HYPERTENSIVE RISK FACTOR BEHAVIOR AND BORDERLINE HYPERTENSION

by

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CHAPTER I

INTRODUCTION

Statement of the Problem

If American culture continues its present trends, more than half its citizens can expect to die of heart disease - a quarter of whom will be less than 65-years old. In 1974 cardiovascular disease claimed 54% of the death total, two-thirds of which were associated with high blood pressure (American Heart Association, 1977). This "silent killer" has been identified as the single most important risk factor in the incidence of cerebrovascular disease and heart failure (Framingham Study, 1970; Veteran's Administration Cooperative Study, 1968). Indeed, hypertension may be the greatest threat to the health of the American public.

Chronic, sustained high blood pressure in 90-95% of all cases is classified as essential hypertension (Gifford, 1976; Robbins & Angell, 1976). Though completely asymptomatic for up to 30 years, high blood pressure can be detected and medically treated long before overt symptoms develop (Henry & Cassel, 1969). Effectively treated hypertension can reduce mortality from cardiovascular disease (Veteran's Administration Cooperative Study, 1968).

Specific etiology for essential hypertension is unknown, though numerous factors have been shown to contribute to its development. Genetic determinants and familial tendencies seem to play a role in individual predisposition to hypertension and in one's physiological ability to cope with exogenous factors which place one at risk of developing hypertension (Henry, 1976; Page, 1976). More significant to the present study, however, are several behavioral patterns epidemiologically represented in American life-style, and which have been suggested as being precursory to high blood pressure. Behavioral factors implicated in the development of essential hypertension are: high salt intake, dietary mismanagement resulting in obesity, and high levels of anxiety (Ball & Turner, 1975; Gifford, 1974; Henry, 1976; Kannel, 1976). Smoking usually contributes to transient elevations in blood pressure but its effect is not sustained over long periods of abstinence (Aronow, 1973; Wright, 1975). Each behavior appears to make an individual and additive contribution to the development of hypertension and is known as a risk factor behavior.

The 1973 National Heart and Lung Institute task group on hypertension emphasized that the behavioral aspects are as important as medical treatment in reducing threat of hypertension. Their recommendations included an appeal for more research

into the cultural and social determinants of high blood pressure, and preventive health education aimed at reducing hazards by improving health habits and altering risk behavior (Weiss, 1975). Specifically mentioned were dietary and weight control, minimization of stress, and increased knowledge and awareness about hypertensive disease, its precursors and sequellae.

At the time of the task group report, it was estimated that 52% of hypertensive persons were unaware of their status. In the ensuing years much moneys and effort were allotted to programs of high blood pressure detection, and education of the public regarding possible risk factors in the genesis of hypertension. In 1976 it was postulated that only 20% were unaware of their high blood pressure, yet the incidence of hypertension had not decreased (Gifford, 1976).

In the evaluative phase, one might ask the following questions : What is the real outcome of this intense thrust in public health education? Is the public aware of risk factor behavior and its relationship to hypertension? Are people acquiring knowledge which will lead them to re-evaluate their life-style with regard to hypertensive risk factors? Does the public consider high blood pressure of significant threat to alter individual behavior patterns to avoid its onset? And ultimately, will reduction in risk factor behavior lead to a reduction in blood pressure?

Purposes of the Study

The primary purpose of the present study was to evaluate changes in risk factor behavior in a sample of borderline hypertensive persons subsequent to identification, information exposure, and discussion of hypertensive risk factor behavior.

A second purpose was to evaluate blood pressure change in the presence of risk factor behavior change.

A third purpose was to determine the relationship between changes in behavior with numerous factors thought to intervene in the learning process, i.e., knowledge, health locus of control, and threat of disease.

Limitations

The present study was limited to the group of individuals identified as borderline hypertensive during a 1976 employment site screening. Demographic data and data collected regarding risk factor behavior were ascertained solely on the basis of subjective, verbal reporting by the client. No attempt was made to validate or further quantify responses.

The interviewer effect and reactivity of one questionnaire may have influenced subject response, though the year interval between assessments probably dampened the effect.

It was difficult to assess, or even to suggest, the intervening variables which may have played a role in client learning, reinforcement, or behavior change in the interval between the initial screening and follow-up one year later.

Assumptions

For the purposes of the present study the following assumptions were made:

1) Elevated blood pressure readings exist in the presence of risk factor behavior.

2) The prevalence of risk factor behaviors, their suggested relationship to hypertension, and the epidemiological significance of hypertension indicate that risk factor behavior is an important focus with regard to blood pressure control.

3) Learned behaviors can be unlearned, or repatterned into new behaviors.

4) Hypertension is of sufficient consequence that those persons at increased risk of developing hypertension will be motivated to reduce that risk.

5) Each individual strives for cognitive harmony. Knowledge, belief, and behavior are congruent and compatible in a unified, effectively functioning human being.

Operational Definitions

Operational definitions of the following terms were used:

Adaptation means biological adjustment; defense mechanisms occuring beheath cognitive levels (Lazarus, 1969).

Anxiety level is the amount of "nervous tension" subjectively determined by an individual to be present in his/her daily life (Worker Health Program Questionnaire).

Cognition is any knowledge, opinion, or belief about the environment, oneself, or one's behavior (Festinger, 1957).

Coping is the organization of cognitive behavior in response to environmental stressors; implies mastery of psychological adjustment (Lazarus, 1965).

Family history means that a subject's parent had diagnosed hypertension, stroke, or heart attack before the age of 65 years.

Follow-up blood pressure reading is the mean systolic and mean diastolic measurement obtained 1 year after the initial reading and after educational instruction.

Hypertension means essential or systemic arterial hypertension which exists indipendent of any underlying pathology, i.e., renal artery stenosis, coarctation of the aorta, or adrenal tumor. High blood pressure measures greater than 160/95 mm Mercury (Hg). Borderline hypertension means blood pressure readings

are consistently within the range of 140-160/90-95 mm Hg. Normal blood pressure readings fell below 140/90 mm Hg (World Health Organization).

Initial blood pressure reading is the average of 6 blood pressure measurements obtained by the researcher in a 1976 employment site screening.

Learning: Cognitive learning means the acquisition, retention, and recall of facts and information about a specific topic. Effective learning means an integration of cognitive learning to affect a new behavior and/or extinguish an old behavior (Young & Simmons, 1967).

Obesity is more than 20% above weight recommended by the United States Food and Drug Administration (Grande, 1975).

Overweight is up to 20% above weight recommended by the United States Food and Drug Administration (Grande, 1975).

Prevention is activity suggested by health personnel to potential patients for the purpose of avoiding the onset, or in early detection of disease (Susser, 1975).

Salt intake means the total amount of added salt ingested daily. Salting "everything" or "regularly" is considered to be in excess.

Smoking incidence is smoking at least 10 cigarettes a day.

Justification for Study

Hypertension is an epidemiological problem by virtue of its prevalence, absence of symptoms, and severity of long-term effect (Moser, 1974; Stamler, 1974). Paradoxically, high blood pressure can be detected simply and inexpensively, and then treated long before adverse effects become evident. Convincing evidence for effective treatment of severe elevations in blood pressure was established in the Veteran's Administration Cooperative Study (1968) and the Framingham Study (1970).

Even mild elevations in blood pressure significantly increase risk of premature death (Stamler, 1974). Borderline hypertensives, in a Society of Actuaries' study (1959), demonstrated a 76.4% higher mortality rate in middle age than normotensives (Lew, 1967). Wilbur's (1968) study of 55 persons with borderline blood pressure revealed that 69% of the sample had readings in the high range 12-months later. Borderline blood pressure is indeed, a potent predictor of future hypertension (Julius, 1977; Kirkendall & Nottebohm, 1977).

Epidemiological studies in the United States and throughout the world have identified various social and cultural risk factors which are associated with the incidence of essential hypertension (Weiss, 1975). Most of the suspected risk factors are learned

behaviors. If risk behavior is learned, it can be unlearned; or alternative learning can occur which will obviate the prior behavior.

In a concerted effort to educate the public about risk factor behavior, federal, state, and community programs have been developed. Information on hypertensive disease and development has been dispersed via mass media, public program, small group, and individual learning situations. The aim of preventive health education is to provide sufficient knowledge and awareness as to affect health-related social and behavioral change (Steuart, 1975). Primarily through change in behavior is it anticipated the incidence of high blood pressure can be reduced.

But does learning, indeed, behavior change actually take place? And will a change in behavior result in a reduction in blood pressure? The answer to such questions would be of great value to the preventive health educator, as well as to those persons involved in the management of hypertensive persons and their families. Education concerning those factors which place an individual at risk of high blood pressure may lead to a reduction in risk factor behavior and thence, in the incidence of hypertension.

CHAPTER II

REVIEW OF LITERATURE AND RELATED STUDIES

Hypertension is a chronic disease which affects an estimated 25 million Americans yearly (Stamler, Schoenberger, Shekille, & Stamler, 1974). High blood pressure is asymptomatic in onset and progression, yet can result in disability and death. Though specific etiology for essential hypertension is unknown, genetic, psychological, and behavioral factors have been implicated in its development. On the basis of present knowledge and the prevalence of disease, health education has been directed toward control of those predisposing factors.

The review of literature and related studies includes comment on the epidemiology of hypertension, the pathophysiology of hypertension, identified risk factor behaviors (anxiety, overweight, salt intake, smoking), and factors influencing behavior change.

Epidemiology of Hypertension

Perhaps the most dramatic study to demonstrate the impact and seriousness of high blood pressure on death and morbidity is the 5-year Veteran's Administration study begun by Dr. Fries in 1968 - a study which had to be abandoned after 19 months due to the dramatic mortality rate of hypertensive patients in the

untreated control group. This expansive and well-documented study of 380 hypertensive veterans presented evidence of what had been suspected regarding the relationship between high blood pressure and cardiovascular disease.

Another landmark study, which confirmed the VA results, was a longitudinal follow-up of 5209 adult men and women in Framingham, Massachusettes (1970). Results of that study further suggested that factors other than the inherited characteristics of the vasculature, which determine host susceptibility and resistance, could account for higher blood pressures and concomitant increase in risk of cardiovascular disease (Kannel, Wolf, Verter, & McNamara, 1970). Though blood pressure tended to increase with age, as did the progression of atherosclerosis, there was no evidence that high blood pressure is an innocuous accompaniment of aging (Kannel, et al., 1970). The Framingham study also pointed out that environmental factors (eg., technology, rich food, social pressures...) and behavior patterns predispose most Americans to the development of this threatening and insidious disease.

