AN ANALYSIS OF MEDICAL AND NON-MEDICAL PREDICTORS OF HYPERTENSION IN WOMEN FROM THE MANITOBA HEART HEALTH STUDY

BY

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A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfillment of the Requirements
for the Degree of

DOCTOR OF PHILOSOPHY

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ABSTRACT

Most of the research done to date has been directed toward hypertension and its sequelae cardiovascular diseases in males, because of the relatively high incidence and subsequent high mortality rates when compared to females. However, the prevalence of hypertension in women has been rising consistently and its sequela, cardiovascular disease is the leading cause of death in women. The objective of this study was to further the understanding of hypertension in women by identifying and analyzing biomedical risk factors for their potential risk and for their ability to predict susceptibility to hypertension in a sample population of women, and by identifying and analyzing non-biomedical risk factors for their potential risk and for their ability to predict susceptibility to hypertension. The development of a framework to include both sets of risk factors is guided by the expanded medical paradigm proposed by Foss and Rothenberg (1987). The study uses data which were part of a much larger data set collected by the Manitoba Heart Health Survey.

The data for 447 women in the City of Winnipeg were obtained from a risk factor questionnaire and a clinic visit. Hypertensive status and its relationship with an array of independent variables including: anthropometric measures, blood lipids, cardiac and family history, demographics, lifestyle factors, beliefs and behaviours, were analyzed and interpreted.

The results indicated that strong independent relationships existed between

hypertensive status and a number of biomedical risk factors including: age, short stature, larger waist, larger hips, larger WHR, higher BMI, higher total cholesterol, lower HDL cholesterol, higher LDL cholesterol, higher triglycerides, higher glucose, higher total/HDL cholesterol ratio, and diabetes. Family history of heart disease, hypertension, stroke, and personal history of heart attack, other heart disease and taking medication for heart problems were all predictive of hypertensive status in univariate analysis. Further logistic regression analysis showed that the three biomedical variables; age, history of other heart disease and total/HDL cholesterol ratio, were the strongest predictors of hypertensive status showing unique predictive ability and were most effective in characterizing the impact of biomedical variables on hypertensive status in women.

The results further indicated that strong independent relationships existed between hypertensive status and a number of non-biomedical risk factors. The five predictors of hypertensive status which showed unique predictive ability were: being a homemaker, not exercising strenuously, and the beliefs that high blood pressure causes strokes, high blood cholesterol is not a cause of heart disease, and poor diet is not a major cause of stroke.

Further analysis provided additional information by combining variables which show common themes. These multilevel, multivariate groups showed for example that older age is not alone in predicting susceptibility to hypertension but instead occurs with other variables including higher blood lipid levels, being widowed, being retired, being a homemaker, and having a family history of cardiovascular events.

In conclusion this study identified several variables which combine and interact to increase susceptibility to hypertension. Hypertensive women tended to have several health-compromising beliefs and little intention of making any lifestyle changes. High blood lipids occuring concurrently with hypertension and older age, showed the need for further study into understanding the links between variables. Further studies which indicate time-sequencing of events which precede the development of hypertension are necessary to more fully understand the relationships between hypertension and the predictors of hypertensive status in women.

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LIST OF ABBREVIATIONS

CAD CORONARY ARTERY DISEASE

CHD CORONARY HEART DISEASE

CVD CARDIOVASCULAR DISEASE

FEV FORCED EXPIRATORY VOLUME

HDL HIGH DENSITY LIPOPROTEIN CHOLESTEROL

ISH ISOLATED SYSTOLIC HYPERTENSION

LDL LOW DENSITY LIPOPROTEIN CHOLESTEROL

LVH LEFT VENTRICULAR HYPERTROPHY

CHAPTER 1

INTRODUCTION

1.0   RATIONALE FOR THE STUDY

Over the past several decades there has been a fundamental shift in disease patterns from acute infectious diseases to chronic diseases. In developed countries, acute infectious diseases and their contribution to mortality and morbidity rates are nearly gone and barring any unforeseen catastrophies will be much reduced (Evans & Stoddart, 1990). To date, little effort has been made to alert the public of this shift and the new challenges which now are being faced in chronic disease management. Chronic diseases now cause over 80 percent of all premature deaths and over 90 percent of all disability (Fries & Crapo, 1981).

Cardiovascular diseases in 1990 alone account for about 39 percent of all deaths, and are the leading cause of mortality in Canada (Reeder et al, 1993). This is despite a decline in mortality rates for both stroke and ischemic heart disease through the 1970's and the 1980's at a rate of about 2 percent per year (Reeder et al, 1991). This decline may be partially attributed to a reduction in the prevalence of smoking, a reduction in the consumption of dietary fat, improved identification and control of hypertension, and improved medical and surgical care of individuals who have developed cardiovascular disease. A decline in mortality rates for

stroke may be related to improved public awareness of high blood pressure leading to earlier detection and treatment, a reduced prevalence of smoking, as well as improved therapy for stroke patients.

Studies done in both Canada and the United States estimate that up to 30 percent of cardiovascular mortality can be attributed to elevated blood pressure (Reeder et al, 1991). "Hypertension is a powerful independent contributor to cardiovascular morbidity and mortality, on average conferring a threefold increase in risk at all ages and in both sexes. Coronary heart disease is now the chief lethal sequela of hypertension, occurring at a rate two to three times higher in hypertensives than in normotensives. The risk of cardiovascular disease in the hypertensive patient varies widely depending on the associated risk factors" (Kannel & Higgins, 1990:S3). At any age, and in either sex, raised blood pressure increases the risk of a cardiovascular event whether the blood pressure elevation is labile or fixed, or in the systolic or diastolic component (Kannel, 1986).

The story of hypertension and its management would appear to be a classic example of biomedical progress (Alderman, 1980). It has evolved as follows: the epidemiological observation that blood pressure was linked to cardiovascular disease, followed by the development of effective antihypertensive therapy, the application of this therapy which saved lives,

and its quick acceptance as standard medical practice. There are, however, some oversights in this account.

Hypertension is a physical finding but it is not unanimously accepted that it is a disease or an etiological agent, but perhaps is strictly a risk factor for other diseases. Hypertension may have life threatening consequences, mainly due to its role as a risk factor in cardiovascular diseases, but hypertension, defined as elevated blood pressure, is neither a disease nor injury by current definitions. The current definition of disease implies the inclusion of a distinctive natural history and identifiable cellular changes. Hypertension may be a quantitative disease which does not have causative cellular change distinguishing normal from abnormal (Pickering, 1972). It is often used ambiguously in medical literature, with dictionaries defining it as raised arterial pressure and text books referring to it as a complex disease (Jennings & Netsky, 1991). Hypertension also faces the same dilemma as `psychogenic diseases', if there is no evidence of biomedical disease, then there is no pathological diagnosis (Pickering, 1972). Even when viewed as a chronic disease in itself, hypertension does not have a known etiology and there is no accepted primary prevention of hypertension. Therefore, in addressing the issues surrounding hypertension, most of the efforts are in the secondary prevention of asymptomatic hypertension, or in the primary prevention of its major sequelae, coronary heart disease and stroke.

Hypertension is one of the most common health problems in Canada and in Westernized populations. It also is emerging as a major health problem in the developing world. There has been voluminous research into both the epidemiology and the pathophysiology of hypertension but the basic underlying cause or causes remain obscure. It is presently accepted that hypertension is a main cause of strokes, and one of four important risk factors for coronary heart disease. It is closely related to the development of left ventricular hypertrophy and at extreme levels, to cardiac failure. It is also related to the deterioration of renal function in patients with pre-existing intrinsic renal disease. Whether hypertension causes renal damage in previously undiseased kidneys is uncertain. Hypertension and its sequelae are major factors in the prognosis of diabetes mellitus (Ching & Beevers, 1991).

Hundreds of millions of research dollars have been spent on important large-scale epidemiological studies and clinical trials that have improved understanding of the risks and benefits of treating persons with hypertension. As a result, biomedicine has been successful in providing numerous treatment options to aid in the control of hypertension. These include various pharmacological strategies designed to dilate vessels, reduce cardiac output and decrease blood volume, all with the end purpose of reducing the pressure at which the cardiovascular system operates. Other treatments include dietary restriction of sodium, and

weight reduction. The treatment of hypertension, however, has not led to its control, with only about 50 percent of the hypertensive population remaining controlled (Joffres et al, 1992). The resulting cost of hypertension in morbidity and mortality has led to extensive research to discover the reasons for the large number of identified but uncontrolled hypertensives.

Hypertension is a risk factor for the development of coronary heart disease (CHD), but lowering of blood pressure by antihypertensive drugs has failed to give definite protection against CHD. This dilemma produces a most embarrassing situation as CHD continues to be a major health problem in most countries in the Western world. Of all disease classifications, cardiovascular disease has the greatest costs both direct and indirect. Direct costs refer to the value of resources actually expended that could have been allocated elsewhere. Indirect costs are those equalling the value of lost productivity due to illness or disability, and/or the loss of future earnings by people who die prematurely. In Canada, the direct costs of cardiovascular disease are about $5.2 billion and the indirect costs total about $11.6 billion per year. Although mortality rates are declining, the economic costs of cardiovascular disease are increasing (Reeder et al, 1991). Therefore, as well as continuing to support the declining mortality rates, the incidence of the disease must also be reduced so as to minimize its social and economic costs.

Hypertension is one of the most thoroughly studied problems in biomedical research and it also is the most common problem in the practice of internal medicine. Hypertension is so common, that it accounts for more office visits to internists in the United States than any other reason and is the most common medical intervention by internists (Forrow et al, 1988). The percentages are likely very similar in Canada. Alderman and Lamport (1990) report that as many as 30-40 percent of Americans qualify for the hypertensive label at the present guideline levels. The impetus for this effort by physicians to identify hypertensive persons has been fuelled by the pharmaceutical industry and stimulated further by the National High Blood Pressure Education Program (NHBPEP) which encourages identification and control of hypertension (Roccella & Ward, 1984). This translates into more than 20 million Americans in care at a cost of at least 8-9 billion dollars (Final Report of the Subcommittee on Definition and Prevalence of the 1984 Joint National Committee, 1985) with antihypertensive drugs accounting for six of the top ten selling American drugs (Baum et al, 1985). Hypertension is the most common reason for the use of prescription medications in the United States.

Most of the research done to date has been directed toward hypertension and its sequelae cardiovascular diseases in males, because of the relatively high incidence and subsequent high mortality rates when compared to females (The Hypertension Detection and Follow-up Program,

1984; The Pooling Project, 1978; Multiple Risk Factor Intervention Trials, 1982; and Veterans Administration Cooperative Study, 1982). `Being male' is often referred to as an unmodifiable risk factor for CHD and comparatively little research has been done on women. The mortality rates in all categories of cardiovascular disease except stroke, are almost twice as high for males as for females but for stroke are close to equal in men and women (Reeder et al, 1991). The reason for resistance of women to ischemic heart disease compared to men is still unresolved as is the reason for the loss of this relative resistance after the menopause. However, the incidence of hypertension in women has been rising consistently and its sequela, cardiovascular disease is now the leading cause of death in women, exceeding all neoplastic diseases combined (Eaker et al, 1989).

The clinical trials of antihypertensive therapy do not fully support the current guidelines and practice in the treatment of hypertension in women. Research done to date concerning the adverse effects of anti-hypertensive treatment, have generally excluded women from consideration. There is clear evidence that antihypertensive treatment can reduce cerebrovascular morbidity and mortality, and perhaps cardiovascular morbidity and mortality in men. But the evidence of any reduction in adverse outcomes for women is inconclusive (Anastos et al, 1991). Presently, gender differences are not acknowledged in guidelines for antihypertensive treatment. Research into the relationship between

hypertension and women is needed as the prevalence continues to increase, and present drug therapies have not reached their expectations. The realization that the etiology of hypertension, the treatment of hypertension and the potential outcomes may be different in men and women indicates the results of studies involving men cannot be blindly applied to women. The recent indications that antihypertensive drug therapy may be harmful to some hypertensive women reiterates the need for gender specific research (Anastos et al, 1991).

Despite a number of large-scale longitudinal studies done to try to understand the etiology of hypertension the presently known risk factors account for less than 50 percent of the variance between hypertensive and non-hypertensive subjects. It is apparent that despite this voluminous research that there are other risk factors which make some people more susceptible to hypertension.

The biomedical paradigm has been used throughout the past century to explain much about disease causation, process and treatment. The prominent position of biomedicine as an established paradigm has been maintained by its remarkable successes and its optimism in prospects for improved health in the future. As stated by Lewis Thomas, "we use the hybrid term `biomedical' science as shorthand to describe the whole inquiry that underlies modern medicine... it is biological science that most of us in medicine are betting on for the future, and it therefore seems

natural to attach the words biology and medicine together to name the enterprise" (1977:111, cited by Foss and Rothenberg, 1987:4). As described by George Engel, the biomedical approach has "... a firm base in the biological sciences, enormous technical resources at its command, and a record of astonishing achievement in elucidating mechanisms of disease and devising new techniques" (1977:129).

Biomedicine has led to an increased understanding of the mechanisms of disease, and led the way to breakthroughs in disease management. Despite these outstanding successes, critics both within and outside of the medical community are concerned with its narrow focus in both its research and practice (Antonovsky, 1979,1987; Dubos, 1968; Engel, 1981; Knowles, 1977; McKeown, 1979; Foss & Rothenberg, 1987). Because biomedicine requires all its data to be reducible to a single-level physicalist vocabulary, there will be parts of medicine or in this case causes of diseases which will always be beyond the grasp of its scientific capabilities.

In order to further understand the chronic disease hypertension and discover potential risk factors, it may be necessary to look outside the traditional biomedical model for non-biomedical factors which may be risk factors or play a role in the etiology of hypertension. Data on the traditional biomedical as well as non-biomedical risk factors related to hypertension, including the attitudes, beliefs, knowledge, behaviors and

social environments of the individuals, may help to identify some of the environmental factors which may lead to susceptibility to hypertension.

Current research into fields such as psychoneuroimmunology is showing biological and behavioral links between the nervous system and the immune system that may help to shed some light on the socioeconomic gradient in vulnerability to disease (Jemmott, 1985). The identification of risk factors which are common to those experiencing the morbidity of interest, in this case hypertension, can lead to the provision of interventions or services to prevent or lessen the impact of its occurrence as well as predict future morbidity (Hayes, 1991). By identifying risk factors outside of the present biomedical paradigm and by linking these factors to outcomes, attention could focus on the need for changes in resource allocation, social services or lifestyles. Although there are problems of methodology in this kind of analysis, there is potential to understand and consequently improve health status.

Although hypertension has been treatable and controlled by a number of effective antihypertensive drugs, the most important end points; cardiovascular morbidity and mortality, and all cause morbidity and mortality, have not improved. Although blood pressure has been lowered successfully it has not led to the expected reductions in these end points (MacMahon et al, 1989; Collins et al, 1990; Moser et al, 1991). Therefore, the effectiveness of pharmacological therapy in lowering the risk of

cardiovascular morbidity and mortality is questionable especially for certain subgroups including hypertensive women.

All of these areas present questions which cannot readily be answered without further research. This study attempts to further our knowledge and understanding by presenting potential explanations of women's susceptibility to hypertension. As this is a cross-sectional study, cardiovascular mortality and morbidity were not measurable and therefore hypertension will be considered a chronic disease, rather than a cardiovascular risk factor. Risk factors for hypertensive women both biomedical and non-biomedical will be identified and analyzed as to potential risk as well as their ability to predict hypertension. The contribution of the biomedical risk factors will be combined with the contribution of the non-biomedical risk factors in explaining susceptibility to hypertension. The risk factors will be further analyzed to determine multivariate and multilevel relationships and organization in predicting of susceptibility to hypertension in women. Women only will be studied.

1.1   STATEMENT OF THE PROBLEM

Hypertension is a major chronic disease in Canada, affecting approximately 18 percent of the population (Reeder et al, 1991). Research into the causes of susceptibility to hypertension has been extensive, yet the

etiology of hypertension is still uncertain. It is likely that hypertension is caused by some combination or interaction of environmental and genetic factors rather than just one or the other. Genetic factors cannot be changed, but environmental factors which place one at risk, if identified, can be avoided in an effort to reduce hypertension and its sequela CVD.

The risk factors now known account for less than half of the variance between hypertensive and non-hypertensive subjects in some sample populations (Pooling Project Research Group, 1978), leaving the majority of the variance still unaccounted for and unknown.

The purpose of this study is to further the understanding of the chronic disease hypertension in women by identifying and analyzing biomedical risk factors for their potential risk and for their ability to predict susceptibility to hypertension in a sample population of women, and by identifying and analyzing non-biomedical risk factors for their potential risk and for their ability to predict susceptibility to hypertension. Non-biomedical risk factors present in an individual's culture may impact on susceptibility to hypertension and can be identified by analyzing variables including demographics, beliefs, attitudes, behaviors and social environments. Development of a framework to include both sets of risk factors and predictors will be guided by the expanded medical paradigm proposed by Foss and Rothenberg (1987). The relationships between hypertensive status and individual characteristics including: biomedical

variables, anthropometric variabless, demographics, history, attitudes, beliefs and behaviors will be explored within this expanded medical paradigm.

1.2 RESEARCH QUESTIONS FOR INVESTIGATION

1. What are the identifiable biomedical risk factors which are predictive of hypertension in women?

2. What are the identifiable non-biomedical risk factors which are predictive of hypertension in women?

3. Can the risk factors, biomedical and non-biomedical, be synthesized into an expanded model which is optimally predictive of hypertensive status in women?

4. What is the potential of additional information to be useful in synthesizing risk factors to show multivariate and multilevel relationships?

1.3   LIMITATIONS OF THIS STUDY

1)   The non-biomedical information is determined by data collected by the Manitoba Heart Health Study and is not supplemented by a follow-up interview which may have added valuable cultural information.

2)   The study is cross-sectional and therefore causal relationships between variables and outcome cannot be demonstrated. The data will not be followed up prospectively. Cross-sectional studies collect data in selected populations without any prior knowledge of the frequency of outcomes or suspected causal factors. Their data can reveal association but does not speak to the question of time order, or to the issue of direction. The associations are founded on what is prevalent at a said time and not on what is incident over time (Susser, 1991).

3)   The questions on the survey somewhat limit the possible responses and those responses classified as "other" were not analyzed further.

4)   The generalizability of this data is limited to urban women because data included women only from the City of Winnipeg and not from

all of Manitoba. This was done to prevent confounding the results by combining urban, rural, and northern locations which may affect many of the variables.

1.4   DEFINITIONS OF TERMINOLOGY

To clarify the terminology used in this study, the following definitions are provided:

1.4.0   Biomedicine

Biomedicine refers to today's scientific medicine. It achieves medical advances by means of an increased understanding of the biological mechanisms of the body. It is used interchangeably with: *scientific medicine, Western medicine, cosmopolitan medicine, and modern medicine.*

1.4.1   Culture

Culture in this study refers to one's acquired knowledge that is used to interpret experience and generate social behavior. Rather than emphasizing behavior, customs, objects, or emotions, the focus is on their `meaning' (Spradley, 1979:5). People learn their culture by observing other people, listening to them, and then making inferences. Every culture

provides people with a way of seeing the world. Culture includes assumptions about the nature of reality as well as specific information about that reality (Spradley, 1979:10).

1.4.2 Hypertensive

For the purpose of this study, hypertensive refers to those subjects currently being treated for hypertension with drugs, salt restriction or weight reduction and/or mean diastolic blood pressure greater than or equal to 90 mm Hg.

1.4.3 Paradigm

Paradigm describes "...a research group's particular common and shared way of seeing,...it is the particular paradigm of the research tradition that guides their scientific thinking" (Kuhn, 1970). *Paradigm* refers to a model of acceptable scientific practice. A paradigm involves a distinctive way of seeing the world, and its adoption by a scientific community initiates and dominates a period of `normal' science. During such a period, scientific inquiry is said to be essentially a mopping-up operation of solving problems or puzzles prescribed by the paradigm, rather than a search for evidence to test it. (Review of Thomas Kuhn's Structure of Scientific Revolutions in Scientific American, May, 1964)

1.4.4   Postmodern Science

Postmodern science began early in the twentieth century and is still developing. Its disciplines include relativity theory, quantum mechanics, irreversible thermodynamics and ecology. Its characteristics include interactionism, emergence, loop-structure, and mutual causality.

CHAPTER 2

REVIEW OF LITERATURE

Chapter 2 begins by reviewing the theoretical and methodological frameworks which have set the direction of medical research in general and research into hypertension specifically. Current research into new ideas in medical paradigms are reviewed to provide the context for the present study. A review of the relationship of cardiovascular disease and atherosclerosis to hypertension follows providing the setting for hypertension as a chronic disease, as well as a risk factor for other chronic diseases. The risk factors for hypertension are reviewed in sections entitled biomedical risk factors and non-biomedical risk factors to coincide with the research questions and subsequently with the reporting of results and discussion.

2.0   THEORETICAL AND METHODOLOGICAL FRAMEWORKS

2.0.0   The Biomedical Model

The biomedical paradigm has been the prominent paradigm used for most medical research over the past century, and has been used to explain much about disease causation, process and treatment.

In the years which preceded the advent of scientific medicine, disease causation and cure were based on folk wisdom, ad hoc procedures, and subjective explanations. With the emergence of scientific medicine which developed from `modern' science from 1650 AD on for the next 250 years, the orderly search for physical causes and cures of disease relied almost exclusively on a physical approach to disease. The dominant premises of this scientific medicine or biomedicine as it is now referred, include Newtonian physics and Cartesian dualism. These premises assume that the body can be considered separate from the mind and that the body can be understood by knowing of its parts and how they interrelate.

The conceptual change which initiated scientific medicine was initiated by Descartes in his *Traite de l'homme* which separated reality into two separate realms: the mind *res cogitans* and the body or matter *res extensa*. Descartes referred to the body as a machine, consisting of nerves, muscles, veins, blood and skin, which would continue to have the same functions with or without the mind. Newton's concept of the universe as a great harmonious and materially ordered machine fostered the idea that matter was inert and separate from the scientists themselves. The natural world was seen as "...a complex material whole, which was composed of assembled atomic parts which when interacting with sufficient complexity, could form biological systems as complex as human beings" (Foss & Rothenberg, 1987:24). This reduction of the universe to matter in motion

was confirmed in the development of modern physiology such as the discovery of the circulation of blood.

The result of this scientific thinking was the movement from preservative medicine of the holistic Greek and Roman approaches to health and disease, to curative medicine. The resulting reductionist sciences which now account for present medical strategies and successes include: immunology; bacteriophage and microbial genetics; cell biology; membrane structure and physiology; neurophysiology, and molecular biology. They are central to the contemporary medical community. "By the end of the nineteenth century, with the development of the sciences of physiology, cellular pathology, and bacteriology, the preeminence of a biochemical and physiomaterial approach to disease was consolidated" (Foss & Rothenberg, 1987:27).

In summary, the essence of the biomedical model was that the body became viewed as a machine which malfunctioned and needed repair. The body was recognized as a physiological mechanism, and the patient was recognized as a biological entity whose protection from disease depended on internal intervention. The highest level of organization studied was the individual organism, and patients were considered independently of the psychosocial and environmental forces in which, in a contrasting (prerevolutionary) hygieian view of disease, they must adopt in order to maintain health. This natural science paradigm provided the foundation

for the rise of biomedicine and its subsequent successes. Notable successes include the recognition that certain organic entities (ie. bacteria) caused certain diseases and that their pathogenic effect could be avoided or reversed by certain substances (ie. antitoxins and vaccines) and secondly, Koch's discovery of the tubercle bacillus as the cause of tuberculosis.

By the 1950's a medical strategy was firmly in place, with its underlying premises being: the body can be considered separately from the mind, (dualism) and that the body can be understood through knowledge of its parts and how they interrelate (reductionism). "A consistent theme throughout any discussion of the biomedical model is the philosophical commitment to the separation of mind and body and the methodological commitment to the idea that all bodily events have determinate causes" (Foss & Rothenberg, 1987:36-37). These foundations originated in the seventeenth century conceptual revolution spearheaded by Newton and Descartes and culminated in today's biotechnic successes.

Biomedicine has a scientific and philosophical commitment to the natural science paradigm and has developed at the foundations level from modern sciences such as classical mechanics and statistical thermodynamics. The concepts forming the foundation of biomedicine include:

1) reductionism - disease is explained by referring to the sum of the smallest units of biological functions such as genetics, and cellular pathologies;

2) mechanism - an engineering model of events where a stimulus or pathogen produces a given reaction or disease;

3) causality - disease is the result of external forces or events which act upon the biological organism in a linear time fashion;

4) determinism - fundamental mechanistic laws explaining the process of disease can be determined and will hold across space and time; and

5) dualism - the mind and the body are separate and as such designations such as psychosomatic illnesses, may mean those without a reducible biological explanation (Labonte, 1990).

2.0.1   Medical Countermovements

"The hall mark of a scientific model is that it provides a framework within which the scientific method may be applied. The value of a scientific model is measured not by whether it is right or wrong but by

how useful it is. It is modified or discarded when it no longer helps to generate and test new knowledge" (Engel, 1981:122). The biomedical model is the framework within which most research has been done in recent years. The theories and methods generated within this framework including disease causation, process, and treatment have been consistent with the philosophical foundations of biomedicine. It has continued to be the most useful framework because of its unprecedented successes but as science evolves into its post-modern era, medical countermovements have resulted from trying to extend biomedicine's relatively narrow focus. Some examples of countermovements include: naturopathy, homeopathy, chiropractic, nutritional medicine, Christian Science, psychic healing, and most recently holistic medicine. Dissatisfaction from within the medical community has also arisen as a result of the realization that there is a multicausal base for disease which cannot be dealt with scientifically using the present scientific model.

It is becoming increasingly apparent that disease causation may not be understood totally within the confines of the biomedical paradigm. There are many dimensions of disease which are not susceptible to the scientific approach and if this approach is deemed to be inadequate, then it would appear that broader approaches which are dynamic, holistic, or based on general systems theory would be deserving of consideration. "The triumphs of the biomedical model have been in

the areas for which the model has provided a suitable framework for scientific study. The biopsychosocial model extends that framework to include previously neglected areas" (Engel, 1981:122).

Psychological and Social Structural Models

Other theoretical frameworks have also competed with the biomedical model as alternatives which could possibly explain new observations. These include the psychological model and the social structural model. Veatch (1980) maintains that the biomedical model's premise that disease results from external and uncontrollable causation, is now contradictory to the empirical realities of chronic diseases.

The psychological model maintains that determinism is modulated through psychological imperatives, and perhaps grounded in childhood experiences, which make it impossible for an adult to change compulsive habits (Veatch, 1980). The psychological model focuses narrowly on the links between microlevel stimuli and intrapersonal processes such as perception, cognition, learning, and development. It has typically studied the individual, the mind, or mental and psychological processes. During the 1970's and early 1980's psychologists in every major area of the discipline noted the deficiencies in decontextualized research and called for approaches to behavior that were more holistic and ecologically grounded.

Schwartz (1982) proposed a `biopsychosocial' view of health and illness to replace the `single cause, single effect' model in an effort to address the complex interactions among physiological, psychological, and social dimensions of well-being. Within the fields of cognitive, personality, and social psychology there was an increasing trend toward the contextual analyses of cognition and social behavior. Altman (1982) contended that "we are in the midst of a full-fledged scientific revolution across all areas of psychology, involving a shift from unidirectional, mechanistic analysis of environment and behavior toward transactional and contextually oriented models" (as quoted in Stokols & Altman, 1987:42).

Issues in psychology including: the relationship between persons and environments, the relationship of time and change to psychological processes, and issues associated with philosophy of science, theory, and research methodology are presently being addressed in many fields of psychology. Tyler (1981, as referenced in Stokols & Altman, 1987:8) described recent phenomena in psychology. She portrayed psychology as extending its boundaries in recent years to emphasize the role of context and holistic aspects of human activity. Furthermore, she referred to the growing interest in: the idea of multiple directions of causation and relations between variables, systems approaches involving complex sets of variables, appreciation of the importance of understanding single events,

theoretical approaches that emphasize contextualism and that view phenomena as historical events, and theories centered around ideas.

The social structural model notes the association of poor health habits with lower socioeconomic class and supports the argument that society has the ultimate responsibility for improving the conditions that lead to better health, not the individual. Moos (1984) hypothesizes that one's perception of one's environmental system gives rise to resource and stressor life experiences which in turn affect one's coping and finally one's health outcome. His conceptual framework of illness considers adaptation to stressful life circumstances to be affected by an environmental system and a personal system. For example, factors in the social environment as well as personal factors may cause chronic strain which may lead to depression, which in turn may affect the immune system and increase the risk of physical illness. In a similar manner, environmental factors may lead to cohesion and social approval which in turn promotes stress-resistance or buffering effects by stimulating neuroendocrine mechanisms to produce beta endorphin and other neuropeptides (Broadhead et al, 1983). Factors in the social environment may alter health risk behaviors such as eating, smoking, and drinking patterns and thereby affect health status (Moos, 1984).

Broadhead et al (1983) further suggest that elements of social well-being such as wealth, political power, and social prestige may not

make one healthier but at the same time there are some elements of social environment that do tend to make one healthier. A factor such as social support is just one psychosocial resource which has the potential to sustain healthy functioning and enable a person to act and participate effectively. Veatch (1980) concludes that rather than any one of these models, that the ultimate model should be multicausal and not exclusively the property of medicine, psychology, sociology, or public policy.

Ecological Models

Numerous efforts have been made over the past several decades to expand the health care field away from the traditional reductionist view of biomedical science to a more ecological view (Lalonde, 1974; Ottawa Charter for Health Promotion, 1986). This ecological view says that health is the product of the continuous interaction and interdependence of the individual with his or her ecosphere; that is, the family, community, culture, societal structure and physical environment (Greene & Raeburn, 1988). The ecological model views health and disease as outcomes of a complex system of interactions between the individual worker and multiple levels of environmental influences (Robins & Klitzman, 1988). Within the medical community, there has been increasing recognition of the importance of preventive medical approaches. There also has been

more active involvement by the public in consumer choices and a demand for accurate information on which to base such choices, with changes in personal lifestyles showing the most significant changes (Fries & Crapo, 1981).

Ahmed, Koeker and Coelbo (1979) offer a viewpoint that emphasizes the social and cultural context of the person in conceptualizing health:

> "Our definition of wellness and illness takes into account the specific roles the individual is expected to play in this cultural milieu, as well as the judgements that the individual himself and significant others in his social network, make about the adequacy of his performance. In particular, effective functioning in two social roles, the familial and the occupational, tends to be recorded as crucial to the well-being of the individual and his community. Health, then, must be viewed not merely as a state desirable in itself but it is a means toward the fulfillment of strategic role obligations, and illness as a obstacle to such fulfillment." (1979:13)

This perspective is closely aligned with the Hippocratic conception of medicine, which emphasized maintaining a balance and harmony in lifestyle at home and at work. Health, in turn, becomes a very complex notion, which involves a number of interacting systems that affect a person's capacity to adapt to the demands that are perceived as important in living. Diseases in this context are not so much physiological abnormalities to be cured, as they are chronic responses to the circumstances of living (Thoresen, 1984).

2.0.2   INFOMEDICAL MODEL

Biomedicine has had many successes including increasing understanding of the mechanisms of disease, as well as leading the way to breakthroughs in disease management. Despite these outstanding successes, critics both within and outside of the medical community have expressed concerns with its narrow focus in both its research and practice (Antonovsky, 1979,1987; Dubos, 1968; Engel, 1977,1981; Knowles, 1977; McKeown, 1979; Foss & Rothenberg, 1987). Because biomedicine requires all its data to be reducible to a single-level physicalist vocabulary, there will be parts of medicine or in this case causes of diseases which will always be beyond the grasp of its scientific capabilities.

Hans Selye (1956) introduced the general adaptation syndrome, which suggested that stress may influence susceptibility to disease by altering immunological functioning (Jemmott, 1985). Several researchers have since linked psychological stress to the incidence of a variety of diseases (Dohrenwend & Dohrenwend, 1974; Jenkins, 1976; Robken & Strening, 1976; Minter & Kimball, 1978) and the immune system as the body's principal mechanism of defense against disease. A new field, known as psychoneuroimmunology (Jemmott, 1985) has presented the concept that the `immune system', operating via the central nervous and neuroendocrine systems, may act as a `transducer' between experience and an immunosuppression prone personality, which when it interacts with

particular pathogens leads to disease. Others may be able to mobilize through the central nervous system (CNS), the neuroimmunological and neuroendocrinological resources to prevent damage to the organism.

Research into fields such as psychoneuroimmunology which are showing biological and behavioral links between the nervous system and the immune system may help to shed some light on issues such as the socioeconomic gradient in vulnerability to disease (Jemmott, 1985). The identification of risk factors which are common to those experiencing hypertension, can lead to the provision of interventions or services to prevent or lessen the impact of its occurrence as well as predict future morbidity (Hayes, 1991). By identifying these risk factors outside of the present biomedical paradigm and by linking these risk factors to outcomes, attention could focus on the need for changes in resource allocation, social services or lifestyles. Although there are problems of methodology in this kind of analysis, the importance may be in the potential to understand and consequently improve health status.

Presently, there is no established paradigm available to accommodate non-biomedical factors. Without such a paradigm, the risk factors which are outside the biomedical paradigm which may be quantitatively significant for the overall health status of modern populations will be excluded from disease causation research. The premise of modern scientific medicine as established in the biomedical model is not

adequate to address the multifactorial basis for disease. To discover the factors which account for the presently unexplained variance in hypertension, it is necessary to go beyond the traditional biomedical paradigm because of its limitations which result from the premises on which it is based. The biomedical paradigm, is the conceptual framework that determines in what manner we approach the world in general and a specific area of interest in particular (Foss & Rothenberg, 1988:4).

The biomedical paradigm is based on such modern sciences as classical mechanics and statistical thermodynamics. An expanded medical paradigm proposed by Foss and Rothenberg (1987) is based on post modern sciences such as quantum mechanics, and irreversible thermodynamics, which introduce the principles of interactionism, emergence, loop structure, mutual causality, and self-organization. Once these are in place, neither what is human nor what is ecological can any longer be ignored or excluded by being deemed reducible to something else. The infomedical model is based on interactive principles which reject the reductionist and dualist premises of the biomedical model. As a self-organizing systems model, the infomedical model provides a framework which is scientific and is able to deal with the extrasomatic aspects of disease inaccessible to biomedical treatment. This expanded medical model involves a conceptual shift from a biological systems infrastructure to a self-organizing systems infrastructure.

Foss and Rothenburg (1987) base their `infomedical model' on the above concept and link information from multiple levels of organization including; mind, culture, and body. The need for a paradigm change in medicine is explored by examining "...the loop-structured connections between the individual humanself and its roots in the biological, psychological, and wider cultural milieu and beyond" (Foss & Rothenberg, 1987:2).

"A reformulation of the medical model ought to include medical anthropology's understanding of medicine as a cultural system, as well as our appreciation of the mechanisms by which culture systematically influences disease, illness and healing..." (Kleinman, 1978:90). To move from the medical model on which current health care policy is based, a more complex framework is needed. A framework cannot be developed unless a different paradigm is available from which to work. Many attempts have been made to develop comprehensive, yet flexible frameworks to represent a wide range of relationships among the determinants of health. Most however continue to develop categories into which evidence must fit (Evans & Stoddart, 1990).

Despite the numerous successes of biomedicine we have not reached a state of optimal health and we are still suffering from the continuing burden of illness, disability, distress and premature death. These appear to be less and less sensitive to further extensions in health care, in other

words we may be reaching the limits of medicine. There is voluminous evidence that this burden may be sensitive to interventions and structural changes outside the health care system or the biomedical paradigm. The effect of these risk factors which lie outside the biomedical paradigm are not just a problem of " some poor deprived minority whose situation can be deplored and ignored by the rest of us... we are all (or most of us) affected" (Evans & Stoddart, 1990:1353). These risk factors which are outside of the biomedical paradigm may be quantitatively significant for the overall health status of modern populations. These factors and the issues they involve are not trivial, second-or-third order effects, but to make use of them quantitatively will be challenging because there are presently few methods available. When we are discussing drawing a framework, within the biomedical paradigm, the framework can only be extended to include physical factors in a linear fashion (Evans & Stoddart, 1990).

2.1 HYPERTENSION AND CARDIOVASCULAR DISEASE

Hypertension can be detected by medical signs such as blood pressure in which readings measure the ratio between cardiac output and total peripheral resistance of the vascular system. The currently held hypothesis is that in established hypertension the cardiac output is normal and that the elevation in blood pressure results from an increase in resistance to blood flow at the peripheral arteriolar level (Ching & Beevers, 1991). In a normal vascular system, blood pressure is kept within limits both at rest and during periods of physical stress. When hypertension is present, this ratio is disturbed, and over time this can cause damage to the vessel walls and to the heart itself. The resulting condition can also lead to possible sequelae including kidney disease and blindness, as well as cardiovascular diseases (Williams et al, 1980).

Hypertension is a well established risk factor for the development of CHD. Even though CHD continues to be a major health problem in most countries in the Western world, the incidence of CHD has been reduced over the past few decades (Table 2.1).

Table 2.1 Reduction in the Incidence of CHD in Some Western
          Countries

Country	% Reduction in Incidence of CHD (1970 to 1985)	
	Male	Female
	(ages 30 - 69 years)	
United States	49%	48%
Australia	46%	51%
Canada	41%	43%
Japan	39%	53%

(taken from Leren, 1990:195)

This trend does not hold true everywhere. In countries such as the
Nordic countries, Northern Ireland and Scotland which are traditionally
high coronary heart disease countries, there has been some decrease in
CHD mortality rates ranging from 3 percent to 23 percent and averaging
about 11 percent. There is however, an alarming increase in Eastern
European countries including Romania, Poland, Yugoslavia, Bulgaria,
Hungary, the German Democratic Republic and Czechoslovakia. From
1970 to 1985, the average CHD mortality rate for men aged 30-69 years
was increased by an average of 48 percent with a range of 10 percent to 90
percent (Leren, 1990).

Since 1967-1968, when the incidence of CHD mortality peaked, there
has been a continual reduction in the United States and Canada. It may

be due in part to better medical interventions, or even more so due to the modification of risk factors by the public (Goldman & Cook, 1984; Pell & Fayerweather, 1985).

Epidemiological studies have established that hypertension is an important risk factor in the development of CHD where high cholesterol levels are seen. In some Eastern countries where there are low cholesterol levels it had been found that there is a low incidence of CHD despite the high prevalence of hypertension. This suggests that hypertension may need a metabolic basis of a certain lipid level to become an atherogenic factor. In some countries hypercholesterolemia is endemic and therefore hypertension is defined as an important risk factor (Leren, 1990).

Primary intervention trials have shown that cholesterol-lowering diet alone cannot prevent coronary heart disease. However, cholesterol lowering has been shown to reduce CHD incidence rates. The Oslo Diet Heart Study (Hjermann et al, 1986) was a two-factor study of men only, who smoked and had elevated cholesterol levels. It was able to show that with strict diet intervention cholesterol levels were reduced, leading to reduction in CHD incidence and total mortality. The Lipid Research Clinics Coronary Primary Prevention Trial (Lipid Research Clinics Program, 1984), the WHO Drug Trial (WHO Cooperative Trial, 1980), the Coronary Drug Project (Coronary Drug Project Research Group, 1975), and the Helsinki Heart Study (Frick et al, 1987) were all monofactorial lipid

intervention studies using lipid-lowering drugs, which were able to lower lipid levels and reduce CHD incidence rates, but were not able to significantly reduce overall mortality (Leren, 1990). The Blanckenhorn Study (Blanckenhorn et al, 1987) and the National Heart, Lung and Blood Institute (NHLBI) Study (Levy et al, 1984) showed that lipid-lowering drugs could decrease the progression of coronary atherosclerosis when compared to untreated control groups. Therefore, it appears that improving the lipid profile can prevent CHD to some extent. However, when antihypertensive drugs are being used it is possible that they may have an adverse effect on blood lipids which in turn nullifies or diminishes the beneficial effect of blood pressure reduction.

Data from the Pooling Project (Pooling Project Research Group, 1978) show that rates of cigarette smoking, serum cholesterol levels and blood pressure were of approximately equal importance in predicting coronary events. However, the relative importance of each changed with age. Serum cholesterol levels and perhaps smoking were the strongest predictors of disease in the younger age groups (40-49 years) but blood pressure remains an important predictor throughout the age range (40-64 years). The final Pooling Project report (Pooling Project Research Group, 1978) stated that the relationship between the major risk factors and susceptibility to CHD was consistent, strong, graded, and independent, but there were no female subjects in this study.

There is some concern that the relationship between coronary heart disease mortality and hypertension as measured by achieved diastolic blood pressure is J-shaped. Some large scale studies (Cruickshank et al, 1987; Samuelsson et al, 1987) which treated moderate to severe hypertensives, have demonstrated that for those individuals whose diastolic blood pressure dropped below 80 mm Hg there is a slightly higher CHD mortality (Ching & Beevers, 1991). There are some explanations offered for this J-shaped curve relationship. It is possible that the J-curve relationship between diastolic blood pressure and risk of coronary heart disease, may only be present in those people who have pre-existing coronary heart disease which has further lowered their pressures, and their lower pressures are unrelated to drug therapy (Ching & Beevers, 1991). Perhaps there is a critical level of blood pressure necessary for the diastolic filling of the coronary arteries (Cruickshank, 1988) or perhaps J-curves are particular to patients with some pre-existing coronary disease. Perhaps treatment should be less aggressive with these patients with target diastolic pressures around 90 mm Hg. rather than lower. It is suggested that low diastolic pressure in former hypertensives may be reason to consider underlying coronary heart disease.

Coope and Warrender (1988) observed a J-curve in both treated and the untreated elderly hypertensives encouraging the suggestion that preexisting coronary heart disease is present. The Medical Research

Council (MRC) Trial (MRC Working Party, 1985) studied younger patients with mild hypertension, who had a low incidence of coronary heart disease at entry and throughout the trial, and this study showed no J-curve.

A major overview of all population studies done on the risk of CHD, (MacMahon et al, 1990), confirmed that CHD risk in relation to lower blood pressure extends down to low levels of blood pressure. This is true for the non-elderly at least, and there is presently no evidence that very low blood pressure in patients with no pre-existing CHD has a bad prognosis. However, in a British study of low blood pressure, Wessely et al (1990) found that low blood pressure may be associated with an increasing morbidity from non-specific non-life-threatening ailments.

Although it has been demonstrated that there is a statistically significant association between blood pressure and the subsequent incidence of CHD (Dollery, 1986), this does not prove cause and effect. Evidence of causation would include showing that modification of the predictive factor (hypertension) would then change the incidence of the disease (CHD) in the direction and with the magnitude expected. Dollery (1986b) looked at placebo-controlled trials in hypertension including the Veterans Administration Trial (Veterans Administration Cooperative Study Group, 1967, 1970), the Australian Therapeutic Trial (Report by the Management Committee, 1980), the Medical Research Council Trial (MRC) (Medical Research Council Working Party, 1985) and the trial of the

European Working Party on Hypertension in the Elderly (Amery et al, 1985). The MRC trial was by far the largest of these but the results of trials have been disappointing for preventing coronary events. The multiple risk factor intervention trials (MRFIT) (Multiple Risk Factor Intervention Trial Research Group, 1982) have been inconclusive and antihypertensive therapy trials have shown no change or small reductions in the incidence of myocardial infarction. The slope of the relationship between blood pressure and risk of myocardial infarction would anticipate a reduction of 25-30 percent in the incidence of coronary events. Failure to achieve this reduction in coronary events by reducing high blood pressure is now a major unresolved issue in treatment of hypertension.

