensives with a BMI \geq 30 in their mean improvement.

To test the hypothesis that hypertensives with a BMI < 30 differ significantly than hypertensives with a BMI \geq 30 in their mean improvement, an independent samples t-test was used. The BMI < 30 group was composed of 17 subjects; 3 males and 14 females. The BMI \geq 30 group was composed of 22 subjects; 12 males and 10 females. Compared to BMI < 30 group, subjects with BMI \geq 30 had significantly more males ($p = 0.02$), asthma ($p = 0.05$) and showed trends toward more chronic pain history ($p = 0.06$) and less exercise ($p = 0.09$). The mean actual SBP for the BMI < 30 group (N = 17) was greater than the mean actual SBP of the BMI \geq 30 group of subjects; however this difference was not statistically significant. The BMI < 30 groups' mean actual SBP was 139.5 on day 1 and 138.2 on day 4. The BMI \geq 30 group had a mean actual SBP of 135.1 mmHg on day 1 and 134.5 mmHg on day 4.

An independent samples t-test was calculated to compare the mean improvement score of the BMI < 30 subgroup with the mean improvement score of the BMI \geq 30

subgroup. The mean improvement score of the BMI < 30 subgroup was $.9 \pm 3.9$ mmHg and the mean improvement score for the BMI ≥ 30 subgroup was $.7 \pm 4.9$ mmHg. The mean improvement scores of the BMI < 30 group were not statistically different than the mean improvement scores of the BMI ≥ 30 group ($t = -.158$, $df = 37$, $p = .875$).

Figure 4-3 shows the effects of BMI level on estimation of SBP as measured by absolute differences of actual and estimated SBP for days 1 and 4. As shown in Figure 4-3, both subgroups of BMI had decreases in their mean AD between day 1 and day 4; however, these trends were not statistically significant.

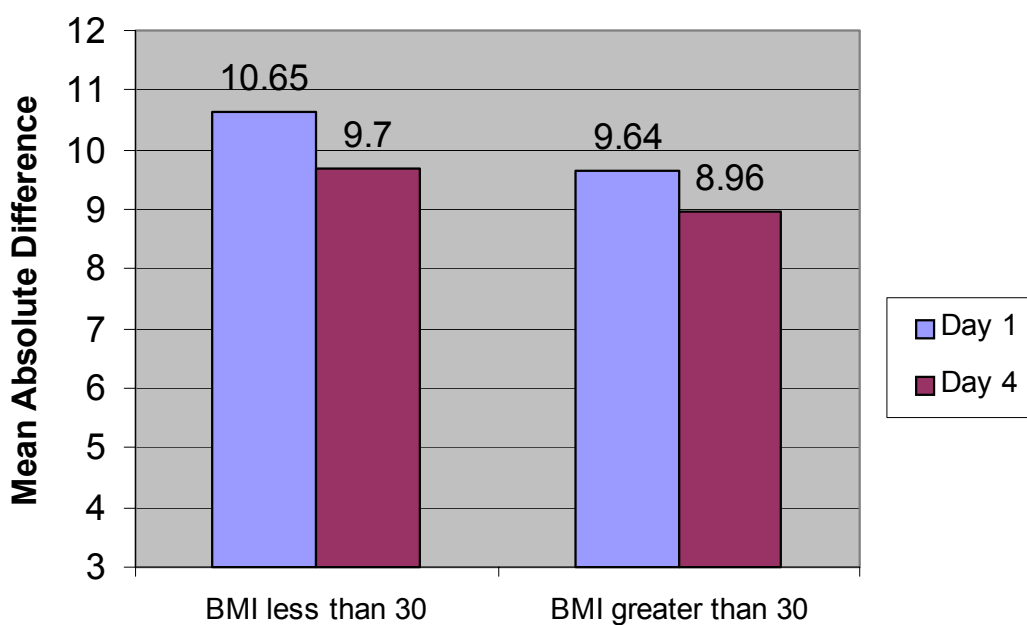


Figure 4-3. BMI Effects on Estimation of SBP in Total Sample (N = 39).

Hypothesis 5. Male hypertensives differ significantly in their mean improvement, compared with female hypertensives.

To test the hypothesis that male hypertensives differ significantly in their mean improvement, compared with female hypertensives, an independent samples t-test was performed. There were 15 male subjects and 24 female subjects. Comparing both groups, male mean age was 45.1 years versus 50.4 years ($p = 0.17$) and mean BMI was

32.3 versus 29.3 ($p = 0.06$). The groups differed significantly in terms of medication use ($p = 0.02$) and medication type ($p = 0.01$). Among females, 96% reported taking one medication on a daily basis compared with 67% males. Seventy percent of females reported taking antihypertension medications, whereas only 40% of males reported taking hypertension medications. Mean BMI for males was higher than females (32.3 kg/m^2 versus 29.3 kg/m^2 respectively) ($p = 0.07$). Actual SBP levels for days 1 and 4 were not significantly different between males compared to females. Interestingly, female subjects mean actual SBP decreased from 136.6 mmHg on day 1 to 134.8 mmHg on day 4. The mean actual SBP was also lower for the females compared with the males. Additionally, the males' mean actual SBP increased between days 1 and 4, while the females' mean actual SBP decreased. These trends in mean SBP were not found to be statistically significant.

An independent t-test was calculated comparing the mean improvement scores of male hypertensive subjects to female hypertensive subjects. No significant differences were found ($t = -.752$, $df = 37$, $p = .457$). The mean improvement of the male hypertensives (0.1 ± 4.4 mmHg) was not significantly different than the mean improvement of the female hypertensives (1.2 ± 4.5 mmHg). Figure 4-4 shows the effects of gender on estimation of SBP among the total sample of hypertensive subjects ($N = 39$).

Hypothesis 6. Hypertensives < 48 years of age differ significantly compared to hypertensives ≥ 48 years and older.

To test the hypothesis that hypertensives < 48 years of age differ significantly compared to hypertensives ≥ 48 years and older, an independent samples t-test was performed. The < 48 years of age group was composed of 24 subjects (8 males and 16

females). The ≥ 48 years of age group was composed of 15 subjects (7 males and 8 females). Both groups were similar for all demographic variables except education level ($p = 0.03$). Sixty percent of younger subjects were college-educated compared with 25% for the older group. The mean actual SBP for the < 48 years of age group was 130.8 mmHg for day 1 and 131.3 mmHg for day 4. The mean actual SBP for the ≥ 48 years of age group was 140.9 mmHg for day 1 and 139.0 mmHg for day 4. The < 48 years of age group had significantly lower actual SBP than the ≥ 48 years of age group on days 1 and 4 ($p = 0.004$ and $p = 0.05$ respectively by independent samples t-test).