Pathophysiology of Hypertension

The etiology of essential hypertension is unknown, though various malfunctions in the physiological mechanism of blood pressure

control have been postulated in disease genesis. The autonomic nervous system seems to play a major role in the development of hypertension (Page, 1974). Sympathetic activity, initiated by stress and/or emotional arousal, stimulates an increase in cardiac output and peripheral vasoconstriction which results in sudden blood pressure rise. Inappropriate sympathetic response or physiological adjustment to excesses in sympathetic activity may have a role in sustaining elevated pressures as well (Henry, 1976; Page, 1974).

Auto-regulatory disequilibrium, baro-receptor depression, hypothalamic feedback disorders, and various interlocking humoral mechanisms have also been postulated as participatory in blood pressure elevation (Henry & Cassel, 1969). A more integrated approach by Page (1974), called the mosaic theory, proposed that the components of blood pressure regulation interact in a kaleidoscope of influence, compensation, readjustment, and effect. A disruption of this dynamic equilibrium results in blood pressure rise.

Increased peripheral resistance is a hallmark of essential hypertension, though the exact mechanism of its development is not clear (Folkow, 1973). Hypertrophy of the intimal layer of the blood vessel wall occurs as an apparent adaptive response

to frequent and prolonged elevations in pressure (Folkow, 1973). Whether as an adaptive change or as an integral affector, the structural changes increase peripheral resistance, and serve to sustain the elevated pressure (Folkow, 1971; Henry & Cassel, 1969).

Atherosclerosis has been demonstrated to be in cyclical progression with hypertension and thus, factors influencing the advancement of one will have at least an indirect effect on the progression of the other (Chobanian, 1976). Atherogenesis is the accumulation of fibro-fatty plaques within the blood vessel wall, which contributes to vessel rigidity and increased resistance (Robbins & Angell, 1976).

Environmental factors are believed to be important in the aggravation of pathophysiological mechanisms of hypertension (Paul, 1977). Accumulated progression of disease may depend upon the presence of risk factor behavior over time (Paul, 1977). Excess weight, smoking, and anxiety are believed to affect blood pressure by increasing peripheral resistance, transiently or for sustained periods of time. Salt excesses tend to increase blood volume and cardiac output, which will also increase blood pressure and may ultimately lead to peripheral vascular changes.

Genetic endowment, suggested as being a multifactorial contribution, is purported to affect the propensity for structural

changes and hypertension to occur (Page, 1974; Paul, 1977; Pickering, 1974). In those persons genetically predisposed to atherogenesis and/or hypertension, participation in risk factor behavior may severely augment disease (Kannel, et al., 1970; Page, 1972; Pfaffenberger, 1968).

Risk Factor: Anxiety

It has been suggested that anxiety is associated with the genesis of hypertension (Dohrenwend & Dohrenwend, 1973; Friedman, 1970; Jenkins, 1971). Jenkins (1970) concluded from a literature review that anxiety is directly reflected in blood pressure elevation. Inferences about the relationship of anxiety to the development of hypertension include psychosocial, biological, and environmental components which affect the character of interaction of the organism with its environment (Weiss, 1975). Most anxiety theories seem to include these three elements, though perspective varies according to which factor the theorist believes to be primary.

Several researchers who favor the psychosocial component emphasize the psychosomatic determinant and the propensity for certain personality types to contract disease (Friedman, 1970; Selye, 1976). As well, various aspects of the personality may

have varying effects on predisposing, initiating, and/or sustaining hypertension (Weiner, 1977). Biological mechanisms, i.e., genetic configuration, are the focus for other researchers, in light of which personality and blood pressure are simply covariants (Weiner, 1977).

Thirdly, defense and adaptive mechanisms have been incriminated in the etiology of hypertension (Henry, 1976; Henry & Cassel, 1969; Lazarus, 1975). It has been postulated that the environmental demand for adaptation causes continual stimulation of humoral, endocrine, and nerual mechanisms responsible for blood pressure elevation, an automated response to stress (Henry & Cassel, 1969). Chronic aggravation of such physiological response by psychological and life-stress occurances could lead to sustained hypertension (Harris & Singer, 1968; Henry & Cassel, 1969).

In summary, symbolic and physical interaction with the environment seems to be directly reflected in systolic blo od pressure elevation (Henry & Cassel, 1969). Sensitivity and tolerance of environmental demands seem to be based on biological mechanisms and learned methods of coping (Weiss, 1975). Ineffective coping and the physiological adaptation which must alternatively occur may subsequently produce pathology (Lazarus, 1969). Prevention of illness, then, involves avoiding stress, building re-

sistance to it, and being able to cope appropriately (Lazarus, 1975).

Risk Factor: Overweight

A second hypertensive risk factor behavior which is of endemic occurance is obesity (Keys, 1975). America is a society of overnutrition (Page, 1976) where fat babies are considered to be healthy babies, and social custom dictates the ingestion of large amounts of rich foods now so readily available to out technologically advanced populace (Kannel, et al., 1970; St.Pierre & Warren, 1975).

As with most risk factors, the degree of risk is related to excesses in risk behavior. An individual who is more than 10% overweight is believed to be at risk of high blood pressure (Grande, 1973; Keys, 1973). The greater the weight gain, and the more rapidly it is acquired, the greater the risk of hypertension (Paul, 1977; Stamler, Stamler & Pullman, 1967). Furthermore, when the obese person loses weight, their blood pressure usually falls respectively (Keys, 1973).

In multiple Americo-European studies of persons with and without coronary disease, Alexander (1973) demonstrated a significant physiological decrease in vascular resistance and mean arterial pressure with weight reduction in both groups. Cross-

cultural studies of populations possessing similar societal risk factors also correlated a drop in blood pressure with simple weight reduction (Alexander, 1973).

Though hypercholesterolemia and carbohydrate metabolism disorders predispose to hypertension, they fail to account for general association between overweight and hypertension (Glueck, Fallot, & Tsang, 1973; Stamler et al., 1974). Obesity is consistently shown in the literature to be a risk factor in the etiology and severity of hypertension; and, weight loss is shown to be important in controlling high blood pressure.

Risk Factor: Dietary Salt

Dietary influence on high blood pressure is gaining further recognition as the role of salt is more strongly implicated in the pathogenesis of hypertension. After more than 20 years of studying the inter-relationship of salt and hypertension, Dahl(1972) concluded that reducing salt intake in those persons with high blood pressure will result in a significant decrease in blood pressure levels. Relative to weight loss, he found that a decrease in weight without concomitant reduction in salt intake failed to result in lower blood pressure.

Cross-cultural studies by Dahl (1972) and others (Prior,

Evans, Harvey, Davidson, & Lindsey, 1968) showed that from population to population blood pressure varied with salt intake. In addition, when populations with low dietary sodium increased their intake of salt, blood pressure also rose. In a study of 1300 adults, Dahl (1972) found that those with minimal salt intake had lower blood pressures and those with high salt intake had higher incidence of hypertension than random distribution would serve to explain.

Family history appears to have an important effect on individual tolerance to sodium intake, as those genetically predisposed to high blood pressure are more severely affected by increases in salt intake (Swaye, Gifford, & Berritoni, 1972). Changes in salt intake affect changes in blood pressure such that regulation of dietary sodium appears at this point to hold the promise for blood pressure control (Porter, 1977).

Risk Factor: Cigarette Smoking

Cigarette smoking has direct and immediate effects on blood pressure and is an undisputed long term risk factor for cardiovascular disease (Aronow, 1973; Kannel, 1976; Stamler et al., 1974) and atherosclerosis (Paul, 1977). The role of cigarette smoking in the etiology of hypertension is less well

defined. Nicotine causes peripheral vasospasm, an effect which will increase peripheral resistance and raise blood pressure for as long as the stimulus is present (Wright, 1975).

In one study, normotensive smokers were found to experience a pressure rise of up to 20 mmHg, which was sustained for approximately 15 minutes after finishing the cigarette (Roth & Shick, 1960). Hypertensive subjects in the same study responded with more acute (30-45 mmHg) elevations. In the Roth and Shick (1960) study, nicotine-induced peripheral vasoconstriction persisted for 30-60 minutes, and heavy smokers did not display a tolerance to the systemic effects of nicotine. The nicotinic residual, however, will almost completely remit after 1 year of smoking cessation (Leve & McGill, 1973; Wright, 1975).

Risk Factor Behavior and Behavior Change

Excess body weight, high salt intake, and anxiety appear as behavioral risk factors in the genesis of hypertension. Smoking is usually regarded as a transient affector . Each of these risk factors is a learned behavior, acquired and patterned over time. Consistent then with the behavioral model, if the behavior can be learned, it can be unlearned. More appropriately, other behaviors

can be learned which will be incompatible with the original behavior and eventually lead to its extinction (Skinner, 1953).

Behavior is a function of belief and attitude (Young, 1967). As behavior or cognitions change, the other is consequently altered. In striving for internal harmony, an individual generally behaves in ways that are internally consistent with what he/she knows and believes (Festinger, 1957). New information is assessed for its value and appropriateness. Acceptance of new information necessitates alteration in belief or behavior in order that homeostasis be preserved. If inconsistencies develop with the acquisition of new information, there is imbalance, discomfort, and a precipitating anxiety termed "cognitive dissonance" by Festinger (1957). Resolution of this dissonance can be accomplished by rejecting the new information, modifying it to be compatible with the present state, or by altering behavior to coincide with the new state of knowledge.

The "at risk" borderline hypertensive who acquires knowledge about his/her status is placed in this dilemma. Health education is action-oriented, and in order to comply with one's own belief about the value of health, one must change behavior, or suffer in discord (Baric, 1969). "The dissonant state itself is assumed to be motivating, in the sense that a person will attempt to reduce

dissonance so as to achieve consonnance in his/her cognition" (Feather, 1963, p. 157). Pre-set cognitions, social, environmental, and personality factors influence behavior as well, and affect the interpretation and assimilation of new information. The message seems to be, however, that risk behavior is self-destructive and to alter risk behavior may be life-saving (Evans, 1974).

A Health Belief Model, based on the sociopsychological theory of Lewin (1935), has been formulated to predict preventive health behavior (Rosenstock, 1975). The model identifies a configuration of intrinsic and extrinsic forces influencing each individual in the formulation of his/her behavior set. In the model, individual perceptions of susceptibility and severity of disease are modified by personal, sociopsychological, and structural variables such as age, ethnicity, social class, and knowledge about disease (Rosenstock, 1975). These modifying factors, plus external cues and referents color the perception of threat of a particular disease. It is this perceived threat of disease which has shown to be central in determining health-related behavior and in predicting response to preventive or treatment incentives.