Dollery (1986b) in a pooling of the results of randomized placebo-controlled trials found that cerebrovascular disease has a strong relationship to elevated blood pressure and a consistent response to blood pressure reduction. The trials showed that antihypertensive therapy reduced the incidence of stroke by an average of 45 percent. The reduction in strokes was approximately of the magnitude expected from the reduction in blood pressure. The Framingham Study (Kannel et al, 1970) found that hypertensive patients had 4.6 times higher incidence of stroke than did normotensive patients. The slope of the relationship between blood pressure and stroke was steep, with blood pressure being the most powerful risk predictor for stroke.

Bonita and Beaglehole (1989) evaluated the effect that recent control of hypertension had on the decline in stroke mortality. Their calculations based on a pooled analysis of nine randomized controlled trials suggest treatment of hypertension is responsible for only about 6-16 percent of the reduction in stroke mortality. Epidemiological observations attribute 16-25 percent of overall decline in stroke mortality to treatment of hypertension but it is possible that clinical trials can underestimate community-wide benefits of treatment. They suggest that factors responsible for the decline in stroke mortality which occurred before the widespread use of antihypertensive therapy continue to be important and that most (at least three quarters) of the recent decline in stroke mortality is due to factors other than antihypertensive treatment.

2.2   HYPERTENSION AND ATHEROSCLEROSIS

The precise causes of hypertension are unknown except in some adrenal and renal pathologic conditions. The underlying feature appears to be recognized as systemic arteriolar constriction with an increase in systemic vascular resistance with elevation of blood pressure and preservation of cardiac output.   This premise underlies most pharmacological efforts to control hypertension (Gorlin, 1991). The arterial system is the primary target organ of hypertension. Initially, small artery

intimal and medial thickening develop and if the process is allowed to proceed unchecked there can be fibrinoid necrosis. This process can be especially damaging to the cerebral, retinal, renal, and to some degree the coronary arteries.

Despite massive research efforts, the relationship between hypertension and atherosclerosis has never been clearly defined. It is suggested that hypertension can predispose the arteries to atherosclerosis by initiating medial thickening. If atherosclerosis is present before hypertension, then hypertension may play a provocative role in the rupture of plaques and can exacerbate already existing atherosclerotic lesions (Gorlin, 1991). The heart itself is affected by prolonged hypertension. Left ventricular hypertrophy (LVH) results from established hypertension but its severity, chronicity and duration is somewhat unclear. Devereux (1990) has suggested that LVH may indeed be part of an underlying disorder which leads to vascular hypertension, rather than the other way around.

Epidemiological studies do not usually measure or see atherosclerosis, but it is inferred from its product - (CAD). One of the limitations of earlier studies of atherosclerosis was the limitation of traditional visualization methods which did not allow the investigation of the early phase of atherosclerosis which is the structural changes in the arterial wall (Ross, 1986). This prevented the study of the association of

atherosclerosis with the three major coronary risk factors - serum lipoproteins, hypertension and smoking. Furthermore earlier studies were done on dead or symptomatic people and were not generalizable to general populations.

The first study of the etiology of atherosclerosis in an unselected human population sample was done by Salonen and Salonen (1990). The data from this cross-sectional study of Finnish men suggested that elevated serum LDL cholesterol was associated with increased risk of common carotid arterial wall thickening and that smoking was more strongly associated with carotid plaques that intimal-medial thickening. However, they did not find evidence to support association between hypertension and either common carotid arterial wall thickening or carotid plaques. In the data analysis, hypertension was kept binary (no versus yes). They suggest that the lack of association between hypertension and both intimal-medial thickening and plagues could mean that hypertension is a weak precursor of common carotid atherosclerosis, or it is a consequence of knowing little about blood pressure levels in early life. A third possibility is that the atherosclerotic process would lower blood pressure in elastic arteries either humorally or by compensatory vasodilation. They cautioned that the lack of association between hypertension and atherosclerosis in common carotid arteries does not necessarily mean that

hypertension could not be relevant in the development of atherosclerosis in other arterial segments.

Evidence that human atherosclerosis is a reversible condition has been accumulating for more than half a century (Blankenhorn, 1990). The early clinically controlled trials demonstrated that lesion progression could be reduced, and there is evidence that diet can reduce progression, and that regression is possible with rigid dietary fat restriction or lifestyle intervention (Ornish et al, 1990). In a 1971 study (McNamara et al) of young American soldiers killed in Vietnam, some degree of atherosclerosis was found in 45 percent of young, healthy American males. These data were in contrast with earlier reports (Enos et al, 1953) of young American soldiers killed in Korea in which 77.3 percent of casualties had some atherosclerosis. In both studies, the patients were male combat troops killed in action, with a mean age of 22.1 years. This disparity in the frequency of atherosclerosis is unexplained but indicated a significant decrease in atherosclerosis.

Ornish et al (1990) in a prospective, randomised, controlled trial (The Lifestyle Heart Trial) tried to determine if comprehensive lifestyle change could affect coronary atherosclerosis after one year. The results showed that 82 percent of the patients showed change in the direction of regression of coronary atherosclerosis and that adherence to the lifestyle changes was related to changes in lesions in a `dose-response' manner.

This trial suggested that comprehensive lifestyle changes may begin to reverse coronary atherosclerosis in only one year.

2.3   BIOMEDICAL RISK FACTORS FOR HYPERTENSION

2.3.0   Introduction

It is apparent that although the etiology of hypertension remains unclear that modification of some identified risk factors can have an impact. The causes of the decline in mortality from cardiovascular disease, the major sequela of hypertension, are not yet well understood and adequate data are not yet available to evaluate these possibilities. Some research indicates that reduced mortality has resulted from reduced incidence of disease (Pell & Fayerweather, 1985; Goldberg et al, 1986; Gillum et al, 1982; Goldman et al, 1982; Goldman & Cook, 1984), other research seems to indicate another possible explanation being an improved case-fatality rate (Higgins & Luepker, 1988). Goldman and Cook (1984) estimate that 54 percent of the decline in the rate of death from ischemic heart disease is related to changes in lifestyle and about 40 percent attributable to medical interventions.

2.3.1   Age

Age is generally defined chronologically in epidemiological studies as it is simpler to define than defining according to physiological characteristics. Both systolic and diastolic blood pressures increase with age. There is no exact blood pressure level below which cardiovascular complications are absent and above which they are present or even sharply increased, for any age group (Amery et al, 1991). There has been some argument as to whether systolic blood pressure or diastolic blood pressure is a better predictor of future adverse occurrences in the elderly.

Amery et al (1991) argue for the measurement of systolic rather than diastolic blood pressures. Staessen (1988) concurs that systolic is more useful because it increases with age in both sexes up to the age of 80. The Framingham Heart Health Study (Kannel, 1981) cohorts showed that systolic blood pressure increased in both men and women up to age 76, and diastolic blood pressure increased up to age 50, levelled off between 50 and 60, and decreased between ages 60 to 76. Kannel (1981) also found that 30 percent of women in the Framingham Heart Health Study, who were over 65 years old had isolated systolic hypertension (ISH). Saltzberg et al (1988) found that 26 percent of Caucasian women aged 65-74 years had ISH, and that 39 percent of women 75-79 years had ISH. The Hypertension Detection and Follow-up Program (Curb, 1985) found a

prevalence of ISH in Caucasian women aged 60-69 years, of 7 percent and in Black women, 8 percent.

Lopez (1991) supports the position that elevated systolic hypertension is a better predictor of future adverse occurrences than is elevated diastolic blood pressure and further suggests that reductions can be accomplished safely and effectively preferably through the aggressive use of non-pharmacologic therapy.

There has been some argument as to treatment for hypertension in the elderly as a well-defined therapeutic program for the treatment of hypertension in the elderly has yet to be advanced or evaluated (Roy, 1986). There is a belief by some clinicians that hypertension in this age group is a normal process associated with the loss of vascular elasticity, and that its treatment is not worthwhile, or that the elderly will suffer more side effects of antihypertensive drugs.

Older patients differ from younger ones in that they have reduced: myocardial reserve, cardiac output, baroreceptor sensitivity, aortic elasticity, intravascular volume, and plasma renin activity. Their total peripheral resistance is increased and regional blood flow is correspondingly reduced. If blood pressure is rapidly reduced, the elderly may be susceptible to orthostatic hypotension and impaired perfusion of vital organs. Older patients more often have concomitant medical conditions which may affect the management of their hypertension and

help to specify which antihypertensive drugs could be useful. Although compliance is often assumed to be less frequent in older patients, clinical trials show that the elderly comply at least as often as younger ones (Hully, 1985).

Lopez (1991) suggests that the Framingham Heart Study affirmed beyond any reasonable doubt that hypertension is not a normal consequence of aging. Hypertensive elderly, ages 65-74 years were at two to three times the risk of developing cardiovascular disease and three times the risk of death from cardiovascular disease when compared to normotensive persons. He states that hypertension in the elderly subjects is not benign and therefore non-intervention can expose the elderly to increased likelihood of cardiovascular disease and death. Burris (1991) concurs with Lopez that reduction of elevated blood pressure is beneficial in older persons but that treatment must be careful. The elderly are at higher risk for end-organ complications from hypertension and less likely than younger counterparts to survive complications such as myocardial infarction and stroke (Burris, 1991). The Subcommittee on the Definition and Prevalence of the 1984 Joint National Committee (1985) stated that excessively elevated blood pressure should not be considered a normal consequence of aging and that although the problem of hypertension may be somewhat overstated in the elderly, the risks of ignoring it are the same as in younger patients. The pathophysiology of

hypertension may differ in the elderly patients when compared to younger counterparts, and so may the therapy. The Joint National Committee on Detection, Treatment and Evaluation of High Blood Pressure (1988) states that: patients in the 65-80 year old range with established diastolic or combined systolic/diastolic hypertension should definitely be considered candidates for pharmacologic and non-pharmacologic antihypertensive therapy.

The 1988 Joint National Committee on Treatment, Evaluation and Detection of high blood pressure (1988) documented the effectiveness of nonpharmacological measures such as: sodium reduction, weight reduction in the obese, and avoiding excessive alcohol consumption. Aggressive blood pressure reduction with potent drugs is not recommended. Therapy should begin with a low dose and increased slowly, to avoid excessive reduction in blood pressure and the development of orthostatic hypotension.

The very elderly patient presents some unique concerns. For people over the age of 85 years, the relationship between mortality and blood pressure level is less consistent and may reverse, with those with higher pressures tending to live longer. It may be that mortality has eliminated most long standing hypertensives, or it may be that silent or low grade myocardial ischemia causes a lowering of pressure and that low blood pressure might mean unhealthy hearts and poor prognosis. In analyses

done to date, there are no firm conclusions as to the benefit of antihypertensive drugs (Burch, 1983: Mattila et al, 1988; Amery et al, 1986) but the results of the SYST-EUR (European Working Party on High Blood Pressure in the Elderly, 1989) and the SHEP (Neaton et al, 1993) studies confirm the benefits of treating the elderly.

Guralnik (1989) found that the absence of hypertension in the elderly was one of the most important indicators of healthy aging. He followed a sample of 65-89 year olds from the Alameda County Study, for 19 years and found predictors of high physical functioning were: race (non-Black), higher family income level, absence of hypertension, absence of arthritis, absence of back pain, being a non-smoker, having normal weight, and consuming moderate amounts of alcohol. Pinsky et al (1985) and Benfante (1985) concur with Guralnik (1989) that blood pressure is highly predictive of physical functioning. As the older population increases in size and the elderly survive longer, it becomes increasingly important to retain the elderly at high levels of functioning for as long as possible to avoid becoming dependent on the health care system.

There is presently little known about the rate of progression of atherosclerosis with increasing age, and this may be particularly important in older women because of the apparent substantial increase in blood cholesterol levels among older women (Kuller, 1989). Longitudinal data from the Framingham Study indicates that most cardiovascular risk factors

declined in impact with increasing age (Kannel & Vokonas, 1986), with the exception of systolic blood pressure and the presence of glucose intolerance.

2.3.2 Gender

From 30 years of data provided by the Framingham Heart Study, Lerner and Kannel (1986) were able to find striking differences in the clinical presentation of, and the prognosis for coronary heart disease according to gender. They found that coronary artery disease occurred at a consistently later age among women than men with the mean age being 10 years older for women. For myocardial infarction, women were at average 20 years older. The reasons for this phenomenon are not yet clear.

However, Wenger (1990) states that it is highly likely that these data are the result of mistaken diagnosis. If both genders have the same underlying pathophysiologic problem; coronary atherosclerosis, then it is suspect that their outcomes are so disparate. Wenger suggests that there is a discrepancy between `chest pain' and `angina pectoris' in women and that it was documented with the publication of the Coronary Artery Surgery Study (CASS) Registry (Kennedy et al, 1982). Data from this registry showed that 50 percent of women who were referred for coronary arteriographic evaluation of chest pain were found not to have any arterial

narrowing. It appears likely that there was a substantial prevalence of women without coronary heart disease who were diagnosed as having angina in the Framingham Study.

The consequences of this poor diagnosis have led to the flawed belief that women with angina have a much more favorable prognosis and therefore, require less attention than do men. This leads to less concern with their preventive care and with modifying their coronary risk factors. It is likely that it has further led to inappropriate decisions when identifying high-risk women. Because of this misdiagnosis of chest pain, the opportunity for many women to utilize preventive therapies and earlier and safer myocardial revascularization may have been lost (Wenger, 1990).

In a study conducted by James et al (1988), the differences in factors which affected daily variation in blood pressure were examined. They found that the patterns of pressure variation differed between the sexes. Using non-invasive ambulatory blood pressure monitoring they were able to record the situation of measurement, the emotional state and posture. Women were found to have elevated systolic blood pressure for the emotions of happiness, anger, and anxiety. Diastolic pressure was most elevated during reported anxiety, and significantly higher than average during anger. This differed from men who tended to be most elevated during anger.

One explanation offered is that men and women experience their environments differently, probably as a consequence of differences in socialization. In other words, the difference in reactions to similar situations may be because of what is perceived to be socially acceptable for each gender. Another explanation offered is that the men and women in the study may come from different socioeconomic conditions or occupations and that their daily environments and daily experiences may be significantly different. Another possibility is that factors which were not accounted for such as caffeine, alcohol and smoking may have confounded the results. In summary, the data from this study indicated that the factors which influence blood pressure variation during the day may differ between the genders, and that the degree of variation may also differ.

2.3.3   Obesity

Donahue et al (1990) found that in most population based studies, adiposity and the distribution of fat are major determinants of blood pressure level. This association has long been known, but the mechanisms behind this relationship are still not known. The relationship between adiposity and blood pressure is well documented with obese men (BMI>27.8) and obese women (BMI>27.3) nearly twice as likely as the

non-obese of similar age to be hypertensive. Cross-sectional studies have shown a positive relationship between obesity and blood pressure in children (Voors et al, 1977), adolescents (Orchard et al, 1980), young adults (Higgins et al, 1980) and middle-aged adults (Havlik et al, 1983). Longitudinal studies have shown weight gain to be independently related to increased blood pressure in both men and women (Ashley & Kannel, 1974) and long term weight loss has been effective in maintaining lowered blood pressure in those with high normal blood pressure (The Hypertension Prevention Trial Research Group, 1990).

The co-existence of obesity and glucose intolerance in many of the hypertensive population suggests common underlying mechanisms. Moden et al (1985) suggest that hyperinsulinemia may be the underlying link between obesity, hypertension, and glucose intolerance.

It is not clear why insulin-resistance occurs in obesity and there are no prospective data indicating that hyperinsulinemia precedes the development of hypertension in otherwise healthy adults (Donahue et al, 1990). It is possible that a genetic predisposition to both conditions exists, but this has not been proven by studies (Ching & Beevers, 1991). It is possible that obese individuals have increased levels of free fatty acids which impair insulin binding and action. Male-type abdominal obesity has been associated with more marked insulin resistance than those with the female-type of gluteo-femoral obesity, with abdominal obesity being a risk

factor for cardiovascular disease and diabetes in both men and women (Lapidus et al, 1984; Larsson et al, 1984).

2.3.4 Height

Few reports from the major epidemiological studies have addressed height as a possible risk indicator for heart disease in women and no studies were found which addressed height as a risk indicator for hypertension.

2.3.5 Cholesterol Levels

Recent studies indicate that cholesterol lowering is associated with a reduced risk of CHD. The Lipid Research Clinics Coronary Primary Prevention Trial (LRCCPPT) (1984) found that for every one percent reduction in total serum cholesterol, CHD risk was reduced by 2 percent. Total or overall mortality was not conclusively demonstrated to show any benefit from reduced cholesterol levels. The National Heart, Lung and Blood Institute Consensus Conference (1985) concluded that elevated blood cholesterol was a major cause of CHD and that reducing elevated blood cholesterol (specifically LDL) could reduce the risk of myocardial infarctions due to CHD. They recommended a cholesterol-lowering diet

for all adults and children over the age of two years, and treatment for blood cholesterol levels in the upper 25 percent of the population distribution.

Because there is a consistent positive relationship between blood pressure and cholesterol level in populations, it is suggested that there is a biological interrelation between these two major risk factors for CHD (Grimm & Hunninghake, 1986). In the Tromso study (Bonaa & Thelle, 1991), the relationship between the two is graded and continuous throughout the usual range of blood pressure. This study consisted of a large population of men and women with a broad age range and this allowed a detailed analysis of the association between total cholesterol level and blood pressure. The population also had a high prevalence of elevated cholesterol levels and elevated blood pressure levels, which may have made it easier to detect biological interactions between risk factors.

Bonaa and Thelle (1991) assessed the relationships of total cholesterol, high density lipoprotein (HDL) cholesterol, non-HDL cholesterol (total minus HDL cholesterol) and triglyceride levels, with blood pressure. They found that total cholesterol and non-HDL cholesterol levels increased significantly with increasing systolic or diastolic blood pressure in both males and females and in women the association between blood pressure and total cholesterol increased with age. They found that smoking, physical activity and alcohol consumption had little influence on

the association. In subgroups with high alcohol consumption, HDL cholesterol levels correlated positively with blood pressure. Their conclusions support the hypothesis that there are biological interrelations between blood pressure and blood lipids and that these interrelations may influence the mechanisms that associate blood pressure with risk of CHD (Bonaa & Thelle, 1991).

This relationship has been found to be inconsistent in other studies but this may be due to the age and sex distribution. In some Eastern countries where there are low cholesterol levels there is a low incidence of CHD despite the high prevalence of hypertension. This suggests that hypertension may need a metabolic basis of a certain lipid level to become an atherogenic factor. In some countries this metabolic basis is endemic and therefore hypertension is defined as an important risk factor (Leren, 1990).

When selecting antihypertensive therapy, not only elevated blood pressure, but also lipid abnormalities which are often present in hypertensive patients should be considered. In hypertensive patients with moderate and high risk cholesterol levels or elevated triglyceride levels, intervention for lowering lipid levels and for maintaining ideal body weight should be vigorously pursued.

2.3.6   **Family History**

Watt et al (1990) measured the blood pressures of offspring (aged 16-24 years) of patients who had participated in the Medical Research Council Mild Hypertension Trial. Of those offspring with a family history of hypertension, described as having at least one parent in the top 10 percent of the distribution, only 29 percent had a blood pressure score in the top 20 percent of the young population and only 38 percent had a blood pressure score in the top 20 percent when both parents were in the top 20 percent. The study concluded that parental blood pressure data has only limited value in predicting high blood pressure in young people. The data did not provide a scientific basis for a high risk strategy of prevention.

A family history of hypertension has often been referred to as an indicator of risk for developing high blood pressure (Watt, 1986). This assumption has evolved from cross-sectional epidemiological studies which show familial aggregation of blood pressure (Miall & Oldham, 1986).

2.3.7   Diabetes

Non-insulin-dependent diabetes mellitus is associated with an insulin-resistant state and with hyperinsulinemia. It is possible that those diabetics on insulin could also have intermittent hyperinsulinemia because of the pulsatile nature of the insulin therapy. It is further suggested that this would explain why there is no convincing evidence of a reduction in cardiovascular mortality in diabetes treated with insulin (Ching & Beevers, 1991).

About one half of all patients with non-insulin-dependent diabetes mellitus are also hypertensive and these patients tend to have a poor prognosis.

2.3.8   Hyperinsulinemia

Insulin is the most important anabolic and anti-catabolic hormone in the body affecting many membrane functions, and influencing the electrolyte balance across cell membranes. Insulin binds onto target tissues in order to be effective and when there is reduced tissue sensitivity to insulin, this is known as insulin resistance. In an insulin-resistant state, plasma glucose is not effectively removed from circulation which causes

a further release of insulin and results in hyperinsulinemia (Ching & Beevers, 1991).

Since 1966 (Welborne et al), the association between hyperinsulinemia and hypertension has been shown. Ferrannini et al (1987) concluded that hypertension is an insulin-resistant state in itself. The mechanisms by which insulin resistance leads to the development of hypertension is uncertain. Within the same individual, hypertension, obesity, and non-insulin dependent diabetes mellitus commonly occur. Insulin-resistance occurs in all of these conditions but it is not clear whether it is the result of environmental factors or of a genetic predisposition. This issue is important because of its possible reversal by dietary means or antihypertensive drugs.

Impaired lipid metabolism which is manifested by hypertriglyceridemia, hypercholesterolemia and low serum high density lipoprotein (HDL) cholesterol levels, is also found in an insulin-resistant state and could predispose to atheroma formation. Or it could be that insulin stimulates directly vascular smooth muscle cell proliferation and plaque formation in arteries. There are many metabolic changes associated with conditions which are associated with insulin-resistance such as hypertension and it is therefore not surprising that they in turn should have an increased risk of cardiovascular events. Although, the mechanisms of the association between these conditions are still uncertain,

it is possible that insulin resistance is a common factor in hypertension and obesity as well as diabetes mellitus (Ching & Beevers, 1991).

2.3.9   Intracellular Sodium and Intracellular Calcium

Epidemiological research, clinical trials and studies of salt-sensitive laboratory animals have all supported the central role of sodium in the pathogenesis of hypertension (Ching & Beevers, 1991). However, the mechanism by which sodium causes hypertension is not certain. Hypotheses that raised cell sodium levels in blood cells postulates the existence of a hormone, or genetic disposition are two of the possible explanations. It is likely that many differing mechanisms maybe required to cause hypertension making it multifactoral, rather than the result of a single cause.

Ching and Beevers (1991) suggest that it is possible that raised intracellular sodium concentration causes hypertension by leading permissively to a rise in intracellular calcium levels, which in turn could lead to increased contractility of smooth muscle cells. Although it is clear that both intracellular sodium levels and intracellular calcium levels have something to do with hypertension it is difficult to see how they are directly related to a rise in blood pressure.

2.3.12   Combined Biomedical Risk Factors

Ferrannini and Natali (1991) report that hypertensive individuals appear to differ from normotensive counterparts in more ways than just blood pressure. They tend to be more obese, less tolerant of glucose, have a higher prevalence of diabetes and cardiac hypertrophy, have higher circulating levels of cholesterol (Total and LDL), triglycerides, uric acid, insulin, and plasminogen activator inhibitor (PAI-1). Each of these has been shown to be an independent risk factor for atherosclerotic vascular disease. It is therefore logical that essential hypertension seems to be a multifaceted disease and that therapeutic intervention which is directed at a single aspect, even if it is successful, does not necessarily improve (and may worsen) the overall risk for atherosclerotic vascular disease. Hypertension is seldom an isolated condition, and is usually (about 80 percent of the time) associated with other metabolic irregularities, with hyperinsulinemia being an inherent feature of hypertension (Ferrannini & Natali, 1991).

Evidence from epidemiological surveys suggests that age, gender, amount and distribution of body fat, glucose tolerance, insulin sensitivity, blood pressure, and lipid metabolism constitute a network of interrelated functions. The question: `why do all these conditions tend to cosegregate'

is not yet sure, but it appears that one common factor may be hyperinsulinemia which is found in hypertension and also in obesity, glucose intolerance, and milder forms of diabetes and hypertriglycerides (Ferrannini & Natali, 1991). It may be that hyperinsulinemia/insulin resistance is a marker of disease. For instance, diabetes, hypertension, obesity and dyslipidemia have a genetic background although the exact mode of transmission is not known. Furthermore, in populations with a high incidence of diabetes, fasting hyperinsulinemia is a strong predictor of subsequent diabetes development. It is believed that insulin sensitivity is at least partly genetically controlled, and that individuals who are nondiabetic but have a positive family history of diabetes tend to show higher fasting plasma insulin levels and a higher incidence of hypertension and dyslipidemia (Haffner et al, 1988; Haffner et al, 1989). Ferrannini and Natali (1991) suggest that genes may code for different diseases and that "these genes are distributed in close and interrelated loci together with the genes of insulin resistance (linkage disequilibrium)" (1991:1277).

The alternative view that hyperinsulinemia/insulin resistance is a mechanism in the pathogenesis of one or more other conditions is supported by the following evidence. Insulin resistance appears to worsen glucose tolerance. It also appears to stimulate the hepatic production of very-low-density lipoproteins. Furthermore, it remains present in subjects which were formerly obese, but have regained normal body weight. The

hyperinsulinemia could induce blood pressure to rise through different mechanisms such as: sodium-water retention, sympathetic nerve stimulation, changes in transmembrane ion traffic, and direct stimulation of smooth muscle cell growth (Ferrannini & Natali, 1991).

In terms of primary prevention, a better understanding of the interaction between environmental factors and genetic pressure in the natural history of atherosclerotic disease and in the selection of high risk patients, would be helpful. In the meantime, any interventions should take into account the entire hemodynamic-metabolic profile (Ferrannini & Natali, 1991). In summary, despite the voluminous research done on the biomedical risk factors for hypertension, the mechanisms of how they lead to hypertension are still unclear, and the pathophysiology is still unknown.

2.4 NON-BIOMEDICAL RISK FACTORS FOR HYPERTENSION

2.4.0 The Social Class Gradient

Despite a great deal of accumulated evidence on the relationships between risk factors and disease, most disease, mortality and morbidity rates follow a social class gradient with lower income and/or lower social status being associated with higher rates of disease. The reasons for this relationship may be due in part to a greater prevalence of risk factors such as obesity and smoking in the lower socioeconomic stratum of society, or

to unexplored environmental factors common to lower stratas (Reeder et al, 1991).

This relationship between income and/or social status and disease is often misinterpreted as being due to the lack of the basic necessities in life. However, it is apparent it does *not* indicate deprivation at the lower end of the scale for the following reasons. First is the fact that the socioeconomic gradient in health status has been relatively stable over time (Townshend, 1982). This despite the marked rise in average income levels in all developed societies. The proportion of persons who are deprived of the necessities of life in a biological sense has clearly declined. But even more important, the relationship between mortality and/or morbidity and disease is a *gradient* not a step function. In other words, the top people appear to be healthier than those on the second rung, even though the latter are above the population averages for income, status or whatever the critical factors are (Marmot, 1986).

Therefore, we can not assume that the determinants of health that lie outside the narrow boundaries of the health care system, are just a problem of " some poor deprived minority whose situation can be deplored and ignored by the rest of us... we are all (or most of us) affected" (Evans, 1990:1355). The effects of these factors or determinants of health which are outside of the health care system, may be quantitatively very significant for the overall health status of modern

populations. These factors and the issues they involve are not trivial, second or third order effects.

Education, income and occupation are all highly interrelated but there is some debate as to which is the most reliable indicator of socioeconomic status. Miller and Wigle (1986) cite education, European research cites occupational status (WHO/ERO, 1984) and most Canadian and American research cites income levels as most reliable indicators (Labonte, 1990). Wilkinson (1986) states that all three can be considered proxy measures of social class. Much data are available on the relationship between health status and various socioeconomic indicators but most of the data to date have been correlational. Causal inferences have been difficult to make (Labonte, 1990).

A comprehensive review of the health status of Canadians by socioeconomic indicators (income, gender, geographic location) was done by Wilkins and Adams (1983). They concluded there was a 7.7 year quality of life differential between the top and bottom income quintiles. The 1981 Canada Health Survey also found differentials in the prevalence of morbidity, including hypertension and heart disease, with the lowest quintile persons almost twice as often affected as the highest quintile persons (Canada Health Survey, 1981). The Canada Health Promotion Survey (1985) found gradients in health status according to level of education. Those with elementary or less education were less likely to

report themselves as having excellent health when compared to those with university education. A similar gradient appeared with income, with only 17 percent of the poorest reporting themselves in excellent health compared to 30 percent of the wealthiest (Wilkins, 1988).

Miller and Wigle (1986) compared prevalence rates of several cardiovascular risk factors among persons of different educational levels. Among women 20-69 years, the largest relative differences were found in the prevalence rates for smoking, overweight, obesity, elevated diastolic blood pressure and physical activity, with the lowest education group having the highest prevalence of each of these risk factors.

Income is one of the major 'markers' of socially-related health inequalities. It is a flawed measure because it does not account for wealth or for non-income statutory services. It does not include many non-income forms of wealth such as: real estate, dividends, capital holdings and other assets. Income measures a person's capacity to purchase such prerequisites to health as food, shelter, clothing, education and primary health care. Third world countries often depend on (non-income) forms of agriculture, and several first world nations have complex (non-income) provision of health prerequisites such as education, housing and health care. This makes cross-national comparisons difficult and intranational comparisons problematic (Labonte, 1990).

Social equality (decreased income inequalities, improved women's status, lower birth rates, etc.) appears to be linked to improved health status (Labonte, 1990). Wilkinson (1986) found that nations having the greatest after-tax income equality have the best indicators of health, in terms of lifespan and infant survival rates. Japan has emerged as the health leader in lifespan and infant mortality, and its income equality is among the highest in the developed world. Ceresto and Waitzkin (1986) found that after controlling for per capita income and degree of economic development, `socialist' third world countries scored significantly healthier on most physical quality of life variables when compared to `capitalist' third world countries.

Differentials in heart disease morbidity and mortality between socioeconomic classes exist in data from several other countries (Marmot and McDowell, 1986; Kraus et al, 1980; WHO European Regional Office, 1984; Wilkinson, 1986). A British study (Marmot & Theorell, 1988) examining the link between social class and heart disease, concluded that the organization of work, specifically the freedom to make decisions, impacted on health. From their own research as well as a review of international research they concluded that: "above a threshold of poverty, position on the social hierarchy per se may be a more important determinant of health and disease than material conditions" (1988:674). This study also noted that blood pressure rates were similar at work, but

then fell much more for the upper grade men when at home than for the lower grade men when they were at home.

Other factors in the organization of work have been found to impact on health. These include workplace social supports, as measured by the number and quality of interactions with co-workers, and the stress related factors such as the pace of work, the frequency of deadlines and reporting requirements (Ham, 1990). Johnson and Hall (1988) found cardiovascular disease occurred most often among those with high demand jobs, and a low level of both work control and social supports.

Jones and Cameron (1984) argue the importance of defining "social class" theoretically before attempting to develop social class indicators. The demographic variables such as education, income, and occupation do not reflect the psychosocial dynamics involved in being part of a social class which may in turn affect health behaviors and health status. Psychosocial dynamics associated with an increased risk of cardiovascular disease include affect (happiness, social stressors, anxiety, suppressed hostility and anger, depression and, within the workplace, lack of control, role ambiguity, isolation, underwork/overwork and shift work). These dynamics, in turn, are more prevalent among lower-incomed, less-educated and lower occupation-status persons (Labonte, 1990). Marmot and Scott-Samual (1989) argue for a focus on "social epidemiology" with social

conditions and class structures being the focus rather than disease categories.

There is another interesting, but as yet unexplained quality to the gradient relationship of mortality and morbidity across socioeconomic classes. This relationship has appeared relatively stable over long periods of time, despite the fact that the principle causes of death have changed considerably over time. This implies that these underlying factors or determinants of health, outside of the health care system, influence susceptibility to a whole range of disease, not just a certain disease. Whatever the disease outcome, people in the lower socioeconomic groups tend to get it more often and tend to die earlier. The understanding of the relationship between socioeconomic position and factors outside the health care system will require investigations at a more general level as well as the etiology of specific diseases. It also emphasizes that disease-specific policy responses may not be effective because even if one disease is "cured", another will take its place (Evans & Stoddart, 1990).

2.4.1   Physical Activity

Epidemiological evidence acknowledges that physical activity counters tendencies toward CHD (Paffenbarger, 1990). In the MRFIT study (Leon et al, 1987) of men who were considered to be at relatively high risk of developing CHD, there were three levels of daily physical activity and the most active third had a relative risk of CHD 20 percent below that of the least active level. As a predictor of CHD, physical activity was independent of other predictors. It was determined that levels of CHD risk were related inversely to fitness status, as well as physical activity status and perhaps this was because the CHD benefits of exercise may be mediated in part by its influence on cardiovascular fitness.

Paffenbarger et al (1984) found that in a 10 year study of Harvard alumni (all men) that the relative risk of CHD for the more active group was about 40 percent below that of the less active group. Lie et al (1987) in a study of Norwegian men found that the more fit men had lower blood pressure, lower heart rates, lower serum lipids, higher maximal heart rates and maximal blood pressures during exercise, and more favorable spirometry findings, than had those less fit. Physical fitness was a strong inverse predictor of fatal CHD with the risk of the fit group being one fifth of the least fit group.

Slattery and Jacobs (1988) in a study of U.S. railway workers during a 20-year time period, found exercise heart rates were directly predictive of CHD death rates. This study had a broad range of ages and a lengthy follow-up. As with the MRFIT study, there was a high correlation between physical activity levels and ergometric fitness results, but as with all of these studies the subjects included men only (Slattery et al, 1989).

Ekelund et al (1988) found in the Lipid Research Clinics study, that during the 8.5 year follow-up the CHD mortality in the least fit quartile was 6.5 times that of the most fit quartile. Blair et al (1989) followed middle-aged men for 8 years and found the most fit quintile were 70 percent lower in their all-cause mortality than were the least fit quintile. The differences were present but less dramatic among women.

Paffenbarger et al (1986) were able to show a gradient effect of exercise levels on CHD mortality, as exercise levels increased from below 500 kcal/week to an optimum of 3,500 kcal/week. They estimated years of life to be gained by having favorable lifestyles. They estimated the largest gain was 2.7 years from avoiding hypertension, 2.3 years from not smoking, and 1.3 years by being physically active by expending at least > 2000 kcal/week.

2.4.2   Cigarette Smoking

Although it has been clear for many years that cigarette smoking is associated with increased risk of coronary heart disease in men, evidence of the association between smoking and coronary heart disease in women was not available. Now there are several studies, both case-control and prospective cohort studies which show positive relations between smoking and myocardial infarction, and smoking and fatal coronary disease (Willett et al, 1987). Smoking also has been associated with reduced blood levels of high-density lipoproteins (Pooling Project, 1978).

The Nurses Health Study (Willett et al, 1987) was a large prospective cohort study of almost 120,000 nurses who were 30 to 55 years in 1976 who were followed for 6 years. Willett et al (1987) found that the number of cigarettes smoked per day was positively associated with the risk of fatal coronary heart disease, non-fatal myocardial infarction, and angina pectoris in women. Approximately 30 percent of the women were smokers, which is consistent with current percentage in populations of similar ages. They found that there were no safe levels of smoking even women smoking one to four cigarettes per day were at 2.5 times the risk of both fatal coronary heart disease and non fatal myocardial infarction. Those who smoked >=45 cigarettes per day were at 11 times the risk.

However, the Nurses Health Study found that the relative risks of coronary heart disease associated with cigarette smoking were slightly lower in women with hypertension. Rosenberg et al (1985) found similarly reduced relative risks among women with hypertension in a case-controlled study. Willett et al (1987) also found that the attributable risks for coronary heart disease among women who smoked, were elevated in the presence of other risk factors. Hennekens et al (1984) found that cessation of cigarette smoking resulted in approximately 50 percent decrease in coronary heart disease, and sometimes this decrease happened within a matter of months.

Kannel and Higgins (1990) in examining data from the Framingham Study found an incremental increase of 31 percent in cardiovascular mortality for women for each 10 cigarettes smoked per day. Smoking increased cardiovascular risk at any level of blood pressure. The data show that cigarette smoking affects the risk of coronary heart disease whether hypertension is being treated or not. The data from the Framingham Study show that the risk of coronary heart disease is cut in half with cessation of smoking, regardless of the duration of smoking. They suggest that hypertensives who smoke a pack a day and quit smoking can reduce their risk of coronary heart disease by 35-40 percent by not smoking. Therefore, smoking cessation could be considered as a substitute for therapy because cessation offers greater benefits than any

known therapy for avoiding cardiovascular sequelae. Peripheral arterial disease risk is increased threefold by smoking. In the Framingham Study, the risk of cardiovascular death for former smokers who smoked 20 cigarettes per day for 30 years was reduced within one year, to the same risk as those who had never smoked. Other risk factors accelerate atherosclerosis but smoking does not appear to.

Tuomilehto et al (1991) found that 28 percent of all strokes (fatal and non-fatal) could be attributed to hypertension and 17 percent to smoking. He found that the effect of smoking and hypertension together is multiplicative. He also found that 18 percent of all stroke events in women could be attributed to hypertension and 4 percent attributed to smoking. Both hypertension and smoking were of greater risk for men, with smoking rates for women in his study being very low (between 10 percent and 15 percent). Epidemiological evidence suggests that the relationship between smoking and stroke may be causal because the risk increases for smokers versus non-smokers independently of other risk factors, there is a dose-response relationship, and risk decreases when smoking is stopped (Tuomilehto et al, 1991).

Regardless of other factors, smoking increases the risk of hypertension (Kannel & Higgins, 1990) and therefore, hypertensive smokers should be encouraged to quit smoking. The prevalence of smoking among hypertensive women in the Framingham Study was about

31 percent. Although there are no controlled clinical trials on the advantages of quitting smoking, the epidemiological data suggest a strong benefit. Sometimes when smoking is stopped, blood pressure shows a slight increase but this is likely due to weight gain.

Cigarette smoking causes a rise in catecholamines, lowers the ventricular fibrillation threshold, makes platelets more adhesive and reduces the oxygen-carrying capacity of blood (Kannel & Higgins, 1990). All of these events put the hypertensive person at a disadvantage. To further aggravate these effects, high-density lipoproteins are reduced which changes the cholesterol ratios. In the Medical Research Trials (MRC Working Party, 1985) smoking status had an adverse effect on treatment outcome.

In terms of prevention and making policy decisions the priority in treating hypertensive patients should be in encouraging smoking cessation. Smoking cessation offers greater benefit than any therapy in reducing the risk of myocardial infarction, sudden death, peripheral vascular disease and stroke (Kannel & Higgins, 1990). This can lead to prompt and major reductions in cardiovascular risk without the side effects which come with many antihypertensive therapies. Furthermore, even if antihypertensive drugs are used, their effectiveness can be greatly improved if the person also stops smoking. Therefore, smoking cessation is recommended for the general population, and especially for the hypertensive patients.

2.4.3   Alcohol Consumption

The first noted reference to the relationship between alcohol consumption and blood pressure was made by Lian in 1915 when he reported that French servicemen who drank 2.5 liters of wine or more per day had an increased prevalence of hypertension (Keil, 1990). MacMahon (1987) reports that most studies done to date have reported a positive association between alcohol consumption and blood pressure.

The association between alcohol and blood pressure has been investigated in at least 32 cross-sectional studies, 10 in Europe, 12 in North America, 6 in Australia, 2 in New Zealand, and 2 in Japan (Keil, 1990). Of the 10 European studies all found an association between alcohol and blood pressure independent of other confounding factors. The Munich Blood Pressure Study (Cairns et al, 1984) and the Lubeck Blood Pressure Study (Keil et al, 1989) found the blood pressures of non-drinkers were greater than or equal to those of subjects who consumed 10 to 20 g. of alcohol per day. The Lubeck Blood Pressure Study found that drinkers who consumed >=40 g of alcohol per day had higher blood pressures than non-drinkers. The Munich Blood Pressure study found that male drinkers who consumed >=60 g of alcohol per day, and female drinkers who consumed >=40 g of alcohol per day, had higher blood pressures than non-drinkers. The MONICA Augsburg Survey (submitted as referenced

in Keil, 1990) confirmed the results of the Munich study. These three studies all found that smoking can act as a modifier on the alcohol-blood pressure relationship but a physiological interpretation of this interaction is not yet available.

Of the 12 North American studies, all except the Canada Health Study (Coates et al, 1985) reported a statistically significant positive association between alcohol and blood pressure. The Kaiser Permanente Study (Klatsky et al, 1986) found only small differences between non-drinking men and men consuming 10 to 20 g of alcohol per day. Non-drinking women had higher blood pressures than women consuming 10 to 20 g of alcohol per day thus finding a J-shaped relationship in women. This study suggested that for men and women and all racial groups there may be a threshold of 30 g of alcohol per day for blood pressure elevation. This finding was confirmed in the Lipid Research Clinics Prevalence Study (Criqui et al, 1981) and the Albany Study (Gordon et al, 1986).

The six Australian studies and the two New Zealand studies found linear, J-shaped, or U-shaped associations between alcohol and blood pressure. The National Heart Foundation of Australia Risk Factor Prevalence Study (MacMahon et al, 1984) and the Auckland Study (Jackson et al, 1985) found that drinkers consuming >=30 g of alcohol per day had higher blood pressure levels than non-drinkers.

As well, at least six prospective cohort studies have been done. All found findings consistent with the cross-sectional studies indicating a positive association between alcohol and blood pressure, with the exception of the Honolulu Heart Study (Reed et al, 1982). However, there are still unanswered questions concerning alcohol and blood pressure relationships, including threshold level and shape of the association. Keil (1990) suggests there is a causal relationship between blood pressure and consumption of >30-60 g of alcohol per day. He justifies this view by showing that chance, bias and confounding can not explain the alcohol-blood pressure association. Furthermore, misclassification of the non-drinker group has likely weakened the association. There have been consistent results across cross-sectional, prospective cohort and experimental studies, and the time sequence between cause and effect has been shown. Biological plausibility will require further research.

In measuring alcohol, the exact amount of alcohol consumed over a period of time is hard to obtain in population studies. It is impractical to obtain the blood-alcohol level (which is considered the gold standard) and therefore, most epidemiological studies rely on self-reports. Several studies on self-report measures have reported that heavy drinkers tend to underreport or even deny alcohol consumption. Underreporting in epidemiological studies may also cause the threshold dose for alcohol

consumption to be too low (MacMahon, 1987; Colsher & Wallace, 1989; Chick, 1982).