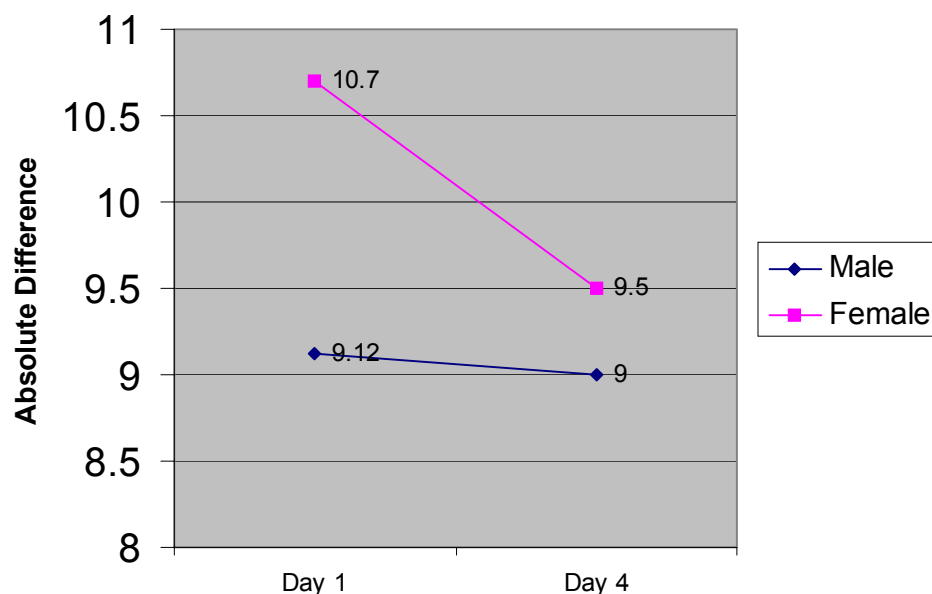


Figure 4-4. Gender Effect on Estimation of SBP in Total Sample (N = 39).

An independent samples t-test was calculated comparing the mean improvement of hypertensives less than 48 years of age to hypertensives aged 48 years and older. The mean improvement of hypertensives less than 48 years of age (0.9 ± 2.9 mmHg) was not significantly different than the mean improvement of hypertensives 48 years of age and older (0.71 ± 5.2 mmHg). No significant difference was found ($t = .117$, $df = 37$, $p = .907$). Refer to Figure 4-5 for graphical presentation of these results.

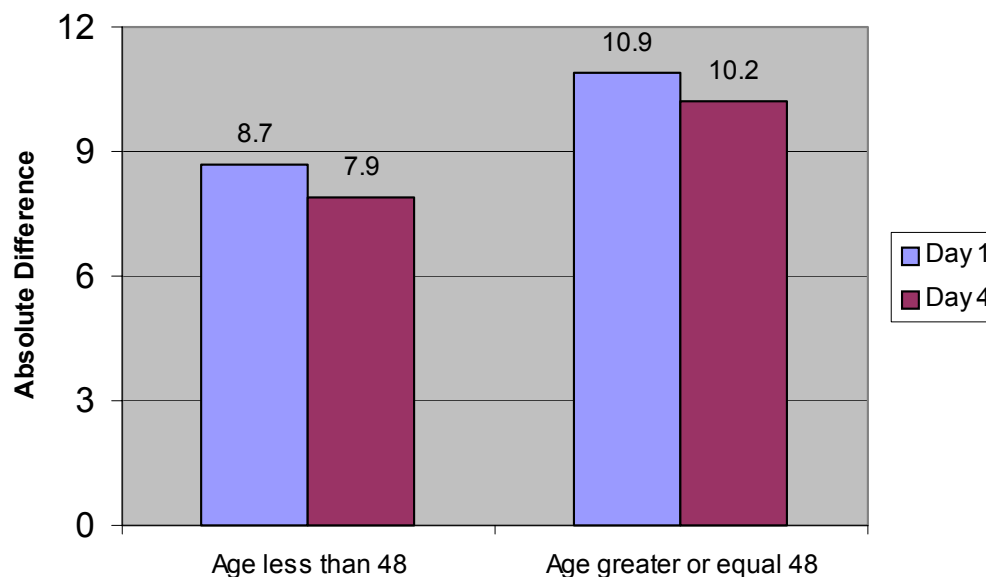


Figure 4-5. Age and Estimation of SBP in Total Sample (N = 39).

Hypothesis 7. Hypertensives using antihypertension medication differ significantly in their mean improvement compared with hypertensives not taking medications.

To test the hypothesis that hypertensives using antihypertension medication differ significantly in their mean improvement compared with hypertensives not taking medications, an independent samples t-test was performed. The HM nonuser and HM user subjects were similar in age, marital status, and education level. The HM nonuser subjects trended to be more overweight, had the diagnosis of hypertension longer, and had higher actual and estimated SBP compared with the HM users; however, these trends were not significant. The HM users mean SBP on days 1 and 4 were similar to the HM nonusers; with a mean actual SBP of 136.5 for HM users and 137.7 for HM nonusers for both days. Refer to Table 4-6 for a description of the HM users and nonusers. Values are expressed as means \pm standard deviations, frequencies, and percentages.

An independent samples t-test was calculated comparing the mean improvement of hypertensives using HM (N = 23) to hypertensives not using medication (N = 16). A significant difference was found between groups ($t = 2.038$, $df = 37$, $p = 0.05$) for a two-

tailed test ($p \leq 0.05$). Figure 4-6 compares the mean improvement between both groups. The mean improvement of the HM nonuser group (2.4 ± 5.2 mmHg) was significantly better than the group using HM ($-.4 \pm 3.4$ mmHg).

Table 4-6. Description of antihypertension medication user and nonusers

	HM user (N = 23)	HM nonuser (N = 16)
Age	49.2 ± 10.7	47.2 ± 12.8
Gender	6 males, 17 females	9 males, 7 females
Education level		
Noncollege educated	15 (65.2%)	9 (56.2%)
College-educated	8 (34.8%)	7 (43.8%)
Race		
Caucasian	18 (78.3%)	14 (87.5%)
African American	5 (21.7%)	2 (12.5%)
Time with diagnosis		
Less than 5 years	14 (60.9%)	13 (81.3%)
5-10 years	3 (13.0%)	3 (18.8%)
11-20 years	4 (17.4%)	0 (0.0%)
21 or more years	2 (8.7%)	0 (0.0%)
BMI	29.6 ± 5.5	31.8 ± 5.3
ASBP day 1, mmHg	136.4 ± 12.0	137.9 ± 9.6
ESBP day 1, mmHg	135.6 ± 9.5	139.7 ± 6.9
ASBP day 4, mmHg	135.1 ± 13.5	137.5 ± 10.0
ESBP day 4, mmHg	135.1 ± 14.0	137.4 ± 7.9
AD day 1, mmHg	9.7 ± 3.3	10.5 ± 3.8
AD day 4, mmHg	10.1 ± 3.1	8.1 ± 3.0*
Mean improvement, mmHg	-.4 ± 3.4	2.4 ± 5.2*
Number of observations day 1	12.0 ± 1.8	13.0 ± 2.8
Number of observations day 4	12.5 ± 1.7	12.0 ± 1.8

* $p < 0.05$

As shown in Figure 4-7, gender has an effect on estimation of SBP among subjects who are not taking HM (N = 16). Female hypertensives that didn't take medications for hypertension (N = 7) were compared to male hypertensives that didn't take medications for hypertension (N = 9) using an independent samples t-test. A significant difference ($p = 0.03$) was found between the two groups using a two-tailed

test. The mean improvement for hypertensives that did not take HM was 0.06 ± 5.4 mmHg for males and 5.5 ± 3.1 mmHg for females. This improvement is also interesting given the fact that similar findings occurred when comparing CE males and females. Refer to Table 4-7 for a description of hypotheses and major findings.