Superimposed on the interpretive aspects of behavior prediction, important association has been found between locus of

control and activities undertaken to prevent or recover from illness (Weiss, 1975). Internal and external controls shade perceptions of all factors involved in decision-making, and influence the prediction of behavior change in the presence of risk.

Again, behavior, belief, and attitude (adapted knowledge) are mutually effecting (Hartley, 1961). A change in behavior occurs with the belief that one's own actions affect one's susceptibility to a disease. Educational efforts are generally aimed at primary prevention and generic control of risk factor behavior, i.e., at altering one's susceptibility. Perceived threat of disease and health locus of control are important considerations in evaluating change, or failure to change, health-related behaviors.

Summary

Risk factor behaviors are endemic to American culture and with the accompanying hypertension, contribute significantly to the incidence and mortality of cardiovascular disease. The thrust of most health education programs is to decrease the prevalence of risk behavior, thereby decreasing the incidence of hypertension (Steuart, 1975). Conclusive evidence supporting that progression is limited, yet movement toward change of what

is known and suspected is necessary because the problem will not exhaust itself (Ball & Turner, 1975).

Primary prevention has as its goal the reduction of death and disease through modification of destructive behavior. Health education is based on the belief that new knowledge will result in new behavior. The National Heart and Lung Institute task force on hypertension emphasized that altering risk factor behavior is of prime importance in reducing the threat of hypertension. Dietary control, minimization of stress, and increased knowledge and awareness were suggested objectives for health education programs (Weiss, 1975).

Overweight, dietary salt intake, anxiety, and smoking have been identified as possible risk factors in the presence and development of high blood pressure. It is these four factors which were assessed, according to the guidelines of the present study, in determining degree of risk with regard to hypertension. Elevated blood pressures have been associated with the presence of risk factor behavior. Further exploration of that relationship was the purpose of the present study.

Hypotheses

The hypotheses were as follows:

1) Subsequent to information exposure concerning risk factors related to hypertension, persons with borderline blood pressure will alter behavior to reduce their risk of disease.

2) There will be a significant relationship between a change in risk factor behavior in persons with borderline hypertension and change in blood pressure.

3) Greater perception of threat, high levels of knowledge, and internality of control will significantly correlate with risk factor behavior modification.

CHAPTER III

METHODOLOGY

Selection of Sample

A sample population of 1200 employees of three banks and a laundry in Kalamazoo, Michigan was randomly selected by volunteering to participate in a community hypertensive screening program. All employees were initially screened for hypertension by this investigator between January and June, 1976, under the auspices of CATCH (Community Action to Control Hypertension), a federally funded community health organization. Worksite screening consisted of a private interveiw with each client at a time allotted by the employer. Initial classification as borderline hypertensive was made on the basis of 6 blood pressure readings taken on two separate occasions, 1 to 4 weeks apart. On each occasion, 2 out of 3 readings of 140-160/90-95 mmHg sufficed for classification of the client in the borderline category. Forty adults were identified as borderline hypertensive. No client claimed illness or disability from disease or its sequellae.

The Sample

Approximately 1 year after the CATCH screening, 30 borderline hypertensive adults were available for study. Five subjects from the original sample of 40 had terminated employment; 3 persons were on vacation, and 2 refused to participate. No subjects had died within the year interval and no subject had instituted an antihypertensive medication regime. One individual had begun meditating twice daily in the 2 weeks before follow-up.

There was one Black male and one Black female in the otherwise Caucasian sample of 19 men and 11 women. Mean age for the men of the sample was 35 years (Range 19-53); for females, 47.5 (Range 20-61). Fifty percent of the sample declared a family history positive for hypertension.

Design and Procedures of the Study

The structure of this correlational investigation was a one-group pretest/posttest design. This format facilitated the measurement and evaluation of behavior and blood pressure change over time. As an evaluative tool for the educational program, per se, the utility of the one-group pretest/posttest design is limited by the time lapse between testing and the influence of intervening

variables on client learning. Subject maturation and history threaten the internal validity of the selected design, though the time interval of the present study probably strengthened internal validity by diminishing the reactivity of the screening questionnaire (Campbell & Stanley, 1963). Also, blood pressure regression was countered by multiple blood pressure measurements, i.e., 3 readings on each of 2 separate occasions. A random order administration of questionnaires was intended to minimize systematic error of the interactional effect between questions about risk factor behavior and the behavior itself.

Initial Screening Interview

At the initial screening each client volunteered to have his/her blood pressure measured by the investigator and agreed to answer questions related to high blood pressure. A screening questionnaire (WHPS) was orally administered and 3 blood pressure measurements were taken during the private, structured interview. Blood pressure was measured on the client's left upper arm while in the sitting position (see Appendix A for specific technique). Readings were taken 5-7 minutes apart and immediately recorded.

After the questionnaire was completed, the client was

informed of his/her blood pressure readings and their relative significance. Normotensive persons were advised to have their blood pressure checked yearly. Clients exhibiting high blood pressure readings were referred to the physician of their choice. Clients with 2 out of 3 readings in the borderline category were scheduled for a re-screening appointment 1-4 weeks later.

At the conclusion of each interview, the client was presented with printed literature from the American Heart Association concerning basic facts about high blood pressure, its cause, treatment, and sequellae. Clients were specifically made aware of five major factors contributing to the development of hypertension: family history, obesity, high salt intake, anxiety, and smoking. Each client was counseled as to his/her personal risk of high blood pressure, and as to adjustments in life-style recommended to avert further ascension in blood pressure. Explicit advice was given to reduce excess body weight, limit salt intake, quit smoking, and refine coping skills, or develop alternative mechanisms for coping with stress.

Re-screening Appointment

At the scheduled worksite re-screening appointment, the clients who exhibited initial borderline readings were again in-

terviewed by the researcher. Three blood pressure readings were taken 5-7 minutes apart, demographic data were elicited, and selected questions from the WHPS were asked. Subjects who exhibited normotensive readings at this time were advised to have their blood pressure rechecked every 6 months. Clients whose blood pressure remained in the borderline range were again instructed in the importance of blood pressure control and control of those behavioral factors which place one at risk of hypertension. It is this group of clients which was selected as the study sample.

An average of the 3 blood pressure readings from the initial screening and the 3 blood pressure readings from the re-screening appointment yielded a mean systolic and mean diastolic pressure reading for each subject. For the purposes of the present study, these pressure means were identified as the initial systolic and initial diastolic readings.

Follow-up

One year later, sample subjects were recontacted and reassessed for blood pressure and risk factor behavior changes. Clients were again privately interviewed at the worksite by this researcher or by another nurse, familiar with the study questionnaires and with research procedings. The WHPS,

a Cognition Assay (CA), and Wallston's Health Locus of Control (HLC, 1975) were administered in a random order. The WHPS was orally administered, as in the initial interview. Verbal and writte instructions were provided for the client to complete the CA and HLC indipendently. No time limit was imposed, but all clients completed written sections within 10 minutes. With subjects in the sitting position, 3 blood pressure readings were taken on the left upper arm. The average systolic and average diastolic pressures were identified as the subject's follow-up blood pressure readings.

Initial and follow-up systolic and diastolic readings were then compared. Data indicating age, sex, race, family history, height, weight, smoking incidence, amount of salt intake, and average anxiety level were educed from initial and follow-up WHPS. CA and HLC scores were tabulated. Data were analyzed and relationships among data were interpreted by means of linear regression analysis and significance testing.

Data Collection Instruments

Instruments selected for use in the present study were: Worker Health Program Screening Questionnaire (WHPS; Erfurt & Foote, 1975), Health Locus of Control (HLC; Wallston, Wallston, Kaplan, & Maides, 1975), and a Cognition

Assay (CA) developed by the researcher for the purposes of the present study (see Appendix A for samples of each instrument). Blood pressure was measured with a new Nelkin mercury sphygmomanometer which had been tested to be leak-free. All blood pressures were measured according to a specific technique (see Appendix A for detailed description). First and fifth phase Korotkoff sounds were used to identify systolic and diastolic pressure readings respectively.

All initial assessments and 70% of the follow-up readings were completed by this investigator. This investigator and the assistant had been tested for measurement reliability and precision by an independent observer. With the use of a teaching stethescope (one with dual ear sets and a simgle bell piece), 2 blood pressure readings were measured on each of 3 volunteers. No greater than 2 mm discrepancy was allowed to "pass" the screener. The variability introduced to study results by the investigators is assumed to be randomly distributed; no systematic differences were noted in the responses obtained by each interviewer in collection of data for the present study.

Worker Health Program Screening Questionnaire

The only questionnaire utilized in collection of both initial and follow-up data was a selection questionnaire developed by

the Worker Health Program Institute of Labor and Industrial Relations of the University of Michigan and Wayne State University. The WHPS questionnaire was consistent with guidelines of the Michigan Association for Regional Medical Programs. The self-report method of data collection was tested for reliability 1-4 weeks after the initial interview. Elicited at the second interview were demographic data and the answers to 8 of the 20 WHPS questions. Agreement between respective responses was 100%.

For follow-up data collection an addendum was added to the WHPS battery. Questions on the addendum asked each client to estimate the difference between his/her present behavior and behavior exhibited a year earlier. Agreement between estimated behavior change and change computed by the researcher by comparing initial and follow-up WHPS responses was 98%. Reliability of response and concurrent validity of the questionnaire was accepted.

Health Locus of Control

The HLC is a standardized, area-specific questionnaire with scaled responses, designed to measure degree of external and/or internal control over health (Wallston et al., 1975). The scale consists of 11 cross-validated items regarding locus

of control related to health. Using a Likert-type format, responses are scored in the external direction from 1 to 6, and reverse scored for internally worded items. Possible scores range from 11 to 66, higher scores indicating greater externality of control.

HLC shows an alpha-reliability of .72. Concurrent validity of the HLC poses a .33 correlation (significant at .01) with the traditional Internal-External scale of Rotter, purposefully, states Wallston, to enhance its discriminant validity, i.e., specificity for health behaviors. The HLC has previously been used to show relationships between locus of control beliefs and health care related behaviors (Wallston, et al., 1975). Its use in the present study was to aid in evaluating risk factor behavior change.

Cognition Assay

A Cognition Assay (CA) was designed by the investigator and consultants to elicit knowledge and perception of threat concerning hypertensive disease. Questions on knowledge and threat were formulated, then presented to a panel of health professionals whose task was to identify the focus of each question, i.e., would a response reveal knowledge, belief, etc. Questions accepted for inclusion in the CA were those for which within-panel

variability was limited to 1%. The final assay was believed to exhibit clarity, discrimination, and construct validity.