Secondly, the group which drinks no alcohol at all needs to be further defined. It consists of lifelong abstainers, former drinkers, drinkers who deny they drink, and those who are too sick to drink (Keil, 1990:44). The association between alcohol consumption and blood pressure may be altered if these groups are separated. This lack of separation may lead to an underestimate of the relationship between alcohol and blood pressure and perhaps dispel the finding that non-drinkers have greater risk than low and moderate drinkers.

The physiological mechanisms of how long-term regular alcohol consumption leads to elevated blood pressure is not yet clear, Maheswaran et al (1986) suggest: "the most attractive theory on present evidence to explain the mechanism of alcohol-induced hypertension is that of a direct effect of alcohol on vascular smooth muscle perhaps mediated by calcium influx" (as quoted in Keil, 1990). Based on the information presently available, Keil concludes "It is conceivable that a major improvement in the assessment of the exposure variable alcohol will contribute to a stronger alcohol-blood pressure and alcohol-coronary heart disease association and will transform the frequently found J-shaped curves to a more linear relationship with a threshold dose of approximately 30 to 60 g of alcohol per day for hypertension and a possibly higher threshold for coronary

heart disease" (Keil, 1990:50). Ching and Beevers (1991) state that it is hard to deny a close direct association between a consumption of 3-4 units of alcohol per day and blood pressure. The mechanisms are uncertain but it appears that the pressor effects of alcohol are very reversible. Both epidemiological and clinical studies indicate that reducing alcohol intake for as little as a few days is associated with a rapid fall in blood pressure (Potter & Beevers, 1984; Puddey et al, 1987; Ueshima et al, 1987).

2.4.4 Stress

To date there have been few prospective epidemiologic studies conducted which use psychosocial measures such as stress to predict the prevalence of hypertension. Selye's (1956) initial characterization of the stress response is now known to involve the secretion of about a dozen hormones and the suppression of various others. Studies also indicate that chronic activation of the stress response can impair health, with some people appearing to be more susceptible to stress-related disorders than others, although it is not understood why.

The work being done by Sapolsky (1990) suggests that peoples' psychological and social characteristics (for example, their emotional makeup, personality and position in society) can profoundly influence their physiological response to stress. During the stress response, glucose which

provides the body's main source of energy is mobilized from storage sites. Blood carrying glucose and oxygen is diverted from organs such as the skin and intestines which are not used in physical exertion, and delivered directly to organs which are crucial - the heart, the skeletal muscles and the brain. To create this shift in blood flow some blood vessels constrict, others dilate, and the heart rate increases. Cognition sharpens, and the perception of pain in blunted. Physiological activities which are not immediately needed are inhibited including: growth, reproduction, inflammation and digestion. By chronically activating the stress response, glucose is constantly mobilized rather than being stored. This causes healthy tissues to atrophy and allows fatigue to set in. Given enough time, cardiovascular changes promote hypertension, in turn causing damage to the heart, the blood vessels and the kidneys (Sapolsky, 1990).

These physiological events which translate personality traits into diseases have taken years to develop but are nearly impossible to trace in human beings because of their complex emotional lives, and the fact that people cannot be caged in laboratories for controlled, long-term study. Sapolsky's findings are consistent with advice of stress management advocates that is, being able to predict and control the outcome of social interactions and finding ways of blunting the long-term effects of stress is beneficial. He found the number of social stressors to which a person is subjected is less important than the emotional style with which one

perceives and copes with the stressors. One should assess patients' control and predictability in difficult circumstances.

The Israeli ischemic heart disease study (Kahn et al, 1972) identified a cohort of 3829 men with initial blood pressure of <140/90 mm Hg. Five years later those with blood pressures of >165/95 mm Hg were identified. It was found they were more likely to be men who suppressed their feelings when faced with interpersonal conflict.

Murphy (1991) did a retrospective study which identified job dimensions which were associated with cardiovascular disability. From these job dimensions he identified occupations which had high scores on these job dimensions, indicating they were more likely to be associated with higher rates of cardiovascular disability. Some of the occupations he identified were: transportation jobs such as air traffic controllers; airline pilots and attendants; bus drivers; locomotive engineers; truck drivers; preschool, adult education, and physical education teachers; physicians; factory workers; and craftsmen/foremen such as machinists, carpenters, and foremen. Most of the job dimensions associated with high rates of cardiovascular disability were also associated with non cardiovascular disability. Only airline attendants, physical education teachers and preschool teachers are predominantly female occupations. These results are consistent with other work done in which work factors associated with the pathogenic process link stress to disease outcomes (Kasl, 1978).

In intervention studies done by Patel et al (1981, 1985) stress management has been used to reduce blood pressure and has had encouraging results. However, other studies such as Van Montfrancs et al (1990) have had negative results. Ching and Beevers (1991) suggest that perhaps stress management is effective in reducing blood pressure for office visits, but not for home blood pressure readings.

2.4.5 Dietary Salt

Epidemiological studies have often compared high and low salt-consuming societies, but have not taken into account the confounding effects of age, body mass index (BMI), alcohol intake, and other known confounding variables. The INTERSALT Project (Intersalt Cooperative Research Group, 1988) was a major international epidemiological comparison of 52 populations in 32 countries to assess dietary salt intake. It found a weakly significant correlation between median sodium excretion and systolic blood pressure. Thus the INTERSALT Project lends support for the concept that salt intake is related to blood pressure, but the association is weaker than the relation between body-mass index and blood pressure (Dyer & Elliot, 1989), or between alcohol intake and blood pressure (Rose & Stamler, 1989). The INTERSALT Project supported the hypothesis that salt intake plays a role in the development of hypertension.

It showed a highly significant correlation between the median salt intake and the slope of the regression line between pressure rise and age. Epidemiologists have noted that hypertension is usually only seen in populations that show a close association between a rise in blood pressure and advancing age. In populations where blood pressure does not rise with age, hypertension is not seen.

At the community level, two studies in which the populations were subjected to intensive advice on dietary salt intake were then compared to a control population. The Belgian study (Staesson et al, 1988) was a longitudinal study which found a statistically significant reduction in sodium excretion but no significant reduction in blood pressure. The second longitudinal study was done in Portugal (Forte et al, 1989) and found that in the population which was given advice, there were significant reductions in both mean systolic and mean diastolic blood pressures. Portugal is a country where salt intake is notoriously high and this study suggested that reduced salt intake can reduce blood pressure in non-hypertensives as well as hypertensives.

In a clinical study of hypertensive outpatients, MacGregor et al (1982) reported that by reducing sodium excretion, blood pressure could be reduced and then more recently reported (MacGregor et al, 1989) that the degree of salt depletion has a 'dose-response curve' relationship to reduced blood pressure. However, a study by Watt et al (1983) in South

Wales found no reduction in blood pressure with a reduction in salt. In a recent study by Parker et al (1990), the effects of alcohol reduction, salt restriction, and a combination of both were compared. They found that alcohol reduction both alone and combined with salt restriction resulted in reduced blood pressure, but salt restriction alone had no significant effect on blood pressure.

In conclusion, there appears to be no major disadvantage to salt reduction as a method of reducing blood pressure; however, studies have shown an increase in blood pressure as a result of salt depletion. It may be that hypertensives are more salt sensitive than normotensives, or it may be that the achieved salt intake in some studies, is not significantly different than what is presently being consumed by healthy people (Ching & Beevers, 1991).

2.4.6 Calcium

The relationship between calcium intake and blood pressure is controversial and requires further trials. The NHANES Study (Harlan et al, 1984) found that calcium intake was negatively associated with blood pressure in the populations studied. This prompted McCarron et al (1985) to examine whether calcium supplementation could be used as a non pharmacological method of reducing blood pressure. At this point in time

there is not enough evidence to encourage calcium intake or supplementation.

2.4.7 Potassium

The INTERSALT Project (INTERSALT Cooperative Research Group, 1988) was not able to confirm that low potassium intake was related to raised blood pressure. Some clinical trials (Khaw & Thom, 1982; MacGregor et al, 1982; Patki et al, 1990) which used potassium supplementation to manage mild hypertension, were able to demonstrate a significant reduction in blood pressure. Although the use of potassium supplementation is controversial, it is still wise for hypertensives and non-hypertensives to increase their intake of potassium rich foods such as fruits and vegetables.

2.4.8 Anger and Anxiety

There have been few prospective epidemiological studies using psychosocial measures to predict the prevalence of hypertension. Markovitz et al (1991) in a three year prospective study of middle-aged women examined the hypothesis that feelings of anger and anxiety increase the risk for essential hypertension. Both independently predicted

an increase in systolic blood pressure (p<0.01) offering support to the hypothesis that increased feelings of anger and anxiety can lead to an increase in blood pressure over time. The results could have been affected by the small number of hypertensive cases but this limitation aside anxiety seemed more likely to lead to hypertension than did anger and anger expression. The advantages of this study were that it was prospective, and that the psychosocial factors were assessed as independent predictors of blood pressure change while biological and genetic variables were controlled for. Thus methodological problems did not confound the conclusions. This study also found that menopause did not contribute to blood pressure change, a finding consistent with a similar longitudinal study by Beresteyn et al (1989).

James et al (1986) in a study of men and women with borderline hypertension found that emotional arousal significantly increased systolic and diastolic pressure (p<0.00001) independently of posture or location of subject during measurement. They found blood pressures which were taken during reported angry or anxious states were higher than those blood pressures taken during a happy state (p<0.01). Furthermore, happiness scores were inversely related to systolic pressure (p<0.01) and anxiety was, positively associated with diastolic blood pressure (p<0.02). Of the variables measured, which included; position, situation, emotional state, the interaction of position and situation, and the interaction of all

three, emotional state was the most statistically significant source of variation in blood pressure!

In another study done on borderline hypertensives all of whom were undergraduate students (3 females, 30 males), Schneider et al (1986) divided them into two groups: one group who maintained high blood pressure outside the clinic, and a second group whose average blood pressure returned to normal at home. The first group who maintained their high blood pressures reported a greater intensity of anger although they suppressed their anger to a greater extent. The two groups did not show differences in their anxiety or in their blood pressure variability.

Cottington et al (1986) in a study of male blue-collar workers, examined the modifying effects of suppressed anger on the job stress-hypertension association. The prevalence of hypertension was greater for men who suppressed their anger and who reported more job stress related to inadequate opportunities for promotion, security, and satisfying co-worker relationships. This study was limited by its design as it was a cross-sectional study which did not follow the variables over time.

2.5   MANAGEMENT OF HYPERTENSION

2.5.0   Measurement of Hypertension

The standard procedure for measuring blood pressure consists of a static blood pressure measurement in a clinical setting. There are several issues in the measurement of blood pressure which are cause for concern because it is now becoming apparent that blood pressures vary extensively through the day so that virtually all normotensive persons sometimes have hypertensive levels, and all hypertensives sometimes have normal levels (Pickering et al, 1984). Since the advent of effective antihypertensive therapy, classification of hypertension has generally been done by static blood pressure measurement. This method of classification has resulted in a reduction of cardiovascular events overall by 20 percent but has had little demonstrable effect on incidence of myocardial infarction (Hypertension Detection and Follow-up Program Cooperative Group, 1979; Medical Research Council Working Party, 1985; James et al, 1988). Several studies have been done to test variation in measured blood pressure. Some have shown substantial intraindividual variation in blood pressure over the course of the day (Pickering et al, 1986). Sources of this variation have been identified as activity (Mann et al 1979; Clark et al, 1987; Van Egran & Madarasmi, 1988); emotional state (James et al, 1986; James et al, 1988); changes in posture (James et al, 1986; Pickering et al, 1982); and situation

of measurement (James et al, 1986; James et al 1988; Pickering et al, 1982; Harshfield et al, 1982). It also has been suggested by James et al (1988) that these factors may in turn be dependent on the sex of the individual.

In the study done by Clark et al (1987) the variation in blood pressure was studied by recording activities as well as blood pressures during the day. A portable blood pressure recorder was used to take blood pressure readings every 15 minutes and the patients recorded their activity and location in a diary. The results showed that the overall effect of activities on blood pressure accounted for about 40 percent of the observed variation in blood pressure for systolic, and 39 percent in the diastolic blood pressure, with the time of day accounting for about 33 percent of the observed variation in both systolic and diastolic blood pressure. There was little variation in blood pressure once the effect of activity was allowed for and it was concluded that there is no important circadian rhythm of blood pressure which is independent of activity.

James et al (1990) also assessed whether the effects of factors such as: emotional state; posture, situation of measurement; and gender, on blood pressure variation were different when measured during summer months (May-Sept.) and winter months (Nov.-Mar.). Their results indicated that more factors had greater effects on blood pressure during the winter months than the summer months. Giaconi et al (1989) also investigated the seasonal influences on blood pressure measurements and

found that readings were generally significantly higher in the cold season for mean diastolic daytime blood pressure. Their observations agreed with James et al (1990), indicating that blood pressure may be strongly influenced by environmental temperature.

These studies into the variation in blood pressure may have important implications for the interpretation of blood pressure in population studies. We can deduce from these studies that hypertensives are a prognostically heterogeneous group because blood pressure varies according to time of day, activity, time of year, setting, means of observation, and emotional state.

Another issue in the classification of individuals by blood pressure is that mensuration is used to produce a number at which blood pressure can be treated (Herman, 1991). The cornerstone upon which our understanding of hypertension has been based is the measurement of blood pressure in the clinic. As well as increasing evidence that this measurement may not be representative of blood pressure at other times, there are other apparent dangers in evaluating blood pressure this way. Mejia et al (1990) encountered two phenomena which would lead to wrongly classified hypertension. The first one was hardening of the arteries that gave a falsely high reading. Although this has long been known, its significance has grown because of its particular application to the elderly. The second one was "cuff-induced" hypertension which was

measured by comparing an intra-arterial determination in one arm, with a sphygmomanometer measurement in the other. The brachial artery pressure rose when the cuff was inflated, but was otherwise normal.

A further criticism is made by Kleinman (1988) who questions whether human beings should be measured in any sense at all when in fact there are no metric measures in biomedicine or behavioral research for the existential qualities of suffering, such as hurt, desperation, moral pain and triumph. These qualities can however be evaluated by ethnography, biography, history and psychotherapy, and not by thinned-out images of patients and families arising from symptom scales and survey questionnaires.

Ambulatory Monitoring of Blood Pressure

The decision to initiate antihypertensive treatment and the evaluation of ongoing therapy is usually based on blood pressure readings taken in a physician's office. As mentioned previously it is now well documented that these measures do not accurately reflect average 24 hour pressures (Harshfield et al, 1982). Therefore, 24 hour ambulatory recording is by far more useful in evaluating mildly hypertensive patients. Ambulatory blood pressure monitoring can determine whether blood

pressure is sustained outside of an office setting and can measure blood pressure variability.

The term 'borderline or mild hypertension' refers to a heterogeneous group of patients, only a few of whom will progress to essential hypertension or suffer cardiovascular consequences (Pickering et al, 1986). There are several problems inherent in the observation of borderline hypertension, with only 20 percent of young patients going on to develop sustained hypertension in later life. The Hypertension Detection and Follow-up Program (1984) found that treatment of borderline hypertension may reduce cardiovascular morbidity, and has put impetus on physicians to treat such patients. If all those with diastolic blood pressures over 90 mm Hg are treated, then it is possible that large numbers of patients will be treated for relatively little benefit (Pickering, 1983). The adverse side-effects of this medication means that the cost-benefit ratio of treating these patients appears to be very poor.

Ambulatory blood pressure provides detailed information on the levels and variability of blood pressure that occur over a 24 hour period. Paratti et al (1987) suggest that ambulatory blood pressure monitoring be used to assess the blood pressure load that acts on the heart, as well as the peripheral circulation throughout the day and night. It has further use in the evaluation of antihypertensive drugs for their efficacy and duration of action.

The two ways of measuring ambulatory blood pressure are: by direct intra-arterial measurement or by non-invasive portable recorders. The use of invasive techniques is questioned because of its high cost, and its risks which have made some countries consider it unethical. Ambulatory blood pressure monitoring is considered superior to office blood pressure measures in diagnosing hypertension, predicting cardiac end organ damage and cardiovascular morbidity and mortality (Pickering, 1987).

There are some problems encountered in the use of ambulatory blood pressure monitors. Broadhurst et al (1990) state that ambulatory blood pressure monitors are accurate for resting blood pressures but not for blood pressures taken while exercising. Furthermore, they are expensive which limits their availability and some do not meet standards of accuracy and reliability (O'Brien et al, 1990). A further concern is with the patient keeping a behavioral diary on the day of monitoring. The patient's acceptance of the diary is often far from "the ideal" and the gathering and the processing of behavioral data are so time-consuming that they do pose a formidable barrier to wider use of ambulatory monitoring. To overcome these problems, Van Egeren and Madarasmi (1988) suggest the logging of the information must be interesting enough to motivate the patient, and be manageable and accessible to the technician or clinician responsible for report writing and data analysis. To address

these problems, Van Egeren and Madarasmi have developed a computer-assisted diary which has several advantages over the typical ambulatory monitoring. There is considerably less technician and clinician time required with the information available immediately for data analysis and clinical reports. It also eliminates the problem of lack of standardization which exists in present diaries. Furthermore, the computer-assisted diary has the potential benefit of better quantification of behavioral information and allows for greater comparability of data gathered at different centres. It also allows for the possibility of developing activity-standardized blood pressure norms for use with patients. The results of their study provide evidence that blood pressure fluctuates moment to moment as influenced by the pattern of their daily life, expressed externally by behavior and internally in mood states. Van Egeren and Madarasmi (1988) do not comment on the costs of the computer-assisted diary, and its acceptance may largely depend on its costs as well as its benefits.

White-Coat Hypertension

White-coat hypertension is generally defined as a persistently elevated clinic blood pressure and a normal ambulatory blood pressure (Pickering, 1990). It is believed that white-coat hypertension may be a conditioned response to the physician because the stress appears to produce a transient elevation in blood pressure only while in a clinical setting. Pickering (1990) also found that behavioral factors such as stress can significantly influence the diurnal profile of blood pressure. However, there is a problem in linking behavioral factors to hypertension, in that the duration of their effect on blood pressure is unknown, and he was therefore unable to show whether behavioral factors played a role in the development of sustained hypertension.

Pickering et al (1990) found that technicians' (usually female) readings were not only lower than physicians' (usually male) readings, but they were closer to the daytime average recorded during ambulatory monitoring. Although 21 percent of hypertensive patients had white-coat hypertension, in those patients with more advanced hypertension the percent of white-coat hypertension was only 5 percent. They further suggested that white-coat hypertension is a learned response to the environment of the physician's office, but is not necessarily a generalized abnormality in blood pressure regulation. There is only limited evidence

but it does suggest that the subgroup of patients who have white-coat hypertension are at a relatively low risk of cardiovascular morbidity. Further work is needed to determine whether antihypertensive treatment can safely be withheld. Pickering et al (1988) also found that white-coat hypertension was more likely in those patients who were female, younger, and not obese. The possibility that white-coat hypertension is a marker for a patient's response to the stresses of everyday life has been used as an argument for defending the use of clinic pressures, but Pickering et al (1988) were unable to verify this in their study.

Julius et al (1990) reported a high prevalence (58 percent of the hypertensive population) of white-coat hypertension in Tecumseh, Michigan. Previous studies by Pickering et al (1988), Krakoff et al (1988), Lerman et al (1989) and Borkowski (1989) estimated prevalence levels of between 21 percent and 39 percent. However, Julius et al (1990) used different procedures than previous studies. They used clinic measurement which was taken just once and home blood pressures. Ambulatory monitoring was not used and it is possible that home blood pressures which were normal would have ambulatory pressures which were in the hypertensive range. Julius et al (1990) suggest that ambulatory blood pressure monitoring (as used by Pickering) has not been accepted as a gold standard against which everything else must be measured and in fact ambulatory monitoring is a very expensive technique, and one whose

advantages have not yet been well evaluated. Perhaps as Julius et al (1990) suggest one should look at other possible methods to establish white-coat hypertension including the use of home monitoring rather than only considering ambulatory monitoring.

Pickering (1991) suggests that elevated clinical readings could result from: 1) generalized hyperreactivity to arousal or 2) the white-coat effect which is when the pressor response is more specific to the clinic setting. These can be distinguished by ambulatory monitoring but not by home monitoring. The inclusion of the hyperreactors in the Tecumseh Study (Julius et al, 1990) could explain the relatively high prevalence of white coat hypertension. Pickering (1991) goes on to suggest that if different populations are to be compared then some agreed upon criteria need to be established. Criteria should include: the definition of white-coat hypertension as a persistently raised clinic blood pressure and a normal ambulatory blood pressure; more than one office visit to establish definition; evaluation of 24-hour ambulatory blood pressure to establish definition; and definition of raised blood pressure (140/90 mm Hg is acceptable) and normal blood pressure.

Myers and Reeves (1991) studied the existence of a white-coat effect in treated hypertensive patients and found a marked white-coat effect in treated hypertensive patients whose office blood pressures were substantially higher than ambulatory values. The difference between office

and ambulatory readings was at least 20 mm Hg systolic and/or 10 mm Hg diastolic in 73 percent of the seventy-one patients (37 were women). This study showed that white-coat hypertension in treated patients is possible and brings into question the treatment of some patients on chronic antihypertensive drug therapy. Despite repeated office blood pressures some patients' blood pressures will fall spontaneously without therapy and some patients receiving therapy may not need drugs or instead they may be managed satisfactorily on less medication. Despite these findings ambulatory blood pressure monitors are generally only available in specialized centers and therefore most decisions on whether to treat hypertension are made on the basis of office readings alone.

Furthermore, there is no evidence that white-coat hypertension is a "prehypertensive" condition (Pickering, 1990:S146). It is thought that the stress-related effects on blood pressure are transient and the role of behavioral factors in sustained hypertension remains unclear. It is possible that chronic low grade stress may lead to sustained elevation of blood pressure. Pickering's (1990) data are cross-sectional and will need to be supported by prospective studies before any conclusions can be made.

In Canada, blood pressure is usually measured in clinical settings for diagnosis of possible hypertension, this despite studies expressing the now commonly held belief that blood pressures are influenced by the physician and the setting. For the past 25 years, published articles have

expressed this belief as Kain et al (1964) concluded: "single casual pressures are apt to reflect temporary rises in pressure resulting from stimulus from the central nervous system due to the presence of the physician, the medical environment, or other personal factors, and therefore may not reliably indicate the net daily load because pressures may be lower at other times" (quoted in Pickering et al, 1990:S142).

Reactivity Blood Pressure

Hypertensives are a prognostically heterogeneous group because blood pressure varies according to time of day, setting and means of observation. Alderman et al (1990) studied "reactivity blood pressure" by systematically recording pretreatment nurse administered diastolic blood pressure and subtracting it from pretreatment physician administered diastolic blood pressure. Over 14 years of follow-up the incidence of myocardial infarction, stroke, and cardiovascular disease mortality and all-cause mortality were determined using Cox survival analysis (Cox, 1972 as cited in Alderman et al, 1990). It was found that only age, sex, and diastolic blood pressure reactivity remained predictive of myocardial infarction or total cardiovascular disease. They suggest that blood pressure reactivity is possibly a centrally-medicated phenomenon, which identifies

a subgroup of hypertensives with an increased risk of myocardial infarction despite successful blood pressure control.

Alderman et al (1990) hypothesize that patients whose clinic blood pressure was higher when measured by a physician than by a nurse would be similar to "white-coat" hypertensives who were found by 24-hour blood pressure monitoring to be hypertensive only in the physician's office. Those patients who had greater blood pressure reactivity (their pretreatment blood pressure taken by a physician was significantly higher than the blood pressure taken by a nurse) had more heart attacks despite successful antihypertensive therapy, when compared to those with lower blood pressure reactivity (their pretreatment blood pressure taken by a physician is similar to or less than the blood pressure taken by a nurse). These results are in agreement with those of Perloff et al (1989) which found that when ambulatory systolic blood pressure varied widely from clinic systolic blood pressure it correlated best with subsequent cardiovascular complications. Alderman et al (1990) accounted for cardiac risk factors not considered in Perloff's analysis.

The study by Alderman et al (1990) is longitudinal compared to the white-coat studies which are cross-sectional. In this prospective longitudinal study it was found that the reactivity blood pressure was a more effective prognostic tool than a physician's measure of pretreatment blood pressure in its ability to predict cardiovascular disease occurrence

among successfully treated hypertensive patients. Specifically, it was a greater blood pressure response to the physician in comparison to the nurse that predicted myocardial infarction occurrence. It is difficult to determine whether the blood pressure reactivity in Alderman's study is causally related to myocardial infarction or just marks those who are for some reason at greater risk. Generally, females did not display the reactivity gradient that was so strikingly evident among males.

The issue raised by Alderman's study is that the measurement of blood pressure by a physician in a single office visit is only modestly predictive of prognosis and may in fact not be an appropriate means of diagnosis and subsequent treatment by antihypertensive therapy. From the results of this study, it appears that this single physician measurement would over diagnose the number of hypertensives and cause those who are truly not hypertensive to begin unnecessary, costly, and possibly harmful therapy.

2.5.1 Screening for Hypertension

If screening programs are to be part of a public health initiative, Berwick (1985) proposes some guidelines for health screenings to encourage quality, accuracy and efficacy in detecting disease or identifying and reducing risk factors for chronic disease. He states that the disease

involved should be of sufficient prevalence and unlikely to be detected without screening, the tests must have acceptably low costs and error rates, and if health problems are identified, presymptomatic treatment should be available, advantageous, and acceptable to the general public. There is some debate whether blood pressure screening meets these criteria and further debate on whether labelling as hypertensive may outweigh any benefits derived from early intervention through screening programs.

2.5.2   Control of Hypertension

Contemporary biomedicine provides numerous treatment options to aid in the control of hypertension. These include various pharmacological strategies designed to dilate vessels, reduce cardiac output and decrease blood volume, all with the end purpose of reducing the pressure at which the cardiovascular system operates. Dietary restriction of sodium, and weight reduction are used also. Control of hypertension often requires active participation by the patient and his/her family in the form of self-treatment, self-medication, the adoption of risk-reduction behaviors, and disease prevention behaviors. Chronic diseases can not be managed by the physician alone and in fact are often controlled by the patient with the assistance of the health care provider. The potential to

control hypertension and thereby reduce the rates of morbidity and mortality lies mainly with the patient and the understanding of the patient's attitudes, beliefs and behavior is critical.

The crucial missing link in hypertension control is compliance. It is generally estimated that the magnitude of non-compliance in the general population is about one-half, meaning, those who are known hypertensives under biomedical treatment who do not adhere to their regime or who are uncontrolled. There is a vast literature which spans several decades, of research into compliance with biomedical treatment, but demographic, socioeconomic and organizational correlates have offered little insight into patient behavior. Compliance is difficult to determine as monthly pill counts are not fool proof, nor are self-reports. Control of hypertension seems to be a more reliable outcome for measurement. Problems in management of hypertension, specifically compliance with biomedical treatment are often due to personal health beliefs.

The Health Belief Model was first used to predict preventive health behaviors. It has successfully extended to research on compliance with antihypertensive treatment (Hershey et al, 1980; Nelson et al, 1979; Kirscht & Rosenstock, 1977). Patient perceptions have generally been found to be significantly associated with compliance including severity of disease, locus of control over treatment, and efficacy of treatment. In these studies, approximately 15 percent of the variance in compliance behavior was

explained. This type of research has some limitations by virtue of its origins. The Health Belief Model was deductively derived from theory in cognitive psychology and has generated research on beliefs that are theoretically significant. It becomes problematic if there is sole reliance on theoretically generated survey questions because this can mean that entire domains of health cognition may not be tapped. This point was emphasized by Blumhagen (1982) in his discussion of Health Belief Model applications to compliance in hypertension. He expressed the problem as one of depending too much on what theory deems important, and not on what the people think is important. This realization leads to the pursuit of inductive, ethnographic research to compliment and provide a social and cultural context to the relationship between health beliefs and compliance to hypertensive medications.

The concept of compliance is usually defined as the extent to which a patients' behavior coincides with that prescribed by a physician for treatment of a disease (Sackett, 1976). However, it is reasonable to be suspect of the label `noncompliant behavior' because this label often only addresses the issue of obedience to medical authority. In looking at behavior from a patients perspective, the problem of noncompliance is reconceptualized as a problem in illness management. This shifts the focus from obedience of the medical authority, to a focus on personal behavior and actions within a personal context. This in turn allows the client to be

viewed as a cognizant person, making choices within the context of everyday life.

2.5.3 Antihypertensive Treatment

The first antihypertensive drugs were introduced in the 1950's for the treatment of malignant hypertension or symptomatic disease, which was potentially lethal if left untreated (Dollery, 1987). The advent of safer drugs has led to offering treatment to asymptomatic people and a progressive redefinition of `need' in terms of lower levels of blood pressure requiring treatment (Kawachi & Wilson, 1990). The National Heart and Blood Pressure Education Program (1980) stated that all patients with diastolic blood pressures above 90 mm Hg ought to be treated, even in uncomplicated cases. Defined this way, 20-30 percent of the population in many Western countries now had levels of blood pressure requiring drug treatment (Wing, 1984). About two thirds of people over age 65 have blood pressure higher than 140 mm Hg systolic or 90 mm Hg diastolic (Schoenberger, 1991). "This conception of an arbitrarily defined level of blood pressure as `diseased' has led to the successive redefinition of the level of blood pressure `needing treatment', as well as to far-reaching consequences for the labelling of large sections of the population, the

creation of demand for medical treatment and for the supply of drugs to treat it" (Kawachi & Wilson, 1990:1240).

There has been a gradual retrenchment of the guidelines for treatment since 1984, when the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure gave widely received recommendations on the use of drug therapy for hypertension. It advised that "the benefits of drug therapy appear to outweigh any known risks from such therapy for those with a diastolic pressure persistently elevated above 95 mm Hg..." (1049) and recommended that stepped-care drug regimens be used with these patients. In 1986, the World Health Organization (WHO) and the International Society of Hypertension (ISH) guidelines raised the cutoff level to 100 mm Hg (or above 95 mm Hg only after 3-6 months of observation). With recent evidence from antihypertensive trials, these recommendations are now being disputed.

The clinical trials of antihypertensive therapy do not fully support the current guidelines and practice in the treatment of hypertension in women. The research which has been done to date concerning the adverse effects of antihypertensive treatment have generally excluded women from consideration. The evidence is clear that antihypertensive treatment can reduce cerebrovascular morbidity and mortality, and perhaps cardiovascular morbidity and mortality in men. But the evidence of any reduction in adverse outcomes for women is inconclusive (Anastos et al,

1991). Presently gender differences are not acknowledged in guidelines of the treatment of hypertension.

The benefits of treating hypertension can be reduced further by the labelling of a person as hypertensive. Evidence from many studies, although somewhat inconsistent, suggests that labelling is harmful. Patients tend to be off work more and have a lower perception of their own health once labelled than before diagnosis (Alderman & Lamport, 1990.

Although the shape of blood pressure-risk curves differ for different end points, increasing more gradually for CHD than for stroke, several facts indicate that elevated blood pressure is related to these end points (Wilhelmsen, 1989). There are some other well established risk factors for CHD, but lipid disturbances, tobacco smoking, and elevated blood pressure remain the most important. However, in some cases normalization of these other risk factors may be more important than treating blood pressure especially if the antihypertensive agent being used has adverse effects on other CHD risk factors like blood lipids (Wilhelmson, 1989). The indication from trials such as the Multiple Risk Factor Intervention Trial (MRFIT) is clearly that controlling blood pressure, if smoking status and serum cholesterol levels are neglected, is like fighting for a better outcome with one arm tied behind the back (Stamler et al, 1989).

In a summary of issues raised at the National Heart, Lung, and Blood Institute (NHLBI) Workshop on Antihypertensive Drug Treatment (Cutler et al 1989), it is suggested that although blood pressure is a continuously distributed risk variable - the higher the blood pressure, the greater the cardiovascular risk - evidence suggests that not all groups at the same level of blood pressure have the same risks for the various adverse cardiovascular sequelae. Depending on demographic characteristics such as age, sex, and socioeconomic status, and other cardiovascular risk factors like hypercholesterolemia, smoking and left ventricular hypertrophy, some groups will be more prone to develop stroke, others to develop myocardial infarction depending on their risk factors. Therefore, drug interventions should "... not only lower blood pressure but also confer additional benefits relative to specific disease processes or morbid outcomes" (Cutler et al, 1989:167).

Hypertension is a major risk factor for cardiovascular morbidity and mortality, and the most important risk factor in cerebrovascular disease. One of the most complex assumptions of reducing CHD mortality, is that this reduction will result in reductions in total mortality. The Lipid Research Clinic Trials (1984) and the Helsinki Heart Study (Frick et al, 1987) both failed to show reductions in total mortality in clinical trials of cholesterol lowering. In a meta-analysis (Muldoon et al, 1990) of cholesterol reduction studies, there was a net reduction in CHD deaths but

the reductions in total mortality were nonsignificant. However, the compensatory deaths were from nondisease causes such as accidents and suicide and not from other diseases.

Cutler et al (1989) also suggest that gender is an important risk factor to consider when making treatment decisions. Women with few or no other risk factors have low absolute risk if blood pressure is only mildly elevated. Evidence from the Medical Research Council (MRC Working Party, 1985) also showed that women tend to have more side effects from at least one of the drugs they used. Therefore, if the relative benefit of antihypertensive drugs is about the same as for men, at least for stroke, then the absolute benefit-harm ratio is probably less and it would seem reasonable to use a higher blood pressure threshold for women when initiating treatment (Cutler et al, 1989).

Although the evidence is weak that treatment of hypertension can positively affect CHD mortality, it can be assessed quantitatively. The estimated benefits according to the trials which have been done, can be applied to an estimate of the increase in the proportion on treatment since 1968 which is when the decline in CHD mortality began. This is the approach used by Goldman and Cook (1984) when they estimated that from 1968 to 1976, approximately 8.7 percent of the decline in mortality could be attributed to the benefits derived from antihypertensive treatment (McKinlay et al, 1989).

Beaglehole (1986) estimated that in Auckland, New Zealand, between 1974 and 1981, approximately 12 percent of the observed decline in CHD mortality could be attributed to antihypertensive treatment. However, McKinlay et al (1989) concluded that medical interventions including antihypertensive treatment, pharmacological management of hypercholesterolemia, improvements in emergency management outside of hospitals, the introduction of coronary care units in hospitals, and coronary bypass surgery have had little impact on population mortality rates.

The contribution of antihypertensive therapy to stroke mortality has been somewhat greater. Between 1970 and 1980, stroke mortality declined by 40 percent for people 35-74 years of age, in the United States. It is estimated that approximately 12 percent of the decline can be attributed to the increase in antihypertensive therapy (Bonita & Beaglehole, 1987, as referenced in McKinlay et al, 1989). This indicates that medical intervention in hypertension explains only a limited amount of the dramatic decline in stroke mortality and that a satisfactory explanation would have to address some other issues. These issues include: the decline in stroke mortality which occurred prior to the availability of antihypertensive drugs, the contribution of changes in other known risk factors, and the reasons for the increases in stroke mortality in other industrialized countries during the same time period.

The use of antihypertensive therapy has been hotly debated since its introduction. In an effort to synthesize all the evidence of its benefits and of its questionable effects, at least three meta-analyses have been carried out. The advantage of carrying out meta-analyses is to reduce the role of chance in interpreting the findings. Individual trials may be too small to show significance, whereas an overview can be more informative. The evidence from these meta-analyses indicates little disagreement in the findings that pharmacologic therapy is effective in decreasing strokes and stroke deaths, in reducing the occurrence of congestive heart failure, in reversing left ventricular hypertrophy , and in reducing the rate of progression of renal disease (Moser et al, 1991).

The meta-analysis conducted by Collins et al (1990) suggests that in addition to the above findings, there is a significant reduction in coronary heart disease. Other meta-analyses have suggested a similar reduction but were not able to achieve statistical significance. However, when all the meta-analyses were evaluated by Moser et al (1991), it was found that they were strikingly similar in showing approximately 40 percent reduction in the risk of stroke, and a range of 8 percent to 14 percent reduction in the risk of coronary heart disease. The differences in risk can be attributed to differences in the inclusion criteria for each meta-analysis and in the variability in end points used.

The meta-analysis conducted by MacMahon et al (1989) included community-based as well as some hospital-based trials, focusing on mild to moderate hypertension. It also included the Multiple Risk Factor Intervention Trial (MRFIT) which was not included in Collins et al (1990). The MRFIT was confounded with respect to its evaluation of blood pressure reduction by antihypertensive therapy because it was not designed to do this evaluation, but rather to test men with various combinations of elevated cholesterol level, smoking, and elevated blood pressure levels. The meta-analysis conducted by Hebert et al (1988) included only community-based trials, including more severe hypertension. Collins et al (1990) used both community-based and hospital-based trials, all of which were unconfounded drug trials, and evaluated cardiovascular morbidity or mortality in both mild and more severe hypertension.

Moser et al (1991) suggest that one of the main reasons for the differences in findings of the three meta-analyses is the variability in end points. For example, in the Hypertension Detection and Follow-up Program Study (HDFP) the non-fatal myocardial infarctions (MIs) were identified by: medical history, electrocardiogram or Rose Questionnaire, and thereby contributed a large proportion of nonfatal MIs to the totality of end points. The meta-analysis by MacMahon et al (1989), considered the non-fatal MIs diagnosed by ECG only, and Hebert et al (1988) considered the non-fatal MIs diagnosed by either ECG or clinical history. The

meta-analysis by Collins et al, (1990) considered only clinical history. Moser et al (1991) further suggest that if HDFP results considered only the non-fatal events identified by clinical history, the ratio of nonfatal compared to fatal CHD events, would be similar to the ratios for the other trials.

MacMahon et al (1989) further found that in observational studies, prospective cohort results suggest similar reduction in the risk of stroke when compared to clinical trials, but a lower incidence of CHD in clinical trials than would be anticipated based on observational data. A possible explanation would be that the meta-analyses are based on short terms (3 to 5 years) and the observational data is based on usual, life-long levels of blood pressure. In 8.3 year data from the HDFP, it is suggested that after 5 years of treatment some decrease in CHD mortality was found, but when an additional 3.3 years of follow-up was done the CHD mortality had been reduced even further and that this reduction was the result of accrued benefits over the course of the first 5 years of the study.

In summary, Moser et al (1991) present three issues when comparing meta-analyses: 1) the MRFIT trial was confounded with respect to reducing blood pressure and was therefore not included in Collins et al (1990) study; 2) the Collins et al (1990) report included trials with data on severe or complicated hypertensive subjects, but the sample size was too small to have an influence on the conclusions; and 3) the difference in

findings of the meta-analyses can be explained by the definition of non-fatal MI end points used in the HDFP trial.

Moser et al (1991) suggest that "a single randomized, controlled clinical trial of sufficient size and power to detect clinically important treatment effects..." (1278) would be more effective than any meta-analysis but in its absence Collins et al (1990) meta-analysis serves as " the most meaningful summary of the available data and has identified aspects of therapy that may require further elaboration" (1278). The meta-analysis calls for a re-examination of the conclusions formerly presented which conclude only a minor effect on CHD events from antihypertensive therapy. However even a 10-14 percent reduction in CHD events is of major importance in terms of both the clinical and public health impact.

Yusuf et al (1989) speculate that perhaps antihypertensive treatment does not completely prevent congestive heart failure but postpones its development by several decades. By extrapolating from the available data the reduction in congestive heart failure (CHF) might be smaller yet in trials of mild hypertension which are not yet complete. Thereby the impact of antihypertensive treatment in preventing congestive heart failure in an unselected population (which will consist of mainly mild hypertensives) will be more modest yet. They further suggest that diuretics used in most clinical trials may indeed delay or mask the diagnosis of CHF by preventing the development of signs of congestion

without actually affecting cardiac dysfunction and that the treatment of hypertension leads to postponement rather than actual prevention of CHF. The exact reasons for these trends are not readily apparent but they speculate that once CHF develops, treatment with diuretic drugs may delay deterioration of ventricular function and ultimate death. This in turn would reduce CHF-related deaths in middle age and increase similar deaths in old age.

Schoenberger (1991) suggests that use of different drugs in these clinical trials may have had a better effect on prevention of myocardial infarction. Most have relied on the use of diuretics and beta blockers which have known adverse effects on electrolytes, lipid metabolism, glucose metabolism, insulin resistance, and quality of life (Schoenberger, 1991). He states that the goal of antihypertensive therapy should be to extend the life expectancy of hypertensive patients to that of subjects without high blood pressure. He suggests that therapy with alpha blockers, angiotensin-converting enzyme (ACE) inhibitors and calcium antagonists will become increasingly common. Presently the treatment of hypertension is based on the guidelines of the Joint National Committee for the Detection, Evaluation, and Treatment of High Blood Pressure (1988).

Evidence that antihypertensive therapy affects quality of life was found by Farmer et al (1990) in a study of the relationship between longitudinally measured blood pressure and cognitive performance. Data

extrapolated from the Framingham Study which included participants aged 55-89 years with no clinically diagnosed cerebrovascular disease was used, and no association was found. However, there was a highly significant graded relationship between cognitive impairment and the probability of being off antihypertensive medication. This raises the possibility of an undesirable outcome from lack of compliance to prescribed drug treatment regimens in elderly hypertensives or as the authors suggest, that the cognitive impairment may be associated with reduced compliance to antihypertensive treatment.

Miall (1990) summarizes that even the larger trials were too small to detect what may have been a major public health benefit, such as a 10 to 15 percent reduction in hypertension-related events. MacMahon et al (1986) found that even when the major trials were pooled, the sample size would be insufficient to detect a modest reduction in coronary artery disease (CAD) events. The average length for the trials was 5.6 years and perhaps this was too short a duration. CAD incidence is influenced by many factors, hypertension is just one, and controlling it for 5 or 6 years may have been insufficient to modify CAD which had been developing for a lifetime.

In summary, the current literature supports the conclusion that hypertension in women and its related cardiovascular outcomes continue to be a major health problem in Canada and in other industrialized

nations. A review of recent epidemiological surveys of hypertension and related risk factors, and of clinical trials of antihypertensive therapies leads to the following conclusions.

Modification of some identified biomedical risk factors have had a positive impact on reducing hypertension and subsequent morbidity and mortality. Interventions to reduce obesity, elevate blood lipid levels and elevated blood pressure levels have shown potential benefits. Other biomedical risk factors such as increasing age and family history cannot be changed.

Despite continuing efforts, the metabolic basis of hypertension remains unknown. It has been observed that within the same individual, hypertension, obesity, and non-insulin dependent diabetes mellitus, commonly occur (Ferrannini et al, 1987). It is possible that an insulin-resistant state is a common factor in hypertension, obesity and diabetes mellitus, but the mechanisms of such an association are still uncertain (Ching & Beevers, 1991).

There has been voluminous research done on the biomedical risk factors for hypertension, yet the etiology remains uncertain, and the majority of the variance between hypertensive and non-hypertensive groups remains unknown. Factors outside of the present health care paradigm may be quantitatively significant and these non biomedical risk factors may further the understanding of hypertension. Hypertension is

more prevalent in lower socio-economic classes. Active lifestyle, moderate consumption of alcohol, cessation or reduction of cigarette smoking, stress management, and low salt consumption appear to reduce the prevalence of hypertension and support the need for intervention of these risk factors. Many of the studies done on these risk factors are cross-sectional and look at only a single risk factor. The need for more comprehensive longitudinal research into non biomedical risk factors is apparent.