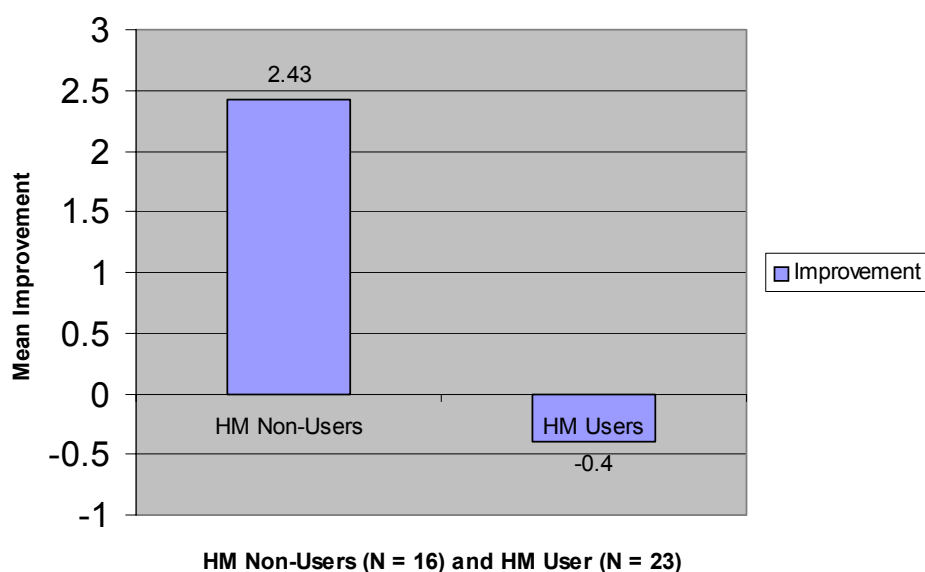


Figure 4-6. Comparison of improvement in SBP estimation between HM nonusers and HM users. * $p \leq 0.05$ by independent sample t-test.

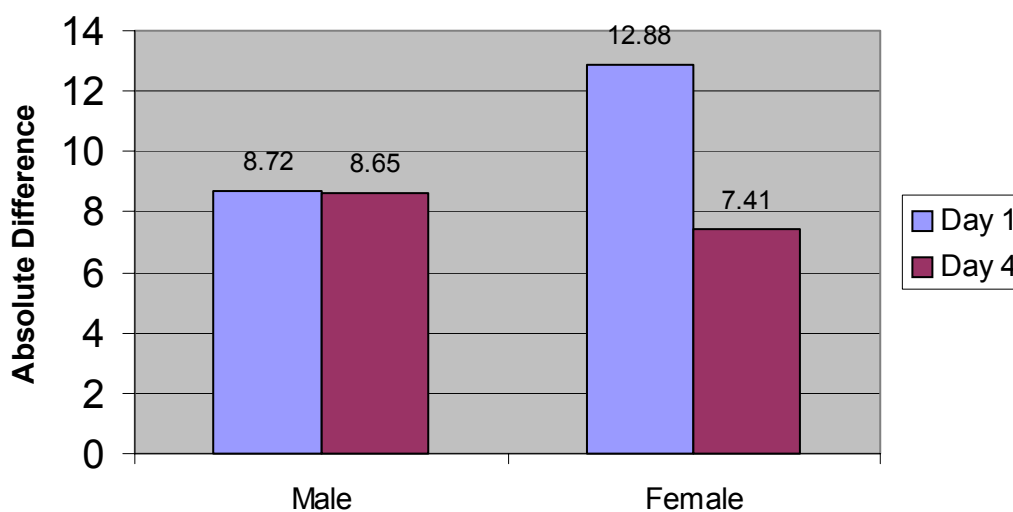


Figure 4-7. Gender effects on estimation of SBP among HM nonusers (N = 16). * Female scores statistically different than male scores at $p \leq 0.05$ level by independent sampled t-test.

Table 4-7. Summary of major outcome measures for each hypothesis

Hypothesis	Focus	Outcome measure	Analysis method	Results
H1	TS (N = 39)	Day 1 mean AD compared with day 4 mean AD	Paired-samples t-test	Mean AD D1 = 10.1 mmHg Mean AD D4 = 9.3 mmHg MI = 0.8 mmHg
H2	CE (N = 15) & NCE (N = 24)	Compare both groups mean improvement	Independent samples t-test	MI CE = 2.0 mmHg MI NCE = 0.04 mmHg
H3	CE (N = 15)	Day 1 mean AD compared with Day 4 mean AD	Paired-samples t-test	Mean AD D1 = 9.74 mmHg Mean AD D4 = 7.8 mmHg MI = 2.0 mmHg *p = 0.042
H4	BMI < 30 (N = 17) & BMI ≥ 30 (N = 22)	Compare both groups mean improvement	Independent samples t-test	MI BMI < 30 = 0.9 mmHg MI BMI ≥ 30 = 0.7 mmHg
H5	Males (15) & females (24)	Compare both groups mean improvement	Independent samples t-test	MI males = 0.1 mmHg MI females = 1.2 mmHg
H6	Age < 48 (N = 15) & Age ≥ 48 (N = 24)	Compare both groups mean improvement	Independent samples t-test	MI age < 48 = 0.9 mmHg MI age ≥ 48 = 0.7 mmHg
H7	HM nonuser (N = 16) & HM user (N = 13)	Compare both groups mean improvement	Independent samples t-test	MI HM nonuser = 2.43 mmHg MI HM user = -0.4 mmHg * p = 0.05

* Note: TS = total sample, CE = college educated, NCE = noncollege-educated, MI = mean improvement, HM = hypertension medication, BMI = body mass index.

Analysis of Covariance

It is useful to determine if there are any covarying factors that are significantly related to mean improvement of estimating SBP. A one-way between subjects ANCOVA (analysis of covariance) allows the investigator to remove the effect of a known covariate, thereby providing a method of statistical control. An ANCOVA was performed to examine the effects of gender and hypertension medication use on the total

sample mean improvement scores, covarying out the effects of BMI and age. The corrected model was significantly related to mean improvement in estimation of SBP between days 1 and 4 ($p = 0.05$). The main effect of hypertension medication use was significantly related to mean improvement ($p = 0.05$), with nonusers of hypertension medication having greater improvement (2.4 ± 5.2 mmHg) than users of hypertension medication ($-.4 \pm 3.4$ mmHg). The interaction between hypertension medication use and gender was also significantly related to mean improvement ($p = 0.03$). These effects were seen after taking into account BMI and age. Both BMI ($p = 0.5$) and age ($p = 0.9$) were not significantly related to mean improvement scores.

Reporting Symptoms and Estimating SBP

Among the total hypertensive sample, 14 (36%) participants reported symptoms associated with high BP. Reported symptoms included tenseness, flushing, and headache. A greater improvement (1.14 ± 4.9 mmHg) was seen in the 14 subjects who reported symptoms associated with elevated BP compared to the 25 subjects who did not report symptoms (0.6 ± 3.5 mmHg). An independent samples t-test was performed to compare the mean improvement scores of subjects who reported experiencing symptoms with those who did not. No significant differences were found ($t = -.38$, $df = 37$, $p = 0.71$). Refer to Figure 4-8 for a description of this data.

Among hypertensives that do not take HM, 13 subjects reported not experiencing symptoms relating to their high BP levels and 3 reported experiencing symptoms relating to their high BP levels. The mean improvement of those subjects is shown in Figure 4-9. The mean improvement of subjects who did not take HM and reported symptoms associated with high BP levels ($N=3$) improved an average of 4.14 mmHg after training. Subjects who did not report symptoms associated with their SBP and who were not using

HMs (N = 13) improved an average of 2.03 mmHg after training. The small numbers of participants in each group and the uneven distribution of subjects per group make this comparison difficult and more inquiry is needed.