Questions 1 through 6 of the CA tested objective knowledge about hypertension and its sequellae and affectors. Content included information presented at the CATCH screening and relevant information not presented at the time of screening. Questions 7 through 10 are knowledge questions written in personalized form. They were designed to reveal the accepted, or adapted knowledge of the client. Questions 11 through 14 were adapted from the Semantic Differential for Health (Jenkins, 1966) and concern severity and susceptibility to disease, i.e., threat of hypertension. CA responses were marked on a graphic rating scale, polar responses being true-false, agree-disagree, or 0%-100%. Scoring was achieved with the use of a plastic ruler which divided the scale into 5 equal categories. Responses were scored from 5 to 1 (left to right) for questions 1 to 10, and from 1 to 5 (left to right) for questions 11 to 14. CA score was computed by adding total score for knowledge to threat score total. Higher scores indicated greater knowledge and perception of greater threat.

Analysis of Data

Several investigators at the Johns Hopkins Center for Health Care Services, Research, and Development have designed a study to assess risk factor behavior and its affect on blood pressure (Green, Levine, & Deeds, 1975). Diet, stress, smoking, blood pressure, and weight were measured and assigned a value which was computed to determine a numerical value for degree of risk for each patient. An expert panel quantified a value for each behavior change according to presumed association with medical outcome. The assigned value was multiplied by 1 if the patient complied with theraputic instructions to alter behavior; if not, it was multiplied by zero. Scores were tallied and each patient received a pre and post score from 0 to 10.

While this rating scale is helpful in that it is the first attempt to quantify risk factor behavior and behavior change, it is limited in that it does not discriminate between elimination of risk behavior and a behavior change in the appropriate direction, but which still leaves the individual at risk. Thus a more sophisticated matrix was designed for the present study.

A risk factor rating scale was designed with the following purposes in mind: 1) to encapsulate the present state of knowledge concerning risk factor behavior and high blood pressure.

2) to provide a reasonable means of assessing degree of risk and risk alteration.

3) to quantify behavior into meaningful degrees of risk. Categorization and assignment of values in the proposed risk factor rating scale was derived from the literature review and expert opinion, but is at best, conjecture (McDonald, 1977).

TABLE 1

Risk Factor Rating Scale

Risk Factor Behavior	Risk Factor Value				
	0	1	2	3	4
Weight	normal	+10%	+15%	+20%	+25%
Anxiety	little	-	average	-	great
Added Salt	none/ substitute	cook only	add occasionally	-	use lots
Smoking (pk/day)	none	1	1½-2	2½-3	+3

Information was quantified in the following manner:

1) From the WHPS, each identified behavior was assigned a numerical value indicating the degree of risk as determined by the proposed risk factor rating scale. Risk factor scores were totaled for each subject. Systolic and diastolic blood pressure data were educed from the initial WHPS and computed to determine the mean and standard deviation for initial blood pressure readings.

2) From the WHPS administered at follow-up, risk factor behavior values were determined and tallied. Follow-up systolic and diastolic pressure means and standard deviations were also computed.

3) Mean change between initial and follow-up readings and scores were computed for blood pressure and risk behavior.

4) The significance of risk factor behavior change was determined by the Wilcoxon Sign Rank Test, a statistical technique which accounts for magnitude as well as direction of change (Haber & Runyon, 1963).

5) Significance of risk factor behavior reduction was determined by comparing initial and follow-up mean risk factor scores according to Student's *t* Test.

6) Linear regression analysis, with the aid of Pearson's correlation coefficient, was utilized to determine the strength of relationship between risk factor change and mean blood pressure change.

7) HLC and CA scores were tabulated. Threat perception, knowledge, and locus of control scores were individually correlated with risk behavior change scores, utilizing Pearson's coefficient of correlation.

8) Confidence levels greater than 95% were accepted as indicating statistical significance in all statistical testing.

CHAPTER IV

RESULTS

Blood pressure was measured and information was obtained from each of the 30 subjects in the manner described. Risk factor behavior was initially exhibited by all subjects in the borderline hypertensive sample. A composite description of the subjects at the initial screening was: Subjects were 12% overweight, experienced above average levels of anxiety, and regularly added salt to their table food. Half the sample smoked cigarettes - an average of 23 cigarettes a day.

A year subsequent, the sample composite was: 14 subjects evidenced normal blood pressure readings, 2 were high, and the remaining were borderline. As an aggregate, the sample smoked as much, was as anxious, and added as much salt as they did a year prior. Mean weight for the sample was reduced to 11% excess.

Risk Factor Behavior Change

Initial and follow-up risk factor score totals and the distribution of change among the identified risk behaviors are presented in Table 2. Within a possible range of 1 to 16, initial risk factor

scores ranged from 4 to 12, Mean 7.46, SD 2.06. Scores tallied a year subsequent ranged from 1 to 12, Mean 7.06, SD 2.56. Nineteen subjects (63%) modified risk behavior to produce a change in total risk factor score. Change scores ranged from -6 to 4, Mean -0.4, SD 2.13. Eleven of the 19 scores showed a decrease of 1 to 4 points. Increases of 1 to 6 points affected risk factor scores of 8 subjects. Eleven subjects (36%) demonstrated no change in total risk factor score.

TABLE 2

Risk Factor Score Change

Risk Factor Behavior	Risk Factor Scores		
	Initial	Follow-up	Change
Total Score			
Mean	7.46	7.06	-0.40*
Std. Dev.	2.06	2.56	2.15
Weight			
Mean	1.63	1.42	-0.17
Std, Dev.	1.46	1.39	0.77
Anxiety			
Mean	2.36	2.40	+0.04
Std. Dev.	0.57	0.20	0.75
Added Salt			
Mean	2.30	2.00	-0.30
Std. Dev.	1.08	1.12	0.90
Smoking			
Mean	0.70	0.76	+0.06
Std. Dev.	0.98	0.98	0.36

* $p < .05$, Wilcoxon Sign Rank Test

Tabulation of follow-up risk factor scores revealed that weight and anxiety change occurred with greater frequency. Eighty percent of the sample experienced a weight change, though only 30% registered a change in risk factor-weight score. An average weight loss of 17 pounds reduced the risk factor score of 6 (20%) subjects; a mean increase of 8.6 pounds affected 3 risk factor scores. Mean change for risk factor-weight score was -.17, SD 7.7. The difference between initial and follow-up means was not statistically significant.

Alteration of anxiety state affected 40% of risk factor scores. Six subjects (20%) claimed a decrease in their anxiety over a year's period of time, and an equal number claimed an increase. Mean change in risk factor-anxiety score (-.04) was not statistically significant. Though 18 subjects (60%) did not alter their risk factor-anxiety score, 6 of those subjects stated their "high" anxiety levels to be greater than the "high" reported a year earlier. Three subjects described anxiety levels as "average" at each interview, but stated the follow-up level to be comparatively less than the year prior.

Smoking and dietary salt intake proved the more recalcitrant risk factor behaviors. Salt intake was reduced below the "add regularly" level by only 5 subjects (16%). one subject switched

to a salt substitute following initial risk factor education by the researcher. The remaining 25 subjects maintained dietary sodium intake at acquired levels.

Non-smokers comprised 50% of the study sample. Those who smoked cigarettes consumed 10 to 50 cigarettes a day, Mean 23/day. Four subjects (13%) altered their smoking habits enough to affect risk factor value; 1 subject reduced the number of cigarettes smoked, 2 increased, and 1 started smoking. No one quit smoking.

Total risk factor change for the study group was significant at a .05 level, using the Wilcoxon Sign Rank Test; change occurred in the negative direction. The difference between means of initial and follow-up risk factor scores was not statistically significant, when compared utilizing Student's *t* Test. Therefore, the first hypothesis is rejected. For those subjects who decreased total risk factor score, however, that reduction was significant at a .05 level, according to Student's *t*.

Blood Pressure Change

Blood pressure changes varied from -24.6 mmHg to 11.3 mmHg systolic, and -17.3 mmHg to 10.6 mmHg diastolic.

Mean decrease in systolic pressure (-8.12 mmHg) was significant at a confidence level greater than 99%. Mean diastolic decrease (-3.18 mmHg) was significant at a confidence level greater than 95%.

TABLE 3

Systolic and Diastolic Pressure Changes

	Blood Pressure			
	Systolic		Diastolic	
Readings	Mean	SD	Mean	SD
Initial	143.45	8.99	88.22	5.67
Follow-up	135.33	10.43	85.04	8.83
Change	-8.12**	9.76	-3.18*	7.48

* $p < .05$; ** $p < .01$

Forty-seven percent (N=14) of subjects initially classified as borderline hypertensive evidenced normal readings at follow-up. Two subjects (6%) had blood pressure higher than 160/95 mmHg. Fourteen subjects (47%) maintained readings within borderline margins.

Risk Factor Behavior Change: Blood Pressure Change

Blood pressure change data were evaluated in relation to risk factor behavior change data (see Table 4). The correlational relationship between changes in blood pressure and total risk factor behavior score changes, when tested by Pearson's correlation coefficient were: $r=.27$ (systolic), and $r=.06$ (diastolic). The weak statistical relationship does not support the second hypothesis which must be rejected accordingly.

Linear regression analysis of those who decreased risk factor score revealed a weak relationship with blood pressure change, as did analysis of those who did not reduce risk factor score. However, it is noted that a risk factor score reduction accompanied a much greater reduction in blood pressure than had no risk factor decrease occurred.

TABLE 4

Risk Factor Score Change: Blood Pressure Change

Risk Factor Score Change	Mean Blood Pressure Change			
	Systolic	r	Diastolic	r
Decrease	-11.20*	-.16	-4.8	.103
No Decrease	-6.31*	.38	-2.05	-.115
Total	-8.12**	.27	-3.18*	.065

* $p < .05$; ** $p < .01$

When blood pressure change data were analyzed in relation to risk behavior changes and the assigned variables, several trends were noted (see Table 5).

TABLE 5

Blood Pressure Change of Assigned Groups

Assigned Groups	N(hx)[a]	Mean Blood Pressure Change	
		Systolic	Diastolic
White Males < 40yrs.			
Decrease RFB	4(1)	-10.2	-6.2
No decrease	8(4)	- 0.8	+1.7
Total	12	- 4.1	-0.9
White Males > 40yrs.			
Decrease RFB	3(2)	- 7.2	-2.8
No decrease	3(2)	- 6.3	-4.9
Total	6	- 6.75	-4.6
White Females < 40yrs.			
Decrease	1(1)	-20	-15.3
No decrease	1	- 8.6	- 0.3
Total	2	-14.3	- 7.8
White Females > 40yrs.			
Decrease RFB	2(1)	-12	-10.7
No decrease	6(2)	-13.75	- 6.6
Total	8	-13.3	- 7.62
Black Male < 40yrs.			
No decrease	1	- 4	+ 2
Black Female > 40yrs.			
Decrease RFB	1	-17	+ 6

a (hx) indicates number of subjects with family history positive for hypertension

The most dramatic trend noted in the relationship between risk factor score change and blood pressure change was a mean systolic pressure decrease in all assigned groups. A second trend was that mean pressure reduction was usually greater for groups that also reduced risk factor score. Thirdly, mean change in women's pressures showed a decrease of more than twice the amount of decrease in the mean pressures of men.