As well as re-examining the need to understand more about risk factors for hypertension, there appears to be a need to re-examine the measurement of hypertension. To account for variations in blood pressure levels and to decrease the chance of over diagnosis due to white coat hypertension or reactivity blood pressure, the use of ambulatory blood pressure monitoring may be more valid than the current clinic procedure.

Hypertension appears to be a multifaceted disease and therefore therapeutic intervention which is directed at a single aspect, even if it is successful, does not necessarily improve (and may worsen) the overall risk for cardiovascular disease. Both pharmacological and non-pharmacological interventions are currently being studied for their ability to maintain quality of life and reduce overall cardiovascular risk, as well as lowering blood pressure.

Despite the significant prevalence of hypertension in the female population, and the place of its major sequela cardiovascular disease as the

leading cause of death in women, relatively little research has been done on the epidemiology, natural history or results of antihypertensive treatment in women, as compared to men. Current guidelines were developed based on several large studies. Three of these: the Veteran's Administration Cooperative Study (1970); the Oslo Study (1980); and the Multiple Risk Factor Intervention Trial (1982), included no women subjects. The Hypertension Detection and Follow-Up Program (1979) with 46 percent of the subjects being women, found no decrease in mortality in the subset of white women receiving stepped-care treatment, but instead found a slight increase. The British Medical Research Council (1985) supported this finding when analyzing the data by genders and found that all-cause mortality in treated women increased by 26 percent. These two studies which supported the possibility that white women may be harmed by antihypertensive therapy, must be interpreted with caution because the analysis by gender was a post-hoc subgroup analysis and the studies were not designed for such an analysis.

Despite the fact that these major intervention trials were used to rationalize current treatment guidelines, it is not clear whether women benefit at all from therapy. The prevalence of hypertension is about equal in white women and white men, but because women outnumber men in the population and even more so in the older population, there are more hypertensive women than men. Also, the attributable risk for

cardiovascular complications of hypertension is higher for women than men (Anastos et al, 1991). The need for a gender specific research to guide appropriate treatment of hypertensive women is apparent, but currently aggressive non-pharmacological intervention appears to be a safe recommendation.

CHAPTER 3

METHODS & PROCEDURES

3.0   DATA SOURCE

This present study uses data which were part of a much larger data set collected by the Manitoba Heart Health Survey in 1989. The survey consisted of three separate components: a risk factor questionnaire; a food frequency nutrition questionnaire; and a clinic visit. The subjects for the survey were recruited from the registry of beneficiaries of the Manitoba Health Services Commission (MHSC) as on June 1, 1989. Adult residents of Manitoba aged 18-74 were targeted, with those living in institutions and members of the armed forces and RCMP being excluded. Those living in the "unorganized territories" were excluded for logistical reasons. The total eligible adult population was 754,664.

A stratified sampling design was used. Subjects were categorized by geographic strata to allow for comparison of three main types of communities in Manitoba. Stratum 1 consisted of the City of Winnipeg and adjacent rural municipalities. This stratum had an eligible adult population of 464,195 or 62 percent of the total eligible adult population of Manitoba. Stratum 2 consisted of six regional centers plus their respective adjacent rural municipality, and larger towns with adult

populations greater than 2000 people. This stratum contained 15 percent of the eligible adult population of Manitoba. Stratum 3 had 23 percent of the population and consisted of small rural communities including Indian Reserves, local government districts, rural municipalities, and small towns and villages.

From each of the geographical strata approximately 1,333 adults were sampled, and they were subdivided into 6 age-sex groups of equal size (ages 18-34, 35-64, and 65-74 in both sexes). MHSC used a random number generator to generate the appropriate number of individuals according to age and sex. In Stratum 1, there were 1,350 eligible subjects, of these, 908 subjects were interviewed (Table 3.1).

Table 3.1 Distribution of Survey Participants in Stratum I by Age and Gender

	Ages 18-34		Ages 35-64		Ages 65+		All Ages	
	Total Inter-viewed	Seen in Clinic	Total Inter-viewed	Seen in Clinic	Total Inter-viewed	Seen in Clinic	Total Inter-viewed	Seen in Clinic
Male	188	139	137	111	136	127	461	377
Female	192	120	135	105	120	98	447	323
TOTAL	380	259	272	216	256	225	908	700

Taken from: Young et al 1991, MHHS Technical Report (Table 3.3:23)

For the present study only data on the female subjects in Stratum I, which included the City of Winnipeg and adjacent rural municipalities, were analyzed. As indicated by the stated research questions, the topic of interest involves women and hypertension specifically, thereby excluding all males. Stratum I was chosen to avoid confounding the data with variables which may be unique to rural or northern residents. All data from the risk factor questionnaire and the clinic visit were analyzed for all women in Stratum I.

3.0.0   Response Rates

Of the 4080 individuals generated by MHSC, a large number were not located, mostly the result of outdated or incomplete addresses or low response rates in particular age-sex groups such as young males. An additional 510 individuals were then generated and those who agreed to participate were added to the first sample. Among those who were successfully located, the overall response rate was 77 percent for home interviews, and of these 84 percent kept their clinic appointments (Figure 3.1).

Figure 3.1 Manitoba Heart Health Study Response Rates

First
Sample
4080

+

Second
Sample
510

Eligible
Subjects
4590

Not
Located
967

Located
3623

Refused
831

Interviewed
at Home
2792

77%

Not seen
in Clinic
453

84%

Seen in
Clinic
2339
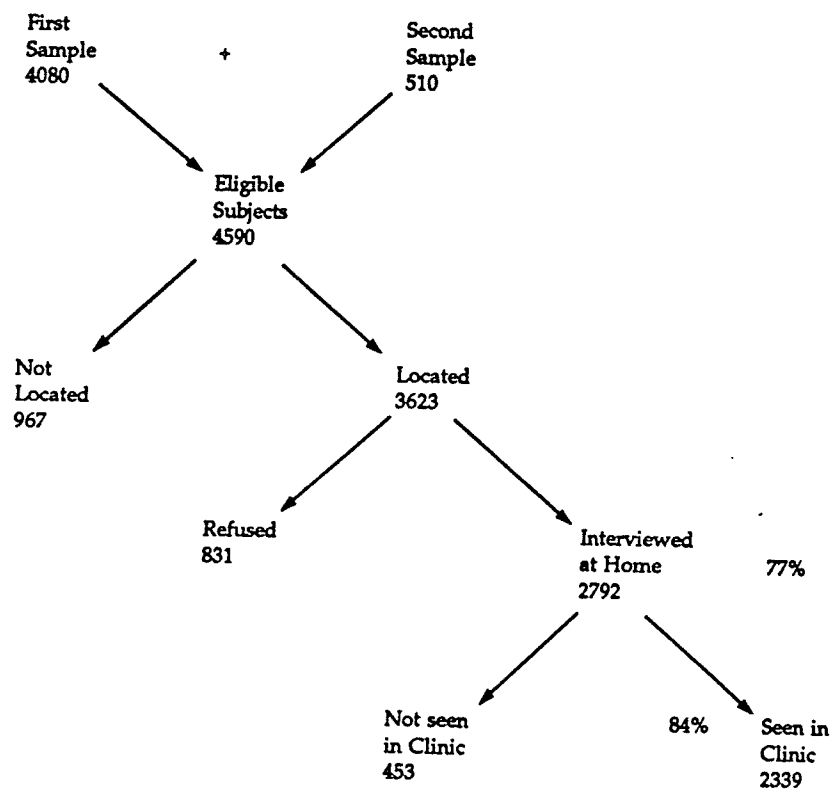
Taken from: Young et al 1991, MHHS Technical Report (Fig. 3.2:23)

Of the 1350 eligible subjects in Stratum 1, plus the 430 subjects added from the second sample, 1336 subjects were located. Of these, 428 subjects refused to participate, leaving a total of 908 who were interviewed, a response rate of 68 percent. Of those interviewed 700, or 77 percent were subsequently seen in clinic (Figure 3.2).

Figure 3.2 Stratum I Response Rates


```
Eligible                        Second
Subjects          +             Sample
1350                            430
         ↘                ↙
            Located
            1336
         ↙                ↘
32%  Refused                      Interviewed
     428                          at Home        68%
                                  908
                              ↙          ↘
                  Not seen              Seen in      77%
                  in Clinic            Clinic
                  208                  700
```
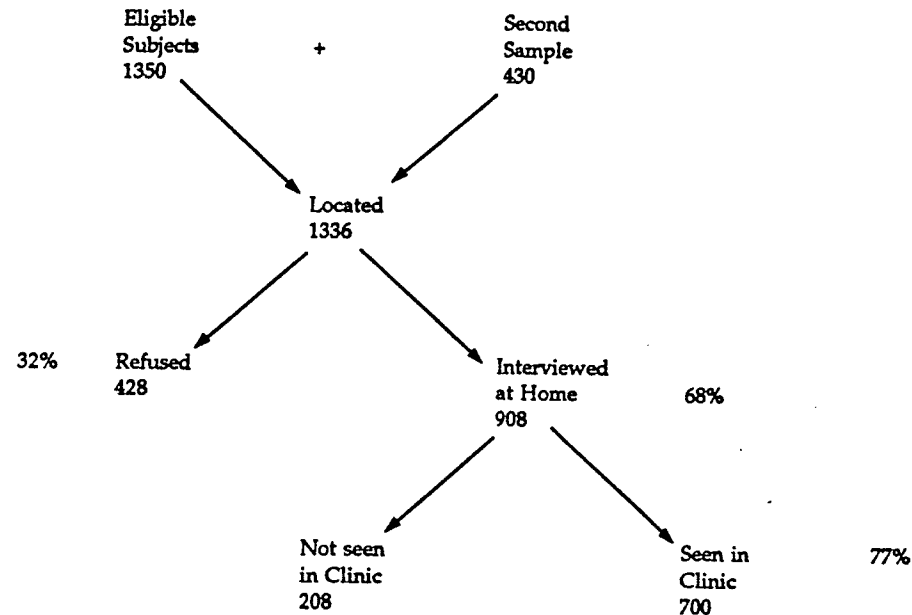
Taken from: Young et al, 1991, MHHS Technical Report (Table 3.2:22)

3.0.1 Selection Bias

Weighted sample data of the sociodemographic characteristics of survey participants were compared to the 1986 Canada Census and were found to be representative of the Manitoba population. Selection bias was evaluated by comparing the health care utilization pattern of survey participants with those who were eligible but did not participate. No significant differences were found between participants and non-participants thus making a selection bias unlikely. A second check was done by comparing variables of those who attended clinic and those who did not. There were significant differences

between these, with the non-attenders being younger, and more likely in Stratum 1. After adjusting for differences in age, sex, stratum, education, income and occupation, only regular smoking was significantly different between clinic attendees and non-attendees. Non-attendees were 1.5 times more likely to be regular smokers than attendees.

Subjects were selected using a stratified sampling design from the three geographic strata which were divided into 6 age-sex groups of equal size. To improve response rates, preliminary work was done to increase acceptance of the survey and create public support. Introductory letters to explain the survey and its purpose were sent to Manitoba physicians, Health and Community Services Regional Directors, and local police detachments. Information about the survey was placed in various newsletters including the Manitoba Medical Association, the College of Physicians and Surgeons and the Manitoba Association of Registered Nurses. Media coverage included daily and weekly newspapers, radio and television throughout the province.

Subjects were sent letters to inform them of their selection for the survey. Following the mailing of the letters to participants, media coverage encouraged non-respondents to take part in the survey. The participants were not given additional information about the survey at this point, but were notified they would be contacted by a survey nurse within 3 weeks of receiving the letter. There was regular contact with the Provincial public health system which was not formally involved.

Following the signing of the consent form, each subject was given an identifier consisting of 10 digits which would identify geographic location, name, age, and interviewer. A separate file was kept in the Survey Office which could link names and addresses with identifiers.

3.1 STUDY DESIGN

This investigation used cross-sectional and descriptive data. The primary focus was hypertensive status and the relationship to an array of independent variables including risk factors, demographics, attitudes and beliefs, and anthropometric measures.

The investigation was based on data from a large sample of subjects and, therefore, attained a sufficient number of subjects in each outcome category to provide precise or reliable prevalence estimates. The subjects were all randomly selected from the female population of Winnipeg and surrounding rural municipalities therefore the results will have decreased variability and increased generalizability.

A cross-sectional investigation of hypertension and an array of independent variables may not be as accurate as a prospective study which would enable the analysis of the effects of independent variables over time. However, time and financial limitations lead to cross-sectional study as an acceptable method of identifying risk factors and predictors of hypertension.

If the subjects could be followed over time, the longitudinal data collected could confirm this cross-sectional data. The number and random selection of subjects makes the sample representative and the data generalizable to women in the City of Winnipeg and its rural municipalities. All measurements were taken between October and December 1989 so the time between measurements was small.

The interviewer data included some self-reported data concerning personal health practices such as physical activity, smoking, diet and alcohol consumption and thus there may have been some inter-individual variation. The use of self-reported data also is subject to recall bias which may lead to incorrect classification of individuals into risk factor categories. Categories were formed for the dependent variable, hypertensive status, to allow for data analysis that would answer the research questions.

The first research question: *What are the identifiable biomedical risk factors which are predictive of hypertension in women*, was addressed by analyzing data from the Manitoba Heart Health Survey (Appendix A). Initially factors collected on the risk factor questionnaire and the clinic visit were analyzed using descriptive analysis and then grouped and analyzed using logistic regression models. The analysis provided information on real differences in hypertensive status by using univariate analysis to determine which variables were predictive of hypertensive status in women. The variables which were predictors of hypertensive status were then grouped and

multivariate analysis was used to determine which variables were optimally predictive of hypertensive status in women. A final logistic regression model indicated the biomedical risk factors which were optimally predictive of hypertensive status in women.

The second research question: *What are the identifiable non-biomedical factors which are predictive of hypertension in women,* was addressed by analyzing data from the Manitoba Heart Health Survey, specifically data collected from the subjects themselves including their attitudes, knowledge and beliefs concerning hypertension and cardiovascular health, as well as risk factors and selected demographic information. The interviews had been conducted by trained nurse interviewers and the interviews had been standardized, structured and elicited only information requested by the interviewer.

The advantage of analyzing this data is that it may provide a context of a subject's life which may influence their hypertensive status. One possible explanation is that such non-biomedical factors act as `triggers' for specific biological changes which then act as specific pathogens. Another possibility is these non-biomedical factors could each be causal factors which would expand the quantity of risk factors and predictors for hypertension, while maintaining the single-level reductionist biomedical model. The analysis provided information on real differences in hypertensive status by using univariate analysis for each of the non-biomedical factors, followed by

multivariate analysis of those found to be predictive of hypertensive status in women. A final model included the non-biomedical factors which were found to be optimally predictive of hypertensive status.

The third research question: *Can the risk factors, biomedical and non-biomedical, be synthesized into an expanded model which is optimally predictive of hypertensive status in women,* was addressed by combining the factors, both biomedical and non-biomedical, which were found to be optimally predictive of hypertensive status. The resulting model encompassed these factors and pulled them into an integrated whole allowing the various influences to be seen in the context of the model. The model has the potential to expand our understanding of hypertension in women by including the physical, social and behavioral factors which together relate to hypertensive status.

The fourth research question: *What is the potential of additional information to be useful in synthesizing risk factors to show multivariate and multilevel realtionships,* was addressed by taking the expanded model from research question 3 which encompassed both biomedical and non-biomedical factors, and using factor analysis to identify some common factors which interconnected fundamental themes through the sets of variables. This synthesis of the biomedical and non-biomedical factors allowed the commonalities between beliefs, behaviors, demographics and health status to be defined. It also provided an expanded framework within which the final

predictive model could be discussed and relationships between factors and hypertensive status could be explored in terms of characteristics including interactionism, emergence, loop structure and mutual causality.

3.2 DESCRIPTION OF PROCEDURES USED IN THE MANITOBA HEART HEALTH SURVEY

3.2.0 Blood Pressure Measurement

The data from the Manitoba Heart Health Survey provided four blood pressure measures taken on two different occasions; the initial interview and the clinic visit. Two readings were taken on each occasion; at the beginning and end of the home interview, and at the beginning and end of the clinic visit. Hypertension was defined as those with mean diastolic blood pressure greater than or equal to 90 mm Hg. (based on the mean of all four measurements or two measurements where subjects did not come to clinic) and or currently on antihypertensive medications, salt-restricted diet or a weight reduction program.

Measurement of blood pressure was done using the indirect method, using a mercury sphygmomanometer. The subject was seated comfortably and rested quietly for at least 5 minutes before the measurement commenced. Subjects had been asked not to eat or smoke for at least 30 minutes before the measurement. The right arm was relaxed and the forearm supported at heart

level. The cuff was inflated and the stethoscope placed over the brachial artery. The first and fifth Korotkoff sounds were recorded for the systolic and diastolic blood pressures, respectively (Memorandum from the WHO/ISH, 1986). If sounds continued to 0 mm Hg, the fourth Korotkoff sound was used (Joffres et al, 1992). Most current data have been related to the phase V diastolic blood pressure.

The reliability of the blood pressure measurements was assessed by doing fifty replicate measurements of blood pressure on designated days at randomly selected clinics. All diastolic blood pressure measures were found to be ± 6 mm Hg, except one.

3.2.1 Survey Questionnaire

The questionnaire (Appendix A) was developed by the Manitoba Heart Health Survey on the basis of modules which were already available from other survey studies done in Canada (Canada Blood Pressure Study, 1989; Smoking Habits of Canadians, 1988; Stevens et al, 1986). The questionnaire was reviewed by an expert panel and subsequently tested for reliability.

The data were collected in two phases. The first phase was a 40 to 60 minute home interview where subjects were asked to respond to the questionnaire. It included information on demographics, lifestyle, diabetic status, hypertensive status, knowledge and awareness of cardiovascular risk

factors. At both the beginning and end of the interview, blood pressure was measured. At the end of the home interview, the participants were invited to attend a clinic. The clinic visit was usually within two weeks of the home interview. Blood pressure readings were taken at the beginning and end of the visit as well as a fasting blood sample for lipid analysis and anthropometric measures including height, weight, and waist and hip circumference (MacLean et al, 1992).

All participants were informed of their results at the time of the visit with the exception of plasma lipid results which were sent by letter. Those with elevated levels of blood pressure or blood cholesterol were referred to physicians with public health nurses notified to ensure this follow-up.

3.2.2 Training and Quality Control

Quality control measures used by the Manitoba Heart Health Survey included the development of a training manual of standard procedures; training of coordinators and interviewers; and scheduled replication of measurements by survey supervisors. Training workshops for coordinators and interviewers averaged 4 days and were run by the same instructor to provide consistency across the country. To insure reliability of results, every 50th person had all clinical measurements repeated blindly by another

interviewer, and two blood samples were submitted to the lab to check the quality of laboratory procedures.

Risk factor questionnaires and clinic data were edited by the nurse interviewers and then further reviewed by the Survey office before data entry. Following data entry, preliminary frequency counts and descriptive statistics were used to find inconsistencies and inaccuracies. The responses were then checked against the original questionnaires, and if errors could not be reconciled, the values were taken out of the data set.

The reliability of the data entry of the questionnaires was assessed by randomly selecting fifty risk factor questionnaires and comparing them to computer records of each of the fifty questionnaires. There were three errors found, out of the 305 variables entered for each of the 50 records selected, or 0.02 percent of the entries.

3.2.3 Anthropometric Measures

The anthropometric measurements from the Manitoba Heart Health Survey included height, weight and body girths. These measurements were taken in the morning, on fasting participants dressed in light indoor clothing without shoes. Height (in cms) was measured using a square and tape measure fastened to the wall. Weight (in kgs) was measured using a

calibrated balance beam scale. Body mass index (BMI) was calculated as weight (in kgs) divided by the square of height (in meters).

Waist circumference was measured by measuring tape placed horizontally at the level of noticeable narrowing, and done at the end of a normal expiration. If narrowing was not evident, measurement was taken at the estimated lateral level of the twelfth or lower floating rib. Hip circumference was measured by measuring tape placed around the hips at the level of the symphysis pubis and the greatest gluteal protuberance. All measurements were made to the nearest centimetre. Waist hip ratio (WHR) was calculated as waist circumference (in cms) divided by hip circumference (in cms) (Reeder et al, 1992).

Replicate measurements of height and weight were done on fifty subjects on designated days, at randomly selected clinics. All replicate height measurements were within one centimeter and all weights were within one kilogram.

3.3   ETHICS

The survey design and instruments received approval from the University of Manitoba, Faculty of Medicine Ethics Committee. Written informed consent for participation in this study was obtained from all subjects prior to the interview and clinic procedures.

3.4 STATISTICAL ANALYSIS

For this investigation, all statistical analyses of the Manitoba Heart Health Survey data were performed using Number Cruncher Statistical System (NCSS), series 5.1 software. Significant differences between hypertensive and non-hypertensive women were identified in order to find risk factors and predictors of hypertensive status.

Descriptive statistics of the complete data set according to hypertensive status, as a binary outcome were done. Means and standard errors were calculated for all demographic variables and clinical measurements which were continuous. Frequencies and percentages were calculated for all categorical variables. Unpaired t-tests (two-tailed) were utilized to determine whether there were significant differences between the means of the continuous variables according to hypertensive status of the women. Chi-squared tests were the univariate analysis utilized to determine significant differences in the categorical variables according to the outcome variable, hypertensive status.

Multiple logistic regression analyses were used to investigate groups of factors for ability to predict hypertensive status. Logistic regression analysis was chosen because of its ability to accommodate both categorical and continuous variables in a model with a binary outcome. "The power of a logistic regression lies in its ability to incorporate and control for the effects

of a variety of explanatory variables simultaneously" (Hassard, 1990:95). Logistic regression models probabilities and as such there is no true equivalent of percent of variation explained in logistic regression. As a form of multiple regression, it has become the standard method of analysis when the outcome being measured is binary (Evans, 1988). Since it was first published in the Framingham Heart Health Study, the logistic regression model has become the standard method for regression analysis of dichotomous data in many fields, especially in the health sciences (Hosmer, 1989).

In each multiple logistic model, each variable has a logistic co-efficient (beta estimate) which measures or quantifies the unique impact of particular variable on the outcome variable. The model is able to untangle the effects of the various factors and assess the impact of each on the outcome variable. The effects of other confounding factors are controlled for, or eliminated (Hassard, 1990).

Factor analysis was used to find commonalities within each group of variables such as: demographics, beliefs, behaviours, biomedical, lifestyle, and cardiac and family history. This statistical method is applied to a set of variables to seek underlying factors or groups of variables with a common theme. Each of the identified factors is based on a common theme which influences or shows commonalities amongst variables. The variables from different levels which occur together demonstrate multivariate interactions within their level as well as multilevel interactions between factors in various

levels. Finally, these multilevel factors were analyzed in a logistic regression model to identify which multilevel factors were optimally predictive of hypertension in women. Identifying each of these predictive factors, which contain multilevel variables which share a common theme, may help to further understand hypertension in women and the relationships between the multiple risk factors.

CHAPTER 4

BIOMEDICAL PREDICTORS OF HYPERTENSIVE STATUS: RESULTS AND DISCUSSION

Chapter 4 begins with a description of the subjects and their characteristics. Next it addresses the first research question: *what are the identifiable biomedical risk factors which are predictive of hypertension in women,* by including the results and discussion of cross-sectional data on risk factors of hypertensive status which are within the parameters of the biomedical model.

4.0 DESCRIPTION OF SUBJECTS

The subjects were 447 women of the City of Winnipeg aged 18 to 74 years. Seventy of the women were identified as hypertensive with the remaining 377 non-hypertensive.

Hypertensive status was used as the outcome variable rather than either systolic blood pressure level or diastolic blood pressure level. Some hypertensive women who were able to control their hypertension would appear to be normotensive if either their systolic or diastolic blood pressure level were used as the outcome variable. This raises the

possibility of misclassification of some of the subjects and may lead to questionable results. The categorical outcome, hypertensive status, was therefore used as the most reliable outcome variable. A subject was classified as hypertensive if she was presently being treated for hypertension with drugs, salt restriction or weight reduction and/or her mean diastolic blood pressure was greater than or equal to 90 mm Hg.

The data on characteristics of the 70 hypertensive women in the City of Winnipeg stratum were compared to data from the Manitoba Heart Health Survey for all hypertensive women in Manitoba (Table 4.1).

Table 4.1 Characteristics of Hypertensive Women

Variable	Hypertensive Women in Stratum I N = 70, N (%)	Hypertensive Women in Manitoba (Young et al, 1991)
Hypertensive Status	70 (16%)	(15%)
Hypertension Controlled*	48 (77%) controlled	N/A
Hypertension Treated*	48 (77%) treated	N/A
Current Treatment***	47 (89%) anti-hypertensive drugs	53% anti-hypertensive drugs
Initial Treatment**	50 (91%) anti-hypertensive drugs	71% anti-hypertensive drugs
Awareness of Hypertension	62 (89%) aware	87% aware

* 8 values missing
** 15 values missing
*** 17 values missing

The prevalence of hypertension is similar, but the initial and current treatment appear to be quite different in the City of Winnipeg stratum as compared to women in all of Manitoba. The City of Winnipeg women are more likely to have had antihypertensive drugs as both initial treatment and current treatment.

4.1   BIOMEDICAL RISK FACTOR RESULTS

The first research question: *what are the identifiable biomedical risk factors which are predictive of hypertension in women,* is addressed by analyzing the data from the risk factor questionnaire and the clinic visit. The data are analyzed initially using descriptive and then univariate analysis to determine significant differences by hypertensive status.

Multivariate analysis is then used to analyze each group of predictive factors and then the entire group of biomedical predictors together. A final model which is optimally predictive of hypertensive status is then determined.

The following analyses relate to Research Question 1.

4.1.0   Anthropometric Measurements

The anthropometric measures of the subjects are presented in Table 4.2.

Table 4.2 Anthropometric Measures of 447 Hypertensive and Non-Hypertensive Women

Variable	Total Women N = 447 Mean	Non-Hypertensive Women N = 377 Mean ± S.E.	Hypertensive Women N = 70 Mean ± S.E.
Height (cm.)	162.71	163.25 ± 0.40	160.73 ± 0.79
Weight (kg.)	66.6	65.9 ± 0.84	69.89 ± 2.01
Waist (cm.)	79.63	78.35 ± 0.69	85.8 ± 1.78
Hip (cm.)	99.76	99.02 ± 0.57	103.35 ± 1.40
Waist/Hip Ratio	.796	0.79 ± 0.004	0.83 ± 0.01
Body Mass Index (BMI) kg/m^2	25.16	24.82 ± 0.31	27.05 ± 0.71

Using .05 level of probability, a series of unpaired t-tests found significant differences in means of several anthropometric measurements by hypertensive status.

The relationship between height and hypertensive status (t=2.65, p<0.008) indicated that hypertensive women were significantly shorter than non-hypertensive women.

The relationship between waist measurement and hypertensive status (t=-4.32, p<0.0000) indicated the hypertensive women had significantly larger waist circumferences than the non-hypertensive women.

The relationship between weight and hypertensive status (t=-1.94, p<0.053) is not significant but closely approaches significance.

The relationship between hip measurement and hypertensive status (t=-3.09, p<0.002) indicated that hypertensive women have significantly larger hip circumferences than non-hypertensive women.

The relationship between waist/hip ratio (WHR) and hypertensive status (t=-3.62, p<0.0003) indicates that hypertensive women have significantly larger waist/hip ratios.

The relationship between body mass index (BMI) and hypertensive status (t=-2.95, p<0.003) indicates that hypertensive women have significantly higher BMI than non-hypertensive women.

Each anthropometric variable (except weight) indicated a significant difference between the group means of hypertensive and non-hypertensive women. Using a logistic regression model, the six items comprising anthropometric measures were considered simultaneously to address the question: *do body dimensions defined in a broad sense as anthropometric measures, bear any relationship to hypertensive status.* A significant relationship between anthropometric measures and hypertensive status was observed (Model chi square = 26.63 with 6 d.f., p<0.0002).

A downward step-wise regression was done which eliminated any variable which did not reach statistical significance at the conventional 0.05 level and the results are shown in Table 4.3.

Table 4.3 Anthropometric Measurements Predictive of Hypertensive Status in Women in Reduced Logistic Model

Anthropometric Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Height	-.066	.0246	7.24
Waist Measurement	.049	.012	16.66

In univariate analysis, all anthropometric variables were predictive of hypertensive status, except weight, which closely approached significance. In a reduced logistic model, only height and waist measurement had unique predictive ability. The other variables do not appear because their predictive power is effectively duplicated by either height or waist measurement. Height and waist measurement were found to be the anthropometric measurements which most effectively characterize the impact of body dimensionality on hypertensive status in women.

4.1.1   Blood Lipid Levels

The blood lipid levels of the subjects are presented in Table 4.4.

Table 4.4   Blood Lipid Levels of 447 Hypertensive
and Non-Hypertensive Women

Variable	Total Women N = 447 Mean	Non-Hypertensive Women N = 377 Mean ± S.E.	Hypertensive Women N = 70 Mean ± S.E.
Total Cholesterol	5.22	5.09 ± 0.06	5.88 ± .017
High Density Lipoproteins	1.41	1.43 ± 0.02	1.31 ± 0.06
Low Density Lipoproteins	3.19	3.09 ± 0.05	3.70 ± 0.15
Triglycerides	1.36	1.25 ± 0.04	1.94 ± 0.16
Total/HDL Ratio	3.89	3.72 ± 0.07	4.75 ± 0.23

A series of unpaired t-tests found significant differences in means of some measurements of blood lipid levels, by hypertensive status.

The relationship between total cholesterol levels and hypertensive status, ($t=-4.89$, $p<0.0000$) indicated that hypertensive women had significantly higher cholesterol levels than non-hypertensive women.

The relationship between high-density lipoproteins (HDL) and hypertensive status, ($t=2.19$, $p<0.029$) indicated that hypertensives had significantly lower HDL cholesterol levels.

The relationship between low-density lipoproteins (LDL) and hypertensive status, ($t=-4.29$, $p<0.0000$) indicated that hypertensive women had significantly higher LDL cholesterol levels.

The relationship between triglyceride levels and hypertensive status ($t=-6.29$, $p<0.0000$) indicated that hypertensive women had significantly higher triglyceride levels.

The relationship between total cholesterol/HDL cholesterol ratio and hypertensive status ($t=-3.617$, $p<0.0003$) indicated that hypertensive women had significantly higher cholesterol ratios.

Each blood lipid level variable indicated a significant difference between group means of the hypertensive and non-hypertensive women. The five items comprising blood lipid level measurements were considered simultaneously using a logistic regression model to address the question: *do blood lipid levels defined in a broad sense, bear any relationship to hypertensive status.* A significant relationship between blood lipid levels and hypertensive status was observed (Model chi square = 31.82 with 5 d.f., $p<0.0000$).

A downward step-wise regression was done which eliminated any variable which did not reach statistical significance at the 0.05 level of probability and the results are shown in Table 4.5.

Table 4.5 Blood Lipid Variables Predictive of Hypertensive
Status in Women in Reduced Logistic Model

Predictive Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Total/HDL Ratio	.917	.195	22.06
HDL	1.566	.653	5.75

The logistic regression model indicated that Total/HDL cholesterol ratio and HDL emerged as the predictive variables, with triglycerides being not quite significant at the 0.05 level of probability. In univariate analysis, all blood lipid levels were predictive of hypertensive status. However, in a reduced logistic model, only Total/HDL cholesterol ratio and HDL cholesterol level had unique predictive ability. The other blood lipid levels do not appear because their predictive power effectively duplicated by cholesterol ratio or HDL. Therefore, Total/HDL cholesterol ratio and HDL cholesterol level were found to be the blood lipid measurements which most effectively characterize the impact of blood lipid levels on hypertensive status in women.

4.1.2 Cardiac History

The cardiac history of the subjects is presented in Table 4.6.

Table 4.6 Previous Cardiac History of 447 Hypertensive and Non-Hypertensive Women

Variable	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Have had a heart attack	15 (3%)	8 (2%)	7 (11%)
Have other heart disease	46 (10%)	25 (7%)	21 (30%)
Take medication for heart	31 (7%)	9 (2%)	22 (31%)
Have had a stroke	11 (2.5%)	7 (2%)	4 (6%)

Chi square tests found significant differences in the frequency of history of cardiac events, by hypertensive status. The relationship between hypertensive status and history of heart attack (x^2=12.26, p<0.0005) indicated that hypertensive women were significantly more likely to have had a heart attack than non-hypertensive women.

The relationship between history of any other kind of heart disease and hypertensive status (x^2=34.65, p<0.0000) indicated that hypertensive women were significantly more likely to have other kinds of heart disease than were non-hypertensive women. Hypertensive women also were

significantly more likely (x^2=83.08, p<0.0000) to be taking medication prescribed by the doctor for heart trouble.

The relationship between stroke and hypertensive status (x^2=3.64, p<0.05) is not significant but closely approaches significance.

Each cardiac history variable except stroke indicated that hypertensive women were significantly more likely than non-hypertensive women to have experienced that variable. Using the logistic regression model, the four cardiac history variables were considered simultaneously to address the question: *does previous cardiac history bear any relationship to hypertensive status*. A significant relationship between cardiac history and hypertensive status was observed (Model chi square = 34.38 with 4. d.f., p<0.0000).

A downward step-wise logistic regression was done which eliminated the variables that did not reach statistical significance at the 0.05 level of probability. Table 4.7 provides the beta estimate and standard error of those variables which are statistically significant.

Table 4.7 Cardiac History Variables Predictive of Hypertensive
Status in Women in Reduced Logistic Model

Cardiac History Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Other heart disease	-1.254	.407	9.50
Take medication for heart	-1.165	.474	6.04

In univariate analysis, all the cardiac history variables except history of stroke, were predictive of hypertensive status. In a reduced logistic model, only history of other heart disease and presenting taking medication for heart, had unique predictive ability. The predictive power of the other variables was effectively duplicated by these variables. Therefore, history of other heart disease and presently taking medication for heart were found to be the cardiac history variables which most effectively characterize the impact of cardiac history on hypertensive status in women.

4.1.3 Family History

The family history of health problems of the subjects is presented in Table 4.8.

Table 4.8 Family History of Health Problems of 447 Hypertensive
          and Non-Hypertensive Women

Health Problem	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Heart Disease	187 (42%)	140 (37%)	47 (67%)
High Blood Pressure	259 (58%)	211 (56%)	48 (69%)
Stroke	118 (26%)	84 (22%)	34 (49%)
Diabetes	106 (24%)	86 (23%)	20 (29%)
High Cholesterol	113 (25%)	97 (26%)	16 (23%)

Chi square tests found significant differences in the frequency of some family history of health problem variables, by hypertensive status. The relationship between family history of heart disease and hypertensive status (x^2=21.85, p<0.0000) indicates that hypertensive women are significantly more likely to have family with heart disease.

Hypertensive women are significantly more likely than non-hypertensive women (x^2=3.85, p<0.05) to have family history of high blood pressure, but both groups have a high frequency (56 percent for non-hypertensives and 69 percent for hypertensives) of family history of hypertension.

Hypertensive women are far more likely to have a family history of stroke (x^2=21.00, p<0.0000) than non-hypertensive women.

The relationship between hypertensive status and family history of diabetes (x^2=1.08, N.S.) indicates no real differences exist between hypertensive and non-hypertensive women. Similarly, there are no real differences between hypertensive and non-hypertensive women in family history of high cholesterol (x^2=0.26,N.S.).

Some of the family history variables indicated a significant difference in hypertensive status. Using a logistic regression model, the family history variables were considered simultaneously to address the question: *does family history of heart-related disease in a broad sense bear any relationship to hypertensive status in women.* A significant relationship between family history and hypertensive status was observed (Model Chi square = 32.52 with 5 d.f., p<0.0000).

A downward stepwise logistic regression was done as shown in Table 4.9. The table provides the beta estimate and standard error of those variables which were statistically significant at the 0.05 level of probability.

Table 4.9 Family History Variables Predictive of Hypertensive Status in Women in Reduced Logistic Model

Family History Variables	Beta Estimate	Standard Error	Chi Square Beta = 0
Heart Disease	.997	.289	11.87
Stroke	.892	.283	9.89

In univariate analysis, some of the family history variables were predictive of hypertensive status. In a reduced logistic model, only family history of heart disease and family history of stroke had unique predictive ability. The other variables which were predictive of hypertensive status in univariate analysis, do not appear because their predictive power is effectively duplicated by family history of heart disease or family history of stroke. These two variables were found to be the family history variables which most effectively characterize the impact of family history on hypertensive status in women.

4.1.4 Diabetes

The distribution of subjects by diabetic status is presented in Table 4.10.

Table 4.10 Distribution of 447 Hypertensive and Non-Hypertensive
Women by Diabetic Status

Variable	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Told you are diabetic	16 (4%)	10 (3%)	6 (9%)

In univariate analysis, hypertensive women were found to be significantly more likely to be diabetic than non-hypertensive women (x^2=6.068, p<0.01).

However, when the variable diabetic status was put into a logistic regression model to address the question: *does diabetic status bear any relationship to hypertensive status*, a non-significant relationship between diabetic status and hypertensive status was observed (Model Chi square = 0.01 with 1. d.f., p<0.938).

The results of analyzing the types of treatment of the diabetic women are presented in Table 4.11. The numbers were too small to do any further analysis.

Table 4.11 Distribution of 16 Diabetic Women by Treatment

Treatment	Total Women N = 16	Non-Hypertensive Women N = 10	Hypertensive Women N = 6
No current treatment	3 (19%)	1 (10%)	2 (33%)
Insulin	4 (25%)	4 (40%)	
Pills to control blood sugar	5 (31%)	2 (20%)	3 (50%)
Diet	6 (38%)	4 (40%)	2 (33%)
Weight loss	1 (6%)		1 (17%)
Other	1 (6%)	1 (10%)	

4.1.6 Glucose

The distribution of subjects by glucose level is presented in Table 4.12.

Table 4.12 Glucose Levels of 447 Hypertensive
and Non-Hypertensive Women

Variable	Total Women N = 447 Mean	Non-Hypertensive Women N = 337 Mean ± S.E.	Hypertensive Women N = 70 Mean ± S.E.
Glucose	5.48	5.378 ± 0.61	5.954 ± .144

An unpaired t-test found a significant difference in the means, indicating that hypertensive women had significantly higher glucose levels than non-hypertensive women (t=-3.820, p<0.0001).

Using a logistic regression model, a significant relationship between glucose level and hypertensive status was observed (Model chi square = 11.11 with 1 d.f., p<0.009). Therefore, glucose level was found to have predictive ability in relation to hypertensive status in women.

4.1.6   Age

The age distribution of the subjects is presented in Table 4.13.

**Table 4.13 Age Distribution of 447 Hypertensive and
Non-Hypertensive Women**

Variable	Total Women N = 447 Mean	Non-Hypertensive Women N = 377 Mean ± S.E.	Hypertensive Women N = 70 Mean ± S.E.
Age	44.74	41.50 ± 0.92	62.17 ± 1.41

An unpaired t-test found a significant difference in mean age of the hypertensive and non-hypertensive women. The relationship between hypertensive status and age (t=-9.34, p<0.0000) indicated that hypertensive women were significantly older than non-hypertensive women.

Using a logistic regression model, a significant relationship between age and hypertensive status was observed (Model chi square = 77.85 with 1 d.f., p<0.0000). The logistic regression analysis indicated that age had a unique predictive ability in relation to hypertensive status in women.

To further examine the relationship between age and hypertensive status, a multiple regression analysis was run with age and mean diastolic blood pressure values. A highly significant relationship (t=5.47, p<0.0000) was found and age was found to explain more than 19 percent of the variance in diastolic blood pressure.

A review of recent research has suggested that the relationship between diastolic blood pressure and age was not necessarily linear, but may level off after the age of 60. To find if there were indeed any possible curves in the relationship, the age variable was squared and included in a multiple regression analysis. The multiple regression model indicated that age squared was significant (t=-4.23, p<0.0000) and added to the relationship between age and diastolic blood pressure. By placing the values from the multiple regression model into a linear equation, a line can be drawn to show the relationship between diastolic blood pressure and age as shown in Figure 4.1.

**Figure 4.1 Relationship between Diastolic Blood Pressure and Age
in 447 Hypertensive and Non-Hypertensive Women**

Distribution of diastolic blood pressure by age
X – AGE by   Y – MEANSBP = O
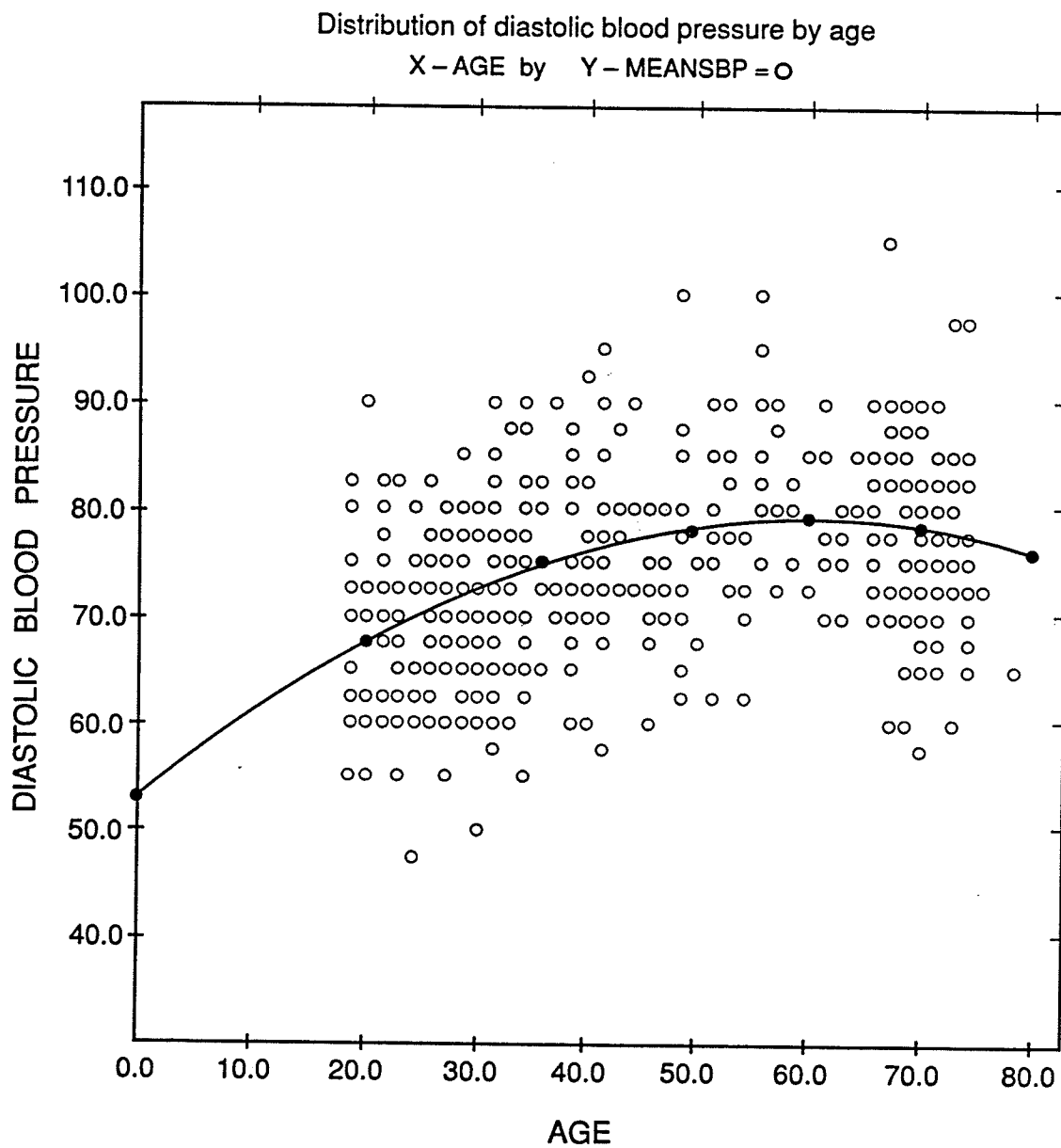
The linear model indicates that diastolic blood pressure reaches its maximum level at age 61.52 years and slowly decreases from then on.