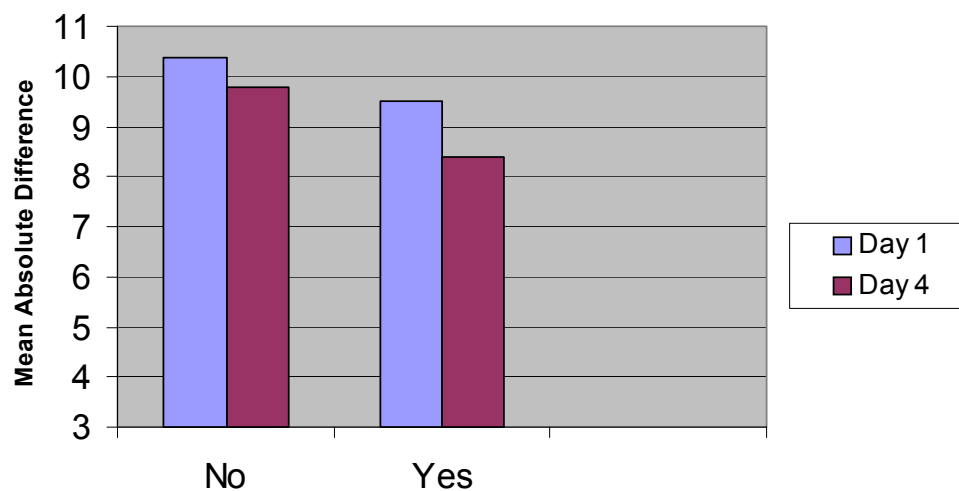


Figure 4-8. Reporting of symptoms associated with high BP and SBP estimation.

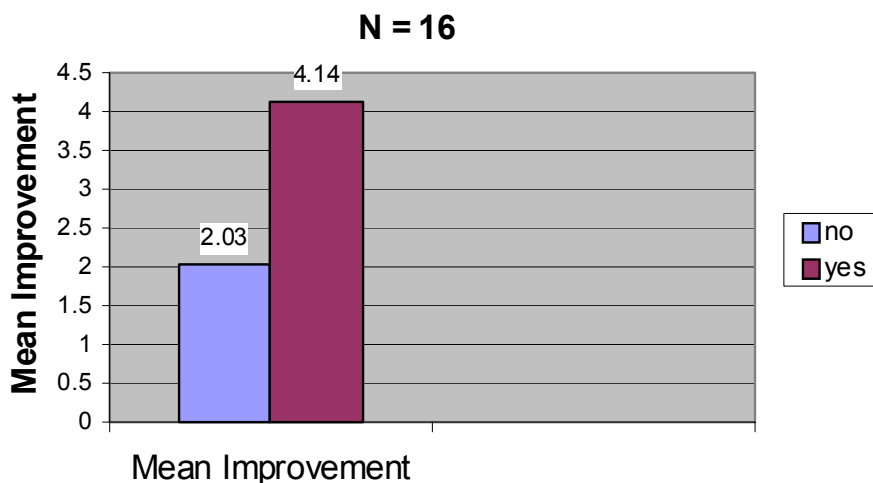


Figure 4-9. Reporting of symptoms among nonusers of HM (N = 16).

High BP Estimation

Because of the repeated measures design of the study, absolute differences can be computed for repeated measures of actual SBP and estimated SBP. A total of 1847 BP measurements/estimations were analyzed. Using SPSS, a filter variable was created by selecting only cases that were less than 140 mmHg (N = 1095). The mean AD across all

days was compared between cases that were < 140 mmHg compared with cases that were ≥ 140 mmHg. There were statistically significant differences between the two groups using an independent samples t-test ($t = 4.13$, $df = 1845$, $p = 0.001$). The mean AD across all days for the ≥ 140 mmHg group of cases was 9.37 ± 7.4 and the mean AD across all days for the < 140 mmHg group of cases was 8.0 ± 6.7 mmHg. The less than 140 mmHg group of cases ($N = 1095$) had a mean AD on days 1, 2, and 3, and 4 of 9.9 mmHg, 6.6 mmHg, and 8.8 mmHg, respectively. The greater than or equal to 140 mmHg group of cases ($N = 752$) had a mean AD on days 1, 2, and 3, and 4 of 10.0 mmHg, 8.7 mmHg, and 9.9 mmHg, respectively.

CHAPTER 5 DISCUSSION AND RECOMMENDATIONS

All descriptive and analytic results that addressed each research hypothesis will be discussed in this chapter. Conclusions and implications for clinical practice as well as recommendations for future research will also be provided.

Discussion of Results

This study was unique in its design and attempts to directly focus on hypertensives and provide both physiological and self-awareness feedback, especially using a repeated measures design and ambulatory BP monitoring. Similar studies have undertaken the task of determining the effects of feedback on BP estimation (Barr et al., 1988; Cinciripini et al., 1979; Greenstadt et al., 1988; Luborsky et al., 1976). However, the present study is the first to examine the effects of biosituational feedback using ambulatory monitoring on the estimation of SBP in adult hypertensive persons. Prior studies were composed of normotensive, younger, male samples. In addition, this study sought to uncover differences in estimation of SBP among a population of adult hypertensives and also between different sub-groups of the sample.

Hypothesis 1

For hypothesis 1, hypertensive subjects' day 1 mean absolute difference between actual and estimated SBP was compared to their day 4 mean absolute difference using a paired-samples t-test. The mean absolute difference on day 1 was 10.1 mmHg. The mean absolute difference on day 4 was 9.3 mmHg. These findings indicate that subjects

were accurate in estimating their SBP within ± 10.1 mmHg on day 1 and that they improved an average of 0.8 mmHg from day 1 to day 4. These results were not statistically significant using a two-tailed test ($t = 1.09$, $df = 38$, $p = 0.28$). These results occurred for several reasons. First, because there has been limited research hypertensive persons and estimation of SBP, it was unclear what, if any, factors would have more influence on estimation of SBP. Therefore, data were collected that captured potential influencing factors. In this study, education level, gender, BMI, age, and hypertension medication usage were examined to assess any differences between groups of hypertensives. In testing hypothesis 1, these factors were not accounted for or controlled. However, the results of hypotheses 3 and 7 indicate that college education and hypertension medication usage are important factors to consider when training hypertensive persons to become more aware of their SBP.

The mean estimated SBP for the total sample was 137.3 ± 8.6 mmHg on day 1 and 136.1 ± 11.8 mmHg on day 4. It is interesting to note that while the mean actual and mean estimated SBP were fairly similar on day 1 and day 4, the standard deviation was not as similar. For example, the standard deviation on day 1 for actual and estimated SBP was 11.0 mmHg and 8.6 mmHg respectively. The actual variation in SBP was greater than the variation of estimated SBP. On day 1, subjects were given their mean SBP level for the first study day. This information was given to assist the subject in having a better idea of where to start the estimation process. The data indicate that on the first study day, subjects tended to estimate their SBP within a tight range of numbers and tended to stay within this range. Additionally, one estimate was highly correlated with the immediately following estimate. For example, using the SAS statistical software package, a mixed model procedure was performed to assess the relationship between one

estimate and the next. This relationship was 0.80, indicating that the two measurements were highly correlated. This means that subjects also tended to guess their SBP fairly consistently, and changed their estimation based on some knowledge/intuition. This finding has been seen in other studies (Brondolo et al., 1999). Although subjects tended toward improving improved their mean absolute difference between day 1 and day 4, the improvement was not significant.