Genetic predisposition to hypertension seemed to exert important influence on male subjects. Diastolic pressure increased in 6 out of 9 men with a positive family history. Coincidently, of the 9 men who increased diastolic readings, 6 claimed a positive family history . No woman with a family history of hypertension increased diastolic pressure.

Cognition and Health Locus of Control

Knowledge and threat scores were collectively tabulated in Cognition Assay score determination. Mean CA score was 53.7, SD 6.3. Linear regression analysis of CA scores and risk factor behavior change scores revealed a weak relationship, utilizing Pearson's r. Correlation between behavior change and CA was -.07; for knowledge, r=.13; and r=.20 for threat.

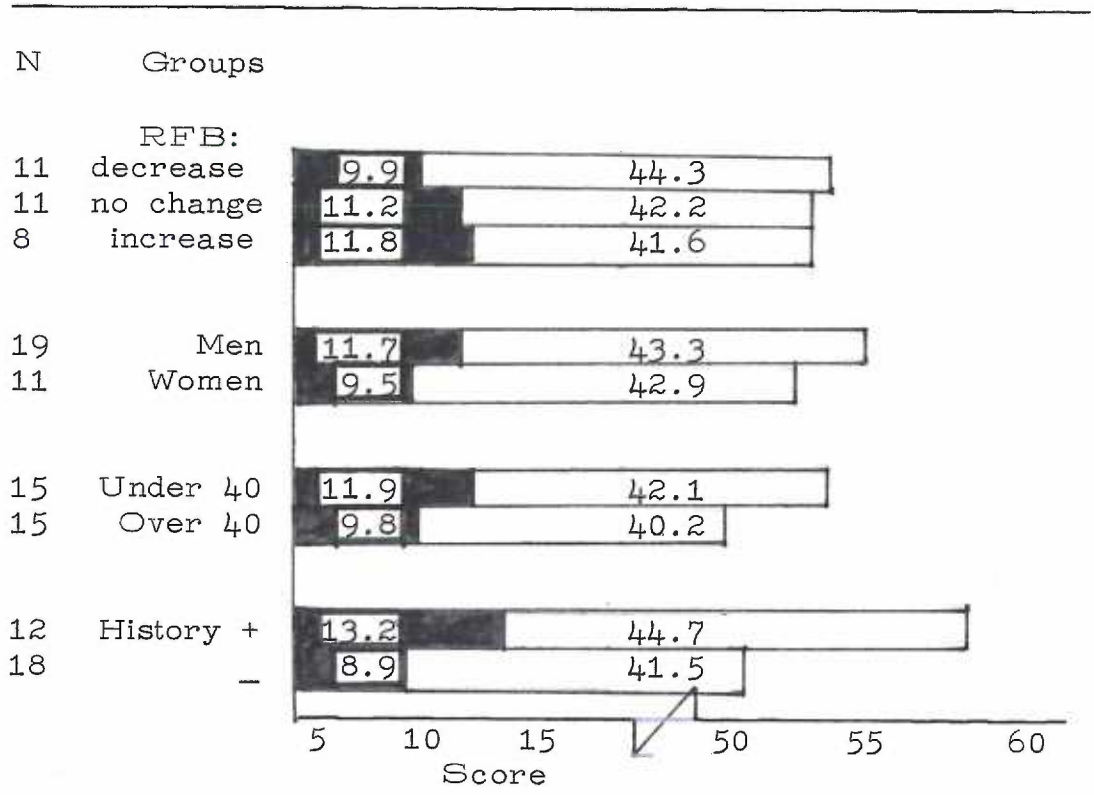
HLC scores (Mean 35.6, SD 7.3) also evidenced weak statistical correlation with risk factor behavior score change ($r=.23$). No correlations were statistically or clinically significant. Thus, the third hypothesis is summarily rejected.

The HLC did provide some descriptive information concerning sample subjects. Those who did not alter risk factor behavior were greater internalizers than those who either increased or decreased risk factor behavior. Mean HLC score for those who decreased risk factor score was 32.8; for those who showed no risk factor change, 29.2; and 38.4 for those whose risk factor score increased.

Several trends were noted in CA scores among assigned groups (see Figure 1). Males, subjects under 40 years old, and those with family history of disease perceived greater threat of hypertension than their respective counterparts. The difference between mean threat scores of those with and without family history was highly significant. Secondly, those who lowered risk factor scores perceived less threat of disease, and evidenced slightly higher levels of knowledge than those who did not decrease risk factor score. A high degree of knowledge, however, was prevalent among all sample subjects.

FIGURE 1

Knowledge and Threat Among Assigned Groups

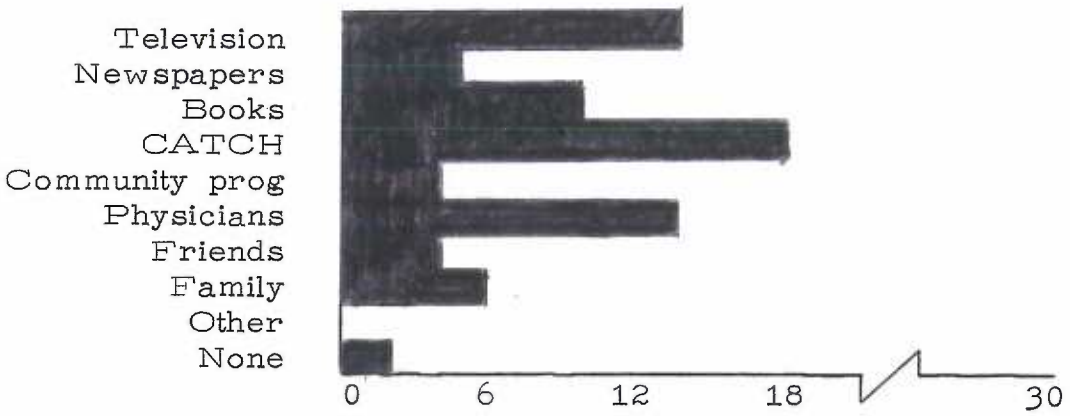


threat score; knowledge score

Subjects attributed their acquisition of knowledge concerning high blood pressure to the sources listed in Figure 2. The CATCH program (initial interview and accompanying instruction) was credited with having the greatest influence on knowledge states. Private physicians, television, and available literature contributed substantially, but secondarily.

FIGURE 2

Sources of Information



CHAPTER V

DISCUSSION

Alterations in Risk Factor Behavior

The first hypothesis of the present study is not accepted because behavior did not change in accordance with educational directives. The ultimate test of educational success is behavior change, and the fact that no real differences occurred between initial and follow-up risk factor scores addresses the question of educational effectiveness. Moreover, total risk factor score change was significant (Wilcoxon) partially because of behavior change which occurred contrary to educational intent.

Knowledge and threat levels are also indicators of educational effectiveness, and are believed to be critical in behavior motivation as well (Lazarus, 1969; Rosenstock, 1975). Knowledge was high among all participants. Those experiencing the greatest threat of disease, however, were not necessarily the ones experiencing the greatest diminution in risk factor score. Knowledge and threat measurements may reflect some cognitive aspects of sample subjects, but alone did not accurately predict risk behavior modification in the present study. Although the third hypothesis was rejected because CA and HLC scores

did not correlate as predicted, they may still aid in interpretation of behavior change.

There appear to be three possible explanations why risk factor behavior was not significantly decreased in the present study. Assuming the necessary consonnance on knowledge, belief, and behavior in an effectively functioning hyman being, lack of knowledge may be a reason for failure to modify behavior. Mean knowledge score was fairly high. If subjects were not knowledgeable about hypertension and risk factor behavior, then they must have achieved high CA scores by some other means. One such possibility is that even with random administration of questionnaires, the subjects found "clues" to the "right" answers from preceding questions. That mean scores were so high seems to preclude that possibility. A second possibility stems from the fact that only pertinent information was included on the CA. No "trick questions" or irrelevant data were included to "check" the respondent. A good guesser may have presumed all CA questions were about hypertension and answered them all positively. Again, the prevalence of high scores seems to preclude that possibility as well.

If, however, CA scores were a true reflection of client knowledge, as is asserted by the researcher, then high scores in

absence of behavior modification implies that subjects are suffering high degrees of dissonance. The interpretive aspect of the cognitive dissonance theory mitigates this seeming incongruence (Feather, 1963). Evaluation of the information received is what determines the behavioral effect. Pre-existing cognitions and firmly held beliefs affect the interpretation of acquired information. Dissonance is more likely to occur when information is in conflict with pre-established belief. If information is obtained before committment to a belief, no dissonance results, though new knowledge may be contrary to existing behavior.

Thus, acquired knowledge may cement with a belief and then associate with a desired behavior; or, knowledge may simply be suspended in a belief system which has not yet jelled. In this case, knowledge may not be congruent with behavior, but no dissonance would result.

Belief is another important element in ascertaining why more subjects did not decrease risk behavior. The CA revealed that those persons aware of their genetic predisposition to hypertension were significantly (4.3, $p < .01$) more threatened by the possibility of disease development. Yet this acknowledgement of susceptibility made them no more successful at behavioral

risk reduction than those who lacked the genetic component. Fear plays a significant role in the perception of threat, and is perhaps the most potent behavior motivator (Kirscht, Becker, & Eveland, 1976). Fear is most effective when immediate results are required, however; as a long-term affector its impact is mollified by adaptive, rationalization processes engaged to defend against the threat (Mausner, 1971). Those with family history of disease probably have well-developed rationalizations enabling them to tolerate that life-long threat.

Greater threat of disease, perceived by those deemed genetically susceptible was apparently not shared by those susceptible because of behavioral attributes. Only those uncontrollable factors (inherited proclivity) elicited greater threat. Within the paradigm of cognitive dissonance, a low perception of threat indicates that the belief of behavioral risk is not firmly held in the cognitive matrix of sample subjects. Thus, high levels of knowledge can be sustained in absence of belief commitment, i.e., perception of threat, without stimulating dissonance, and without directly affecting behavior.