To further examine the relationship between blood pressure and age, a multiple regression was also run between age and systolic blood pressure. The relationship was highly significant (t=18.13, p<0.0000) and age was found to explain almost 43 percent of variance in systolic blood pressure.

To determine if the relationship between systolic blood pressure and age was linear, the age variable was squared. A multiple regression model indicated that age squared was significant (t=4.50, p<0.0000) and added to the relationship between age and systolic blood pressure.

A scatter plot was used to show the relationship between systolic blood pressure and age. The values from the multiple regression model were placed into a linear equation and the results were used to draw a regression line through the scatter plot. The relationship between systolic blood pressure and age is shown in Figure 4.2.

Figure 4.2 Relationship Between Systolic Blood Pressure and Age in 447

Hypertensive and Non-Hypertensive Women

Distribution of systolic blood pressure by age
X – AGE by   Y – MEANSBP = O
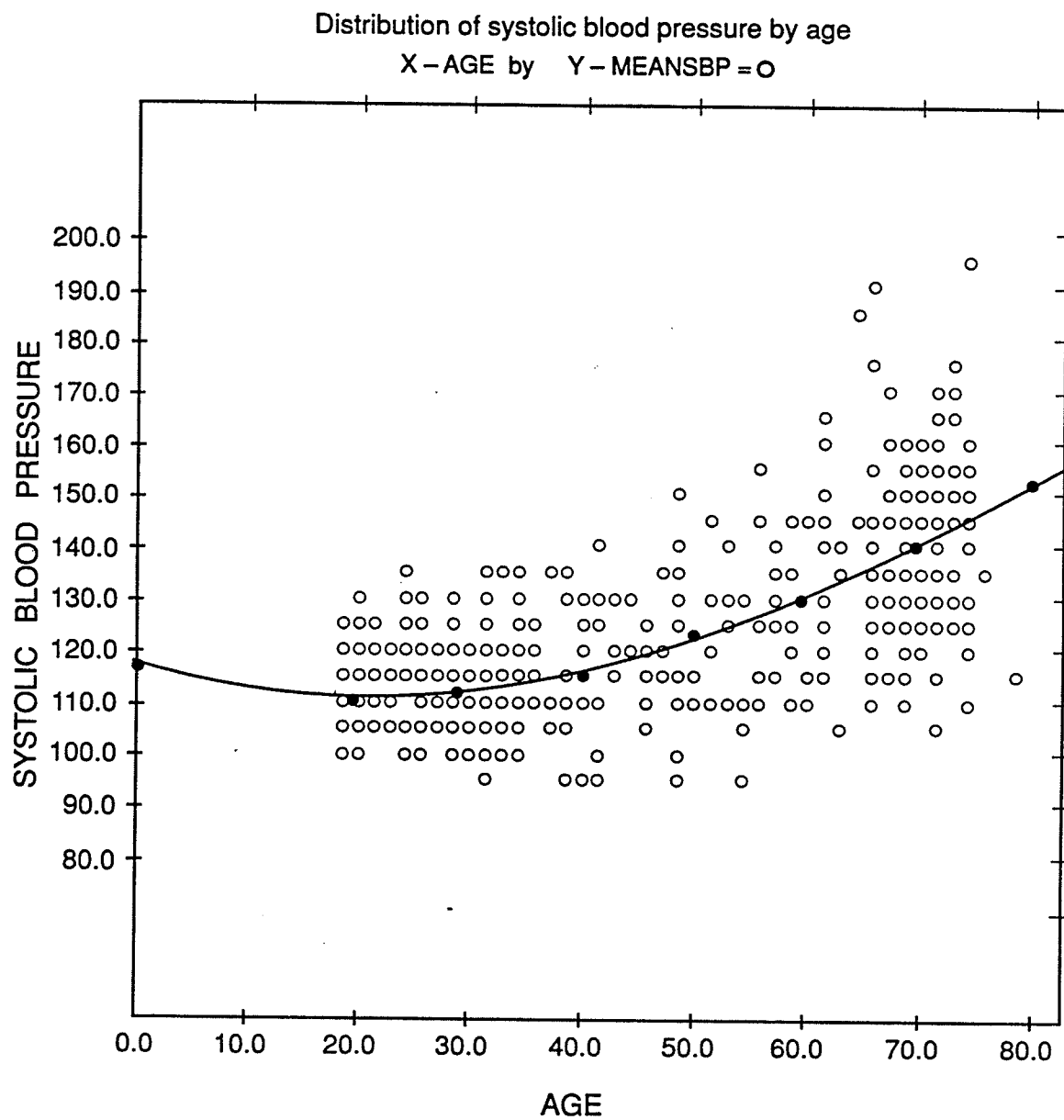
The systolic blood pressure for this sample is at its minimum at 20.43 years. The regression line then curves upward as systolic blood pressue increases, increasing at a faster rate in the older age groups.

4.1.7 Combined Biomedical Risk Factors

The biomedical variables which were shown in univariate analysis to be predictive of hypertensive status in women are presented in Table 4.14.

Table 4.14 Biomedical Risk Factors Which are Predictive of Hypertensive Status in Women in Univariate Analysis

Variable	Risk	Probability Level
Age	$t = -9.34$	0.0000
Height	$t = 2.65$	0.008
Waist	$t = -4.23$	0.0000
Hip	$t = -3.09$	0.002
WHR	$t = -3.62$	0.0003
BMI	$t = -2.95$	0.003
Total Cholesterol	$t = -4.89$	0.0000
HDL Cholesterol	$t = 2.19$	0.029
LDL Cholesterol	$t = -4.29$	0.0000
Triglycerides	$t = -6.29$	0.0000
Total/HDL Ratio	$t = -3.62$	0.0003
History of heart attack	$x^2 = 12.26$	0.0005
History of other heart disease	$x^2 = 34.65$	0.0000
Take medication for heart	$x^2 = 83.08$	0.0000
Family history of heart disease	$x^2 = 21.85$	0.0000
Family history of high blood pressure	$x^2 = 3.85$	0.05
Family history of stroke	$x^2 = 21.00$	0.0000
Diabetes	$x^2 = 6.07$	0.01
Glucose	$t = -3.82$	0.0001

The relationship between each of these risk factors and hypertensive status, is linear and independent as shown in Figure 4.3. Univariate analysis accounts for the relationship between one risk factor and the dependent variable. It does not account for the relationships between the other risk factors and the same dependent variable.

Figure 4.3 Biomedical Risk Factors Which are Predictive of Hypertensive Status in Women in Univariate Analysis
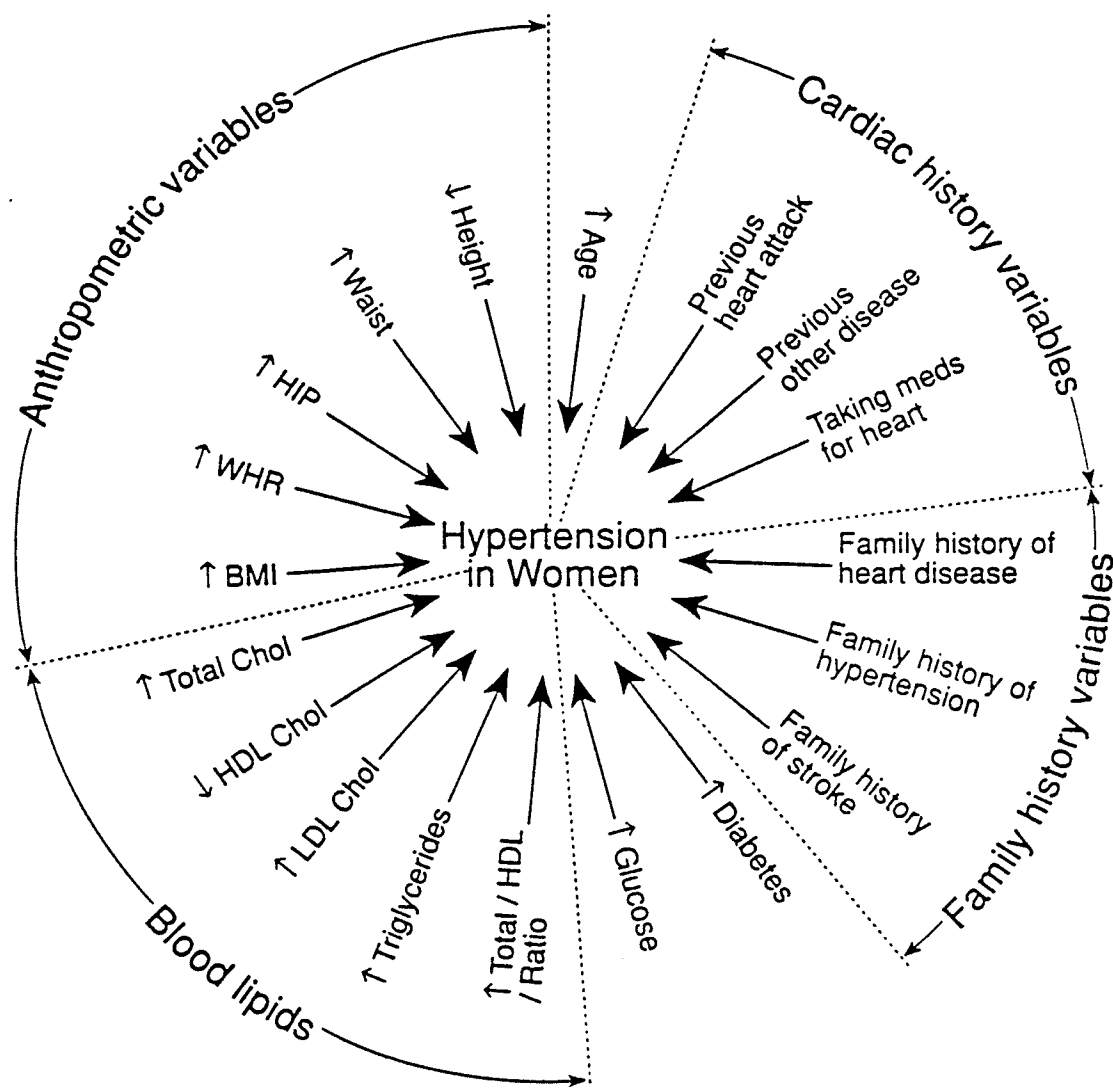
The biomedical variables which were shown to have unique predictive ability when each group of variables such as anthropometrics, blood lipids, cardiac history and family history was analysed using multivariate analyses, are presented in Table 4.15. The other predictors identified in univariate analysis did not appear because their predictive power is effectively duplicated by one or more of the variables left in the model. The variables left in the model, most effectively characterize the impact of each of the biomedical groups of variables on hypertensive status in women.

**Table 4.15 Biomedical Predictors of Hypertensive Status
in Women from Reduced Logistic Models**

Predictive Variable	Chi Square Beta = 0
Age	54.50
Height	7.24
Waist	16.66
Total/HDL Ratio	22.06
HDL	5.75
History of other heart disease	9.50
Take medication for heart	6.04
Family history of heart disease	11.87
Family history of stroke	9.89
Glucose	10.40

The relationship between each of these risk factors and hypertensive status is linear, but takes into consideration the effects of the other risk factors present in that specific logistic model and their relationship with the dependent variable. Each of the risk factors is the most optimally predictive risk factor from each of the reduced logistic models which were representative of the groups of biomedical variables. These risk factors are each representative of a group of variables, but most are independent of each other. For example: Total/HDL cholesterol ratio and HDL cholesterol level represent the blood lipids group of variables, but are independent of the other biomedical predictors from other logistic models. They are however not independent of each other or the other lipid measures. These relationships are shown in Figure 4.4.

Figure 4.4 Biomedical Predictors of Hypertensive Status in Women in Reduced Logistic Models
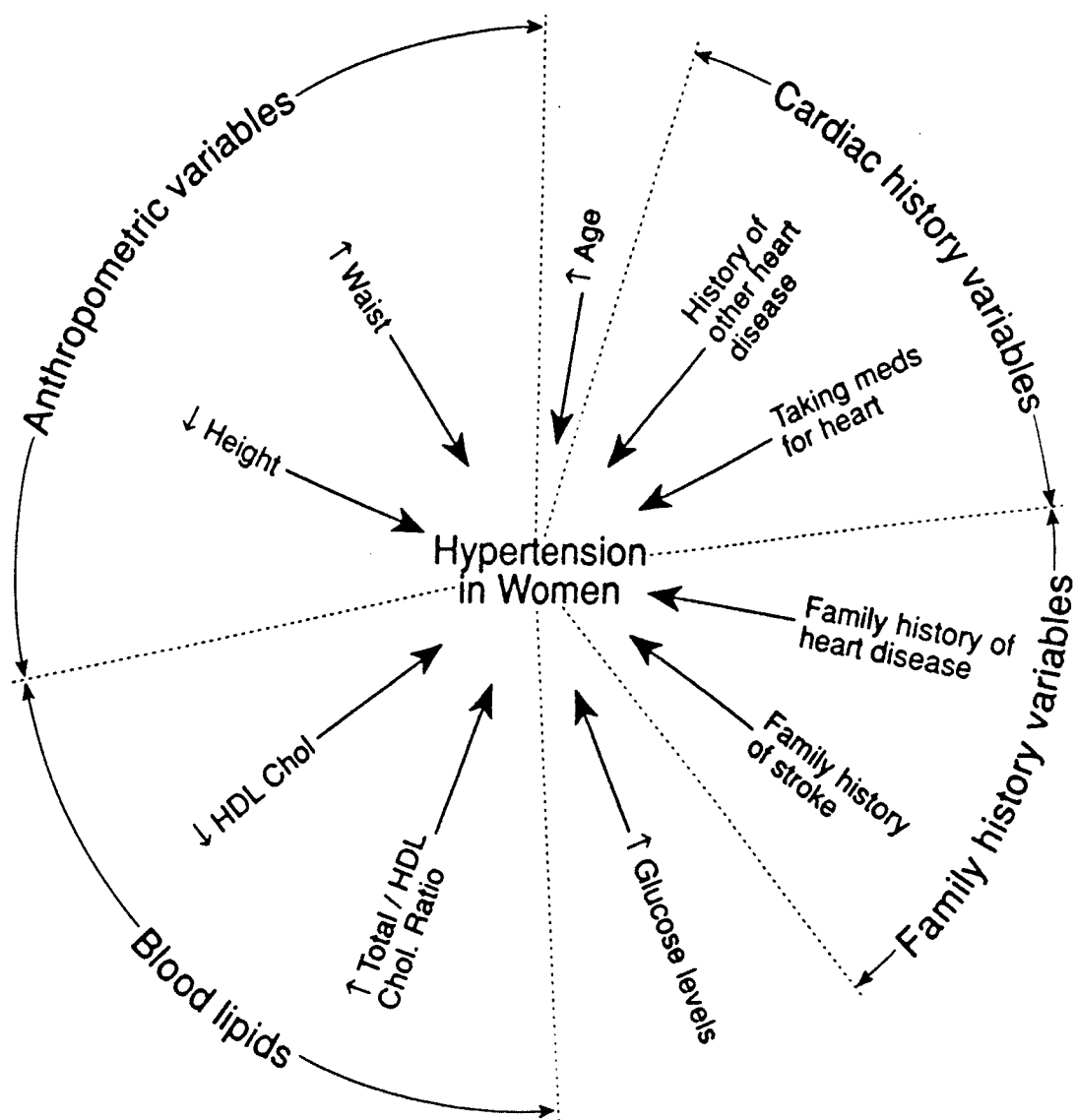
To address the question: *which biomedical variables are optimally predictive of hypertensive status in women,* all the variables which were predictive of hypertensive status in reduced logistic models were considered simultaneously using a logistic regression model. A significant relationship between these predictive variables and hypertensive status was again observed (Model chi square = 63.96 with 10 d.f., $p<0.0000$).

A step-wise logistic regression eliminated the variables which did not reach statistical significance at the 0.05 level of probability and is shown in Table 4.16.

Table 4.16 Combined Biomedical Variables Predictive of Hypertensive Status in Women in Reduced Logistic Model

Predictive Variables	Beta Estimate	Standard Error	Chi Square Beta = 0
Age	.048	.012	14.84
History of other heart disease	-1.23	.446	7.65
Total/HDL Ratio	.493	.142	12.09

A reduced logistic model included: age, history of other heart disease and Total/HDL cholesterol ratio as the variables which showed unique predictive ability. These variables were most effective in characterizing the impact of biomedical variables on hypertensive status in women when all predictive variables from reduced logistic models were considered as shown in Figure 4.5.

Figure 4.5 Biomedical Predictors of Hypertensive Status
in Women in Final Reduced Logistic Model
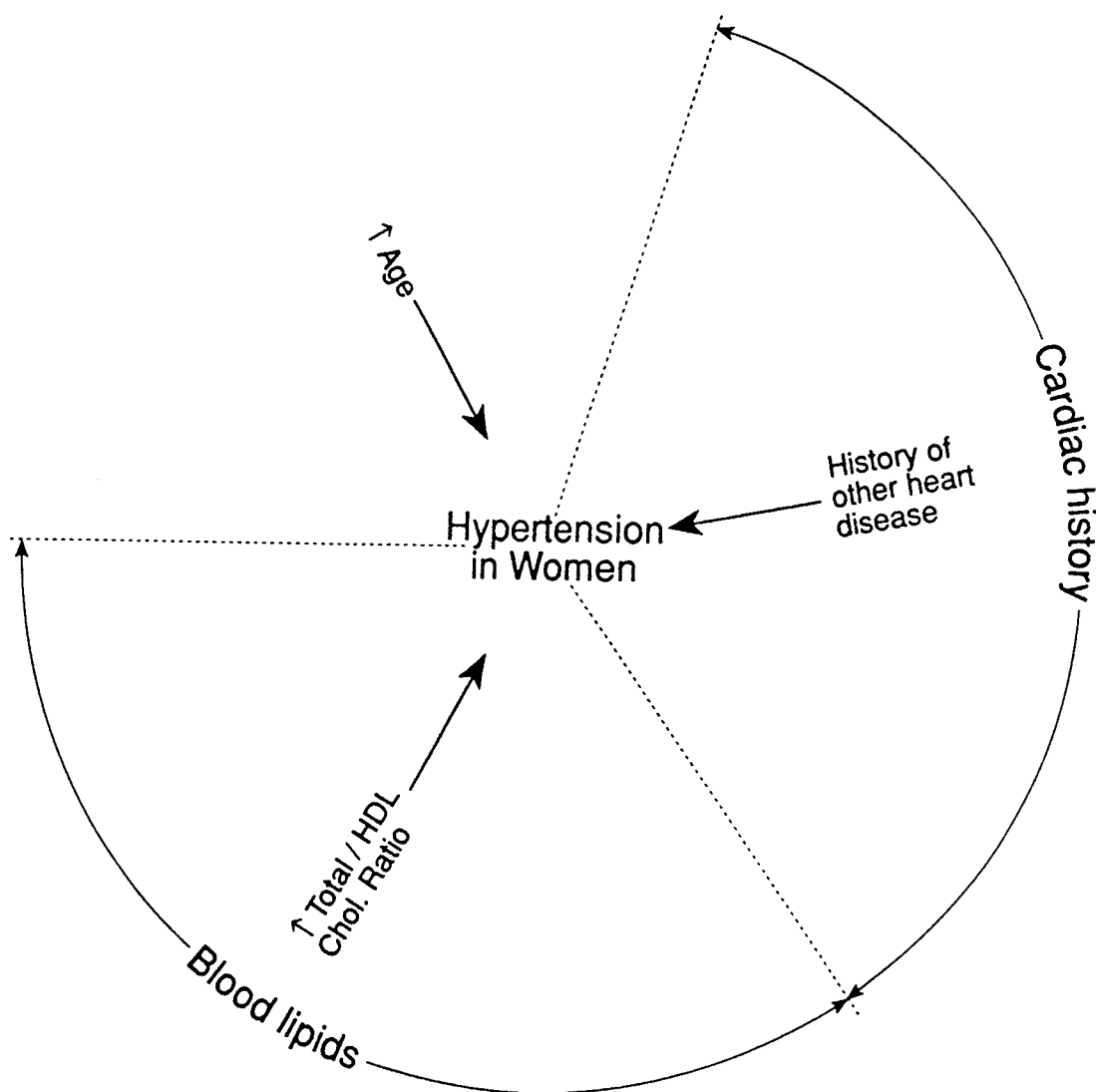
The relationships between each of these variables and hypertensive status in women is linear, but takes into consideration the effects of the other predictive variables from reduced logistic regression models.

4.2   DISCUSSION OF BIOMEDICAL RISK FACTORS

4.2.0   Description of Subjects

The estimated prevalence of hypertension among women in the Manitoba population was reported in the Manitoba Heart Health Study to be about 15 percent (Young et al, 1991). The present data estimate a similar prevalence of about 16 percent for women in the City of Winnipeg who are hypertensive. Approximately 77 percent of the hypertensive women were found to be controlled which is higher than the approximately 50 percent controlled suggested by the literature (Evans & Stoddart, 1990). However, women tend to be controlled more often than men and as this study includes only women, this may account for the discrepancy.

About 77 percent of the hypertensive women in Winnipeg were found to be treated for hypertension, with the vast majority (89 percent) being treated with antihypertensive drugs. This is in contrast with only 53 percent of treated hypertensive women in the Province of Manitoba

being treated with antihypertensive drugs. There is also a discrepancy between the percentage of hypertensive women in Winnipeg who were initially treated with antihypertensive drugs (91 percent), and the percentage of hypertensive women in Manitoba who were initially treated with antihypertensive drugs (71 percent). A majority of women (89 percent in the City of Winnipeg, 87 percent in the Province of Manitoba) were aware of their hypertension.

4.2.1 Discussion of Biomedical Predictors of Hypertension in Women

In addressing the first research question: *what are the identifiable biomedical risk factors which are predictive of hypertension in women*, the results of the present study support the research that both systolic and diastolic blood pressure increase with age. There is no exact blood pressure level below which cardiovascular complications are absent and above which they are present or even sharply increased, for any age group (Amery et al, 1991). The Framingham Heart Health Study cohorts (Kannel, 1981) show that systolic blood pressure in both men and women increased up to age 76, and diastolic blood pressure increased up to age 50, levelled off between 50 and 60, and decreased between ages 60 to 76. When blood pressure was viewed as a continuous variable, the relationship between systolic blood pressure and age indicated that systolic blood pressure

increased up to age 80, increasing at a faster rate in the older groups of women. Diastolic blood pressure reached its maximum at age 61 and decreased slightly from then to age 80. Despite the finding that age is a strong predictor of hypertensive status, studies such as the Framingham Heart Health Study affirm that hypertension is not a normal consequence of aging (Lopez, 1991). Research further suggests that absence of hypertension in the elderly is an important indicator of healthy aging (Guralnik, 1989; Pinsky et al, 1985; Benfante, 1985).

The mean blood lipid level measurements in the sample population (Total cholesterol:5.22; HDL:1.41; LDL:3.19; Triglycerides:1.36) are similar to the mean blood lipid levels published by the Canadian Heart Health Surveys (Total cholesterol:5.13; HDL:1.41; LDL:3.08; Triglycerides:1.38) (Connelly et al, 1992). Compared to non-hypertensive women, those with hypertension were significantly more likely to have higher total cholesterol, LDL cholesterol, triglycerides and Total/HDL cholesterol ratios. Hypertensive women also had lower HDLs.

A consistent positive relationship between blood pressure and blood lipid level in populations has been found (Grimm & Hunninghake, 1986; Bonaa & Thelle, 1991) and the relationship between blood pressure and total cholesterol level has been found to be graded and continuous throughout the usual range of blood pressure (Bonaa & Thelle, 1991). The present data confirm these findings and suggest that HDL and Total/HDL

cholesterol ratio are the blood lipid measures which most effectively characterize the impact of blood lipids on hypertensive status in women. HDL also has been found to be inversely related to CAD in men and women, and in statistical terms has been found to have the strongest relationship of any lipid studied so far (Castelli et al, 1990).

The anthropometric measures published by the Canadian Heart Health Survey (Reeder et al, 1992) included: mean BMI (25.0) and mean WHR (0.78). These were similar to the mean measures in the present data: BMI (25.2) and WHR (0.796). Waist measurement, hip measurement, WHR, BMI, and height were found in the present data to be predictors of hypertensive status in univariate analysis. Hypertensive women were found to have significantly larger waist and hip measurements, significantly larger WHR's, significantly higher BMI's and to be significantly shorter in height than non-hypertensive women. These results were consistent with the research.

In most population based studies, adiposity and the distribution of fat are major determinants of blood pressure levels (Donahue et al, 1990) and cross-sectional studies have shown a positive relationship between obesity and blood pressure in all ages (Voers et al, 1977; Orchard et al, 1980; Higgins et al, 1980; Havlik et al, 1983). Palmer et al (1990) found that shorter women were at greater risk for a first myocardial infarction, in a study of women under age 65.

However, the present data also indicated that the anthropometric measures which were optimally predictive of hypertensive status in women were waist measurement and height. BMI is an index of body ponderosity calculated from height and weight measurements and is often used as a representative measure of anthropometry. The present data show BMI as predictive of hypertensive status in univariate analysis but, in multivariate analysis, it does not contribute any additional information to the variables: waist measurement and height, and thereby does not make a significant contribution to our ability to predict hypertensive status in women.

Hypertension is recognized as one of the three major risk factors for CHD (Leren, 1990) with cigarette smoking, serum cholesterol levels and blood pressure being of approximately equal importance in predicting coronary events (Pooling Project, 1978). Yet the present data on cardiac history variables indicated that history of previous heart attack was predictive of hypertensive status in univariate analysis, but did have unique predictive ability when combined with other cardiac history variables in multivariate analysis. A possible explanation is that this study included only women and the Pooling Project included only men.

The relationship between blood pressure and CHD has been shown to be statistically significant (Dollery, 1987) but research indicates that reduction in high blood pressure has failed to achieve reduction in

coronary events, as would have been expected if hypertension were a predictive factor in CHD (MacMahon et al, 1990; Collins et al, 1990; Hebert et al, 1990). Research further indicates that the hypertensive patients in the Framingham Study had 4.6 times higher incidence of stroke than non-hypertensive patients (Dollery, 1987). The present data on cardiac history variables indicate no significant difference in history of stroke by hypertensive status in women.

The present data indicate that history of heart disease and presently taking medication for heart were optimally predictive of hypertensive status in multivariate analysis of cardiac history variables. Hypertensive women have had significantly more other heart diseases, but what diseases these are, is not specified. Some may have specified hypertension in this category. Likely, taking medication for heart, is optimally predictive of hypertensive status because almost all hypertensive women (91 percent) in the present data are taking medication for their hypertension.

Current epidemiological research suggests that hypertension is related to an interplay of genetic and environmental factors (Ching & Beevers, 1991) and the genetic theory is supported by the high frequency of positive family histories in hypertensives. The present data indicate a family history of heart disease or a family history of stroke are the variables which are optimally predictive of hypertensive status in multivariate analysis of family history variables. Research has shown that

a family history of hypertension is an indicator of risk for developing high blood pressure (Watt, 1986; Miall & Oldham, 1986). The present data found a family history of hypertension to be predictive of hypertensive status in univariate analysis but not to contribute additional information in multivariate analysis of family history variables.

The present data indicate that hypertensive women are significantly more likely to be diabetic than non-hypertensive women, as well as have significantly higher glucose levels. This is consistent with the current research which concludes that hypertension is an insulin-resistant state in itself (Ferrannini et al, 1987). These data may have been confounded by the fact that hypertensive women who were on anti-hypertensive therapy were classified as hypertensive, and insulin-resistance can possibly be reversed by antihypertensive drugs as well as by dietary means (Ferrannini et al, 1987). Although the mechanisms of the association between these conditions are uncertain, it is possible that insulin resistance is a common factor in hypertension as well as obesity and diabetes mellitus (Ching & Beevers, 1991).

The variables which were most effective in characterizing the impact of biomedical variables on hypertensive status in women were age, history of other heart disease, and Total/HDL cholesterol ratio. Age is the strongest independent predictor of hypertensive status, indicating the need for research in the risks of high blood pressure in older women. The mean

age of the hypertensive women was 62 years, considerably older than the non-hypertensive women who had a mean age of 44 years.

Total/HDL cholesterol ratio as an effective predictor of hypertension indicated that cholesterol levels appear to be associated closely with blood pressure levels. The benefit of determining this ratio rather than other cholesterol level measurements is apparent, but it is also expensive compared to doing only a measure of total cholesterol. History of other heart disease is an effective predictor of hypertension in the present data, but its significance in developing future policy is unclear because "other heart disease" was not specified, and the results may have been confounded by subjects who considered hypertension as an "other heart disease".

Some research questions which require further study include:

1. What is a "safe" blood pressure level for older women and how much does the "safe" level change with increasing age?

2. Are antihypertensive drugs effective in reducing cardiovascular morbidity and mortality, or do the adverse effects such as affecting cholesterol levels, outweigh any benefits in hypertensive women?

3.   Would treatment for cholesterol levels be of more benefit to hypertensive women than antihypertensive treatment?

4.   Is comprehensive lifestyle change a feasible alternative to antihypertensive therapy in elderly hypertensive women?

CHAPTER 5

NON-BIOMEDICAL PREDICTORS OF HYPERTENSIVE STATUS:
RESULTS AND DISCUSSION

5.0   NON-BIOMEDICAL RISK FACTOR RESULTS

The second research question: *What are the identifiable non-biomedical risk factors which are predictive of hypertension in women* is addressed by analyzing the following data from the risk factor questionnaire and the clinic visit. The data are analyzed initially using descriptive and then univariate analysis to determine significant differences by hypertensive status. Multivariate analysis is then used to analyze each group of predictive factors and then the entire group of non-biomedical predictors together. A final reduced logistic model which is optimally predictive of hypertensive status is then determined.

5.0.0   Demographics

The demographic characteristics of the subjects are presented in Table 5.1 to Table 5.11.

5.0.0.0 Current Employment Status

The current employment status of the subjects is presented in Table 5.1.

Table 5.1 Distribution of 447 Hypertensive and
Non-Hypertensive Women Subjects
by Employment Status

Current Employment Status	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Full time 35+ hours	170 (38%)	161 (43%)	9 (13%)
Part time < 35 hours	65 (14.5%)	60 (16%)	5 (7%)
Unemployed*	6 (1.0%)	-	-
Laid off*	2 (N/A)	-	-
Retired	97 (22%)	60 (16%)	37 (53%)
Other*	11 (2.5%)	-	-
Homemaker	74 (17%)	58 (15%)	16 (23%)
Student	22 (5%)	22 (6%)	-

- Data suppressed due to small numbers
* Approximately 4 percent of women were: unemployed, laid off, or other

Chi square tests found significant differences in some items of current employment status, by hypertensive status. The relationship between hypertensive status and full-time work (x^2=22.32, p<0.0000) indicated that non-hypertensive women are significantly more likely to work full-time than hypertensive women. The relationship between hypertensive status and part-time work (x^2=3.65, N.S.) indicated that non-hypertensive women appear to be more likely to work part time, but the difference is not significant. The relationship between hypertensive status and being retired (x^2=47.42, p<0.0000) indicated that hypertensive women are significantly more likely to be retired than non-hypertensive women. The relationship between hypertensive status and homemaker status (x^2=2.39, N.S.) indicated that although more hypertensive women are homemakers, the difference is not significant. The relationship between hypertensive status and student status (x^2=4.30, p<0.038) indicates that significantly more non-hypertensive women are likely to be students.

Using a logistic regression model, the employment variables were considered simultaneously in order to address the question: *does employment status bear any relationship to hypertensive status?*. A significant relationship between current employment status and hypertensive status was observed (Model chi square = 57.11 with 4 d.f., p<0.0000).

A step-wise logistic regression was then done which eliminated the variables that did not reach statistical significance at the 0.05 level of probability. Table 5.2 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.2 Employment Variables Predictive of Hypertensive
Status in Women in a Reduced Logistic Model

Employment Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Retired	2.240	.326	47.18
Homemaker	1.436	.377	14.47

In univariate analysis, not working full time, being retired, and not being a student were all found to be predictive of hypertensive status. In a reduced logistic model, only being retired and being a homemaker had unique predictive ability. The other variables did not appear because their predictive power is effectively duplicated by these variables. Being retired and being a homemaker were found to be the employment status variables which most effectively characterize the impact of employment status on hypertensive status in women. Odd ratios indicate that retired women are nine times more likely to be hypertensive than women who work full time, and homemakers are four times more likely to be hypertensive than women who work full time. Age appears to confound these results.

5.0.0.1   Marital Status

The marital status of the subjects is presented in Table 5.3.

Table 5.3 Distribution of 447 Hypertensive and Non-Hypertensive Women by Marital Status

Marital Status	Total Women N = 445[*]	Non-Hypertensive Women N = 375[*]	Hypertensive Women N = 70
Single	117 (26%)	107 (29%)	10 (14%)
Married/ Common Law	280 (63%)	240 (64%)	40 (57%)
Widowed	48 (11%)	28 (7%)	20 (29%)

[*] 2 missing values

Using a chi square test, the relationship between hypertensive status and single status (x^2=6.07, p<0.01) indicated that there are significant differences in single status between hypertensive and non-hypertensive women, with non-hypertensive women being far more likely to be single.

The relationship between hypertensive status and married or common-law status (x^2=1.07, N.S.) indicates no significant difference exists between hypertensive and non-hypertensive women in married status, with most women in each group being married or common-law.

The relationship between hypertensive status and widowed status (x^2=27.54, p<0.0000) indicated a significant difference exists between hypertensive and non-hypertensive women, with hypertensive women being far more likely to be widowed. This result is consistent with the fact that hypertensive women are significantly older and therefore, are more likely to be widowed than younger women.

The marital status variables were considered simultaneously using a logistic regression model in order to address the question: *does marital status bear any relationship to hypertensive status?* A significant relationship between marital status and hypertensive status was observed (Model chi square = 24.45 with 2 d.f., p<0.0000).

A step-wise logistic regression model was then done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.4 provides the beta estimate and standard errors of the statistically significant variables.

**Table 5.4 Marital Status Variables Predictive of Hypertensive
Status in Women in Reduced Logistic Model**

Marital Status	Beta Estimate	Standard Error	Chi Square Beta = 0
Widowed	1.607	.329	23.77

In univariate analysis, being single and being widowed were both found to be predictive of hypertensive status. In a reduced logistic model,

only being widowed had unique predictive ability. Therefore being widowed was the marital status variable which most effectively characterized the impact of marital status on hypertensive status in women. An odds ratio indicates that widowed women are five times more likely to be hypertensive than non-widows.

5.0.0.2   Education

The education level completed for the subjects is presented in Table 5.5.

Table 5.5 Distribution of 447 Hypertensive and Non-Hypertensive Women by Education Completed

Education Completed	Total Women N = 439[*]	Non-Hypertensive Women N = 371[**]	Hypertensive Women N = 68[***]
No schooling	1 (-)	--	0 (0%)
Elementary (Grade 6)	75 (17%)	56 (15%)	19 (28%)
Secondary (Grade 12)	197 (45%)	167 (45%)	30 (44%)
Post-Secondary	166 (38%)	148 (40%)	18 (26%)
Not sure/ No response	--	--	--

--   Data suppressed due to small numbers
[*]   8 values missing
[**]   6 values missing
[***]   2 values missing

Chi square tests found significant differences in some items of education completed, by hypertensive status.

The relationship between hypertensive status and grade 6 education completed (x^2=6.39, p<0.01) indicated a significant difference between hypertensive and non-hypertensive women. Hypertensive women were more likely to have grade 6 as the highest grade completed in school. The relationship between hypertensive status and grade 12 education completed (x^2=.05, N.S.) indicated no real differences exist between the two groups of women in grade 12 education completed. Both groups have nearly half their subjects in this group.

The relationship between hypertensive status and post-secondary education (x^2 = 4.64, p<0.03) indicates real differences exist between the two groups, with non-hypertensive women significantly more likely to have completed post-secondary education. Using a logistic regression model, education completed variables were considered simultaneously to address the question: *does education level completed bear any relationship to hypertensive status?* A significant relationship between education and hypertensive status was observed (Model chi square = 9.33 with 2 d.f., p<0.009).

A step-wise logistic regression model was then done which eliminated the variables that did not reach statistical significance at the 0.05

level of probability. Table 5.6 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.6 Education Level Variables Predictive of
          Hypertensive Status in Women in
          Reduced Logistic Model

Education Level	Beta Estimate	Standard Error	Chi Square Beta = 0
Grade 6	-.681	.318	4.59
Post-Secondary	-1.071	.352	9.25

In univariate analysis, grade 6 education and post-secondary education were found to be predictive of hypertensive status. In a reduced logistic model, grade 6 education and post-secondary education were found to have unique predictive ability.

Odds ratios indicate that women with only grade 6 completed were almost twice as likely to be hypertensive as women with grade 12 completed, and three times more likely than women with post-secondary education completed.

5.0.0.3 Language First Spoken

The language first spoken by the subjects is presented in Table 5.7.

Table 5.7 Distribution of 447 Hypertensive and Non-Hypertensive Women by Language First Spoken

Language	Total Women N = 446[*]	Non-Hypertensive Women N = 376[*]	Hypertensive Women N = 70
English	316 (71%)	272 (72%)	44 (63%)
French	21 (5%)	18 (5%)	3 (4%)
Other	109 (24%)	86 (23%)	23 (33%)

[*] 1 missing value

The majority of subjects in both groups report English as the language first spoken. The relationship between hypertensive status and French language first spoken (x^2=.03, N.S.) indicated no real difference between the groups in French first spoken. Both groups had small numbers of subjects who used French as the language first spoken.

The relationship between hypertensive status and other languages than English or French spoken first (x^2=3.23, N.S.) indicated no real differences exist between the groups. The hypertensive women tend to be more likely to have spoken other languages first, but the difference is not significant.

Using a logistic regression model, the language first spoken variables were considered simultaneously in order to answer the question: *does language first spoken bear any relationship to hypertensive status?* A significant relationship between language first spoken and hypertensive status was not observed (Model chi square = 3.06, 2 d.f., p<0.22).

No further analysis was done as the model did not help to explain real differences in hypertensive status.

5.0.0.4   Number of People in the Household

The number of people in the household in which the subjects live is presented in Table 5.8.

Table 5.8   Distribution of 447 Hypertensive and Non-Hypertensive Women by Number of People in the Household

Number of People	Total Women N = 443[*]	Non-Hypertensive Women N = 376[**]	Hypertensive Women N = 67[***]
1 person	68 (15%)	47 (13%)	21 (31%)
2 people	160 (36%)	131 (35%)	29 (43%)
3 people	95 (21%)	81 (22%)	14 (21%)
4 people	82 (19%)	80 (21%)	2 (3%)
5 people	29 (7%)	28 (7%)	1 --
6 people	7 (2%)	--	--
7 people	--	--	--

[*]   4 missing values
[**]   1 missing value
[***]   3 missing values

The relationship between hypertensive status and number of people in the household was done using a composite of the number of people in the household. The analysis indicates (t = 5.23, p<0.0000) that there is a significant difference between the mean values of the groups. The hypertensive women (mean value 2.0) tend to have smaller households than the non-hypertensive women (mean value 2.84).

When the number of people in household variables were considered simultaneously using a logistic regression model, a significant relationship between the number of people in household and hypertensive status was observed (Model chi square = 30.06 with 1 d.f., p<0.0000).

Table 5.9 Multiple Logistic Regression Analysis of Number of People in the Househould Variable

Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Number of people in household	-.698	.143	23.80

The logistic regression model indicated that number of people in household is an independent predictor of hypertensive status.

5.0.0.5 Income

The income category of the subjects is presented in Table 5.10.

Table 5.10 Distribution of 447 Hypertensive and Non-Hypertensive Women by Income Category

Income Category	Total Women	Non-Hypertensive Women N = 372[**]	Hypertensive Women N = 69[***]
Under $12,000	36 (8%)	26 (7%)	10 (14%)
$12-$24,999	100 (23%)	80 (22%)	20 (29%)
$25-$49,999	151 (34%)	130 (35%)	21 (30%)
$50-74,999	75 (17%)	70 (19%)	5 (7%)
$75,000+	24 (5%)	23 (6%)	1 (0%)
Refused to answer	55 (12%)	43 (12%)	12 (17%)

Univariate analysis using a chi square test was done for each income category. Chi square tests found significant differences in some income categories, by hypertensive status.

The relationship between hypertensive status and income under $12,000. ($x^2$=4.35, p<0.03) indicates that there are real differences between

the means of the two groups. The hypertensive women tend more often than non-hypertensive women to have household incomes of under $12,000. Income categories $12,000 - $24, 999. ($x^2=1.84$, N.S.) and $25,000. - $49,999. ($x^2=0.53$, N.S.) indicate no real difference exist between these groups. The relationship between hypertensive status and income $50,000. - $74,999. ($x^2=5.52$, P<0.019) indicates that non-hypertensive women are significantly more likely to be in this income group. In the income category $75,000. and up there is only 1 hypertensive woman compared to 23 non-hypertensive women, but the relationship did not show significance at the 0.05 level of probability and therefore is not significant ($x^2=2.537$, N.S.).

Using a logistic regression model, the income category variables were considered simultaneously to address the question: *does income level bear any relationship to hypertensive status?* A significant relationship between income category and hypertensive status was observed (Model chi square = 15.05 with 5 d.f., p<0.01).

A stepwise logistic regression was then done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.11 provides the beta estimate and standard error of the variable which was statistically significant.

Table 5.11 Income Category Variable Predictive of Hypertension
Status in Women in Reduced Logistic Model

Income Category	Beta Estimate	Standard Error	Chi Square Beta = 0
$50-$74,999	-1.087	.483	5.07

In univariate analysis, income under $12,000 and income in the $50,000-$74,999 range were found to be predictive of hypertensive status. In a reduced logistic model, only income in the $50,000-$74,999 range had unique predictive ability.