Hypothesis 2

Because recent literature suggests that there are differences in health status and greater health disparities among CE versus NCE persons, educational level was treated as a subgroup and divided into two categories; CE and NCE. Hypothesis 2 compared CE hypertensives with NCE hypertensives to assess differences in their mean improvement scores for day 1 and day 4. The mean improvement was calculated by taking the absolute value of the mean difference between day 1 and day 4 for each subject and then averaging this by the total number of subjects in that group. There were no statistically significant differences in mean improvement between the two groups using an independent samples t-test.

NCE hypertensives had significantly more family cardiovascular disease ($p = 0.05$), were older ($p = 0.05$), and used more hypertension medications ($p = 0.05$). The mean actual SBP for the CE group was significantly lower compared to the NCE group on day 1; 132.8 mmHg and 139.6 mmHg, respectively ($p = 0.05$). Similarly, the mean SBP on day 4 was lower for the CE group, however not significantly ($p = 0.108$). These findings are not surprising considering that older age and lower educational level have been associated with higher BP and greater morbidity and poorer outcomes (de Gaudemaris et al., 2002).

While the findings of the present study indicate that the educational groups were not significantly different from one another in their ability to estimate their SBP, it was interesting to note that CE subjects had fairly good improvement (2.0 ± 4.1 mmHg) after the training intervention. Because of this finding, hypothesis 3 examined the effects of training on CE hypertensives.

Hypothesis 3

For hypothesis 3, the mean absolute difference between actual and estimated SBP on day 1 was compared to the mean absolute difference between actual and estimated SBP on day 4 among CE hypertensives. The mean AD on day 1 was 9.7 ± 3.4 mmHg and the mean AD on day 4 was 7.8 ± 2.0 mmHg. CE subjects were able to estimate their actual SBP within ± 7.8 mmHg, after the training intervention. A significant decrease was found in the mean AD from day 1 to day 4, suggesting that CE hypertensive subjects significantly improved their accuracy in estimating their SBP after training ($p = 0.04$, one-tailed-test). These findings are not surprising considering that educational level affects learning and outcomes (Myllykangas et al., 1995; Winkleby et al., 1992). These findings are similar to published BP estimation research that found that subjects did not have good prediction of their BP prior to feedback or training (Baumann & Leventhal, 1985; Fahrenberg et al., 1995). In addition, these findings support the results from feedback intervention type studies that reported improvement in estimating SBP. Similarly to the current study findings, Luborsky et al., (1976) reported that subjects improved from 11.5 mmHg to 7.4 mmHg after a feedback intervention.

In a study performed by Wizner, Gryglewska, Gasowski, Kocemba, and Grodzicki (2003), hypertensive subjects, compared with normotensives, were less aware of normal BP values. The authors concluded that poor awareness of normal BP values in

hypertensives could be an important factor hindering better BP control. Furthermore, according to the goals delineated by Healthy People 2010, it is important for people to know their BP range and be able to state whether their BP was normal or high (Healthy People 2010, 2003). Therefore, ± 7.8 mmHg is a reasonably good level of accuracy considering it would be clinically important if a subject could assess his/her high SBP within 7.8 mmHg. This finding supports the hypothesis that CE hypertensives decrease their mean AD after training compared to before training. While these results are promising, additional research is needed to test these findings, such as using a larger sample of CE hypertensives age-matched with equal numbers of both genders and controlling for HM use.

Gender analysis was performed to assess mean improvement among male compared to female CE subjects. Gender differences were noted, as female CE hypertensives ($N = 9$) improved 3.3 ± 4.76 mmHg while the male CE hypertensives actually got worse after the training, with a mean improvement score of -0.09 ± 1.2 mmHg. The difference between the two gender groups approached, but did not reach statistical significance ($p = 0.07$). Differences between males and females exist which are emotional, psychological, and physiological and can affect learning patterns (Asai et al., 2001; Hyman & Pavlik, 2001). In a recent study, health disparities in relation to hypertension were examined. Females with less education had almost a 100% increase in hypertension compared with females with more education (de Gaudemaris et al., 2002). Potentially these factors may play a role in females having greater improvement over males.

Clinical characteristic differences between the two groups were found as the CE group was thinner than the NCE group; BMI = 29kg/m^2 and 31.4 kg/m^2 respectively

(nonsignificant difference) and had lower mean actual and estimated SBP levels (significant for day 1, $p = 0.05$). Similar to the total sample of hypertensive subjects, nonsignificant trends in standard deviations of actual and estimated SBP were seen in both the NCE and CE groups. On day 1, subjects in both groups had less variability in their estimation of their SBP compared with day 4. This occurrence, however, was not statistically significant. The findings of hypothesis 3 are in line with research linking intervention success and health outcomes with education level.

Hypothesis 4

Hypothesis 4 assesses whether there are differences in mean improvement between hypertensives with a BMI less than 30 compared to hypertensives with a BMI greater than or equal to 30. There were no significant differences between the BMI groups mean improvement scores, by independent samples t-test ($t = 0.16$, $df = 37$, $p = 0.88$). It would seem possible that people with increased BMI, who may potentially have elevated SNS activity, would have more improvement in or accuracy in estimating their SBP. However, this did not occur. The mean improvement score of the BMI < 30 group was $.9 \pm 3.9$ mmHg and the mean improvement score for the BMI ≥ 30 group was $.7 \pm 4.9$ mmHg. Therefore, on average, subjects in both groups only improved 0.8 mmHg after training.

The mean actual SBP for the BMI less than 30 group ($N = 17$) was greater than the mean actual SBP of the BMI greater than or equal to 30 group of subjects. The BMI < 30 groups' mean actual SBP was 139.5 on day 1 and 138.2 on day 4. The BMI ≥ 30 group had a mean actual SBP of 135.1 mmHg on day 1 and 134.5 mmHg on day 4. At first glance, these findings are surprising considering that a greater BMI has been correlated with higher BP in several studies (Masuo, Mikami, Ogihara, & Tuck, 2000;

Uehara, Miyazaki, Kanase, Sugano, & Toyo-Oka, 1996). However, a majority (85%) of the total sample took some type of medication, while 59% used antihypertensive medication regularly. This probably accounts for the lower SBP levels in the BMI group.

No differences between groups may have occurred for several reasons. First, the BMI ≥ 30 group had significantly more males than the BMI < 30 group ($p = 0.02$). This may have played a part in training and awareness of SBP patterns and estimation. As previously described, there were significant differences among males compared to females in the HM nonuse group with regard to mean improvement. Similarly, there were nonsignificant trends in females having better improvement than males in the CE subgroup ($p = 0.069$). It is clear that females improved greater than males in CE and HM nongroups. More research is needed to assess the impact of gender and BMI groups on improvement of estimating SBP.

In addition, hypertension medication use is probably an important factor in whether people can improve the estimation of SBP. A larger sample size is needed to assess the covariates hypertension medication use, gender and BMI.

Interestingly, subjects who weighed more (BMI ≥ 30) had significantly more asthma ($p = 0.05$) and chronic pain ($p = 0.05$) compared to the thinner (BMI < 30) hypertensive subjects. These findings are similar to research on BMI and health status. People who weigh more and have greater BMI's are at greater risk for developing health problems and high BP (Wizner et al., 2003; Uehara et al., 1996).