Contrary to the study prediction, threat perception among those who reduced risk behavior was lower (-1.6) than among those who did not modify behavior. A plausible interpretation

is that having modified risk behavior, subjects felt less threatened by disease, and thus were able to accept more information about disease pathology. Alternatively, knowledge itself may have been instrumental in risk factor reduction. Validation of that conjecture would have been possible had a CA pretest been administered.

The degree of acquired knowledge, according to Feather (1963), is not a function of personality type. Neither internalizers nor externalizers scored consistently high or low on the CA (r=.20). Although locus of control has in other studies (Weiss, 1975) correlated with propensity to alter health behavior, the association between HLC and behavior change in the present study is dubious.

A third reason risk behavior may not have significantly reduced is that subjects may have felt they were "unable to quit," or that "it wouldn't do any good". Subjects who maintained initial risk factor scores were the greater internalizers. Subjects who altered risk factor scores were greater externalizers - some attempting to manipulate those factors which placed them at risk, while others succumbing to the extrinsic pressures to indulge in acquired, though detrimental activity. Those professing control over their health (internalizers) and failing to

alter risk behavior may be stating the "mind over matter" philosophy. That is, "thinking" one has control may be wherein the control lies and not in the influenced activity.

In the present study, behavior modification occurred least frequently among the appetitive behaviors - adding salt and cigarette smoking. The difficulty of altering long-established habits may have deterred some subjects from reducing risk behavior. Appetitive behaviors develop in response to a complex system of psychological, social, and physiological needs (Bernstein, 1969).

Educational instruction offered information and reasons to eliminate behavior, but suggested no alternative for need satisfaction. Selye (1976) clearly states that an activity "deviant" from the patterned response needs to be built into the subject's response repertoire, such that the cue to detrimental behavior will elicit an equally satisfying, but benign response. Identification of cues initiating behavior, the need it fulfills, and options for meeting that need are necessary to modify appetitive behavior (Mausner, 1971).

Each patterned behavior may be initiated and maintained by a myriad of cues and reinforcers specific to that behavior. Thus impetus to alter behavior may be as diverse. For

example, the cause of weight reduction in a majority of sample subjects is not clearly identified in the results of the present study. Extraneous social influences, intrinsic psychological motivators, appropriate reinforcers, and an acknowledged strength of weight:blood pressure association probably synergistically affected weight change - in both directions.

Smoking, another social behavior and undeniably recalcitrant, may have actually reduced after the teaching sessions. Recidivism after any method of smoking cessation, however, is about 80% within a year (Hunt & Belspac, 1974), and within the structure of the present study would have accounted for smoking rate persistence.

Degree of perceived anxiety changed in 40% of persons interviewed. Change in situational influence, psychological adaptation, or refinement of coping skills may account for altering anxiety states. Situational anxiety is easily and accurately assessed by those prone to anxiety and having prior experience of the anxiety state (Speilberger, 1966). Thus a change in anxiety would have been rightly reported and reflected in physiological consequence, i.e., WHPS and blood pressure readings.

Secondly, mastery of stress-management skills provides

one with control over one's reaction to anxiety-producing situations (Farquhar, 1977). Environmental stressors remain, but as those stressors are perceived, the selected response is relaxed and effective. Effective coping would have reduced the amount of anxiety perceived and experienced by sample subjects; again, WHPS and blood pressure compatibility.

The process of adaptation is less-well delineated. The "cognitive consequences of an anxiety experience...take on a pattern and organization that will affect the nature of subsequent experience at the same time that it will itself be changed by it" (Speilberger, 1966, p. 78). Classic example of the adaptive process occurring among sample subjects is the three reports of a follow-up "average" less than the initial "average". The adaptive phenomenon is an equalizer (Selye, 1976). In the process of adaptation, stress is distributed throughout various organs and systems in the body (Selye, 1976). With stress more widely distributed, perception of stress is altered, though its effect on the body remains.

The weak association ($r=.01$) between anxiety change scores and blood pressure may partially be attributed to adaptation. Subjects perceived less stress, so reported lower risk factor scores. Physiologically the original stress load was

sustained, and reflected in borderline blood pressure.

The higher than "high" anxiety reported by 6 subjects is compatible with this explanation as well. Perhaps perception of anxiety is more accurately assessed in its "present" state. Once the anxiety has been resolved, the fact that one survived, or adapted to, the anxiety may be enough to diminish its impact. Upon recurrence of a stressful situation, a doubtful outcome may create the illusion of ultimate stress, thus a "higher than high" reported anxiety.

In addition, Speilberger (1966) explains that autonomic measures of anxiety reveal a physiological adaptation to specific stressors over time. Adaptation does not occur when one is subjected to constantly varying types and degrees of stress; but the autonomic stress response will be the same. Again, levels in excess of a previously reported "high" could be perceived.

Blood Pressure Change

Comparison of present study results with those of the Wilber and Barrow (1972) study clearly indicates that some intervening factor(s) were responsible for the dramatic differences between respective blood pressures. In the Wilber study 69% of the subjects progressed to high blood pressure after one

year, and 20% returned to normal. Only 2% in the present study had become hypertensive, and 46% reverted to normal.

In light of study similarities (sample size, composition, and time frame) and the marked difference in outcome, the variability of blood pressure or the exposure to information may claim effect, though it is impossible to measure the real educational effect. Although a knowledge pretest was not administered, the credit given CATCH for instruction, the high level of acquired knowledge, and the fact that education was the most universally occurring influence, support the strength of the educational effect. Considering the variability of blood pressure, however, a casual blood pressure reading, as in the Wulber study, may have allowed some frankly hypertensive subjects to be classified as borderline. The following year, regression toward mean pressure would have placed subjects in a more appropriate category. Either or both effects may have contributed to differences in study results.

Some trends noted in blood pressure response seem accountable to 2 major assigned variables: sex and family history. Epidemiologically, the incidence of high blood pressure among women is equal to that among men but mortality rate is much lower among the women, as they are less prone to cardiac disease (Kaplan, 1973). The incidence of borderline

hypertension among women in the present study was half that of men, though their mean age was greater. All women reduced their blood pressure.

In the present study, male subjects with a family history of hypertension responded in a manner well-demonstrating the importance of genetic effect. The propensity for diastolic pressure to increase in accordance with genetic influence was particularly obvious among men less than 40 years old. Systolic pressure was not as markedly affected nor did women show the same proclivity for effect of family history as men.

The mechanism of genetic predisposition to hypertension is unknown, and thus the importance of environmental factors upon persons with the genetic link is also in question. Pickering (1973) believes that the tendency for higher pressures is inherited via a multifactorial gene association, but that the rate of increase with age is environmentally dictated, and may be dependent upon risk factors over time (Paul, 1977).

Blood Pressure Change:Risk Factor Behavior Change

The statistical correlation between risk factor behavior change and blood pressure change did not support the study hypothesis. Even in those who decreased both risk behavior

and blood pressure, the correlation was a weak one. Possible reasons for the weak association are fourfold.

First, heredity may have intervened in the relationship between behavior and blood pressure. Genetic factors seem to determine the physiological propensity for blood pressure elevation, vascular reactivity, and/or structural change, and may affect the character of one's response to environmental stressors as well. Greater sensitivities on some persons and greater resistance to similar risks in others serve to modify the effect of each behavior on blood pressure. Some subjects experienced dramatic alterations in blood pressure in accordance with their efforts to modify risk factor behavior. Some who expended similar energy found their efforts almost futile. Thus, individual idiosyncracy must partially account for the weak correlation ($r=.27$) between risk factor behavior change and blood pressure change.

Secondly, certain behaviors and genetic factors may affect blood pressure in tandem. As with salt and weight, one may decrease without the other, and not afford the necessary reduction in pressure. Anxiety and heredity have also been suggested as being in such association (Weiner, 1977).

Strength of statistical association between risk behavior and blood pressure change could also be muted by the extreme

variability of blood pressure. Blood pressure may be temporally influenced after eating, smoking, exercise, or by a full bladder, physical discomfort, or reaction to the researcher (Maxwell, 1974). These influences were neither identified nor eliminated. Diurnal variance was not controlled for in blood pressure assessment, and situational stressors which may have activated a hyperactive vascular sympathetic system were not eliminated. Though 3 readings taken over a 20-minute period was a design to insure accuracy, 24-hour mean readings could vary considerably from study means, and spurious readings could have occurred which were contrary to behavioral indicators (Mueller, 1930).

A fourth reason for the lack of correlational significance in the present study is that the actual association between risk factor behavior and blood pressure may be a weak one. Despite that possibility and the suggested interference of other factors, blood pressure and risk factor behavior change appear to be related with respect to direction, if not degree. Though statistical correlations did not support the relationship, gross inspection of blood pressure and risk factor data, tabulated according to the guidelines of the present study, reveals obvious trends which deserve further, more sophisticated exploration.

Subjects who were found to have normal blood pressures at follow-up also revealed lower risk factor scores in all risk factor behavior categories. Subjects who remained borderline fluxuated in risk behavior but essentially maintained initial risk factor scores. An increase in all risk behaviors except salt intake describes the scores of those whose blood pressure had also increased.

That education intervened, and that factors believed to influence blood pressure were significantly modified in those who evidenced greatest blood pressure reduction, indeed, suggests a relationship between health education and disease outcome, and between behavior and blood pressure.

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Hypertension occurs as the most widespread and lethal public health problem in America today. Deceptively asymptomatic, hypertension can progress undetected in any individual until target organs, such as the heart, kidney, or brain are suddenly stricken with disability or death. Controlled hypertension can decrease morbidity and lengthen life.

Family history of hypertension is a potent predictor of later development of high blood pressure. Several behavioral factors are also believed to increase one's predisposition to disease. Excesses in salt consumption, weight, anxiety, and cigarette smoking may contribute to the development of this insidious disease.

Community health programs have been created to identify those persons at risk of hypertension. Educational efforts have been concerned with reducing risk behavior, particularly among those persons most susceptible. Borderline hypertension is an omonous sign of likely progression of disease (Kirkendall, & Nottebohm, 1977). For persons with borderline high blood

pressure, behavior modification may be the only alternative in averting the development of essential hypertension.

Summary

The study, Borderline Hypertension and Hypertensive Risk Factor Behavior, was a pretest/posttest design intended to measure blood pressure and risk factor behavior in a group of borderline hypertensive persons, before and after information exposure. The hypotheses tested were: 1) Subsequent to information exposure concerning risk factors related to hypertension, persons with borderline high blood pressure will alter behavior to reduce their risk of disease; 2) Change in risk factor behavior in persons with borderline hypertension will have a significant relationship with change in blood pressure; 3) Perception of threat, internality of control, and high levels of knowledge will have a significant correlation with risk behavior modification.