An odds ratio indicates that women with household incomes under $50,000 per annum are almost three times more likely to be hypertensive than women with household incomes in the $50,000-$74,999 range.

5.0.0.6 Combined Demographic Variables

To address the question: *which demographic variables are optimally predictive of hypertensive status in women,* all of the variables which were predictive of hypertensive status in univariate analysis were considered simultaneously using a logistic regression model. A significant relationship was observed between these demographic variables and hypertensive status (Model chi square = 77.60 with 18 d.f., $p < 0.0000$).

A stepwise logistic regression model was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.12 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.12 Demographic Variables Predictive of Hypertensive
Status in Women in Reduced Logistic Model

Demographic Variables	Beta Estimate	Standard	Chi Square Beta = 0
Number of people in household	-.489	.159	9.43
Homemaker	1.738	.428	16.49
Retired	1.828	.356	26.40

A reduced logistic regression model indicated that number of people in household, being a homemaker, and being retired are the demographic variables which showed unique predictive ability. These variables were most effective in characterizing the impact of demographic variables on hypertensive status in women.

5.0.1   Physical Activity

More than half of the women in the sample (54%) reported engaging in physical activity regularly. However, when the physical activity variable was considered using a logistic regression model to address the question: *does regular physical activity bear any relationship to hypertensive status,* a non-significant relationship between engaging in physical activity regularly and hypertensive status was observed (Model chi square = 0.54 with 1 d.f., p<0.462).

The type of physical activity in which subjects engage and how it compares to others their age is presented in Table 5.13.

Table 5.13 Type of Daily Physical Activity of 447 Hypertensive and Non-Hypertensive Women

Variable	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Usual day time activity:			
Sit/not much walking	87 (19%)	77 (20%)	10 (14%)
Stand/walk a lot	210 (47%)	166 (44%)	44 (63%)
Lift or carry light loads	140 (31%)	126 (33%)	14 (20%)
Heavy work	8 (2%)	7 (-)	1 (1%)
Feel you get the exercise you need	123 (28%)	103 (27%)	20 (29%)
Feel you are as active as others your age	169 (38%)		26 (37%)
More active	144 (32%)		22 (31%)
Less active	114 (26%)		17 (24%)

Chi square tests found significant differences in some variables, by hypertensive status. There is a significant difference in the groups in those who usually stand or walk a lot in their daytime activity with hypertensive women tending to have this type of daily activity more often than non-hypertensive women (x^2=8.40, p<0.004). Non-hypertensives are significantly more likely to do daytime activity which involves lifting or carrying light loads or climbing stairs or hills (x^2=4.94, p<0.03). There was no significant difference between the groups who sit or don't walk much in their daytime activity.

There was no significant difference between the groups in the number who felt they get as much exercise as they need (x^2=0.05, N.S.). Both groups had less than a third of the group (hypertensive women 29%, non-hypertensive women 27%) who felt they were getting the exercise they need.

There were no significant differences between the groups whether they felt they were more active, less active or about the same as others their age (x^2=1.39, N.S.).

Using a logistic regression model, the usual daily physical activity variables were considered simultaneously to address the question: *does type of physical activity in which subjects engage bear any relationship to hypertensive status?* A significant relationship between daily physical activity and hypertensive status was observed (Model chi square = 8.76 with 2 d.f., p<0.01).

A stepwise logistic regression was then done which eliminated the variables that did not reach statistical significance at the 0.05 level of probability. Table 5.14 provides the beta estimate and standard error of the only variable which was statistically significant.

Table 5.14 Daily Physical Activity Variables
          Predictive of Hypertensive Status
          in Women in Reduced Logistic Model

Daily Activity	Beta Estimate	Standard Error	Chi Square Beta = 0
Stand/walk a lot	.766	.268	8.15

In univariate analysis, stand/walk a lot as a daily activity and lifting or carrying light loads as a daily activity were predictive of hypertensive status. In a reduced logistic model, only stand/walk a lot had unique predictive ability. Women are twice as likely to be hypertensive if their daily activity involves standing/walking a lot as usual daily activity compared to the other daily activities.

The nature of physical activity of the subjects is presented in Table 5.15.

Table 5.15 Nature of Physical Activity of Hypertensive
and Non-Hypertensive Women Who Engage in
Physical Activity

Nature of Physical Activity	Total Women N = 243	Non-Hypertensive Women N = 203	Hypertensive Women N = 40
1. Most physical activity is strenuous enough to cause sweating/heavy breathing	91 (37%)	86 (42%)	5 (13%)
2. Exercise for:			
<15 minutes	24 (10%)	16 (8%)	8 (20%)
15 - 30 minutes	66 (27%)	54 (27%)	12 (30%)
30 - 60 minutes	115 (47%)	97 (48%)	18 (45%)
More than 1 hour	35 (14%)	33 (16%)	2 (5%)
3. Exercise at least 15 minutes:			
Daily	50 (21%)	41 (20%)	9 (23%)
5 - 6 times a week	27 (11%)	21 (10%)	6 (15%)
3 - 4 times a week	87 (36%)	76 (37%)	11 (28%)
1 - 2 times a week	68 (28%)	58 (29%)	10 (25%)

Chi square tests found significant differences in some items of

nature of physical activity, by hypertensive status. There is a significant

difference between the hypertensive and non-hypertensive women who

exercise with non-hypertensive women more likely to exercise strenuously

enough to cause sweating or breathing heavily most of the time (x^2=14.25, p<0.0008).

Of those who exercise, the hypertensive women are more likely than the non-hypertensive women to exercise for less than 15 minutes at a time (x^2=9.66, p<0.014), but there is no significant difference between the two groups in numbers who exercise for 15 to 30 minutes (x^2=0.37, N.S.), 30 to 60 minutes ($X2$ = 0.00, N.S.), or more than one hour (x^2=2.84, N.S.).

There is no significant difference between the groups in those who exercise daily for at least 15 minutes (x^2=0.233, N.S.), 5 - 6 times per week (x^2=0.94, N.S.), 3 - 4 times per week (x^2=0.74, N.S.), or 1 - 2 times per week (x^2=0.055, N.S.).

Using a logistic regression model, all the nature of physical activity variables were considered simultaneously to address the question: *does the nature of physical activity which is done bear any relationship to hypertensive status?* A significant relationship between these physical activity variables and hypertensive status was observed (Model chi square = 19.22 with 7 d.f., p<0.008).

A stepwise logistic regression was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.16 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.16 Nature of Physical Activity Variables
Predictive of Hypertensive Status
in Women in a Reduced Logistic Model

Nature of Activity	Beta Estimate	Standard Error	Chi Square Beta = 0
Most physical activity is strenuous enough to cause sweating/heavy breathing	-.836	.234	12.77

In univariate analysis, exercising strenuously and exercising for less than 15 mintues were predictive of hypertensive status. In a reduced logistic model, only exercising strenuously had unique predictive ability. Non-hypertensive women are significantly more likely to do this type of physical activity. Doing physical activity strenuously enough to cause sweating/heavy breathing is the variable which most effectively characterizes the impact of physical activity on hypertensive status in women.

The self reported reasons of the subjects for not being more active are presented in Table 5.17.

Table 5.17 Reasons For Not Being More Active

Reasons	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
1. Lack of time	160 (36%)	147 (39%)	13 (19%)
2. Lack of transportation	26 (6%)	17 (5%)	9 (13%)
3. Lack of money	44 (10%)	35 (9%)	9 (13%)
4. Lack of available facilities in community	59 (13%)	50 (13%)	9 (13%)
5. Lack of interesting/relevant activities	79 (18%)	65 (17%)	14 (20%)
6. Illness or disability	72 (16%)	49 (13%)	23 (33%)
7. Lack of incentive	213 (48%)	184 (49%)	29 (41%)
8. No one to exercise with	121 (27%)	103 (27%)	18 (26%)
9. Other reasons	45 (10%)	40 (11%)	5 (7%)

Chi square tests found significant differences in some stated reasons for not being more active, by hypertensive status. Non-hypertensive women are significantly more likely to find lack of time prevents them

from being more active (x^2=10.71, p<0.001), whereas hypertensive women are more likely than non-hypertensive women to find lack of transportation prevents them from being more active (x^2=7.51, p<0.006).

Lack of money (x^2=0.85, N.S.), lack of easily available facilities in community (x^2=0.01, N.S.), and lack of interesting or relevant activities as reasons for not being more active, are not significantly different between hypertensive and non-hypertensive women. Hypertensive women are more likely to give illness or disability as a reason for not being more active (x^2=17.23, p<0.0000). No significant differences exist between reasons for not being more active such as lack of incentive or no one to exercise with.

Using a logistic regression model to address the question: *do stated reasons for not being more active, bear any relationship to hypertensive status,* the reasons for not being more active were considered simultaneously. A significant relationship between the reasons and hypertensive status was observed (Model chi square = 34.24 with 8 d.f., p<0.0001).

A stepwise logistic regression was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.18 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.18 Reasons for Not Being More Active Variables
Predictive of Hypertensive Status in
Women in Reduced Logistic Model

Reasons	Beta Estimate	Standard Error	Chi Square Beta = 0
Lack of time	-1.044	.333	9.80
Lack of transportation	1.180	.471	6.28
Illness or disability	1.092	.307	12.66

In univariate analysis, lack of time, lack of transportation and illness or disability as a reason for not being more active, were variables which were predictive of hypertensive status. In a reduced logistic regression model, these three variables each had unique predictive ability.

Non-hypertensive women are significantly more likely not to be more active because of lack of time and hypertensive women cite lack of transportation or illness/disease as reasons for not being more active.

5.0.2 Smoking

The smoking behaviors of the subjects are presented in Table 5.19.

Table 5.19 Smoking Behavior of 447 Hypertensive and Non-Hypertensive Women

Smoking Behavior	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Have you ever smoked?	256 (57%)	219 (58%)	37 (53%)
At the present time, do you smoke?	113 (44%)*	103 (47%)*	10 (27%)*
At the present time, do you smoke regularly?	92 (81%)**	82 (80%)**	10 (100%)**

* Out of the number of women in the given category who had ever smoked.

** Out of the number of women in the given category who are present smokers.

Chi square tests were used to analyse smoking behaviors by hypertensive status. The relationship between hypertensive status and those who have ever smoked ($x^2=0.66$, N.S.) indicated that there was no significant difference between hypertensive and non-hypertensive women.

The relationship between those who presently smoke and hypertensive status (x^2=5.25, p<0.02) indicated that non-hypertensive women are significantly more likely to presently smoke than hypertensive women. In fact, only 10 out of the 113 present smokers are hypertensive.

There was no significant difference between the groups in those who smoke regularly (x^2=2.50, N.S.), with most present smokers being regular smokers.

When the variable presently smoke was considered using a logistic regression model to address the question: *does the behavior presently smoking bear any relationship to hypertensive status,* a significant relationship between presently smoke and hypertensive status was observed (Model chi square = 5.48 with 1 d.f., p<0.02).

Non-hypertensive women are significantly more likely to be presently smoking than hypertensive women.

The self reported reasons why subjects gave up smoking are presented in Table 5.20.

Table 5.20 Reasons for Giving Up Smoking

Reason	Total Women N = 113	Non-Hypertensive Women N = 103	Hypertensive Women N = 10
To improve fitness			
Very important	60 (53%)	57 (55%)	3 (30%)
Somewhat important	39 (34%)	35 (34%)	4 (40%)
Not important	14 (12%)	11 (11%)	3 (30%)
To prevent disease/ ill health			
Very important	92 (81%)	87 (84%)	5 (50%)
Somewhat important	19 (17%)	15 (15%)	4 (40%)
Not important	2 (2%)	1 (1%)	1 (10%)
To set a good example to family			
Very important	45 (41%)	41 (40%)	4 (40%)
Somewhat important	32 (29%)	29 (28%)	3 (30%)
Not important	32 (29%)	29 (28%)	3 (30%)
To save money			
Very important	58 (51%)	54 (52%)	4 (40%)
Somewhat important	31 (27%)	30 (29%)	1 (10%)
Not important	24 (21%)	19 (18%)	5 (50%)
To demonstrate self control			
Very important	33 (30%)	29 (28%)	4 (40%)
Somewhat important	46 (41%)	44 (43%)	2 (20%)
Not important	30 (27%)	26 (25%)	4 (40%)
To respect the wishes of non-smokers			
Very important	41 (37%)	36 (35%)	5 (50%)
Somewhat important	39 (35%)	35 (34%)	4 (40%)
Not important	31 (28%)	30 (29%)	1 (10%)
To be sociable			
Very important	12 (11%)	10 (10%)	2 (20%)
Somewhat important	34 (30%)	34 (33%)	0
Not important	66 (59%)	58 (56%)	8 (80%)

There were no significant differences between hypertensive and non-hypertensive women in the importance of the following reasons for giving up smoking: to improve fitness ($x2=3.94$, N.S.), to set a good example to family ($x2=0.21$, N.S.), to demonstrate self control ($x2=2.25$, N.S.), to respect the wishes of non smokers ($x2=1.85$, N.S.), to be sociable ($x2=5.04$, N.S.), or to be more attractive ($x2=0.64$, N.S.). However, there were significant differences in giving up smoking to prevent disease/ill health, with non-hypertensive women more likely to think this is an important reason for giving up smoking ($x2=9.04$, $p<0.01$). Non-hypertensive women were more likely to think saving money is an important reason for quitting smoking ($x2=5.76$, $p<0.056$) although the difference was close but not significant.

Using a logistic regression model, all the reasons for giving up smoking were considered simultaneously to address the question: do reasons for giving up smoking bear any relationship to hypertensive status? A non-significant relationship between reasons for giving up smoking and hypertensive status were observed (Model chi square = 13.75 with 8 d.f., $p<0.09$). Reasons for giving up smoking do not have any unique predictive ability in relation to hypertensive status.

5.0.3   Alcohol

The present data indicate that most of the women have taken an alcoholic drink (non-hypertensive, 97 percent and hypertensive, 96 percent).

Most have also consumed alcohol in the past 12 months (non-hypertensive, 86 percent, and hypertensive, 80 percent).

The number of drinks consumed on average throughout the day is presented in Table 5.21.

Table 5.21 Daily Alcohol Consumption of 447
Hypertensive and Non-Hypertensive Women

Variable	Total Women N = 369	Non-Hypertensive Women N = 314 Mean +-S.E.	Hypertensive Women N = 55 Mean +-S.E.
Number of drinks consumed per day		2.487+-0.101	1.709+-0.137

Using a logistic regression model, the number of drinks consumed per day was considered to address the question: does daily alcohol consumption bear any relationship to hypertensive status? A significant

relationship between number of drinks/day and hypertensive status was observed (Model chi square = 13.54 with 1 d.f., p<0.0002).

The logistic regression model indicated that number of drinks per day was an independent predictor of hypertensive status in women.

5.0.4   Health Beliefs

5.0.4.0   Health Beliefs Concerning Heart Disease and Stroke

The belief that heart disease can be prevented was shared by about half the women in each group (non-hypertensive, 54%, and hypertensive, 56%).

The subjects' beliefs of the major causes of heart disease/heart problems are presented in Table 5.22.

Table 5.22 Beliefs of the Major Causes of Heart Disease/
Heart Problems

Causes	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Poor Diet	256 (57%)	232 (62%)	23 (34%)
Overweight	166 (37%)	138 (37%)	28 (40%)
Excess Fat	108 (24%)	94 (25%)	14 (20%)
Excess Salt	61 (14%)	53 (14%)	8 (11%)
High Blood Cholesterol Level	99 (22%)	83 (22%)	16 (23%)
Foods with High Cholesterol	51 (11%)	43 (11%)	8 (11%)
Excess Stress, Worry or Tension	220 (49%)	183 (49%)	37 (53%)
Overwork or Fatigue	42 (9%)	35 (9%)	7 (10%)
Lack of Exercise	214 (48%)	194 (51%)	20 (29%)
Smoking	212 (47%)	187 (50%)	25 (36%)
Heredity	194 (43%)	166 (44%)	28 (40%)
High Blood Pressure/ Hypertension	81 (18%)	70 (19%)	11 (16%)
Atherosclerosis/Hardening of Arteries	16 (4%)	15 (4%)	1 (1%)
Other	148 (33%)	130 (34%)	18 (26%)
Not Sure	21 (5%)	17 (5%)	4 (6%)

Chi square tests found significant differences in some beliefs, by hypertensive status. The relationship between hypertensive status and the belief that poor diet is a major cause of heart disease ($x2=17.92, p<0.0000$) is highly significant and indicated that non-hypertensive women are more likely to believe poor diet is a major cause of heart disease.

No significant difference was found between the groups in the belief that overweight ($x2=0.29$, N.S.) or excess fat ($x2=0.78$, N.S.), or excess salt ($x2=0.35$, N.S.), or high blood cholesterol level ($x2=0.02$, N.S.) or food with high cholesterol ($x2=0.00$, N.S.) is a major cause of heart disease.

The relationship between excess stress, worry or tension and hypertensive status is not significantly different between the hypertensive and non-hypertensive women ($x2=0.44$, N.S.), but approximately half of the women in each group believe that excess stress, worry or tension are causes of heart disease. This percentage is higher than any other mentioned cause.

No significant difference between the groups is found in belief that overwork or fatigue ($x2=0.04$, N.S.) is a major cause of heart disease.

A highly significant difference was found between the groups in the belief that lack of exercise is a major cause of heart disease ($x2=12.39$, $p<0.0004$), with the non-hypertensive women being far more likely to believe lack of exercise is a major cause of heart disease.

The non-hypertensive women are significantly more likely to believe smoking is a major cause of heart disease ($x2=4.57$, $p<0.03$).

There is no significant difference between the groups in the belief that heredity ($x2=0.39$, N.S.), hypertension ($x2=0.32$, N.S.), artherosclerosis ($x2=1.11$, N.S.) or other causes not listed ($x2=2.05$, N.S.) were major causes of heart disease. There was also no significant difference between the hypertensive women and non-hypertensive women in those who were unsure of the major causes.

Using a logistic regression model, all the beliefs of major causes of heart disease variables were considered simultaneously to address the question: do beliefs of the major causes of heart disease bear any relationship to hyertensive status? A significant relationship between beliefs and hypertensive status was observed (Model chi square = 31.16 with 15 d.f., $p<0.008$).

A step-wise regression was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.23 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.23 Beliefs of Major Causes of Heart Disease
which are Predictive of Hypertensive Status
in Women in Reduced Logistic Model

Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Poor Diet	-1.119	.274	16.61
Smoking	-.568	.275	4.27

In univariate analysis, the beliefs: poor diet, lack of exercise, and smoking were causes of heart disease, were predictive of hypertensive status, but in a reduced logistic model, only poor diet and smoking had unique predictive ability. Therefore, the beliefs that poor diet and smoking were major causes of heart disease, were the most effective variables to characterize the impact of beliefs about causes of heart disease, on hypertensive status in women. Non-hypertensive women are significantly more likely to believe that poor diet and smoking are causes of heart disease.

Strokes

The belief that stroke can be prevented was shared by less than half the women in each group (non-hypertensives, 43%, hypertensives, 47%).

However, the data are somewhat unreliable because a large number of women responded: sometimes (over 30%) and not sure (16%). Therefore, only half responded yes or no.

The subjects' beliefs of the major causes of strokes are presented in Table 5.24.

Table 5.24 Beliefs of the Major Causes of Strokes

Causes	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Poor Diet	154 (35%)	144 (38%)	10 (14%)
Overweight	94 (21%)	83 (22%)	11 (16%)
Excess Fat	65 (15%)	57 (15%)	8 (11%)
Excess Salt	36 (8%)	33 (9%)	3 (4%)
High Blood Cholesterol Level	99 (22%)	81 (21%)	18 (26%)
Foods with High Cholesterol	32 (7%)	30 (8%)	2 (3%)
Excess Stress, Worry or Tension	162 (36%)	138 (37%)	24 (34%)
Overwork or Fatigue	36 (8%)	33 (9%)	3 (4%)
Lack of Exercise	112 (25%)	104 (28%)	8 (11%)
Smoking	110 (25%)	100 (27%)	10 (14%)
Heredity	80 (18%)	68 (18%)	12 (17%)
High Blood Pressure/ Hypertension	133 (30%)	100 (27%)	33 (47%)
Atherosclerosis/ Hardening of Arteries	71 (16%)	63 (17%)	8 (11%)
Other	136 (30%)	119 (32%)	17 (24%)
Not Sure	77 (17%)	65 (17%)	12 (17%)

A series of chi square tests found significant differences in some beliefs of the major causes of stroke, by hypertensive status.

A significant difference between the hypertensive women and non-hypertensive women ($x2=14.95$, $p<0.0001$) indicates that non-hypertensive women are far more likely to believe that poor diet is a major cause of stroke.

No significant difference was found between the two groups in their beliefs that overweight ($x2=1.41$, N.S.), excess fat ($x2=0.65$, N.S.), excess salt ($x2=1.59$, N.S.), high blood cholesterol level ($x2=0.61$, N.S.), foods with high cholesterol ($x2=2.31$, N.S.), or overwork or fatigue ($x2=1.59$, N.S.) are major causes of stroke.

No significant difference was found between the groups in their belief that excess stress, worry or tension was a major cause of stroke, but both groups have more than a third of the group who believe that excess stress, worry or tension was a major cause of stroke.

A significant difference was found between the hypertensive and non-hypertensive women in their belief that lack of exercise is a major cause of stroke ($x2=8.21$, $p<0.004$) with non-hypertensive women being more likely to hold this belief.

Non-hypertensive women are also significantly more likely to believe that smoking was a major cause of stroke ($x2=4.77$, $p<0.03$).

No significant difference was found between hypertensive and non-hypertensive women in their belief that heredity ($x2=0.03$, N.S.), atherosclerosis ($x2=1.23$, N.S.) other causes not listed ($x2= 0.00$, N.S.) are major causes of stroke.

The hypertensive women are significantly more likely to believe that hypertension is a major cause of stroke ($x2=12.01$, $p<0.0005$). This is possibly due to the influence of the medical explanatory models presented to the hypertensive women by the health care providers.

Using a logistic regression model, all the beliefs of major causes of stroke were considered simultaneously to address the question: do beliefs of the major causes of stroke bear any relationship to hypertensive status? A significant relationship between beliefs and hypertensive status was observed (Model chi square = 43.18 with 15 d.f., $p<0.0001$).

A step-wise logistic regression was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.25 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.25 Beliefs of the Major Causes of Stroke which are
Predictive of Hypertensive Status in Women
in Reduced Logistic Model

Variable as Major Cause of Stroke	Beta Estimate	Standard Error	Chi-Square Beta = 0
Poor Diet	-.356	.362	14.06
Hypertension	.956	.273	12.28

In a reduced logistic model, only poor diet and hypertension had unique predictive ability. Therefore, the beliefs: poor diet and hypertension are major causes of stroke, were the most effective of the beliefs about causes of stroke, in characterizing the impact on hypertensive status in women. The non-hypertensive women are significantly more likely to believe that poor diet is a major cause of stroke, whereas hypertensive women are significantly more likely to believe that hypertension is a major cause of stroke.

5.0.4.1 Health Beliefs Concerning Dietary Fat and Cholesterol

The health beliefs of the subjects as to the health problems related to the amount of fat people eat are presented in Table 5.26.

Table 5.26 Beliefs of the Health Problems Related
            to Amount of Fat People Eat

Health Problem	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Overweight/ Obesity	239 (54%)	214 (57%)	25 (36%)
Heart Disease/ Problems or Attack	292 (65%)	256 (68%)	36 (51%)
High Blood Cholesterol	130 (29%)	109 (29%)	21 (30%)
High Blood Pressure	163 (37%)	143 (38%)	20 (29%)
Atherosclerosis	123 (28%)	102 (27%)	21 (30%)
Other	151 (34%)	129 (34%)	22 (31%)
Not Sure	28 (6%)	21 (6%)	7 (10%)

Non-hypertensive women are significantly more likely to relate amount of fat eaten to overweight/obseity than are hypertensive women ($x2=10.51$, $p<0.001$), and to heart disease, heart problems or heart attack ($x2=7.08$, $p<0.008$). No significant differences exist between the groups in relating amount of fat eaten to high blood cholesterol ($x2=0.03$, N.S.), high blood pressure ($x2=2.23$, N.S.), atherosclerosis ($x2=0.26$, N.S.) or to other problems not listed ($x2=0.205$, N.S.).

Using a logistic regression, all health beliefs relating to amount of fat eaten variables were considered simultaneously to address the question: do health beliefs related to the amount of fat eaten bear any relationship to hypertensive status? A significant difference between the beliefs and hypertensive status was observed (Model chi square = 20.34 with 7 d.f., $p<0.005$).

A step-wise logistic regression was done and Table 5.27 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.27 Beliefs of the Health Problems Related to Amount of Fat Eaten which are Predictive of Hypertensive Status in Women in Reduced Logistic Model

Health Problem	Beta Estimate	Standard Error	Chi Square Beta = 0
Overweight/Obesity	-8.75	.273	10.31
Heart Disease/ Problems/Attack	-7.12	.267	7.09

In a reduced logistic model, only the beliefs that the health problems overweight/obesity and heart disease/problems/attacks are health problems related to amount of dietary fat, had unique predictive ability. Therefore, these beliefs most effectively characterized the impact of beliefs about health problems related to amount of fat eaten on hypertensive status in women. Non-hypertensive women believe that

overweight/obesity and heart disease/heart attack are related to amount of fat eaten.

Dietary Cholesterol

Almost three-quarters of the subjects (73%) believed cholesterol is found in both food and blood, with an additional 12 percent believing it is found only in food and 9 percent believing it is found only in blood. There were no significant differences in these beliefs between the hypertensive women and the non-hypertensive women.

The beliefs of the subjects as to how dietary cholesterol can affect health are presented in Table 5.28.

Table 5.28 How Dietary Cholesterol Can Affect Health

How Cholesterol Rich Food Can Affect Health	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Hardening/clogging of arteries	158 (35%)	133 (35%)	25 (36%)
Increase blood pressure	108 (24%)	97 (26%)	11 (16%)
Heart attack	223 (50%)	192 (51%)	31 (44%)
Stroke	104 (23%)	88 (23%)	16 (23%)
Angina (pain in chest)	--	--	--
Increase blood cholesterol	--	--	--
Other	119 (27%)	99 (26%)	20 (29%)

-- Data supressed due to small numbers.

There are no significant differences in any of these beliefs although the belief that foods rich in cholesterol can increase blood pressure is held by more non-hypertensive women than hypertensive women. The difference does not quite reach significance at the 0.05 level of probability.

Using a logistic regression, all health beliefs related to beliefs of how foods rich in cholesterol can affect health were considered simultaneously. No significant difference between the beliefs and hypertensive status was observed (Model chi square 8.04 with 7 d.f., $p<0.33$). Therefore, these beliefs do not have any unique predictive ability in relation to hypertensive status in women.

Blood Cholesterol

The beliefs of the subjects as to how blood cholesterol can affect health are presented in Table 5.29.

**Table 5.29 Beliefs of How High Levels of Cholesterol
in the Blood Can Affect Health**

How High Levels of Cholesterol in Blood Can Affect Health	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Hardening/clogging of the arteries	185 (41%)	157 (42%)	28 (40%)
Increase blood pressure	113 (25%)	99 (26%)	14 (20%)
Heart attack/heart disease	255 (57%)	229 (61%)	26 (37%)
Stroke	139 (31%)	120 (32%)	19 (27%)
Angina (pain in chest)	-	-	-
Other	84 (19%)	75 (20%)	9 (13%)

There are no significant differences in the belief that high levels of cholesterol in the blood can affect health, with the exception of heart attack/heart disease. Non-hypertensive women are significantly more likely to believe that high levels of cholesterol in the blood can cause heart attack or heart disease ($x2=13.419$, $p<0.0003$).

Using logistic regression, all health beliefs relating to high levels of cholesterol in the blood were considered simultaneously. A significant difference between the beliefs and hypertensive status was observed (Model chi square = 16.39 with 6 d.f., $p<0.0118$).

A step-wise logistic regerssion model was done and Table 5.30 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.30 Health Problems Affected by High Levels of Cholesterol in the Blood which are Predictive of Hypertensive Status in Women in Reduced Logistic Model

Health Problem	Beta Estimate	Standard Error	Chi Square Beta = 0
Heart attack/ Heart disease	-.958	.269	12.73

In a reduced logistic model, only the belief that the health problem heart attack/heart disease is a health problem related to high levels of cholesterol in the blood, had unique predictive ability. Therefore, this belief most effectively characterizes the impact of beliefs about high levels of blood cholesterol on hypertensive status in women.

The beliefs of the subjects as to what can lower blood cholesterol levels are presented in Table 5.31.

Table 5.31 Beliefs of What Can Lower Blood Cholesterol Level

Variable	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Exercise regularly	190 (43%)	172 (46%)	18 (26%)
Control stress	39 (9%)	35 (9%)	4 (6%)
Control fatigue	-	-	-
Take prescribed medication	38 (9%)	29 (8%)	9 (13%)
Eat food with less cholesterol	253 (57%)	220 (58%)	33 (47%)
Eat less fatty foods	294 (66%)	249 (66%)	45 (64%)
Lose weight	36 (8%)	30 (8%)	6 (9%)
Use low fat dairy products	116 (26%)	100 (27%)	16 (23%)
Other	171 (38%)	148 (39%)	23 (33%)

Non-hypertensive women are significantly more likely to think exercising regularly can lower blood cholesterol levels ($x2=10.200$, $p<0.006$). There were no significant differences between the groups in beliefs that: controlling stress ($x2=0.945$, N.S.), taking prescription medications ($x2=2.025$, N.S.), eating foods with less cholesterol ($x2=3.022$, N.S.) or less

fatty foods (x2=0.08, N.S.), losing weight (x2=0.03, N.S.), using low fat dairy products (x2=0.413, N.S.) or other things (x2=1.024, N.S.), could lower blood cholesterol levels. More than half of the subjects in both groups believed that blood cholesterol levels could be lowered by eating foods with less cholesterol, and eating less fatty foods.

Using logistic regression, all health beliefs relating to what can lower blood cholesterol levels, were considered simultaneously. A significant difference was observed between the beliefs and hypertensive status (Model chi square = 17.58 with 9 d.f., $p<0.0404$).

A step-wise logistic regression was done and Table 5.32 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.32 Variables Which Lower Blood Cholesterol Levels
as Predictors of Hypertensive Status in
Women in Reduced Logistic Model

Variables	Beta Estimate	Standard Error	Chi Square Beta = 0
Exercise regularly	-.900	.290	9.65

In a reduced logistic model, only the belief that exercising regularly lowered blood cholesterol levels had unique predictive ability. Therefore, this belief most effectively characterizes the impact of beliefs about lowering blood cholesterol levels on hypertensive status in women.

5.0.4.2 Health Beliefs Concerning Hypertension

The health beliefs of the subjects as to how high blood pressure can affect health are presented in Table 5.33.

Table 5.33 Beliefs of What Health Problems are Affected by High Blood Pressure

Health Problem	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Stroke	189 (47%)2*	149 (47%)**	40 (65%)***
Kidney trouble	27 (7%)*	22 (6%)**	5 (8%)***
Heart attack/ problems	261 (65%)*	223 (65%)	38 (61%)
Hardening of the arteries	27 (7%)*	24 (7%)	3 (5%)
Eye problems	--	--	--
Nose bleed	--	--	--
Headache	62 (14%)	49 (13%)	13 (19%)
Dizziness	73 (16%)	66 (18%)	7 (10%)
Swelling	--	--	--
Other	149 (33%)	131 (35%)	18 (26%)
Not sure	--	--	--

* 44 values missing (Total = 403)
** 36 values missing (Total = 341)
*** 8 values missing (Total = 62)
-- Values suppressed due to small numbers.

The hypertensive women are significantly more likely to believe high blood pressure can cause strokes ($x2=9.13$, $p<0.0025$). There is no significant difference in beliefs that high blood pressure causes kidney trouble ($x2=0.218$, N.S.), heart attack or heart problems ($x2=0.39$, N.S.),

hardening of the arteries ($x2=0.406$, N.S.), headache ($x2=1.536$, N.S.), dizziness ($x2=2.43$, N.S.), or other problems ($x2=2.17$, N.S.).

Using logistic regression, all health beliefs relating to how blood pressure can affect health showed that they did indeed show significant differences in the outcome variable, hypertensive status (Model chi square = 22.20 with 7 d.f., $p<0.0023$).

A step-wise logistic regression was done and Table 5.34 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.34 Health Problems Affected by High Blood Pressure Pressure which arePredictive of Hypertensive Status in Women in Reduced Logistic Model

Health Problem	Beta Estimate	Standard Error	Chi Square Beta = 0
Stroke	.863	.271	10.18
Headache	.781	.388	4.06
Dizziness	-1.023	.468	4.77

In a reduced logistic model, the beliefs that high blood pressure affects health by causing stroke, headache or dizziness are the only beliefs which had unique predictive ability. Therefore, these beliefs most effectively characterize the impact of beliefs about which health problems are affected by high blood pressure, on hypertensive status in women.

The subjects' beliefs as to what causes high blood pressure are presented in Table 5.35.

Table 5.35 Beliefs As To What Causes High Blood Pressure

Causes	Total Women	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Being overweight	160 (36%)	131 (35%)	29 (41%)
Smoking	138 (31%)	119 (32%)	19 (27%)
Too much salt	131 (29%)	112 (30%)	19 (27%)
Race or ethnic group	--	--	--
Stress/worry/tension	318 (71%)	267 (71%)	51 (73%)
Fatty foods	178 (40%)	159 (42%)	19 (27%)
Drinking coffee	39 (9%)	33 (9%)	5 (7%)
Regular hard exercise	--	--	--
Being pregnant	--	--	--
Heredity	119 (27%)	96 (25%)	23 (33%)
Drinking alcohol	81 (18%)	72 (19%)	9 (13%)
Using birth control pills	--	--	--
Being underweight	--	--	--
Low income, low education	--	--	--
Too much blood in system	--	--	--
Getting little exercise	80 (18%)	75 (20%)	5 (7%)
Old age	--	--	--
Other	163 (37%)	146 (39%)	17 (24%)
Not sure	--	--	--

-- Data suppressed due to small numbers

There are no significant differences in the beliefs that being overweight ($x2=1.147$, N.S.), smoking ($x2=0.54$, N.S.), eating too much salt

(x2=0.188, N.S.), drinking coffee (x2=0.197, N.S.), heredity (x2=1.65, N.S.), drinking too much alcohol (x2=1.550, N.S.) cause high blood pressure. There is no significant difference in the belief that stress/worry/tension cause high blood pressure (x2=0.119, N.S.), but it is a belief shared by over 70% of all subjects! A significant difference was found in the belief that fatty foods cause high blood pressure (x2=5.567, p<0.018) with non-hypertensive women being significantly more likely to believe eating fatty foods can cause high blood pressure. Non-hypertensive women were significantly more likely to believe getting too little exercise can cause hypertension (x2=6.53, p<0.01), and that there are other causes not listed which can cause hypertension (x2=5.314, p<0.02). Not one single subject in either group believed that low income or low education could cause high blood pressure.

Using logistic regression, all health beliefs relating to what causes high blood pressure were considered simultaneously, to answer the question: do beliefs about what causes high blood pressure bear any relationship to hypertensive status in women? A significant difference between the beliefs and hypertensive status was observed (Model chi square = 20.47 with 10 d.f., p<0.0251).

A step-wise logistic regression model was done and Table 5.36 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.36 Beliefs as to what causes High Blood Pressure which
are Predictive of Hypertensive Status in
Women in Reduced Logistic Model

Causes	Beta Estimate	Standard Error	Chi Square Beta = 0
Getting little exercise	-1.095	.477	5.28
Other	-.634	.299	4.49

In a reduced logistic model, the beliefs that: getting little exercise, or other causes not listed cause high blood pressure, are the only beliefs which had unique predictive ability. Therefore, these beliefs effectively characterize the impact of beliefs about what causes high blood pressure, on hypertensive status in women.

The beliefs of the subjects as to what things people eat and drink that are related to high blood pressure are presented in Table 5.37.

Table 5.37 Food and Drink Related to High Blood Pressure

Food/ Drink	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Salt/salty foods	181 (41%)	151 (40%)	30 (43%)
Sodium	25 (6%)	25 (7%)	0 -
Alcohol	264 (59%)	229 (61%)	35 (50%)
Fats	185 (41%)	163 (43%)	22 (31%)
Saturated fats	79 (18%)	69 (18%)	10 (14%)
Cholesterol	101 (23%)	94 (25%)	7 (10%)
Calories/eating too much	--	--	--
Additives/ preservatives, food coloring	--	--	--
Caffeine/coffee	120 (27%)	109 (29%)	11 (16%)
Sugar/sweets	101 (23%)	90 (24%)	11 (16%)
Starch/starchy foods	--	--	--
Pork	--	--	--
Meat other than pork	--	--	--
Meats generally	--	--	--
Fried foods/ greasy/oily	127 (28%)	115 (31%)	12 (17%)
Calcium	--	--	--
Red meats	--	--	--
Fast foods	--	--	--
Other	--	--	--
Not sure	--	--	--

-- Data suppressed due to small numbers

There are no significant differences in the beliefs that eating salt or salty foods ($x2=0.193$, N.S.), drinking alcohol ($x2=2.818$, N.S.), eating fats ($x2=3.393$, N.S.), eating saturated fats ($x2=0.655$, N.S.) or eating sweets or sugar ($x2=2.247$, N.S.) were related to high blood pressure. Non-hypertensive women were significantly more likely to believe sodium is related to high blood pressure ($x2=4.917$, $p<0.027$), but there were only small number of subjects involved. The non-hypertensive women were significantly more likely to believe cholesterol in food was related to high blood pressure ($x2=7.528$, $p<0.0061$), drinking caffeine or coffee was related to high blood pressure ($x2=5.237$, $p<0.02$), and eating fried, greasy or oily foods was related to high blood pressure ($x2=5.182$, $p<0.023$).

Using logistic regression, all health beliefs as to what things people eat and drink are related to high blood pressure, were considered simultaneously to address the question: do beliefs about what things people eat and drink are related to high blood pressure, bear any relationship to hypertensive status in women? A significant difference between the beliefs and hypertensive status was observed (Model chi square = 28.06 with 9 d.f., $p<0.0009$).

A step-wise logistic regression was done and Table 5.38 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.38 Beliefs of Food and Drink Related to High Blood
Pressure which are Predictive of Hypertensive Status
in Women in Reduced Logistic Model

Food/ Drink	Beta Estimate	Standard Error	Chi Square Beta = 0
Drinking caffeine/coffee	-.754	.348	4.69
Cholesterol in food	-1.056	.413	6.53

In a reduced logistic model, the beliefs that: drinking caffeine/coffee, or cholesterol in food are related to high blood pressure, are the only beliefs which had unique predictive ability. The non-hypertensive women were more likely to have these beliefs.

5.0.5 Health Behaviors

5.0.5.0 Behavior Changes

The behaviors which have been done in the last year to improve health are presented in Table 5.39.

Table 5.39 Behaviors Done in the Past Year to Improve Health

Behavior	Total Women N = 447	Non-Hypertensive Women N = 377	Hypertensive Women N = 70
Did nothing	86 (19%)	76 (20%)	10 (14%)
Increased exercise	88 (20%)	80 (20%)	8 (12%)
Lost weight	32 (7%)	29 (8%)	3 (4%)
Improved eating habits	118 (27%)	105 (28%)	13 (19%)
Quit or reduced smoking	30 (7%)	26 (7%)	4 (6%)
Reduced drugs or meds	--	--	--
Drank less alcohol	4(1%)	--	--
Had blood pressure checked	--	--	--
Attempted to control blood pressure	--	--	--
Learned to manage stress	7 (2%)	--	4 (6%)
Reduced stress levels	18 (4%)	--	6 (9%)
Received medical treatment	11 (3%)	--	4 (6%)
Other	39 (9%)	29 (8%)	10 (14%)
Not sure	11 (3%)	--	4 (6%)

-- Suppressed data due to small numbers

A series of chi square tests found significant differences in some of the behaviors, by hypertensive status.

The non-hypertensive women were more likely to increase their exercise (x^2=3.58, p<0.058). There was no significant difference between the groups in losing weight (x^2=1.03, N.S.), improving eating habits (x^2=2.62,N.S.), or reducing smoking (x^2=0.13,N.S.), and the numbers were too small to do any further meaningful analysis on the rest of the behavior variables.

The behavior variables were considered simultaneously using a logistic regression model to address the question: *do behaviors done in the last year to improve health, bear any relationship to hypertensive status in women.* A significant relationship between behavior and hypertensive status was observed (Model chi square = 13.99 with 4 d.f., p<0.0073).

A step-wise logistic regression was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability and Table 5.40 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.40 Behaviors Done in the Last Year Which are
         Predictive of Hypertensive Status in
         Women in Reduced Logistic Model

Behaviors	Beta Estimate	Standard Error	Chi Square Beta = 0
Increased exercise	-.937	.404	5.38
Improved eating habits	-.723	.335	4.67

In a reduced logistic model, the behaviors: increasing exercise and improving eating habits in the past year, are the only behaviors which had unique predictive ability. Therefore, these behaviors effectively characterize the impact of behaviors on hypertensive status in women.

Non-hypertensive women are significantly more likely to have increased exercise and improved eating habits in the last year to improve health, than hypertensive women.

5.0.5.1   Intended Behavior Changes

The behaviors which the subjects intend to change to improve health are presented in Table 5.41.

Table 5.41 Intended Behavior Changes to Improve Health

Intended Behavior Change	Total Women N = 447	Non-Hypertensive Women N = 337	Hypertensive Women N = 70
Do nothing	68 (15%)	53 (14%)	15 (21%)
Increase exercise	211 (47%)	194 (51%)	17 (24%)
Lose weight	99 (22%)	80 (21%)	19 (27%)
Improve eating habits	121 (27%)	107 (28%)	14 (20%)
Quit/reduce smoking	56 (12%)	53 (14%)	3 (4%)
Reduce drugs or meds	--	--	--
Drink less alcohol	--	--	--
Attempt to control blood pressure	--	--	--
Learn to manage stress	17 (4%)	15 (4%)	2 (3%)
Reduce stress level	15 (3%)	13 (3%)	2 (3%)
Receive medical treatment	7 (2%)	--	--
Other	56 (12%)	48 (13%)	8 (11%)
Not sure	18 (4%)	12 (3%)	6 (9%)

-- Data suppressed due to small numbers

A series of chi square tests found significant differences in some intended behaviors, by hypertensive status.

No significant differences were found between hypertensive and non-hypertensive women in their intentions to do nothing to improve health in the next year (x^2=2.49, N.S.). The non-hypertensive women were

significantly more likely to intend to increase exercise (x^2=17.49, p<0.0000) and to intend to quit or reduce smoking (x^2=5.15, p<0.02) than hypertensive women.

There was no significant difference between the groups in their intention to lose weight (x^2=1.20, N.S.) or improve eating habits (x^2=2.10, N.S.)