Hypothesis 5

Hypothesis 5 sought to examine gender differences in estimating SBP. An independent samples t-test was performed to compare the mean improvement scores of male hypertensive subjects with female hypertensive subjects. No significant differences

were found ($t = -.752$, $df = 37$, $p = .457$). The mean improvement of the male hypertensives (0.1 ± 4.4 mmHg) was not significantly different than the mean improvement of the female hypertensives (1.2 ± 4.5 mmHg). Refer to Figure 4-4 for a graph of gender differences.

When controlling for HM use between both genders, the mean improvement for HM nonusers was 0.06 ± 5.4 mmHg for males ($N = 9$) and 5.5 ± 3.1 mmHg for females ($N = 7$). Using an independent samples t-test, female hypertensives that did not take medications for hypertension were compared to male hypertensives that did not take medications for hypertension. A significant difference ($p = 0.03$) was found between the two groups using a two-tailed test. The female HM nonusers were able to estimate their SBP within ± 7.4 mmHg after training, while the male HM nonusers were able to estimate their SBP within ± 8.65 mmHg. The mean improvement for female HM nonusers was ± 5.5 mmHg. The interaction between gender and HM use was significant even after covarying out the effects of BMI and age. These findings are not necessarily surprising considering that male gender has been shown to be a predictor in lack of awareness of hypertension (Asai et al., 2001; Hyman & Pavlik, 2001).

Hypothesis 6

Hypothesis 6 sought to compare two age groups of adult hypertensives to examine differences in mean improvement. An independent samples t-test was performed to compare the mean improvement of hypertensive persons less than 48 years of age with hypertensive persons greater than or equal to 48 years of age. There were no significant differences in improvement of SBP estimation between the two groups of hypertensive persons ($t = 0.117$, $df = 37$, $p = 0.91$). Refer to Figure 4-5 for an illustration of these findings. Both groups were similar in their improvement scores (0.9 mmHg for

older hypertensives and 0.7 mmHg for younger hypertensives). This finding is not surprising given that the literature suggests that there may be several types of persons with elevated SNS activity, including older and younger hypertensives and obese hypertensives.

The mean actual SBP for the younger group was 130.8 mmHg for day 1 and 131.3 mmHg for day 4. The mean actual SBP for the older group was 140.9 mmHg for day 1 and 139.0 mmHg for day 4. The < 48 years of age group had significantly lower actual SBP than the \geq 48 years of age group on both days 1 and 4 ($p = 0.004$ and $p = 0.05$ respectively by independent samples t-test). This has also been shown in other studies and underscores the differences between younger and older hypertensives (Hyman & Pavlik, 2001). Hyman and Pavlik found similar results and reported that an age of at least 65 years accounted for the greatest proportion of risk for lack of control and awareness of SBP among hypertensives.

When comparing the two age groups, the younger hypertensives were significantly more educated ($p = 0.03$) and had lower SBP levels on day 1 ($p = 0.004$) and day 4 ($p = 0.05$). It is unclear if these differences played a role in the results; however, it is clear that education and SBP levels are important in BP estimation. Among both age categories, 67% of younger subjects were HM users and 54% of the older subjects were HM users. Therefore, HM use could have been a confounding factor in why some subjects improved and why others did not. Because of the potential importance of HM use in the ability to estimate SBP, hypothesis 7 sought to uncover any differences in mean improvement between subjects who took HM medications compared with those who did not.

Hypothesis 7

An independent samples t-test was performed to compare the means of hypertensive persons using HM (N = 23) to hypertensive persons not using HM (N = 16). As Table 4-6 shows, the HM nonuser and HM user subjects were similar in age, marital status, and education level. The HM nonuser subjects were more overweight, had the diagnosis of hypertension longer, and had greater levels of actual and estimated SBP compared with the HM Users. The HM nonusers mean weight was 216 pounds and mean height was 69.1 inches. In reviewing the literature on weight and body mass index, people with greater weight may have increased SNS activity and therefore are speculated to possibly have more signs or symptoms related to SNS activation (Masuo, Mkami, Itoh, & Tuck, 2000). It is unclear whether this affects their ability to be more aware of their SBP.

A significant difference was found between groups ($t = 2.038$, $df = 37$, $p = 0.05$) for a two-tailed test ($p \leq 0.05$). HM use continued to be significantly related to mean improvement even after covarying out the effects of gender, BMI, and age. The mean improvement of the HM nonuser group (2.4 ± 5.2 mmHg) was significantly better than the group using HM ($-.38 \pm 3.4$ mmHg). These findings were observed even after performing an ANCOVA, covarying out the effects of BMI and age ($p = 0.05$). These findings are not surprising considering the knowledge regarding the main and residual effects of antihypertensive medications. BP medications attempt to decrease BP by affecting pathways in the body responsible for BP control (e.g., RAAS, slow-calcium channel, and beta-receptors). Even though antihypertensive medications are designed to be selective in lowering BP safely, additional and/or side-effects of antihypertension medications are known to occur. For example, beta-blocking medications are reported to

have adverse effects such as bradycardia, syncope, low BP, asthmatic attacks, congestive heart failure, hallucinations, loss of appetite, headaches, nausea, weakness, and depression (Smith & Reynard, 1995). These adverse effects may impact the potential for people with hypertension to experience symptoms associated with their high BP. It has been reported that adults who are obese have impaired adrenergic and baroreflex function (Grassi et al., 2000). It may be possible that obese hypertensive persons may have signs or symptoms associated with increased SNS activity and/or BP elevation, but this is unreported in the literature.

Reporting of Symptoms

There were also trends noted among subjects who reported symptoms associated with high BP versus those subjects who did not report symptoms associated with high BP. Interestingly, subjects who reported symptoms associated with high BP showed a trend toward greater accuracy before and after training and a trend toward greater improvement in estimating their SBP. Although these findings are not statistically significant, it may be important to continue this inquiry into additional factors that may be related to estimation of SBP. For example, when controlling for HM use, the mean improvement in subjects who reported symptoms and did not take HMs was 4.1 mmHg compared to 2.0 mmHg for HM users. These findings show promise in creating a model that may help distinguish people who could be more easily trained to estimate their SBP, however the sample size was small ($N = 3$) for the subjects who reported symptoms and did not take HMs. In future studies, it may be worthwhile to examine HM users and nonusers improvement after training separately and control for reporting of symptoms.

BP Variability

There are several factors to consider when evaluating BP estimation. First, variability of BP is important because without fluctuations, patients have no comparison experiences to facilitate awareness of different levels of BP. BP variability is influenced by both bio-situational and behavioral factors, presumably through central modulation of autonomic drive to the heart and sympathetic blood vessels (Esler, 2000). People without hypertension or circulatory disease may not display great variations in BP or have symptoms that may be associated with SNS activation. Because the physiological literature suggests that people with high BP may have fluctuations in circulating hormones and changes in SNS activity, it was important to evaluate people who are experiencing typical day-to-day life stressors. Variability was achieved in this study as subjects' mean actual SBP on day 1 was 137.0 ± 11.0 mmHg and 136.1 ± 12.1 mmHg on day 4. This finding was similar to actual SBP data from 10 randomly selected subjects in Dr. Yucha's study that showed an average daytime range in SBP of 33.2 mmHg, ranging from a minimum of 19 to a maximum of 56 mmHg monthly (unpublished BP variability data, 2001).

High BP Estimation

When analyzing the repeated measurements and calculated scores, subjects estimated their SBP within a tight range of numbers and had little deviation compared to the actual SBP measurements. Furthermore, with a large increase in SBP level, one might imagine that potentially subjects may get better in estimating their SBP. However, the opposite was true. As subjects' actual SBP got larger they continued to estimate within their mean range and therefore their absolute difference scores were extremely high.