Twenty-eight adult Caucasions and two Black adults were identified as having borderline blood pressure and coexisting risk factor behavior. A Worker Health Program Screening Questionnaire was utilized to collect demographic and baseline data at a worksite screening. At the initial contact

a series of 6 blood pressure measurements was obtained and each subject received individualized instruction concerning behavioral factors which may contribute to the development of hypertension. Each was advised to alter his/her own risk behavior in attempt to avert the progression of disease.

One year later subjects were recontacted at the worksite for blood pressure and risk factor follow-up evaluation. The WHPS was completed and 3 blood pressure readings were obtained. Wallston's Health Locus of Control and a Cognition Assay, designed by the researcher to test knowledge and threat perception, were utilized in obtaining data to correlate with change in behavior.

Approximately 1 year after educational instruction, computed results revealed a reduction in risk factor behavior which was not statistically significant, and significant reductions in systolic and diastolic blood pressures. Blood pressure reductions were the greatest for those who also decreased risk factor behavior score. Direct correlation between risk factor change and blood pressure change were only suggested. Externalizers were more likely to alter risk behavior than not. Internalizers tended to maintain risk behavior at identified levels. A high level of knowledge was expressed by all subjects. Those having

a positive family history of hypertension perceived greater threat of high blood pressure, but were not necessarily the more likely to alter their behavior. At follow-up, subjects who had reduced their risk behavior perceived the threat of hypertension to be lower than those who maintained or increased risk behaviors.

Conclusions

Definitive steps toward enlightening the public were taken in the initiation of this project and results show that a majority of subjects' knowledge about hypertension is attributed to such an endeavor. Whether solely the effect of the program or a combination of extraneous factors, knowledge was acquired concerning risk behavior and high blood pressure. As well, subjects with family history of hypertension perceived their threat of disease to be great, and those who reduced their risk behavior believed they were less at risk of disease. Behavior change did not correlate significantly with the cognitive outcome of educational instruction, though knowledge was high and behavior change significant. A conclusion, then, is that after informational exposure, risk factor behavior will change, but not necessarily in the direction of educational intent.

Blood pressure reduced in the present study in a manner markedly different from what is usually expected. The dramatic reversion of elevated pressures to normal may be linked to educational instruction and behavior change, though available data for evaluating that relationship was limited. The statistical correlation between blood pressure change and risk factor change was not significant. Results clearly indicate, however, that when risk factor behavior is reduced, systolic pressure falls significantly and the chance for a diastolic reduction is 58% greater than in risk factor is not reduced.

Fear and knowledge assessment, valuable tools for evaluation of health education effectiveness, are incomplete tools for evaluating behavior change motivation. HLC offered interesting but partial perspective of behavior change. Education and/or unidentified intervening factors may affect risk factor behavior in ways other than can be determined by locus of control, knowledge, or threat of disease. As concluded from the findings of the present study, perception of threat, internality of control, and high levels of knowledge do not necessarily correlate with behavior modification.

Recommendations

The results of a comparative study frequently suggest complex relationships, unidentified intervening variables, and unexpected effects which stimulate more questions than answers. Statistical correlations often do not adequately summarixe the results which are the most profound. In that light, the present study has illucidated areas for further research, investigation, and ponder.

Behavior motivation remains one of life's mysteries, despite the innumerable approaches, perspectives, and paradigms with which it is analyzed. Perception of threat of disease and knowledge are important elements in behavior motivation, but should be assessed in conjunction with belief and attitude evaluation as well. Exploration of social cues, reinforcers, and support systems also may illucidate important behavior affectors, and contribute to the understanding of behavioral impetus.

To be totally consistent with the learning theory inherent in this design, alternative behaviors should be offered with new knowledge such that deviance from established behavior would achieve satisfaction for the need which initially stimulated behavior. Specific to risk factor reduction, health

education programs could offer assistance to the "at risk" client as effectively as they presently offer information. Smoking reduction clinics, diet education, weight loss programs, and refinement of coping skills can be offered in structured programs to initiate change.

Social and community involvement is imperative, however, as justification for new behavior needs social reinforcement to be adopted. The family and immediate social support systems are prime targets for confronting prevailing social attitudes about smoking, work-related stress, food consumption, and the social conditions precursory to adoption of risk factor behavior. Generally effective in identifying and educating individuals at risk of disease, health programs now need to focus on those social, cultural, and environmental factors which initiate and support the risk behavior.

Perhaps more central to the prevention of hypertension is the relationship between risk behavior and blood pressure. Research relating the effects of risk factors and change in risk factors with blood pressure change is vital when expecting to control disease with behavior. Primary control of hypertension will be feasible only when its etiological mechanisms are more fully understood.

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APPENDIX A

ASSESSMENT TOOLS

1) Worker Health Program Questionnaire

2) Health Locus of Control

3) Cognition Assay

4) Blood Pressure Measurement Technique

5) U.S.F.D.A. Height-Weight Table

Worker Health Program Questionnaire

Screening Form

ID# _____
site# mo/day/yr client#

BP STATUS

Name: _____ N B H

Address: _____ Aware Y/N

_____ Treat Y/N

Observer: _____ Refrd Y/N

Sex M/F Race B/C/O * Age _____

* BP#1: ___/___ * Height _____

* Weight _____

* Have you ever, at any time in your life, had:

	Client	Parent
a) diabetes?	Y/N	Y/N
b) kidney trouble?	Y/N	Y/N
c) a stroke?	Y/N	Y/N
d) heart problems?	Y/N	Y/N

* Do/Have either of your parents have/had:

* Have you ever had high blood pressure? Y/N
Parents? Y/N

* BP#2 ___/___

Do you have high blood pressure now? Y/N

Are you under a doctor's care for high blood pressure? Y/N

Are you on a low-salt diet or any other type of diet for high blood pressure? 1) low-salt diet
2) other diet
3) No

Are you supposed to be taking any pills or medication for high blood pressure? Y/N

↓ (If No, skip to next topic)

If yes,

Do you know what kind of medicine it is?

_____(name of med)

How often are you supposed to take med?

1) more than 1x/day
2) 1x/day
3) less than 1x/day

Have you actually been taking it that often?

Y/N

When did you last take your medicine for high BP?

_____

At the present time, are you:

1) married
2) single
3) separated
4) divorced
5) widowed

What is your present job:

1) professional
2) managerial
3) administrative
4) sales
5) white-collar service
6) clerical
7) craft or skilled trades
8) transportation worker
9) machine operator
10) assembly worker
11) other type operatives
12) laboror
13) blue collar service
14) other

What was the last grade in school that you completed:

1) 1st-11th
2) HS Diploma
3) 1-3 years of college
4) college degree
5) graduate work
6) Masters degree
7) law degree
8) Ph.D or M.D.

Do you have a regular doctor or clinic which you attend? Y/N

* How long ago was your last visit to a doctor or clinic:

1) less than 6 months ago
2) 6-11 mo. ago
3) 1-2 years ago
4) over 2 years ago

* BP#3 ___/___

* How much do you smoke:
  1) None
  2) 1/2-1 pack/day
  3) 1 1/2-2 pk/d
  4) 2 1/2-3 pk/d
  5) more than 3 pk/d

How much coffee do you drink a day:
  1) none/decaffinated
  2) 1-2 cups
  3) 3-6 cups
  4) more than 6 cups

How much nervous tension are you under, in general:
  1) little
  2) average
  3) much

How much salt do you usually eat:
  1) none/substitute
  2) only cooking
  3) add occasionally at the table
  4) lots; add before tasting

* Are you taking any medications:
  1) none
  2) tranquilizers
  3) sleeping pills
  4) high blood pressure medication
  5) birth control pills / estrogens
  6) other _____

Thank-you. Your blood pressure readings today are:
  BP#1 ___/___
  BP#2 ___/___
  BP#3 ___/___

NOTE: Starred (*) items were included in the abreviated interview of the second screening.

WHPQ Addendum

The amount of salt you eat this year compared to last year is:
1) less
2) the same
3) more

The amount you smoke this year compared to last is:
1) less ___pk/d
2) the same ___pk/d
3) more ___pk/d

The amount of nervous tension you are under is how much , compared to last year:
1) less
2) the same
3) more

Do you meditate or practice relaxation technique? Y/N
If yes, how frequently? _____
How long have you been meditating? _____

How much do you weigh this year, compared to last year?
1) less ____lbs less
2) the same
3) more ____lbs more

You have received information about high blood pressure from the following sources:
1) TV
2) newspaper
3) books/printed literature
4) CATCH program
5)other community programs
6) doctor or other health personnel
7) friends
8) family
9) other

Health Locus of Control

Please indicate your agreement or disagreement with the following statements by circling your answer.

(a) If I take care of myself, I can avoid illness.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(b) Whenever I get sick, it is because of something I've done, or not done.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(c) Good health is largely a matter of good fortune.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(d) No matter what I do, if I am going to get sick I will get sick.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(e) Most people do not realize the extent to which their illnesses are controlled by accidental happenings.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(f) I can only do what my doctor tells me to do.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(g) There are so many diseases around that you can never know how or when you might pick one up.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(h) When I feel ill, I know it is because I have not been getting the proper exercise or eating right.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(i) People who never get sick are just plain lucky.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(j) People's ill health results from their own carelessness.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

(k) I am directly responsible for my own health.
1. Strongly agree
2. Moderately agree
3. Slightly agree
4. Slightly disagree
5. Moderately disagree
6. Strongly disagree

Cognition Assay

Place an "x" anywhere on the line where it indicates your opinion.

1) Persons with high blood pressure have a higher incidence of death from heart disease and stroke.

True _____ False

2) The amount of salt a person eats has a definite effect on their blood pressure.

True _____ False

3) The more pounds overweight a person is, the more likely their blood pressure will be high.

True _____ False

4) Usually people with high blood pressure do not develop symptoms until they have had high blood pressure for many years.

True _____ False

5) The amount of nervous tension in a person's life is likely to affect his/her blood pressure.

True _____ False

6) A combination of smoking and high blood pressure will tripple a persons chance of getting heart disease.

True _____ False

7) The amount of salt I eat has a definite effect on my blood pressure.

Agree _____ Disagree

8) The more weight I gain, the more likely my blood pressure will go up.

Agree _____ Disagree

9) By the time I have physical signs of high blood pressure there probably has been serious damage to some major body organs.

Agree _____ Disagree

10) Some habit I have developed probably contributes to my high blood pressure.