Very few, if any, women in either group intended to: reduce drugs or medication, drink less alcohol, have blood pressure checked, attempt to control blood pressure, or receive medical treatment.

There were no significant differences in the small numbers who intend to learn to manage stress (x^2=0.20, N.S.), reduce stress level (x^2=0.06, N.S.), or make other behavior changes not listed (x^2=0.09, N.S.).

The hypertensive women were significantly more likely to be not sure of their behavior change intentions (x^2=4.44, p<0.04) but the numbers are small in both groups.

When the intended behavior variables were considered simultaneously using a logistic regression model to address the question: *do intended behavior changes bear any relationship to hypertensive status in women.* A significant relationship between intended behaviors and hypertensive status was observed (Model chi square = 28.84 with 7 d.f., p<0.0002).

A step-wise logistic regression was done which eliminated the variables which did not reach statistical significance at the 0.05 level of probability. Table 5.42 provides the beta estimate and standard error of those variables which were statistically significant.

Table 5.42 Intended Behavior Changes Which are Predictive of Hypertensive Status in Women in Reduced Logistic Model

Behavior Change	Beta Estimate	Standard Error	Chi Square Beta = 0
Intends to increase exercise	-1.230	.299	16.93
Intends to quit or reduce smoking	-1.386	.613	5.11

In a reduced logistic model, the intention to increase exercise and intention to quit or reduce smoking are the only intended behaviors which had unique predictive ability. The non-hypertensive women are significantly more likely to intend to increase exercise and to intend to quit or reduce smoking, than are hypertensive women.

5.0.5.2   Losing Weight

Nearly one half (47%) of all women in the present study are trying to lose weight, with no significant difference in the proportion of hypertensive and non-hypertensive women. Of those trying to lose weight, the following methods are being employed: dieting, exercising,

skipping meals, attending weight control programs and other. There are no significant differences between the groups in any of the methods being used to lose weight with dieting (69%) and exercising (63%) being the main methods used. The reasons given for trying to lose weight are presented in Table 5.43.

Table 5.43 Reasons for Losing Weight for Subjects
Trying to Lose Weight

Reason	Total Women N = 212	Non-Hypertensive Women N = 177	Hypertensive Women N = 35
Become more attractive	135 (64%)	118 (67%)	17 (49%)
Improve general health	140 (66%)	113 (64%)	27 (77%)
Decrease risk of heart attack	--	--	--
Maintain blood pressure at acceptable level	--	--	--
Maintain cholesterol at acceptable level	--	--	--
Slow down hardening of the arteries	--	--	--
Decrease risk of diabetes	--	--	--
Other	--	--	--

-- Lack of data

Non-hypertensive women are significantly more likely to try to lose weight to become more attractive than hypertensive women ($x^2=4.14$, $p<0.04$). There was no significant difference between the groups in losing weight to improve general health ($x^2=2.31$, N.S.), and the other reasons did not have enough data to analyze.

5.0.6 Combined Non-Biomedical Risk Factors

The non-biomedical variables which were shown in univariate analysis to be predictive of hypertensive status in women are presented in Table 5.44 and Figure 5.1. Figure 5.1 shows the linear relationship which exists with each of these variables and hypertensive status in women. When non-biomedical variables are considered, there are a large number of risk factors identified.

Table 5.44 Non-Biomedical Risk Factors Which are Predictive of Hypertensive Status in Women in Univariate Analysis

Variable	Risk (t or x^2)	Probability Level
Demographics:		
Employment: Full time	$x^2 = 22.32$	0.00
Employment: Retired	$x^2 = 47.42$	0.00
Employment: Student	$x^2 = 4.30$	0.04
Marital Status: Single	$x^2 = 6.07$	0.01
Marital Status: Widowed	$x^2 = 27.54$	0.00
Education: Grade 6 completed	$x^2 = 6.39$	0.01
Education: Post-secondary	$x^2 = 4.64$	0.03
Number of people in household	$t = 5.23$	0.00
Income: Under $12,000.	$x^2 = 4.35$	0.03
Income: $50,000-$74,999.	$x^2 = 5.52$	0.02
Lifestyles:		
Daily Physical Activity: Stand/walk a lot	$x^2 = 8.40$	0.00
Daily Physical Activity: Lift/carry light loads	$x^2 = 4.94$	0.03
Exercise strenuously	$x^2 = 14.25$	0.0008
Exercise for less than 15 minutes at a time	$x^2 = 5.99$	0.014
Reason for not being more active: Lack of time	$x^2 = 10.71$	0.001
Reason for not being more active: Lack of transportation	$x^2 = 7.51$	0.006
Reason for not being more active: Illness/ disability	$x^2 = 17.23$	0.0000
Smoking status: Present smoker	$x^2 = 5.25$	0.02
Reason to quit smoking: Prevent disease/ ill health	$x^2 = 9.04$	0.01
Daily alcohol consumption	$t = 3.12$	0.0002
Health Beliefs:		
Health belief: Poor diet is a major cause of heart disease	$x^2 = 17.92$	0.0000
Health belief: Lack of exercise is a major cause of heart disease	$x^2 = 12.39$	0.0004
Health belief: Smoking is a major cause of heart disease	$x^2 = 4.57$	0.03
Health belief: Poor diet is a major cause of stroke	$x^2 = 14.95$	0.0001
Health belief: Lack of exercise is a major cause of stroke	$x^2 = 8.21$	0.04
Health belief: Smoking is a major cause of stroke	$x^2 = 4.77$	0.03

Table 5.44 (continued) Non-Biomedical Risk Factors Which are Predictive of Hypertensive Status in Women in Univariate Analysis

Health belief: Hypertension is a major cause of stroke	$x^2 = 12.01$	0.0005
Health belief: Overweight/obesity is related to amount of fat eaten	$x^2 = 10.51$	0.001
Health belief: Heart disease/problems/attack are related to amount of fat eaten	$x^2 = 7.08$	0.008
Health belief: High levels of blood cholesterol cause heart disease/attack	$x^2 = 13.42$	0.0003
Health belief: Regular exercise can lower blood cholesterol levels	$x^2 = 10.20$	0.006
Health belief: High blood pressure affects health by causing strokes	$x^2 = 9.13$	0.0025
Health belief: Fatty foods can cause hypertension	$x^2 = 5.567$	0.018
Health belief: Getting too little exercise can cause hypertension	$x^2 = 6.53$	0.01
Health belief: Other cause not listed can cause hypertension	$x^2 = 5.314$	0.02
Health belief: Sodium is related to high blood pressure	$x^2 = 4.917$	0.027
Health belief: Cholesterol in food is related to high blood pressure	$x^2 = 7.528$	0.0061
Health belief: Drinking caffeine/coffee is related to high blood pressure	$x^2 = 5.237$	0.02
Health belief: Eating fried, greasy or oily foods is related to high blood pressure	$x^2 = 5.182$	0.023
Intended behavior change: Increase exercise	$x^2 = 17.49$	0.0000
Intended behavior change: Quit or reduce smoking	$x^2 = 5.15$	0.02
Intended behavior change: Losing weight to become more attractive	$x^2 = 4.14$	0.04

Figure 5.1 Non-Biomedical Risk Factors Which are Predictive of Hypertensive Status in Women in Univariate Analysis
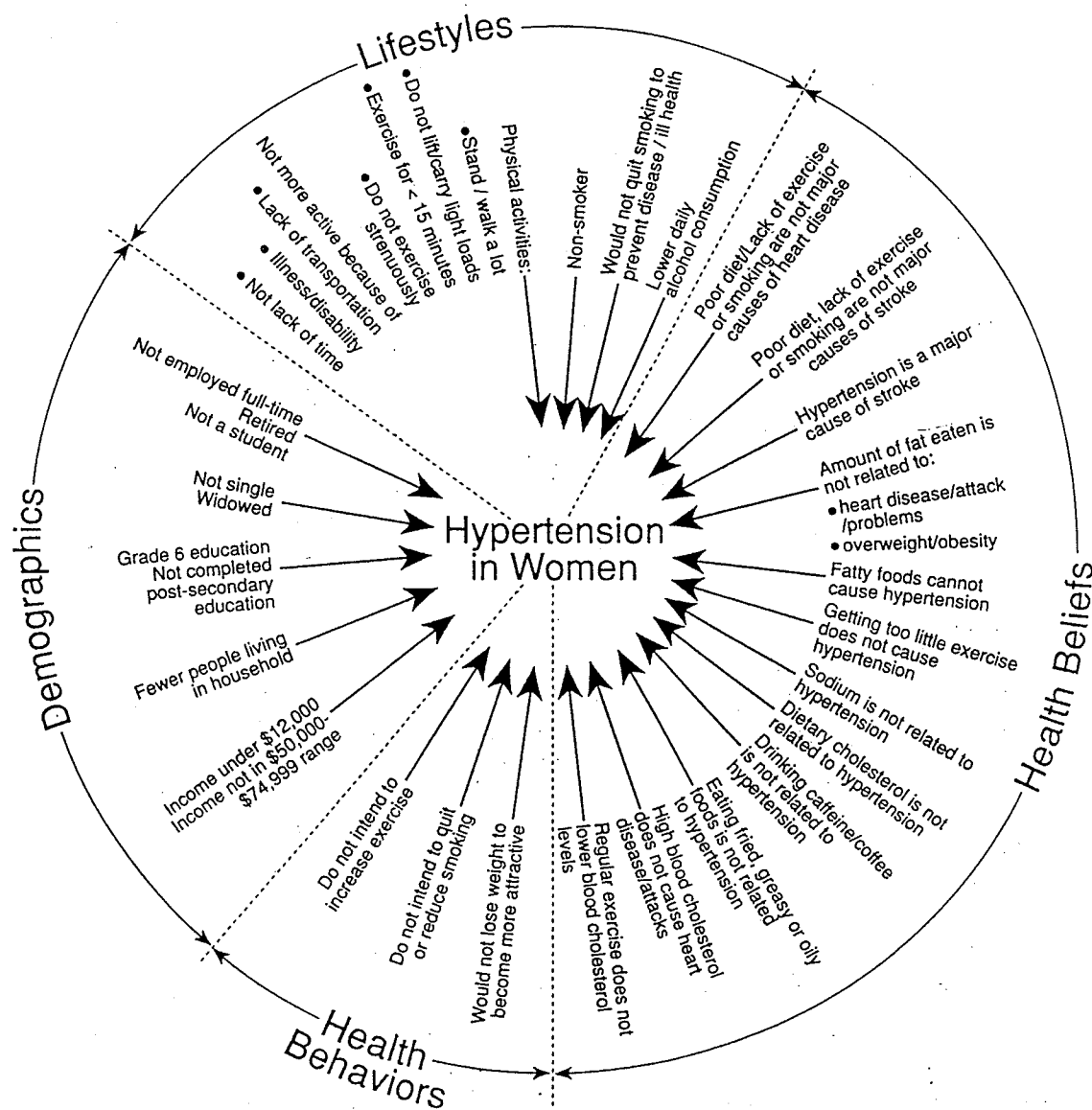
These non-biomedical risk factors are predictive of hypertensive status in women in univariate analysis. The non-biomedical risk factors which were shown to have unique predictive ability when each group of variables such as demographics, lifestyles, health beliefs and health behaviours was analysed using multivariate analysis, are presented in Table 5.45 and Figure 5.2. The other risk factors identified in univariate analysis did not appear because their predictive power is effectively duplicated by one or more of the variables left in the model. The variables left in the model most effectively characterize the impact of each of the non-biomedical groups of variables on hypertensive status in women, taking into account the problem of collinearity with the mathematically strongest being left in the model.

Table 5.45 Non-Biomedical Predictors of Hypertensive Status
in Women in Reduced Logistic Models

Predictive Variable	Chi Square, Beta = 0
Demographics:	
Employment: Retired	47.18
Employment: Homemaker	14.47
Marital Status: Widowed	23.77
Education: Secondary (grade 12)	4.59
Education: Post-secondary	9.25
Number of people in household	23.80
Income: $50,000 - 74,999	5.07
Lifestyles:	
Daily physical activity: Stand/walk a lot	8.15
Exercise strenuously	12.77
Reason for not more active: Lack of time	9.80
Reason for not more active: Lack of transportation	6.28
Reason for not more active: Illness/disability	12.66
Smoking status: Present smoker	5.02
Reason to quit smoking: Prevent disease/ill health	6.63
Daily alcohol consumption	9.66
Health Beliefs:	
Health belief: Poor diet is a major cause of heart disease	16.61
Health belief: Smoking is a major cause of heart disease	4.27
Health belief: Poor diet is a major cause of stroke	14.06
Health belief: Hypertension is a major cause of stroke	12.28
Health belief: Overweight/obesity related to amount of fat eaten	10.31
Health belief: Heart disease/problems/attack related to amount of fat eaten	7.09

Table 5.45 (continued)   Non-Biomedical Predictors of Hypertensive Status
in Women in Reduced Logistic Models

Health belief: High levels of blood cholesterol cause heart disease/attack	12.73
Health belief: Regular exercise can lower blood cholesterol levels	9.65
Health belief: High blood pressure affects health by causing strokes	10.18
Health belief: High blood pressure affects health by causing dizziness	4.77
Health belief: Getting little exercise causes hypertension	5.28
Health belief: Other causes not listed can cause hypertension	4.49
Health belief: Drinking caffeine/coffee are related to high blood pressure	4.69
Health belief: Cholesterol in food is related to high blood pressure	6.53
Behavior change: Increased exercise	5.38
Behavior change: Improved eating habits	4.67
Intended behavior change: Increase exercise	16.93
Intended behavior change: Quit or reduce smoking	5.11

Figure 5.2 Non-Biomedical Predictors of Hypertensive Status in Women in Reduced Logistic Models
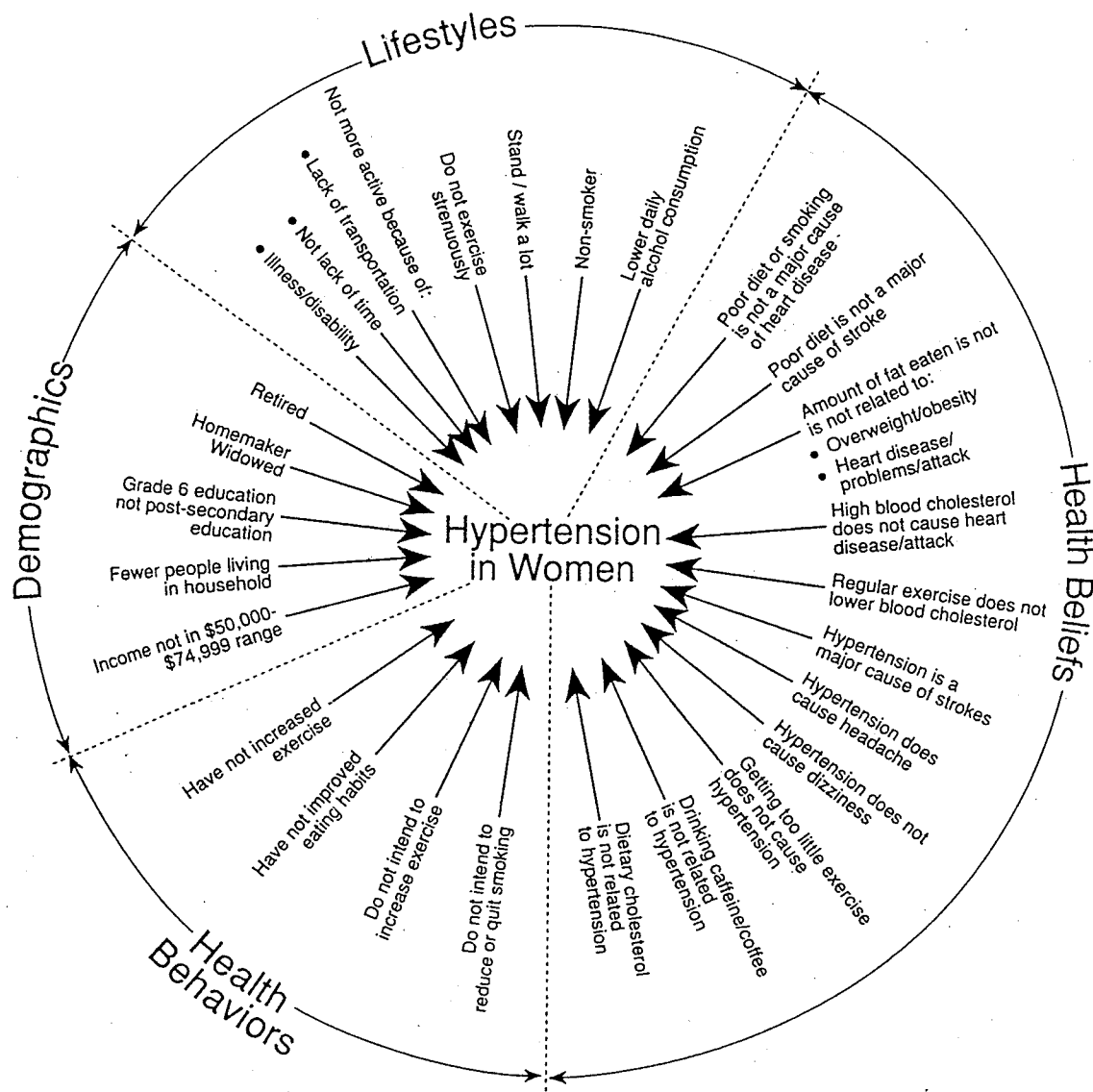
To address the question: *which non-biomedical variables are optimally predictive of hypertensive status in women,* all of the variables which were predictive of hypertension status in reduced logistic models, were considered simultaneously in a logistic regression model. A significant relationship between the predictive variables and hypertensive status was again observed (Model chi square = 67.01 with 29 d.f., p<0.0001). The health beliefs: other causes not listed can cause hypertension, regular exercise can lower blood cholesterol levels, high blood pressure affects health by causing headaches or by causing dizziness, were not included in the logistic regression model as they were not independent risk factors in univariate analysis and as such would not further explain variation by hypertensive status.

The variable: a good reason to quit smoking was to prevent disease/ill health was also omitted as it was redundant with the variable: presently smoking. A step-wise logistic regression eliminated the variables which did not reach statistical significance at the 0.05 level of probability as shown in Table 5.46 and Figure 5.3.

Table 5.46 Non-Biomedical Predictors of Hypertensive Status in
Women in a Final Reduced Logistic Model

Predictive Variable	Beta Estimate	Standard Error	Chi Square Beta = 0
Health belief: High blood pressure causes strokes	2.05	.675	9.24
Health belief: Poor diet is a major cause of stroke	-2.12	.763	7.73
Health belief: High blood cholesterol does not cause heart attack/heart disease	-1.36	.634	4.61
Homemaker	1.44	.695	4.29
Do not exercise strenuously	1.14	.394	8.38

Figure 5.3 Non-Biomedical Predictors of Hypertensive Status in Women in a Final Reduced Logistic Model
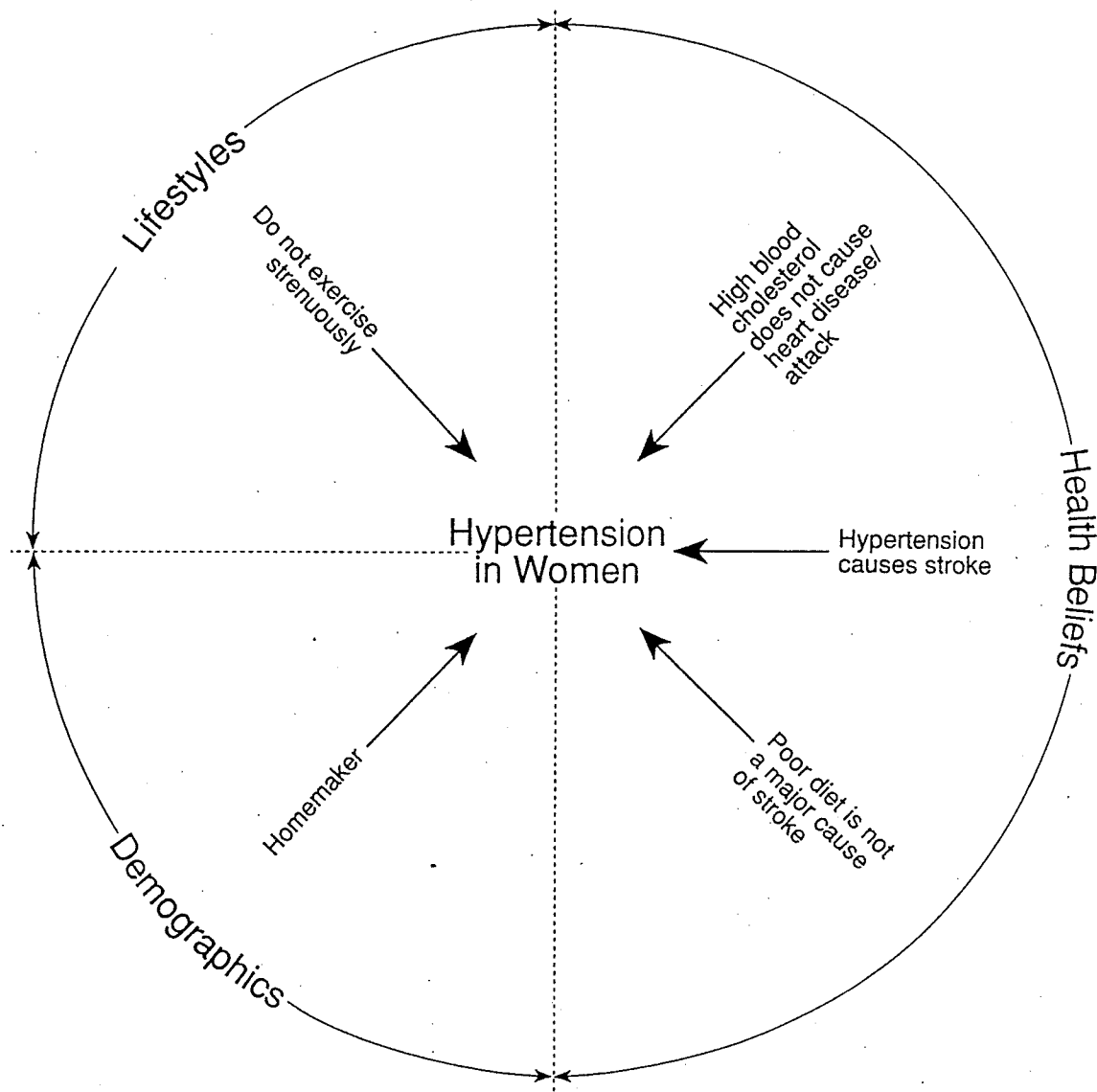
The final reduced logistic regression model included the variables which showed unique predictive ability when considered simultaneously with other non-biomedical variables. Therefore, the non-biomedical predictors which most effectively characterize the impact of non-biomedical factors on hypertensive status in women are: being a homemaker, not exercising strenuously, and having the health beliefs: hypertension causes stroke, poor diet is not a major cause of stroke, and high blood cholesterol does not cause heart attack/disease.

5.1 DISCUSSION OF NON-BIOMEDICAL RISK FACTORS

In addressing the third research question: *what are the identifiable non-biomedical risk factors of hypertension in women*, demographic variables, lifestyle variables, as well as subjects' beliefs and behaviors produced interesting results.

Consistent with current research, employment status follows a social class gradient with women in full-time employment being less likely to be hypertensive than women in other employment status categories. Employment status is not generally used as an indicator of socio-economic status, but it is apparent that those who work full-time often have higher household incomes than those who do not work outside the home

(Labonte, 1990). Being retired or being a homemaker were both found to be independent predictors of hypertension in women.

In the present data, non-hypertensive women were significantly more likely to have professional occupations than hypertensive women. However, occupational status variables analyzed individually or as a composite, were not risk factors or predictive of hypertensive status. Occupational status is sometimes used in European research as a reliable indicator of socio-economic status (WHO/ERO, 1984) and Wilkinson (1986) suggests it could be considered a proxy measure of social class. The present data had many missing values, and also a large percentage of subjects who did not work outside of the home, or were retired from the workplace. These factors may have influenced the results.

Marital status like many of the demographic variables is somewhat confounded by age. Although most subjects are married or in common-law relationships, there are some significant differences in marital status. The hypertensive women are significantly older and significantly more likely to be widowed than the hypertensive women. Widowed status is independently predictive of hypertensive status.

The present data are consistent with research on level of education. Women with grade six as the highest grade completed were significantly more likely to be hypertensive than women with more education. Women with post-secondary education completed had the least risk of being

hypertensive. Completion of grade 12 and completion of post-secondary education were both independently predictive of hypertensive status in women, with women who had completed grade six being twice as likely to be hypertensive as those with grade twelve completed and at three times the risk as those with post-secondary education.

Some possible explanations for these findings may be that again the hypertensive women are significantly older and may have had less opportunity to further their education than younger women. Prevention measures may have missed this group because of their lower reading skills, or perhaps health education in the schools has helped the non-hypertensive women reduce their risk factors. In some research, education has been used as an indicator of socio-economic status (Miller & Wigle, 1986). The Canadian Health Promotion Survey (1985) found gradients in health status according to level of education completed. Miller and Wigle (1986) found relative differences in prevalence rates of cardiovascular risk factors including: smoking, overweight, obesity, elevated diastolic blood pressure and physical activity, with the lowest education group having the highest prevalence of each of these risk factors.

The number of people in the household was independently predictive of hypertensive status. Perhaps more people in the household provides stronger social contacts, or perhaps this variable is also reflecting

age. Older women tend to live alone because they are widowed or with only a spouse because any children have grown up.

The lowest income category was significantly more likely to include hypertensive women and the highest income category was significantly more likely to include non-hypertensive women. These results are consistent with the accumulated evidence that most disease mortality and morbidity rates follow a social-class gradient with lower income and /or lower social status being associated with higher rates of disease (Reeder et al, 1991). Most Canadian and American research cites income levels as the most reliable indicators of socio-economic status (Labonte, 1990). However, it is a flawed measure because it does not account for wealth or for non-income statuatory services, nor does it include many non-income forms of wealth such as: real estate, dividends, capital holdings and other assets (Labonte, 1990). The present data found only the first quintile and the fifth quintile of income status were significantly different by hypertensive status and the middle quintile which contained most of the subjects showed no significant differences. As a composite measure, income showed no significant difference by income status. When all the demographic variables were combined no income categories were predictive of hypertensive status.

Several explanations are possible for this lack of predictive power of income category. The specific household incomes were not recorded,

but rather the broad range within which the income fit. Therefore, the responses themselves were non-specific. A substantial number of subjects (12%) refused to give their income category, 17% of those being hypertensive women. Age again had a possible effect on these results because the hypertensive women were older than the non-hypertensive women and therefore more likely to be retired or on fixed income. The older women were more likely to have non-income forms of wealth such as assets, real estate, and capital holdings; yet be classified into a low-income category.

The present data found that the strongest independent demographic predictors of hypertensive status in women were: being retired, being a homemaker, the number of people in household, and having a office/sales/clerical occupation. This would indicate a primary target group for future prevention efforts.

More than half of the women in the present study reported that they engage in physical activity regularly, with no significant differences by hypertensive status. However, less than one third of each group felt they got as much exercise as they need. The hypertensive women were significantly more likely to stand or walk a lot as their usual daily activity and non-hypertensive women were significantly more likely to lift or carry light loads. Having the daily activity of standing and walking a lot was a predictor of hypertensive status with women who stand or walk a lot

being twice as likely to be hypertensive as those who sit and don't walk much. These results are somewhat surprising but may indicate that daily activity alone is not able to reduce susceptibility to hypertension.

Physical activity status has been found to be inversely related to risk of CHD (Paffenbarger, 1990) and although most research has been done on men (Paffenbarger, 1990, 1984; Leon et al,1987; Lie et al,1987 & Slattery et al,1989). These data were able to show that non-hypertensive women were significantly more likely to engage in physical activity strenuous enough to cause sweating/heavy breathing and that this was also an independent predictor of hypertensive status in this study. It was not able to show a gradient effect of exercise levels, with there being no significant differences in duration of exercise sessions or in number of exercise sessions by hypertensive status. It appears that in this data, moderate exercise did not have any significant effect on hypertensive status. Non-hypertensive women were significantly more likely to report lack of time prevents them from being more active and hypertensive women were significantly more likely to report lack of transportation and illness/disability prevent them from being more active.

These reasons were all independent predictors of hypertension and encourage more research into what is needed to facilitate women to be more active. If the hypertensive women consider their hypertension a disease/disability, then they are obviously not receiving proper health

education which would encourage them to become more active. If they are experiencing another chronic disease, there is a good chance that physical activity would be beneficial also.

The present data found the number of women presently smoking was surprisingly high (44%) compared to approximately 30% in populations of similar ages (Smoking Behavior of Canadians, 1986). Although much of the recent research associates smoking positively with risk of fatal coronary heart disease, non-fatal myocardial infarction, and angina pectoris in women (Willett et al, 1987). However, they found that the relative risks of coronary heart disease associated with cigarette smoking were slightly lower in women with hypertension. Rosenberg et al (1985) found similarly reduced relative risks among women with hypertension but no possible explanations for these findings have been advanced. One of the problems with smoking and hypertension is that these two variables are found simultaneously in only a few cases making further analyses difficult even in large studies.

Furthermore, data from the Framingham study (Kannel & Higgins, 1990) found that smoking cessation could reduce the risk of coronary heart disease by 35-40% and that smoking also increased the risk of hypertension. The prevalence of smoking among hypertensive women in the Framingham study was about 31%, similar to the 27% in the present data.

The higher prevalence of smoking in the non-hypertensive women may account for lack of association between smoking and hypertensive status in this data. The non-hypertensive women are significantly more likely to think prevention of disease/illness and saving money are important reasons for quitting smoking. It is somewhat surprising that the hypertensive women don't consider prevention of disease/illness as an important reason to quit. There also were more non-hypertensive women who wished to quit smoking although the difference was not large enough to be significant.

The vast majority of women in the present study (85%) have taken an alcoholic drink during the past 12 months. However, the hypertensive women drink significantly less alcohol daily than non-hypertensive women and the number of drinks per day was an independent predictor of hypertensive status. This adds fuel to the argument that alcohol can have a protective effect on risk of hypertension. These results have to be taken in the context that the women in both groups drank only moderate amounts of alcohol; (hypertensive women 1.7 drinks per day and non-hypertensive 2.5 drinks per day on average). The results for hypertensive women may have been confounded by some women who are abstainers for health reasons but may have been drinkers in the past. Hypertensive women may drink less because they have been advised to, or know that they should not, and therefore underreport drinking.

Furthermore, these findings concur with the presently available information that the alcohol-hypertension association may be a J-shaped curve with a threshold dose of approximately 30 to 60g. of alcohol per day for hypertension (Keil, 1990).

The health beliefs of the women subjects helped to further explain their susceptibility to hypertension. More than half believed that heart disease can be prevented. Non-hypertensive women were significantly more likely to believe that poor diet, lack of exercise, and smoking are major causes of heart disease/problems and stroke. Belief that poor diet was a major cause of heart disease/problems and stroke, and the belief that smoking was a major cause of heart disease were independent predictors of hypertensive status. Despite these beliefs non-hypertensive women have a high prevalence of smoking.

Approximately one half of the women subjects believed that excess stress, worry or tension were major causes of heart disease (Non-hypertensive women: 49%, and hypertensive women: 53%), far larger percentages than any other suggested causes for the hypertensive women and one of the largest percentages for the non-hypertensive women. More than one third of all the subjects believed excess stress, worry or tension were major causes of strokes. Despite few studies which have measured psychosocial factors such as stress to predict the prevalence of and susceptibility to hypertension, there appears to be a commonly held belief

that stress can lead to hypertension. Researchers (Seyle, 1956; Sapolsky, 1990; Kasl, 1978; Murphy, 1991) suggest that the physiological response to stress can lead to hypertension or high rates of cardiovascular disability. Efforts at interventions such as stress management have had inconsistent results (Patel et al, 1981,1985; Van Montfrancs et al, 1990; Ching & Beevers, 1991). Serious intervention efforts such as those described in the Lifestyle Intervention Trial (Ornish et al, 1990) have shown very promising results. Twice weekly sessions in stress management carried out for one year showed conclusive evidence of atherogenic regression indicating the potential for this preventive action. The potential for further research into the personal contributors to stress could be realized by following up initial interviews with qualitative interviews which allow the researcher to learn more about the subjects life. Knowing more about life events and ability to cope with events, may help to explain some of the presently unknown susceptibility to hypertension.

Hypertensive women were significantly more likely to believe that hypertension was a major cause of stroke, indicating that information on the risk for stroke is being shared with the patients.

Non-hypertensive women are significantly more likely to believe eating fatty foods, and getting too little exercise can cause hypertension. They also were significantly more likely to believe sodium, cholesterol in

food, drinking caffeine/coffee, or eating fried, greasy or oily foods, was related to high blood pressure.

Health beliefs in fact preceded actual changes in health behaviors with non-hypertensive women significantly more likely to have increased their exercise, although actually only 20% did so. Twenty-eight percent improved their eating habits, but not significantly more than the hypertensive women. Very few in either group actually quit smoking (non-hypertensive women 7%; hypertensive women 6%). Both increasing exercise and improving eating habits were independent predictors of hypertensive status with non-hypertensive women significantly more likely to make these behavior changes.

Non-hypertensive women were significantly more likely to intend to increase exercise and quit or reduce smoking and both intended behavior changes were independent predictors of hypertension.

These results highlight the need for more effective health education especially aimed at population at risk for hypertension. The hypertensive women in this study were significantly less likely to believe in or practice preventive health behaviors such as improving eating habits, increasing exercise or quitting or reducing smoking.

Non-hypertensive women are significantly more likely to believe that dietary fat is related to overweight, obesity and to heart disease, heart problems or heart attack and these beliefs were found to be independent

predictors of hypertensive status. More non-hypertensive women believed that dietary cholesterol could increase blood pressure, but the difference did not quite reach significance. Non-hypertensive women are also significantly more likely to believe that high levels of cholesterol in the blood can cause heart attack/disease. They also were significantly more likely to believe exercising regularly can lower blood cholesterol levels. Most women trying to lose weight are doing so to improve general health, but non-hypertensive women are significantly more likely to try to lose weight to become more attractive than are hypertensive women.

The reduced logistic regression model of non-biomedical predictors considered simultaneously showed five which had unique predictive ability. Exercising strenuously was one predictor which was optimally predictive of non-hypertensive status. This is encouraging and indicative of the positive influence of strenuous exercise as a preventive measure for hypertension in women. The variable: being a homemaker is somewhat surprising as a predictor of hypertension and requires further investigation to understand what it is about being a homemaker that leads to susceptibility to hypertension. There are many possibilities not yet explored. In addition to these two predictive variables, there were three health beliefs that emerged. The belief that high blood pressure causes strokes was predictive of hypertensive status, possibly because the hypertensive women learned this from frequent visits with physicians.

The health belief that poor diet is a major cause of stroke, and high blood cholesterol causes heart disease/heart attack were predictive of non-hypertensive status indicated a lack of knowledge or awareness amongst the hypertensive women. One of the most disturbing results of present data has been the health beliefs of the hypertensive women, indicating a great need for raising awareness among this group.

Despite the evidence that benefits of cessation of smoking, moderation of alcohol consumption, weight loss, physical exercise, and reduction of dietary cholesterol should be emphasized as an alternative to pharmacologic measures (Leren, 1990), the present data indicate that the vast majority of the hypertensive women (91%) are being treated by pharmacologic therapy. The data further indicate that the hypertensive women share health beliefs as well as specific demographic characteristics, lifestyles, anthropometric and blood lipid measures, and that these health beliefs indicate that these women do not believe in some of the alternatives to antihypertensive treatment. These beliefs may be due to lack of knowledge, lack of awareness, or lack of information but open up many possibilities for public health approaches. Approaches which include accessible information, individual problem-solving, and supportive health education could be appropriate ways of dispelling these beliefs. Changes in individual beliefs may be necessary before successful lifestyle changes can be adopted by these hypertensive women.

CHAPTER 6

A SYNTHESIS OF BIOMEDICAL AND NON-BIOMEDICAL
PREDICTORS OF HYPERTENSIVE STATUS

By approaching the reduction of hypertension and its sequelae CHD and stroke, through the framework of an expanded medical paradigm, which includes both biomedical and non-biomedical variables, our understanding of the relationships of multiple risk factors may be increased. An expanded medical paradigm has the advantage of considering a comprehensive array of variables including biological, psychological, sociological and ecological. The biomedical paradigm has the disadvantage of looking at the biological mechanisms of the body only, and thereby limiting understanding of hypertension to single level physical explanations. For example, the strongest predictors of hypertension in women, as determined by analysis of the biomedical data, include: age, total/HDL cholesterol ratio, and personal history of other heart disease. We are left with the question; "Are there other predictors of a non-biomedical origin that we don't know about because of the paradigm in which we work?"

If the medical paradigm is expanded to include the non-biomedical predictors such as: being a homemaker, not exercising strenuously, having the health belief that high blood pressure causes stroke, and not having the

beliefs: poor diet is a major cause of stroke or high blood cholesterol causes heart attack/heart disease, then we can move from a single level of explanations (biomedical) to multilevel explanations of hypertension including: biomedical, ecological, psychological and social.

In addressing the third research question, "*Can the risk factors, biomedical and non-biomedical be synthesized into an expanded model which is optimally predictive of hypertensive status in women?*", the biomedical and non-biomedical variables were combined. Figure 6.1 presents a combination of the biomedical and non-biomedical predictors of hypertensive status in women as identified from reduced logistic models, when groups of variables were analyzed. These variables together combine information from multiple levels of organization including mind, culture and body, leading to a much fuller explanation of what predicts hypertensive status in women.

Figure 6.1 Biomedical and Non-Biomedical Risk Factors
Which are Predictive of Hypertensive Status
in Women in Reduced Logistic Models
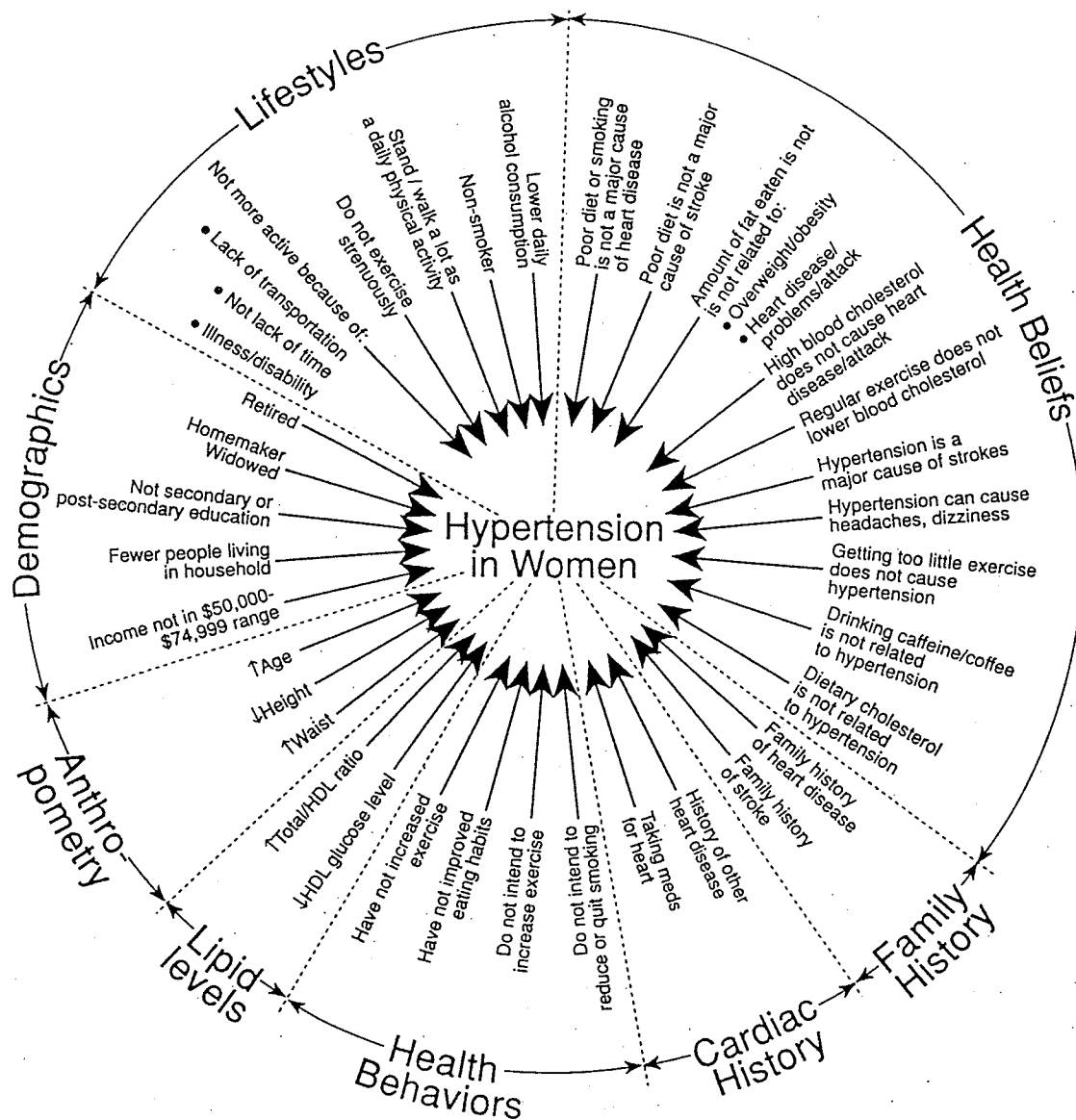
The inclusion of non-biomedical factors in a framework based on an expanded medical paradigm is consistent with frameworks developed by other researchers. The psychological model (Veatch, 1980) focuses on the links between the individual and the mind, and the biopsychosocial model (Engel, 1981) makes an effort to address the complex interactions between the physiological, psychological, and social contexts.

The inclusion of biomedical and non-biomedical factors in a framework is also consistent with the social structural model (Moos, 1984) which takes into account one's environmental system in affecting health outcome. It notes the association of poor health habits with lower socioeconomic class, and hypothesizes that factors in the social environment may alter health risk behaviours such as eating, smoking and drinking patterns and thereby affect health status. Similarly, but referencing the ecosystem as well, the ecological view of health suggests that health outcome is the product of the continuous interaction and interdependence of the individual with his or her ecosphere; that is, the family, community, culture, societal structure and physical environment (Green & Raeburn, 1988).

The framework as presented in Figure 6.1 provides biomedical and non-biomedical risk factors which were predictive when they were included in reduced logistic models along with other variables from their

own level or group such as lipid levels, demographics and anthropometric measures.   It presents numerous possibilities for furthering our understanding of hypertension in women and provides a great deal of information concerning known risk factors for hypertension in women. For example, women who are susceptible to hypertension tend to be older, more likely retired or homemakers, widowed, less educated, with lower incomes and living in smaller households. They also tend to be shorter with larger waist measurements, higher cholesterol ratios, higher glucose levels, and lower HDL cholesterol levels. History of stroke or heart disease runs in the family, and they have history of other heart disease as well as presently take medication for heart problems.