Brondolo et al. (1999) found that higher or lower levels of BP did not account for the individual differences in the ability to estimate BP. In the present study, among the total sample repeated measures data, estimation of SBP lower than 140 mmHg was significantly better than estimating SBP greater than or equal to 140 mmHg. Because subjects estimated their SBP very close to their mean SBP, it was not surprising that when SBP levels were 140 mmHg or higher, subjects were significantly poorer estimators than the times when SBP was less than 140 mmHg ($t = 4.13$, $df = 1845$, $p = 0.000$). This may have occurred for a number of reasons. First, subjects may have not had any cues at all about their SBP and estimated their SBP based on the first few SBP readings and mean score provided to them on day 1. Other studies found that BP estimation was better when subjects were provided with previous BP measurements (Barr et al., 1988; Brondolo et al., 1999; Cinciripini et al. 1979; Greenstadt et al., 1986; Luborsky et al., 1979). Alternatively, they may have had cues of changes in BP, but not have been confident in estimating too much over or under their mean SBP level. It may be prudent to perform a follow-up study to assess the impact of the training intervention without providing subjects their initial mean SBP levels and instead discuss with them their SBP variability and average after the training is completed. This may help them understand SBP variability and have more ability to estimate their high and low ranges and possibly improve overall accuracy.

Conclusions

This study sought to examine the effects of an ambulatory BP, biosituational awareness training intervention on the ability of hypertensive persons to estimate their SBP. The findings of this study are similar to studies that have examined both BP estimation and BP estimation with feedback. Among the feedback intervention-type

studies that have been performed to date, all showed an improvement in BP discrimination after feedback (Barr et al., 1988; Brondolo et al., 1999; Cinciripini et al., 1979; Greenstadt et al., 1986; Luborsky et al., 1976;) and with a similar degree of improvement. This study was unique in that it assessed adult hypertensives using ambulatory BP monitoring coupled with biosituational self-awareness training to improve SBP estimation.

The major findings of this study were four-fold. First, hypertensives that were college-educated had statistically significant improvement in estimating their SBP after the training intervention ($p = 0.04$). Secondly, hypertensive persons not using antihypertensive medications improved at a statistically significant level compared to hypertensive persons who used antihypertensive medications ($p = 0.05$). Third, hypertensive females who did not take antihypertensive medications improved significantly compared with hypertensive males that did not take antihypertensive medications ($p = 0.03$). Finally, as actual SBP increased, estimation of SBP was not as accurate compared with less than 140 mmHg SBP levels ($p = 0.000$).

Implications for Clinical Practice

In the clinical setting, it would be valuable if clinicians could teach patients how to estimate and monitor their BP while they are in their normal environment. As patients become more knowledgeable and responsible regarding their healthcare choices, it is important for clinicians to find alternative and innovative means to improve the care and outcomes of patients. Over the past several years, changes have occurred in health care that have made patients more than mere passive participants of their healthcare. Patients are much more willing and able to learn more about their health and well-being than previous generations (Strohecker, 1999). Teaching people about their BP and BP

patterns is an effective way to improve health of patients and empower people with hypertension to have more control over their own life and health (Healthy People 2010, 2003). This study provides evidence that some hypertensive persons can learn to estimate their SBP at least within an average of ± 7.4 mmHg and that most patients can at least be trained to have more awareness of their mean/range BP information and estimate their SBP within an average of ± 9.2 mmHg. This intervention may also be valuable for patients who need to learn about their SBP patterns. All patients with hypertension should be aware of their BP patterns; patients who are unmotivated to participate in their treatment and/or are unsure of their BP patterns many benefit from learning more about their BP patterns and BP variability. Although the present study did not test this claim, this intervention may improve patients' motivation to seek or continue treatment. More research examining outcomes and SBP estimation is needed to confirm this assertion.

During and after the study, it was clear that some subjects were surprised that their BP levels were as high as they were. In addition, the majority of subjects whose BP levels were outside of the normal range expressed the need to visit their healthcare provider. Some subjects reported visiting their healthcare provider and switching/adding medications because of the knowledge of their actual SBP readings over an extended period of time. It was also valuable for subjects to obtain the 4-day print-out of the results of the ambulatory BP monitoring sessions. Subjects were generally excited to participate and receive the print-out to bring to their healthcare provider.

Although the findings showed significant improvement for certain subjects, it should be noted that this improvement, although significant, was relatively small. Because participants were only aware of their SBP within approximately 7-8 mmHg after training, the findings should not be used to solely treat or directly monitor SBP.

However, the findings of this present study could be used to assist nurses and other healthcare providers in teaching patients about their BP patterns and variability and motivating patients to adhere to prescribed treatments. While this research is promising, more inquiry is needed with larger samples and focused inquiry to see if people with hypertension can improve their awareness of their high BP episodes after participating in ambulatory BP and biosituational feedback training and if this training will ultimately improve healthcare outcomes.

Recommendations for Future Research

A limitation of this study was that the sample size was relatively small to analyze discrete differences among groups of subjects. It was realized during the data analysis phase that there were differences between genders among the CE and HM group. While the subgroups were fairly evenly distributed, the small number of participants in the CE group made it difficult to examine discrete differences in gender among the group of CE hypertensives. Therefore, a large-scale study that includes adult college-educated hypertensives, taking into account HM usage and gender, is recommended. Additionally, because HM use seems to be such an important factor in SBP estimation, a large-scale study examining hypertensive adults who take and do not take antihypertensive medication is also recommended. Although this study consisted of a relatively small group of hypertensive adults (N = 39), it provides useful information that helps elucidate factors that are related to and important in assisting hypertensive persons to be more aware of their SBP levels. Prior to this research inquiry, these factors were largely unknown and mostly speculative.

Another limitation of the study is that subjects were given baseline information about their actual SBP prior to day 1 (pretraining). Actual SBP information was

provided to the subjects because it was thought that subjects may need more information to help them make a guess about their SBP level. While it was true that this information did assist subjects in understanding how to estimate their SBP level, it may be more useful to give subjects broad information about SBP and keep them totally blinded to their actual SBP information until after the pretraining day. Providing this knowledge to them, while useful to the subjects in understanding their SBP and how to estimate their SBP, may have limited the amount of improvement that was captured in this intervention between days 1 and 4. It is recommended to only provide broad SBP range information and to keep the subjects totally blinded to their actual SBP during the pretraining phase.

This research study provides support for using feedback methods to improve the ability to estimate BP in certain populations, especially hypertensives who are borderline hypertensives or newly diagnosed and are not taking HMs, and suggests that BP awareness may be improved in some people using feedback methods. However, the limited number of studies testing feedback methods on hypertensives suggests that more research is needed to further assess the effects of BP awareness feedback training among this group. Research is also needed to evaluate clinical outcomes of BP awareness training, such as BP control, patient motivation and adherence with medical treatment, cost-effectiveness, and morbidity and mortality.