Agree _____ Disagree

11) The chance that I will be hospitalized because of high blood pressure or heart problems before I am 65 is:

0% _____ 100%

12) The chance of my developing heart disease as a result of high blood pressure is:

0% _____ 100%

13) If I continue my present living habits, the chance my blood pressure will go up is:

0% _____ 100%

14) The chance I may die because of problems associated with high blood pressure is:

0% _____ 100%

Procedures for Taking Blood Pressures
(Erfurt & Foote, 1975)

1. The LEFT arm will be used at all times. It should rest firmly on the table, slightly abducted and flexed.

2. The compression cuff should be placed on the upper arm 1-inch above the antecubital fossa. The rubber bladder of the cuff should be centered over the brachial artery which extends along the inner aspect of the arm. Apply the cuff evenly and snugly, but without constriction. The cuff must be at the SAME level as the heart.

3. The nurse should sit on a chair before the client. The manometer should be on the table beside him/her in a vertical position, with the meniscus at eye-level. Read the TOP curvature of the meniscus.

4. (FOR FIRST BP READING ONLY) Palpate the radial artery pulse. Inflate the compression cuff to about 30 mm over the point at which the pulse beat ceases on compression; SLOWLY decompress the cuff (2-3 mmHg/sec) to the point on the manometer which corresponds to the beginning of a palpable pulse. Deflate cuff entirely. (Note: this step in the procedure is designed to give you an estimate of the systolic pressure, as well as to allow the client to experience your technique and alleviate anxiety.)

5. Palpate the brachial pulse in the antecubital fossa and place the stethescope lightly over the artery at this point. The diaphragm of the stethescope should not touch the cuff or the client's clothing.

6. Inflate the compression cuff to 30 mmHg above the determined palpatory systolic pressure. Decompress SLOWLY at a rate of 2-3 mmHg/sec.

7. Note when the first sharp (high tone) sound appears in relation to manometer calibrations and record as the systolic pressure.

8. Continue to deflate slowly to the point where SOUND CEASES. Record calibration as the fifth phase of diastolic pressure.

9. Deflate cuff rapidly and WRITE down pressure readings.

10. Be sure cuff is completely deflated between readings.

11. Repeat steps 5 through 10 for each successive blood pressure reading.

12. Failure of stethescope sounds to disappear at the fifth phase for diastolic reading may necessitate recording diastolic pressure at the fourth phase - onset of MUFFLED sound.

13. If a poor reading is obtained on first attempt, deflate cuff, wait a minute and repeat procedure. Record the second reading but mention the first attempt on the record form.

14. If the client shows an auscultatory gap in manometer reading, do not record the first disappearance of sound as the fifth phase diastolic reading. The sound will return later as decompression continues, and then disappear.

Desirable Weights for Height

Height Inches	Weight in Pounds Men	Women
62		115 ± 9
63	129 ± 11	118 ± 9
64	133 ± 12	122 ± 10
65	137 ± 12	125 ± 10
66	142 ± 12	129 ± 10
67	147 ± 14	132 ± 10
68	151 ± 14	136 ± 10
69	155 ± 14	140 ± 10
70	159 ± 14	144 ± 11
71	163 ± 14	148 ± 12
72	167 ± 15	152 ± 13
73	171 ± 15	
74	175 ± 15	
75	178 ±	

USDA, 1960

APPENDIX B

SUBJECT DATA

1) Consent Form

2) Raw Data

University of Oregon Health Sciences Center
School of Nursing

INFORMED CONSENT

I, _____ agree to serve as a subject in the investigation named HYPERTENSIVE RISK FACTOR BEHAVIOR AND BORDERLINE HYPERTENSION by Janet E. Schmiege, RN, under the supervision of Marie Berger, M.S., Chairperson, Graduate Department at the University of Oregon School of Nursing.

The investigation is concerned with the relationship between hypertensive risk factor behaviors and blood pressure measurement. It also is concerned with factors thought to influence risk behavior change. I agree to 1) have my blood pressure measured in the usual fashion; 2) answer questions about health-related activity, 3) answer questions about high blood pressure and its possible consequences. Questions will be in written and oral form. The entire procedure will last approximately 20 minutes.

I may benefit from these procedures in the reevaluation of my blood pressure, and in the opportunity to learn more about hypertension. There is no physical risk to me, and no experimental procedures will be performed.

The information obtained will be kept confidential. My name will not appear on the records and anonymity will be insured in the use of code numbers.

Janet Schmiege/Sallie Shannon has offered to answer any questions that I might have about my participation in the study. I understand I am free to refuse to participate or to withdraw from participation in the study at any time without effect on my relationship with or participation in the C.A.T.C.H. program.

I have read the foregoing.

SIGN _____

DATE _____

WITNESS _____

Subject Data

	Age*		Ht/Wt[1]	Blood Pressure Sys	Blood Pressure Dia	Risk Factor Score Wt	Anx	Na	Sm	HLC	CA(k/t)
White Males											
DS	19	I.	74/188	142	90	1	4	2	0		
		F.	/170	121	73	0	2	1	0	27	54(47/7)
RW	27*	I.	77/210	145.2	90	1	2	4	0		
		F.	/210	142	94	1	0	0	0	34	57(46/11)
TS	27	I.	74/175	140	69.3	0	2	2	3		
		F.	/180	124	74.6	0	2	2	2	35	49(44/5)
BP	31	I.	71/180	149.3	84	1	4	2	0		
		F.	/182	131	79.3	1	4	2	0	24	58(47/11)
JH	31	I.	73/195	120	90	1	0	4	1		
		F.	/206	124.6	83.3	3	2	4	1	37	42(30/12)
RD	31*	I.	65/150	152.6	84	1	2	2	0		
		F.	/148	146.6	90	1	4	2	0	25	60(43/17)
JC	33*	I.	71/190	152	94	2	2	1	2		
		F.	/190	163.3	104.6	2	2	1	2	33	58(46/12)
JR	33	I.	69/170	140.6	81.3	2	4	2	0		
		F.	/162	140	72	1	4	2	0	36	56(38/14)

* indicates positive family history

[1] height and weight are measured in inches and pounds, respectively

				Blood Pressure		Risk Factor Score					
	Age*		Ht/Wt	Sys	Dia	Wt	Anx	Na	Sm	HLC	CA(k/t)
DB	53*	I.	69/145	148.6	88.6	0	4	4	3		
		F.	/145	140	83.3	0	4	4	3	31	54(42/12)
Black Male											
WW	31	I.	69/186	136	92	3	4	4	1		
		F.	/186	132	94	3	4	4	1	50	53(42/11)
White Females											
CH	20*	I.	65/138	141	82.6	1	2	1	1		
		F.	/138	124	88.6	1	0	1	1	45	50(40/10)
RF	32	I.	68/175	142.6	85.6	4	2	4	0		
		F.	/195	134	85.3	4	2	4	0	39	45(36/9)
GL	40	I.	63/160	148.3	89	4	2	1	0		
		F.	/157	151.3	94.6	4	2	1	0	37	44(37/7)
EI	47	I.	66/115	133.6	91.6	0	0	2	1		
		F.	/113	128.6	84.6	0	2	1	1	44	50(41/9)
BD	54	I.	63/130	152.6	78.6	1	2	2	2		
		F.	/140	128	72	2	2	2	2	46	56(40/16)

	Age*		Ht/Wt	Blood Pressure Sys	Blood Pressure Dia	Risk Factor Score Wt	Anx	Na	Sm	HLC	CA(k/t)
BC	35	I.	68/170	143.3	90.6	1	2	2	0		
		F.	/165	139.3	92	1	2	2	1	34	50(42/14)
GJ	36*	I.	68/180	142.6	89.3	3	4	2	1		
		F.	/170	137.3	98	2	4	2	2	29	67(48/19)
GR	36	I.	73/180	129.3	94.6	1	2	2	2		
		F.	/185	136	92.6	1	4	2	3	35	62(50/12)
TH	37*	I.	72/175	129.3	91.3	0	4	4	0		
		F.	/178	134	91.3	0	4	4	0	42	59(44/15)
RL	41*	I.	75/210	149	92	2	4	2	0		
		F.	/205	137	80.6	2	4	2	0	39	52(40/12)
TT	41*	I.	70/185	126	92	2	4	2	0		
		F.	/170	127.3	93.3	0	4	1	0	31	56(46/10)
BT	41*	I.	72/185	145.3	93.3	1	4	2	1		
		F.	/185	132.6	88.6	1	2	2	1	24	64(50/14)
DS	42	I.	71/205	147.6	91.3	4	2	2	0		
		F.	/210	149.3	93.3	4	4	2	0	31	52(44/8)
DN	42	I.	71/232	160	95	4	2	4	0		
		F.	/160	150	90	2	2	1	0	31	49(41/8)

	Age*		Blood Pressure Ht/Wt	Sys	Dia	Risk Factor Score Wt	Anx	Na	Sm	HLC	CA(k/t)
BJ	55	I.	64/112	156	79.3	0	2	1	2		
		F.	/110	149	69.3	0	4	1	2	49	55(46/9)
MP	57*	I.	63/220	148.3	85.6	4	4	1	0		
		F.	/210	124	72.6	4	4	1	0	41	54(43/11)
LO	58*	I.	65/140	142	93.3	1	4	1	0		
		F.	/145	121.3	76	2	2	1	0	38	64(50/14)
JB	58	I.	68/160	154.6	94.6	0	2	2	0		
		F.	/162	130	86	0	2	2	0	43	53(49/4)
MS	61	I.	66/170	147.3	87.3	4	4	2	0		
		F.	/160	144	83.3	3	4	2	0	22	46(42/4)
Black Female											
GW	40	I.	65/138	141	82.6	1	2	1	1		
		F.	/138	124	88.6	1	0	1	1	45	50(40/10)

ABSTRACT

The present study was a one-group pretest/posttest design which measured and correlated changes in risk factor behavior and changes in borderline hypertensive blood pressure in a group of 30 working adults who had been advised to alter risk behavior. The study also assessed knowledge, threat, locus of control, and the influence of each on behavior change. Blood pressure was evaluated on the basis of 3 consecutive measurements. Risk factor behavior data were elicited by a Worker Health Program Questionnaire and were quantified into degrees of risk according to a risk factor rating scale. Health education information was distributed on a 1:1 basis at the time of the initial interview. Health Locus of Control and a Cognition Assay, designed to test knowledge and threat, were administered to each subject at the follow-up interview. Results indicate that subsequent to information exposure, persons with borderline blood pressure and coexisting risk factor behavior will significantly alter behavior, but not necessarily to reduce risk of disease. The correlation between risk factor change and blood pressure change was not significant, though the group that reduced risk factor also experienced the greatest diminution of blood pressure. Internalizers were the least likely to alter risk behavior. Those with family history of disease were the most threatened by disease, and high levels of knowledge were expressed by most subjects.