APPENDIX A
PRE-/POSTTRAINING SBP ESTIMATION FORM

ID# _____

University of Florida College of Nursing
SBP Estimation Study
Pre-/Posttraining SBP Estimation Form

Category	Systolic BP (mmHg)
Optimal	Less than 120
Normal	121-129
High-Normal	130-139
Stage 1	140-159
Stage 2	160-179
Stage 3	180 or greater

Instructions: Each time the BP cuff starts to inflate:
Write the time in the space provided
Circle an SBP category that corresponds to your estimated SBP
Estimate your SBP and write it in the space provided

Time	SBP category						Estimated SBP
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	
	Less than 120	121-129	130-139	140-159	160-179	180 or greater	

APPENDIX B
SBP ESTIMATION STUDY TRAINING FORM

ID# _____

University of Florida College of Nursing
SBP Estimation Study
Training Form*

Category	Systolic BP (mmHg)
Optimal	Less than 120
Normal	121-129
High-Normal	130-139
Stage 1	140-159
Stage 2	160-179
Stage 3	180 or greater

Instructions: Each time the BP cuff starts to inflate:
Write the time in the space provided
Estimate your SBP and write it in the space provided
Fill out the Self-Awareness checklist
After your BP has been measured, look at the LCD screen on the BP monitor and write down your actual SBP in the space provided

Time	Estimated SBP	Actual SBP

Self-Awareness Checklist: Circle Yes or No for each item

**Current:				**Current:		
Laughing	Yes	No		Headache	Yes	No
Frustrated	Yes	No		Sweaty Hands	Yes	No
Excited	Yes	No		Tense Stomach	Yes	No
Tense	Yes	No		Fast Pulse	Yes	No
Angry	Yes	No		Tingling in face, arms or legs	Yes	No
Rushed	Yes	No		Shortness of Breath	Yes	No
Content	Yes	No		Cold Hands	Yes	No
Annoyed	Yes	No		Warm Hands	Yes	No
Smiling	Yes	No		Happy	Yes	No
Walking	Yes	No		Location at Home	Yes	No
Running	Yes	No		Location at Work	Yes	No
Standing	Yes	No		Location Outdoors	Yes	No

Strenuous Activity	Yes	No		**Within prior 15-minutes: Using Caffeine	Yes	No
Relaxing/Nonstrenuous Activity	Yes	No		Using Alcohol	Yes	No
Lying Down	Yes	No		Using Tobacco	Yes	No
Sitting	Yes	No		Interpersonal Interaction:	Yes	No
Standing	Yes	No		Positive	Yes	No
Eating	Yes	No		Negative	Yes	No
Other Activity:	Yes	No		Neutral	Yes	No

*Adapted from Barr, Pennebaker, and Watson (1988), Brondolo, Karlin, Alexander, Bobrow, and Schwartz (1999), Gellman et al. (1990), JNC VI (1997), and Luborsky et al. (1976).

APPENDIX C
SBP ESTIMATION STUDY TRAINING FORM

University of Florida College of Nursing
SBP Estimation Study
Demographic Data Sheet

ID # _____

D.O.B. _-_-_-_-

Age _____ years

Gender:

- 1.) Male
- 2.) Female

Veteran Status:

- 1.) Yes
- 2.) No

Race

- 1.) Non-Hispanic White
- 2.) Hispanic White
- 3.) Hispanic Black
- 4.) Black or African-American
- 5.) Asian
- 6.) Island Pacific
- 7.) Other

Education

- 1.) Less than 7 years
- 2.) Junior high school (grades 7-9)
- 3.) Some high school (grade 10-11)
- 4.) High school graduate
- 5.) Some college or technical school
- 6.) College graduate
- 7.) Graduate school (master's degree or beyond)

Marital Status

- 1.) Married
- 2.) Never married
- 3.) Widowed
- 4.) Separated
- 5.) Divorced

Height ___cm

Weight ___kg

Length of time since diagnosis of hypertension:

- 1.) Less than 5 years
- 2.) 5-10 years
- 3.) 11-20 years
- 4.) 21 years or more

APPENDIX D
HEALTH HISTORY FORM

ID# _____

University of Florida College of Nursing
SBP Estimation Study
Health History Form

- 1.) Yes No Has a doctor ever told you that you have high blood pressure?
2.) Yes No Do you take medications for high blood pressure?
3.) Yes No Do you experience symptoms associated with your high blood pressure?
4.) If so, list the symptoms:

-
- 5.) Yes No Has a doctor ever told you that you have heart disease?
6.) Yes No Have you ever had a heart attack?
7.) Yes No Have you had cardiac surgery?
8.) Yes No Do you have a cardiac pacemaker?
9.) Yes No Have you ever had a stroke?
10.) Yes No Have you had carotid artery surgery?
11.) Yes No Has your doctor ever told you that you had an aneurysm?
12.) Yes No Have you ever had heart failure?
13.) Yes No Have you ever had an abnormal EKG or irregular heart rhythm?
14.) Yes No Do you have Diabetes?
15.) Yes No Do you have asthma or difficulty breathing?
16.) Yes No Do you suffer from chronic pain?
17.) Yes No Do you have pulmonary, liver, or kidney disease?
18.) Yes No Do you have cancer or tumors?
19.) Yes No Do you have thyroid problems?
20.) Yes No Do you currently smoke tobacco or use tobacco products?
21.) Yes No Have you smoked tobacco or used tobacco products in the past?
22.) Yes No Do you drink alcoholic beverages?
23.) Yes No Do you drink caffeinated beverages?
24.) Yes No Has your doctor ever told you that you have high cholesterol?
25.) Yes No Do you exercise more than one hour per week?
26.) Yes No Are you currently taking any prescription or nonprescription medications?

Name of Medication	Dosage	Frequency Prescribed	Taken as Prescribed? (Yes/No/Sometimes)

Have you or any of your blood relatives (your parents, brothers, sisters, uncles, aunts, cousins, or children) ever had:

- 27.) Yes No Heart attack
 28.) Yes No High blood bressure
 29.) Yes No Stroke
 30.) Yes No Diabetes
 31.) Yes No High cholesterol

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BIOGRAPHICAL SKETCH

Sandra Wolfe Citty was born in Parsippany, New Jersey. She received her Bachelor of Science in Nursing degree in 1995 from the University of Miami School of Nursing, Coral Gables, Florida. Upon completion of her bachelor's degree, Sandra worked as a registered nurse in the area of cardiovascular nursing.

After working as a registered nurse for three years in cardiovascular nursing, Sandra obtained her master's degree in nursing from the University of Miami School of Nursing in 1998. After completion of her degree in nursing, with specialization in family advanced practice nursing, she became board certified by the American Nurse's Credentialing Center as a Family Nurse Practitioner and was licensed as an Advanced Registered Nurse Practitioner. During this period, Sandra was active as an adjunct professor of nursing and continued in clinical nursing practice.

In 1999, Sandra returned to the academic arena as a doctoral student at the University of Florida, College of Nursing. During the course of her doctoral study, Sandra worked as a research assistant in the College of Nursing's Office for Research Support and was awarded a 2-year, predoctoral nurse fellowship at the North Florida/South Georgia Veteran's Health System. Additionally, Sandra has been funded by Sigma Theta Tau International Nursing Honor Society, Alpha Theta chapter, to help offset costs associated with her doctoral research. During the last two years of her doctoral work, Sandra was mentored by Drs. Maude Rittman and Carolyn Yucha during her position as a predoctoral nurse fellow. As a doctoral student, research assistant, and

predoctoral nurse fellow, she has had valuable experiences in learning about veteran's health issues, VA resources, grant writing, proposal development, instrument testing, subject recruitment, data management and analysis, and publishing. Sandra has published a manuscript in the *Journal of Professional Nursing* and has a manuscript (in review) in the *Journal of Applied Psychophysiology and Biofeedback*. Sandra has also presented posters at various regional, national, and international research conferences. Sandra will graduate in May of 2003 with her Ph.D. in nursing and minor area in psychology. She plans to continue her work in the field of nursing as a researcher, educator, and clinician.