These findings are plausible and seem to be consistent with previous research. However, some of the findings in this data appear somewhat surprising. The hypertensive women tend to be non-smokers, consumed less alcohol daily and tended to stand or walk a lot as their daily physical activity, all of these are positive lifestyle behaviours. However, because the time-sequencing of events is unknown, the cause-effect relationship can not be established.   In other words, it is not apparent whether the diagnosis of hypertension happens first and is followed by positive lifestyle behaviours, or whether the positive lifestyle behaviours are present before the diagnosis of hypertension. It was also found that the hypertensive women did not tend to exercise strenuously. However, what

became very apparent when these findings were examined was the risk due to health-compromising health beliefs and behaviours. The hypertensive women did not believe that poor diet, smoking, amount of fat eaten, or high blood cholesterol would lead to adverse cardiac outcomes. Similarly they had not increased exercise or improved eating habits in the past year, and did not intend to do so in the future.

Despite these findings, Figure 6.1 has the same limitation as many of the previous models reviewed in that it is only able to show a linear relationship between each of the predictors and hypertension. It is not able to show any intervariate relationships. Although it provides information from multiple levels of organization, it is not able to link them together to provide multivariate or multilevel interactions or show loop structure. As suggested by Foss and Rothenberg (1987) a more comprehensive framework is desirable which is able to show relationships between risk factors and relationships between combinations of risk factors.

To move to a truly expanded medical paradigm, a more complex framework is needed, a framework which is comprehensive, flexible, and represents a wide range of relationships as well as including variables which are presently beyond the scope of the biomedical paradigm. Methods which have been employed up to now in this study to quantify both the biomedical and non-biomedical factors in this sample assure that

relationships happen in a linear fashion. However, a linear model is limited in explaining relationships, dimensions, and complexity of the connections between factors and outcome, which may be other than linear and independent. In order to gain some additional flexibility and information, factor analysis was employed to identify linear combinations which could include multivariate interactions and multilevel interactions.

In addressing research question 4: "*What is the potential of additional information to be useful in synthesizing risk factors to show other than linear relationships?*" additional statistical methods were employed. The entire set of variables was again assembled and the statistical procedure, factor analysis (Cureton & D'Agostino, 1983), was used to search for commonalities in the variables, some common threads which would tie certain variables together. This study used the technique of principle factor analysis with varimax rotation. All factors with eigenvalues greater than 1.0 were selected for rotation. This procedure initially found commonalities within each group of variables such as: demographics, biomedical and beliefs, and then second-order factor analysis was used to find multivariate interactions between variables, as well as interconnections between the levels of variables including: biomedical, demographic, lifestyle, cardiac and family history, and beliefs and behaviors. This statistical procedure was able to identify individual variables which share a common theme with other variables and together

form a cluster of variables which tend to occur together. The variables from different levels which occur together demonstrate multivariate interactions within their level as well as multilevel interactions between factors in various levels. Finally, these multilevel factors were analyzed in a logistic regression model to identify which multilevel factors were optimally predictive of hypertension in women. Identifying each of these predictive factors, which contain multilevel variables which share a common theme, may help to further understand hypertension in women and the relationships between the multiple risk factors.

Initially, the variables were divided into groups in order to obtain an insight into the relationships and dominant themes within each of these. The presentation of the factor analysis relates major themes in each concept area including: demographics, lifestyle, cardiac and family history, health beliefs, health behaviors, and biomedical variables. Any variables which had more than 10% missing were omitted, as were variables with little variability.

Factor analysis as a statistical method is applied to a set of variables to seek underlying factors or groups of variables with a common theme. Each of the identified factors is based on a common theme which influences or show commonalities amongst variables. All women place or score on a factor, with some scoring very high, others very low and some score in the middle. The very high or very low scorers provide a dramatic

illustration of the 'theme' at its most or its least potent and they clarify the nature of the theme.

The results of the initial round of factor analyses include the following in order: lifestyle variables; demographic variables; biomedical variables; cardiac history/family history variables; health beliefs concerning fats and cholesterol variables; health beliefs concerning hypertension variables; and health beliefs concerning causes of heart disease and stroke variables.

The initial factor analysis of lifestyle variables identified four main factors. The first lifestyle factor is dominated by the variables "number of times per week exercise is done", "whether women are getting as much exercise as they need", and "reasons which prevent them from doing exercise". Women who score highly on this factor exercise fewer times per week, feel they are getting less exercise than they need and if they are prevented from doing exercise it is because of lack of time, lack of incentive or because they have no one to exercise with. The commonality or underlying theme of this factor is **frequency of exercise and barriers to exercise**. It indicates that there is an association between doing less exercise and having barriers which prevent them from exercising.

The second lifestyle factor was dominated by the variables "number of cigarettes smoked daily", "regular exercise" and "reason for not exercising is illness/disability". Women who scored highly on this factor

smoke more cigarettes, do not exercise regularly, and the only reason that they are prevented from doing exercise, is because of illness or disability. The commonality of this factor appears to be **health compromising behaviours**, with smoking being positively associated with inactivity and illness/disability being a further barrier to more activity. This factor appears to be mainly an index of exercise and in addition indicates a positive association exists between inactivity and the health compromising behaviour, smoking.

The third lifestyle factor is dominated by the variables "exercising strenuously", "activity compared to others of the same age", and "reason for not exercising is lack of facilities for exercising". Women who score highly on this factor tend to exercise strenuously, tend to think they are more active than others their own age, and if they are prevented from doing exercise it is due to lack of facilities to exercise. This factor appears to be mainly an **index of regular exercise**, and indicates a positive association between strenuous exercise and a perception of being more active than others of the same age.

The fourth lifestyle factor is dominated by the variables "extent of physically demanding day time activities", "extent of physical activity relative to peers", and (with contrasting sign) "number of alcoholic drinks consumed per day". Women who score highly on this factor have physically demanding day time activities, view themselves as being more

physically active than their peers and have a relatively low daily alcohol consumptioin. This factor appears to be primarily an **index of routine physical activity** and, in addition, indicates that a negative association exists between routine physical activity and a key health compromising behaviour, alcohol consumption.

The initial factor analysis of the demographic variables identified three factors. The first demographic factor is dominated by the variables "worktype" and "years of education". Women who scored highly on this factor were women with less skilled occupations and less schooling. This factor appears to be primarily an **index of low socioeconomic status** with those with little education having less skilled jobs.

The second demographic factor loads heavily on the variables "number of people in the household" and "household income". The women who scored highly on this factor have fewer people living in the household and lower household incomes. This factor appears to be primarily an **indicator of age related circumstances** which include an association between older women, who tend to live with only a spouse or alone, and are retired or on fixed income.

The third demographic factor loads heavily on the variables "employment", "marital status", and "age". Women who scored highly on this factor were women who were retired or homemakers, women who had been widowed and women who were older. This factor appears to be

an **indicator of age** and indicates that a positive relationship exists between older age, being widowed, and being retired or a homemaker.

The initial factor analysis of the biomedical variables identified four factors. The first biomedical factor is dominated by the variables "waist size", "weight", "hip size", and "body mass index". Women who scored highly on this factor tended to be larger is size, had larger waists and hips, weighed more and had a higher body mass index. This factor seems to have **the commonality of body size**. As an index of body size it indicated that a positive association exists between larger waist size, heavier weight, larger hip size and higher body mass index.

The second biomedical factor loads heavily on the lipid variables including "total cholesterol level", "low density lipoprotein level", triglyceride level" and "cholesterol ratio". Women who score highly on this factor have high blood lipid levels as measured by their total cholesterol level, low density lipoprotein level, triglyceride level and cholesterol ratio. This factor is primarily **an index of lipid levels** and indicates that a positive relationship exists between high levels of blood cholesterol as indicated by these measures.

The third biomedical factor also is dominated by lipid measures including "high density lipoproteins" and "total cholesterol level". Women who score highly on this factor have high high-density lipoprotein levels and low total cholesterol levels, both indicating healthy levels of blood

lipids. This factor is **an index of lipid levels** but indicates that a negative association exists between high-density lipoprotein levels and total cholesterol levels.

The fourth biomedical factor is dominated by the variables "height" and "glucose level". The women who score highly on this factor were taller and had lower glucose levels. This factor appears to be primarily an **index of body height** and, in addition, indicates that a negative association exists between height and glucose level, with the taller women having lower glucose levels.

Factor analysis of the cardiac history/family history identified two factors. The first cardiac history/family history factor is dominated by the variables "family history of heart disease", "family history of stroke" and "presently suffering from other heart disease". The women who score highly on this factor have family history of heart disease or stroke and are presently suffering from other heart disease. This factor is primarily an **indicator of family history of cardiovascular disease** and, in addition, indicates that a positive association exists between family history of cardiovascular disease and presently suffering personally from other heart disease.

The second cardiac history/family history factor leans heavily on the variables "family history of high blood pressure" and "family history of high cholesterol". Women who score highly on this factor have a family

history of high blood pressure or high cholesterol. This factor is primarily **an index of family history of risk factors for heart disease**.

The health beliefs of the women were analyzed according to major concepts. The relationships in the major theme areas of "health beliefs concerning fats and cholesterol", "health beliefs concerning hypertension", and "health beliefs concerning causes of heart disease and stroke" were examined.

The initial factor analysis of the health beliefs concerning fats and cholesterol identified five factors. The first belief factor concerning fats and cholesterol loads heavily on the beliefs "dietary cholesterol is related to heart attack", "dietary cholesterol is related to stroke", "blood cholesterol is related to heart attack", and "blood cholesterol is related to stroke". Women who score highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of belief that cholesterol is a major risk factor for cardiovascular disease**, and in addition indicates that a positive association exists between these beliefs.

The second belief factor concerning fats and cholesterol loads heavily on the beliefs "dietary fat is related to arteriosclerosis", "dietary cholesterol is related to arteriosclerosis", and "blood cholesterol can cause arteriosclerosis". Women who score highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of beliefs concerning arteriosclerosis** and, in addition, indicates that a positive

association exists between the beliefs that fat and cholesterol are risk factors for arteriosclerosis.

The third belief factor concerning fats and cholesterol loads heavily on the beliefs "dietary cholesterol is related to other outcomes", "blood cholesterol is related to other outcomes", and "blood cholesterol can be lowered by methods other than exercising and modifying diet". Women who score highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of beliefs concerning cholesterol as a risk factor** and indicates that cholesterol is not believed to be a risk factor for adverse cardiovascular outcomes but may be a risk factor for other adverse outcomes. In addition it indicates a positive association exists between the belief that cholesterol is not a risk factor for adverse cardiovascular outcomes and the belief that dietary cholesterol may be lowered by other methods than exercising and modifying diet.

The fourth belief factor concerning fats and cholesterol loads heavily on the beliefs "fat eaten is not related to heart disease", "fat eaten is not related to other outcomes not listed", and "fat eaten is related to blood cholesterol". Women who score highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of beliefs concerning the relationship between dietary fat and adverse outcomes** and, in addition, indicates that a negative association exists between the beliefs

that fat eaten is not related to heart disease or other outcomes, but fat eaten is related to blood cholesterol.

The fifth belief factor concerning fats and cholesterol loads heavily on the beliefs "fat eaten is not related to blood pressure", "dietary cholesterol is not related to blood pressure", and "high blood cholesterol does not cause high blood pressure. Women who score highly on this factor tend to have these beliefs. This factor appears to be primarily an **index of beliefs concerning fat and cholesterol as risk factors for high blood pressure** and it indicates that a positive association exists between the beliefs that fat and cholesterol are not risk factors for high blood pressure.

The next set of health beliefs analyzed were health beliefs concerning hypertension. The initial factor analysis of the health beliefs concerning hypertension identified five factors. The first belief factor concerning hypertension is dominated by the variables "eating fatty foods causes hypertension", "eating too much sugar and sweets causes hypertension", and "eating too many calories causes hypertension". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an indicator of beliefs that hypertension is caused by dietary habits**, with a positive association between the beliefs that fatty foods, too much sugar and sweets, and eating too many calories, are all causes of hypertension.

The second belief factor concerning hypertension is dominated by the variables "hypertension does not cause stroke or heart attacks", and "hypertension can cause other problems which were not listed". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an indicator of beliefs that hypertension is not a major cause of cardiovascular diseases**, and in addition indicates that hypertension may cause medical problems but that these problems do not include heart attack or stroke.

The third belief factor concerning hypertension is dominated by the variables "eating too much salt causes hypertension" and "a relationship exists between dietary salt and hypertension". Women who score highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of the belief that salt and hypertension are related**.

The fourth belief factor concerning hypertension is dominated by the variables "smoking does not cause hypertension", "drinking too much alcohol does not cause hypertension", and "high blood pressure is not related to the amount of caffeine or coffee consumed". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of beliefs about lifestyle as a cause of hypertension**, and in addition, indicates that the lifestyle choices: smoking, drinking too much alcohol and drinking a large amount of caffeine are not believed to cause hypertension.

The fifth belief factor concerning hypertension is dominated by the variable "heredity causes hypertension". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of the belief that heredity acts alone in causing hypertension.**

The final set of health beliefs analyzed were beliefs concerning the causes of heart disease and stroke. The initial factor analysis identified five factors. The first belief factor concerning the causes of heart disease and stroke is dominated by the variables "poor diet is a major cause of heart disease", "lack of exercise is a major cause of heart disease", "poor diet is a major cause of stroke", "lack of exercise is a major cause of stroke", and "heredity is a major cause of stroke" and "heredity is a major cause of heart disease". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of the belief that cardiovascular diseases are the result of a combination of lifestyle and heredity together** and, in addition, indicates that a positive assocation exists between the beliefs that poor diet, lack of exercise and heredity are causes of heart disease and stroke.

The second belief factor concerning causes of heart disease and stroke is dominated by the variables "excess salt is a major cause of heart disease", "excess fats are a major cause of stroke", and "excess salt is a major cause of stroke". Women who scored highly on this factor tend to have these beliefs. This factor appears to primarily be **an indicator of the**

belief that excess fats and excess salt in the diet can cause cardiovascular disease.

The third belief factor concerning causes of heart disease and stroke is dominated by the variables "high blood cholesterol is a major cause of heart disease", "high blood pressure is a major cause of heart disease", and "high blood pressure is a major cause of stroke". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of the belief that blood pressure and blood cholesterol can cause heart disease and stroke** and in addition indicates the belief that a positive association exists between high blood cholesterol and high blood pressure and adverse cardiovascular outcomes.

The fourth belief factor concerning heart disease and stroke is dominated by the variables "smoking is a major cause of heart disease", "other causes not listed can cause heart disease", and "other causes not listed can cause stroke". Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of the belief that smoking can cause heart disease** and, in addition, indicates the belief that besides smoking other causes of cardiovascular disease are not listed.

The fifth belief factor concerning heart disease and stroke is dominated by the variables "overwork or fatigue are not major causes of heart disease", "overwork or fatigue are not major causes of stroke", "stress,

worry or tension does not cause heart disease", and "stress, worry or tension does not cause stroke. Women who scored highly on this factor tend to have these beliefs. This factor appears to be primarily **an index of the belief that emotional stress or physical stress does not lead to adverse cardiovascular outcomes.**

The results of the initial factor analysis of behaviour variables identified three factors. The first behaviour factor loaded heavily on the variables "presently not trying to lose weight", "presently not dieting to lose weight", "presently not exercising to lose weight", and "not intending to lose weight in the next year". Women who scored highly on this factor tend to have these behaviours. This factor appears to be primarily an **index of weight modification or control** and, in addition, indicates that the individuals not practicing weight modification or control scored heavily on this factor.

The second behaviour factor loaded heavily on the variables "intend to do something to improve health", "intend to exercise", and "intend to improve eating habits". This factor appears to be primarily **an index of intention to improve health behaviour.** The women with a commitment to improvement scored heavily.

The third behaviour factor loaded heavily on the variables "do not intend to control blood pressure" and "do not intend to reduce stress level". This factor appears to be primarily **an index of intention to make**

behaviour changes. Women who scored highly on this factor did not intend to implement these behaviour changes.

A second-order factor analysis (Cureton & D'Agostina, 1983) was done to discover if any correlations existed between factors which belonged to different levels. Up to this point, within each factor the relationships between variables are able to show multivariate interactions and loop structure. In order to find links which can show relationships between factors and relationships between combinations of factors, a second-order factor analysis of the factors from each level of organization was done. The factors from the demographic, biomedical, belief, behaviour and historical levels were factor analyzed together to determine interconnections between the levels. Multilevel commonalities which exist in more than one level of factors, rather than just the single level commonalities present in the stored factors were sought in this analysis.

Correlations between each of the stored single level factors are shown in the correlation matrix (Figure 6.2).

FIGURE 6.2   CORRELATION OF ALL FACTORS MATRIX

The individual correlations between each factor in each level are shown. However, when the stored factors were factor analyzed, eleven multivariable multilevel factors were identified. The number of factors identified was determined by retaining all factors with an eigenvalue of >1.0.. Although research shows this to be an acceptable practice (Cureton & D'Agostino, 1983), in this case it resulted in more factors than may be expected. However, considering the large number of variables in this data set, fewer factors were not able to show the many relationships which became apparent when all eleven factors were retained. Also, as the intention was to use these multilevel factors in a logistic regression model, more precise relationships within each factor would lead to better understanding of the stated outcome: hypertension in women. Although not all of the factors would be relevant to hypertension in women, they each showed some interesting relationships which will be briefly described.

The first multilevel factor is dominated by the factors which have the commonalities or themes "low socio-economic status", "fats and cholesterol can cause arteriosclerosis", and "lifestyle including diet and exercise, and heredity can cause cardiovascular disease". Women who scored highly on this factor were women of low socioeconomic status, who did not have these health beliefs. This factor appears to be primarily an **index of health beliefs held by lower socioeconomic women** and in

addition indicates that a negative association exists between low socioeconomic status and these health beliefs. This factor demonstrates that low socioeconomic status women have these health-compromising beliefs. This multilevel factor demonstrates the underlying theme that women of lower socioeconomic status have many health-compromising health beliefs. They do not share the beliefs that any of the known risk factors such as high fat diet, lack of exercise, heredity or high blood cholesterol can lead to adverse cardiovascular outcomes.

The second multilevel factor is dominated by the factors which have the commonalities or themes "older age", "higher lipid levels", and "family history of cardiovascular disease". Women who scored highly on this factor were older in age, had higher lipid levels, and had family history of cardiovascular disease. This factor appears to be primarily **an index of aging** and, in addition indicates that a positive association exists between aging and higher lipid levels, and family history of disease. This multilevel factor demonstrates the underlying theme that older women have higher lipid levels, are more often widowed, are retired from work or homemakers, and have a family history of cardiovascular events. Their higher blood lipid levels include total cholesterol level, LDL cholesterol level, triglyceride level and total cholesterol/HDL ratio and they also suffer personally from other heart disease.

The third multilevel factor is dominated by the factors which have the commonalities or themes "larger body size", "practicing weight modification or control". Women who scored highly on this factor were women of larger body size, who were practicing weight modification or control. This factor appears to be primarily **an index of weight control behaviours of women with larger body size**. In addition, this factor indicates that a positive assocation exists between weight modification and larger size women, with these women practicing and intending to practice weight control in the coming year.

The fourth multilevel factor is dominated by the factors which have the commonalities or themes "dietary cholesterol is not a risk factor for adverse cardiovascular outcomes but may be a risk factor for other adverse outcomes", "hypertension is a major cause of heart disease, stroke and other health problems", "high blood cholesterol and high blood pressure can cause adverse cardiovascular outcomes", and "intend to make the behaviour changes of controlling blood pressure and reduce stress level. Women who scored highly on this factor were women who believed that hypertension, and high blood cholesterol were risk factors for cardiovascular outcomes, but dietary cholesterol was not, and these women intended to control blood pressure and reduce their stress level. This factor appears to be primarily **an index of beliefs that cardiovascular outcomes are out of the control of the individual,** are external to the

health-enhancing behaviour choices the women could make. In other words, adverse cardiovascular outcomes result from the internal conditions high blood cholesterol and high blood pressure, and lifestyle changes such as diet and exercise will not reduce these levels. Control of blood pressure and stress level, both aimed at hypertension control, are the only intended behaviour changes.

The fifth multilevel factor is dominated by the factors which have the commonalities or themes "smaller households and lower incomes", and "shorter body height". Women who scored highly on this factor were women who lived in smaller households and had smaller incomes, and tended to be shorter in height. This factor appears to be primarily an **index of age related circumstances** and, in addition indicates that a positive association exists between smaller households, lower incomes, shorter stature and higher glucose levels. This factor demonstrates these factors are all age related circumstances.

The sixth multilevel factor is dominated by the factors which have the commonalities or themes "cholesterol, both dietary and blood, is not a major risk factor for cardiovascular diseaase", and "too much dietary salt does not cause hypertension". Women who scored highly on this factor were women who held these beliefs. This factor appears to be primarily **an index of health-compromising beliefs concerning causes of cardiovascular disease and hypertension.**

The seventh multilevel factor is dominated by the factors which have the commonalities or themes "not intending to improve health behaviour", and "not having a family history of risk factors for heart disease". Women who scored highly on this factor did not intend to exercise or improve eating habits and they did not have family history of highl blood pressure or high blood cholesterol. This factor appears to be primarily **an index of intended health behaviours,** and in addition, indicates that a positive association exists between not intending to improve health behaviours, and not having family history of risk factors for cardiovascular disease.

The eighth multilevel factor is dominated by the factors which have the commonalities or themes "smoking and other causes not listed are not causes of cardiovascular disease". Women who scored highly on this factor were women who held these beliefs. This factor appears to be primarily **an index of the belief that smoking does not cause cardiovascular disease,** and in addition, indicates that a positive association exists between the belief that smoking does not cause cardiovascular disease and other causes not listed do not cause cardiovascular disease.

The ninth multilevel factor is dominated by the factor which has the commonality or theme "heredity causes hypertension". Women who scored highly on this factor were women who held this belief. This factor appears to be primarily **an index of the belief that heredity causes**

hypertension, and in addition, indicates that heredity which is external to the individual's control is the sole cause of hypertension. This factor indicates that these women consider hyptertension outside of their control, there is nothing they could do to change their susceptibility as it is caused by a circumstances beyond their control.

The tenth multilevel factor is dominated by the factors which have the commonalities or themes "hypertension is the result of dietary habits." Women who scored highly on this factor tended to believe that eating fatty foods causes hypertension, eating too much sugar causes hypertension, and eating too many calories causes hypertension. This factor appears to be primarily **an index of the belief that dietary habits cause hypertension,** and indicates a positive relationship exists between the beliefs that fatty foods, too much sugar, and too many calories are all harmful dietary habits.

The final multilevel factor is dominated by the factors which have the commonalities or themes " fat and cholesterol are not risk factors for high blood pressure". Women who scored highly on this factor tended to believe that dietary fat is not related to high blood pressure, dietary cholesterol is not related to high blood pressure, and high blood cholesterol does not cause high blood pressure. This factor appears to be primarily **an index of the belief that dietary fat and dietary cholesterol, as well as blood cholesterol are not causes of hypertension.**

These eleven multilevel factors provide a summary of some of the characteristics of the subjects in this sample of women. They provide information on the interconnections which exist between not only individual variables which share common themes to form a factor, but clusters of interconnected variables from different levels which combine to share a common theme in a multilevel factor. Multilevel factors include biomedical and non-biomedical variables together where they share common themes. In order to use this additional information to its best advantage in furthering the understanding of hypertension in women, all eleven multilevel factors were included in a logistic regression model, with hypertensive status in women as the outcome. A significant relationship was observed between these final factors and hypertensive status (Model chi square = 58.55 with 11 d.f., p<0.0000).

A stepwise logistic regression model was done which eliminated the variables that did not reach statistical significance at the 0.05 level of probability. Table 6.1 provides the beta estimate and standard error of those factors which were statistically significant.

Table 6.1 Multilevel Factors Predictive of Hypertensive Status
          in a Final Logistic Model

Multilevel Factors	Beta Estimate	Standard Error	Chi Square Beta = 0
Factor 2	1.444	.289	24.93
Factor 4	1.262	.359	12.37
Factor 11	1.362	.482	7.97
Factor 1	1.073	.383	7.85
Factor 9	.453	.214	4.50

This regression model indicates that multilevel factor 2, multilevel factor 4, multilevel factor 11, multilevel factor 1 and multilevel factor 9 are the factors that show unique predictive ability in predicting hypertension in women. They are the most effective in characterizing the impact of multilevel factors on hypertensive status in women.

The results of this logistic regression of multilevel factors provide additional information to what is already known from the final logistic regression of biomedical and non-biomedical variables in Figure 6.1. In addition to finding the individual variables which are optimal predictors of hypertension in women, the framework can be expanded to include five multilevel factors which are optimally predictive and which bring additional information concerning hypertension in women. Not

surprisingly, the most optimally predictive multilevel factor is multilevel factor 2, which includes four of the individual variables which are optimally predictive in the final logistic model in Chapter 5. In fact all of the single level predictors from the final logistic model in Chapter 5 are included in one of the multilevel factors that are predictive of hypertension. The one exception is the variable "exercising strenuously" that was not included as lifestyle factors were omitted from the multilevel factor analysis due to having too many missing values.

The inclusion of the five multilevel factors which are optimally predictive of hypertensive status does add to our understanding. Multilevel factor 2, which is the most predictive multilevel factor includes many variables which share the commonality of occurring more often in older women. These include the single variable predictors: age, being a homemaker, having a higher Total/HDL cholesterol ratio and having a history of other heart disease which are independently optimally predictive of hypertension, but by occurring within the same multilevel factor, appear to often exist together, not just independently. In addition, as well as occuring together, a number of other variables present in multilevel factor 2, also occur. Multilevel factor 2 indicates that being retired or a homemaker, being widowed, having higher levels of LDL cholesterol, triglycerides, total cholesterol and a higher total/HDL cholesterol level, having family history of heart disease or stroke, and presently suffering

from other heart disease, are all age related variables which occur together in women with increased susceptibility to hypertension.

Figure 6.3 presents the multilevel factors which are predictive of increased susceptibility to hypertension in women. Some single level predictors within multilevel factors are in bold print to emphasize that they were also independent predictors of hypertension. The commonalities which exist within the multilevel factors and their relationships to the independent optimally predictive variables becomes somewhat complex.

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Figure 6.3 Multilevel Factors Predictive of Hypertensive Status in Women
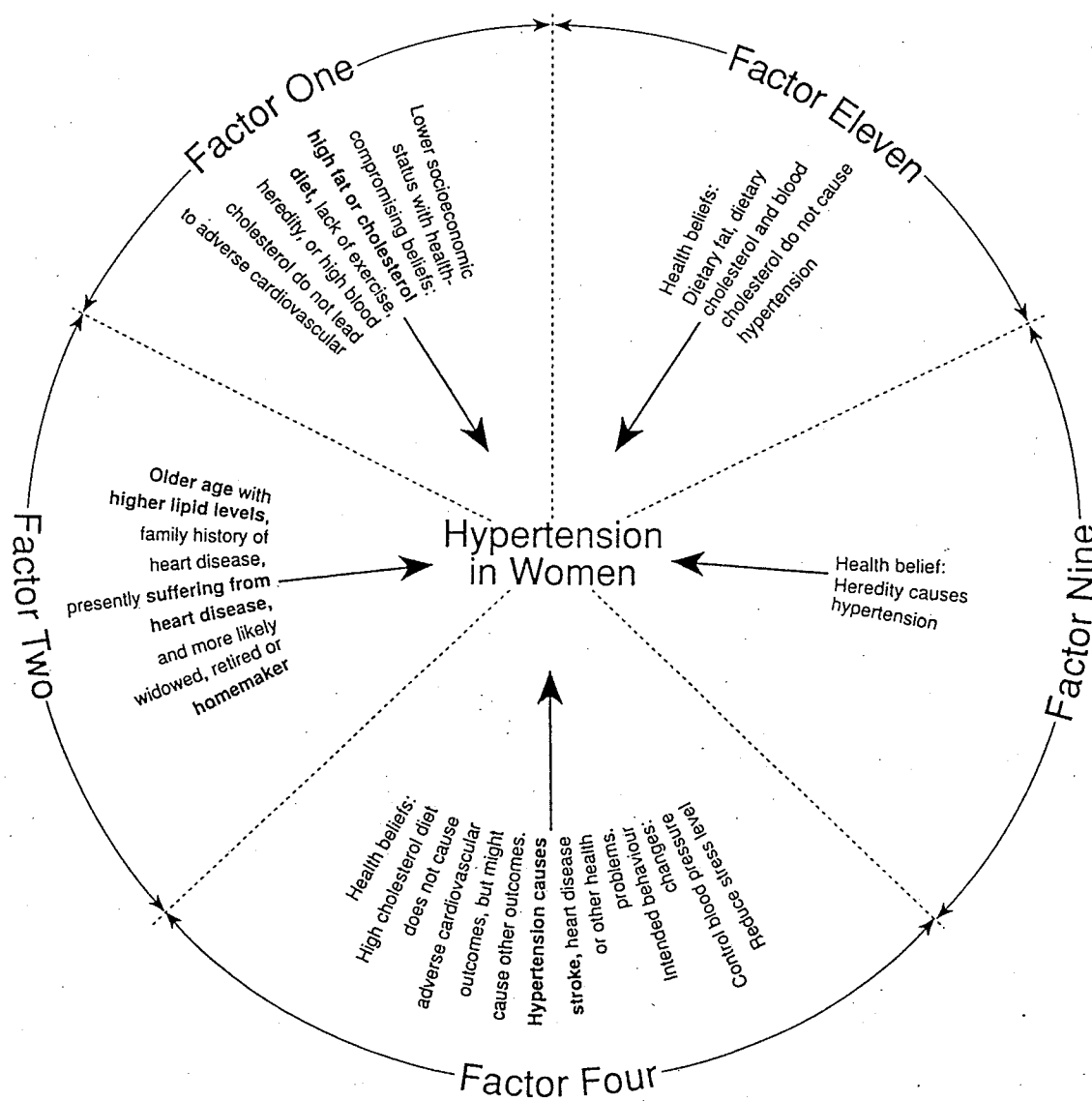
Multilevel factor 1 predicts that women of lower socioeconomic status will be more likely to be hypertensive, but as well, these women will have a number of health-compromising beliefs which put them at risk. They believe that dietary fat, dietary cholesterol and blood cholesterol are not related to arteriosclerosis, and that poor diet, lack of exercise, or heredity do not lead to heart disease or stroke. These beliefs are contrary to what is known of risk factors for adverse cardiovascular outcomes. Therefore, this lower socioeconomic status group of women appear to not know the risk factors for cardiovascular disease and although knowledge itself is not the sole feature of behaviour adoption it is a predisposing factor and may determine what information one pays attention to. The variables in multilevel factor 1 appear to be education related with those with lower education having health-compromising beliefs. It would not be useful to suggest lifestyle modifications such as dietary changes to this group, when they hold these health beliefs. Either the messages about heart health have not been accessed by this group, or perhaps they don't believe or understand the messages. This factor provides some possible reasons for the higher prevalence of hypertension in lower socioeconomic groups of women.

Multilevel factor 2 is strongly predictive of hypertensive status, consisting of older women who have biomedical characteristics which seem to be present more often in hypertensive women. These women are older,

with age being a strong independent predictor of hypertension as well as a component of this factor. This factor contains four of the eight independent predictors of hypertensive status, but shows that they share the commonality of occurring together, as well as together being predictive of hypertension. Older women, as well as being more likely to be hypertensive, are also more likely to have high lipid levels. This supports the concern raised by several researchers (Anostos et al, 1991), that treating hypertension when hypercholesterolemia is also present may prove harmful, by further altering lipid profiles and thereby increasing risk to these women. Many of these women are homemakers or retired and have been widowed. These demographics may be the direct result of being older but may also contribute to lack of access to information about risk factors for heart disease due to social isolation.

Multilevel factor 4 is also shown to be predictive of hypertensive status in women. It consists of a number of health beliefs including two of the three beliefs which were independent predictors from the final logistic model. These health beliefs were: hypertension causes strokes and high blood cholesterol can cause heart disease. These health beliefs provide additional insight into the beliefs held by women who are hypertensive. Several of their beliefs indicate they are not familiar with some of the known risk factors for cardiovascular disease. They believe that dietary cholesterol may cause adverse outcomes, but not

cardiovascular. They believe there are ways to reduce blood cholesterol levels but these do not include modifying diet or increasing exercise. Their intended behaviours are controlling blood pressure and reducing stress, both health enhancing behaviours.

Multilevel factor 9 is predictive of hypertension and consists of the health belief heredity causes hypertension. Although it does not share commonalities with other health beliefs, it is consistent with the health beliefs in the other predictive multilevel factors as it reiterates the belief that hypertension itself is caused by events out of the control of the individual, and the women can do nothing themselves to change these circumstances.

Multilevel factor 11 is predictive of hypertension and further supports the belief held by hypertensive women that hypertension results from external sources outside of the women's control. The theme of this factor is the denial of diet or even blood cholesterol as possible risk factors for hypertension, indicating that diet does not need to be controlled as it is not believed to affect hypertension.

By examining these five multilevel predictive factors identified by a reduced logistic model, more information about hypertensive women becomes apparent, which in turn provides more information with which to direct prevention efforts to reduce the prevalence of hypertension in women. It seems apparent that the information in Figure 6.3 provides the

most comprehensive view of predictors of hypertension in women in the present study. The amalgam in Figure 6.3 is limited by the available data in the present data set. The amalgam is able to show multivariate interactions and multilevel interactions between the variables which are available in the data set. However, the data set does not include all the information about the ecosystem or culture within which each of the women exist so this data cannot be included. The present amalgam does include loop structured connections between predictors and hypertensive status, with the hypertensive women tending to have the predictive characteristics and each predictor making women more susceptible to hypertension. The present amalgam does not however, have the ability to show multilevel organization, due to the lack of knowledge concerning time sequencing. It is not possible from the above information to determine what events take place first. For example, whether a woman is hypertensive first and then develops high lipids, larger body size or certain health beliefs, or whether a woman with preexisting hypercholesterolemia, body size or certain health beliefs later develops hypertension.

An expanded medical paradigm allows for a broader perspective in understanding hypertension than the traditional biomedical paradigm. However, in this study the limitations in the data set made it impossible to totally achieve the comprehensive framework suggested by Foss and Rothenberg (1987). Data obtained from a longitudinal study would allow

time sequencing of events which would be helpful when trying to decipher which events are predisposing and which events are outcomes. Data collection which provides more information about a woman's culture or ecosystem would provide a more complete data set. The more information which is known concerning the predictors of susceptibility, the better chance of achieving a comprehensive framework and more fully understanding the roots of hypertension in women.

CHAPTER 7

SUMMARY AND CONCLUSIONS

The present study has attempted to further the understanding of the chronic disease hypertension in women by identifying and analyzing biomedical risk factors for their potential risk and for their ability to predict susceptibility to hypertension in a sample population of women, and by identifying and analyzing non-biomedical risk factors for their potential risk and for their ability to predict susceptibility to hypertension. The development of a framework to include both sets of risk factors was guided by the expanded medical paradigm proposed by Foss and Rothenberg (1987). The study used data which were part of a much larger data set collected by the Manitoba Heart Health Survey. The survey consisted of three separate components: a risk factor questionnaire; a food frequency nutrition questionnaire; and a clinic visit.

In addressing the first research question: What are the identifiable biomedical risk factors which are predictive of hypertension in women, variables including age, the anthropometric measures; short stature, larger waist, larger hip, larger WHR, higher BMI, and the cholesterol levels; higher total cholesterol, lower HDL cholesterol,

higher LDL cholesterol, higher triglycerides, higher glucose, diabetes and higher total/HDL ratio were found to be predictive in univariate analysis. Also family history variables including; family history of heart disease, hypertension, stroke, and personal history of heart attack, other heart disease and taking medication for heart problems were predictive in univariate analysis. All of these findings seem plausible in light of recent research findings.

To determine whether some of these variables were more predictive than others, the biomedical variables were then analysed in groups such as the group of anthropometrics, the group of blood lipid measures, and the group of cardiac and family history variables. The variables in each group which showed unique predictive ability included: age, short stature, larger waist measurement, higher total/HDL cholesterol ratio, lower HDL level, higher glucose level, and family history of heart disease or stroke, and personal histoy of other heart disease or taking medication for heart problems. The results of this analysis provided some more precise information as to which specific variables could represent a group of variables and provide the most information as to a woman's risk of hypertension. For example; total/HDL cholesterol ratio and HDL cholesterol level represent the

blood lipids group of variables and as such if it is not possible to measure all the cholesterol levels, measuring these two levels would be the most useful of the group in predicting hypertension in women.

To determine which of the biomedical variables are optimally predictive of hypertensive status in women, all of the variables which were predictive of hypertensive status in reduced logistic models were considered simultaneously. The variables age, history of other heart disease and total/HDL cholesterol ratio were the variables which showed unique predictive ability and were most effective in characterizing the impact of biomedical variables on hypertensive status in women.

In addressing the second research question: What are the identifiable non-biomedical risk factors of hypertension in women, variables including demographic variables, lifestyle variables, health beliefs and health behaviours were found to be predictive of hypertensive status in women. Initial univariate analysis of each variable resulted in the demographic variables; not having fulltime employment, being retired or a student, being single or widowed, having a grade six education, not having post secondary education, having fewer people in household, having an income of under $12,000,

and not having an income of $50,000-$74,000, being predictive of hypertensive status. The lifestyle variables; stand/walk a lot as daily activity, not lifting/carrying light loads as daily activity, not exercising strenuously, exercising for less than 15 minutes at a time, not presently smoking, drinking less alcohol, and reasons for presently not exercising including lack of transportation or illness/disability and not including lack of time, were also predictive in univariate analysis.

Variables such as not presently smoking and drinking less alcohol as predictors of hypertension in women were not expected but as little research has been done on women it raises some possible avenues for further research. Several of the health beliefs were predictive of hypertension including the beliefs: poor diet, lack of exercise, or smoking are not major causes of heart disease, poor diet, lack of exercise, or smoking are not major causes of stroke, hypertension is a major cause of stroke, overweight or heart disease are not related to amount of fat eaten, blood cholesterol is not a cause of heart disease, exercise does not lower blood cholesterol levels, fatty foods, lack of exercise or other causes not listed can cause hypertension, and sodium, dietry cholesterol, caffeine, or fried foods are not related to hypertension. Not intending to increase exercise or reduce smoking

or lose weight to look more attractive were also predictive of hypertensive status in women in univariate analysis.

To determine whether some of these variables were more predictive than others, the non-biomedical variables were then analysed in groups such as: demographics, lifestyle, beliefs and behaviours. The variables in each group which showed unique predictive ability provided some more precise information as to which specific variables could represent a group of variables and provide the most information as to a woman's risk of hypertension. To determine which of the non-biomedical variables were optimally predictive of hypertensive status in women, all of the variables which were predictive of hypertensive status in reduced logistic models were considered simultaneously. The variables; high blood pressure causes strokes, poor diet is not a major cause of stroke, high blood cholesterol is not a cause of heart disease, being a homemaker and not exercising strenuously were the variables which showed unique predictive ability and were most effective in characterizing the impact of non-biomedical variables on hypertensive status in women.

In trying to expand our understanding of hypertension the development of a framework including both biomedical and non-

biomedical predictors was guided by the expanded medical paradigm proposed by Foss and Rothenberg (1987). Following the parameters of this expanded medical paradigm allowed for multivariate interactions, loop structure, mutual causality and multilevel organization. It allowed links which could show relationships between risk factors and relationships between combinations of risk factors, rather than being limited to linear relationships between hypertensive status and predictors.

By using the expanded medical paradigm more information about predictors of hypertensive status in women was shown. The final amalgam of predictors showed five multivariate, multilevel groups of variables which shared a commonality or theme, which were predictive of hypertension in women. These groups or factors included: a group with the commonality of aging, a group with the commonality of low socioeconomic status, a group with the common belief heredity causes hypertension , a group with the commonality that they believed that cardiovascular outcomes are out of the control of the individual, and a group with the common belief that dietary fat and cholesterol and blood cholesterol are not causes of hypertension.

The present study has suggested potential explanations for women's susceptibility to hypertension as demonstrated through the use of an expanded medical paradigm. The potential to broaden the focus of both research and practice is demonstrated in this study by the predictive power of variables outside of the biomedical paradigm. To reduce the prevalence of hypertension in women, further understanding is needed and this will depend on learning more about the etiology of hypertension. To do so, the broader focus of the expanded medical paradigm encourages study beyond the present risk factors to other possibilities. Working with an expanded paradigm is one way of providing additional parameters to explore and introducing some new possibilities for primary prevention efforts. This study also presents gender specific findings which can be explored in further research into hypertension and women.

The following conclusions can be made:

1. This study shows that there are a number of biomedical and non-biomedical variables which combine and interact to increase the susceptibility of some women to hypertension. The relationships which have been shown do not prove etiology of hypertensio but do provide a great number of possible areas for further research.

2.   The need for an accepted primary prevention strategy for hypertension in women is apparent in these results. The women who are susceptible to hypertension tended to have several health-compromising beliefs and little intention of making any lifestyle changes. These results would suggest the need for an overall health education strategy targetted at this identified susceptible female population and aggressive non-pharmacological interventions.

3. The strong consistent relationship between high blood cholesterol levels and hypertension in this susceptible population suggests a multifactorial approach is best.

4. The time-sequencing of events which precede the development of hypertension is missing in this study, and will be necessary in future research to more fully understand the relationships between hypertension and the predictors. To determine the order of events which lead up to hypertension, prospective longitudinal studies will be necessary.

5. The strong influence of age as a predictor indicates that age-specific characteristics of older women will need to be determined to establish effective guidelines for measuring cholesterol levels, blood pressure levels and anthropometric measures. It is presently unclear whether

'safe' levels are the same for the elederly as for the young. The definition of hypertension as a raised diastolic blood pressure, may be challanged as more research is done on isolated systolic blood pressure in older women.

6. A prospective longitudinal study which includes qualitative data to determine each woman's story, would further our understanding of susceptibility to hypertension. Acceptance of the expanded medical paradigm would provide a context for expanded research into causes of susceptibility to hypertension in women.

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APPENDIX A

Manitoba Heart Health Survey Questionnaire

THE MANITOBA HEART HEALTH PROJECT

Reference Number

V1	V2	V3	V4
□□□	□□□□	□	□□
1 2 3	4 5 6 7	8	9 10
Cluster	Sample	Group	Interviewer

Interviewer's Name _____

RECORDS OF CONTACTS

Call	Date	Time	Notes/ Comments
1			
2			
3			
4			
5			
6			

Circle the number which you feel indicates the:

Quality of interview   1   2   3   4   5
                            Low   High

Reliability of information   1   2   3   4   5
                            Low   High

Note: 1 is Low, 5 is High

Final status of interview: 1 □ Complete   4 □ Unable to locate
                                    2 □ Refused   5 □ Deceased
                                      3 □ Moved   6 □ Other non-interview(specify) _____

Reference Number ☐☐☐ . ☐☐☐☐ . ☐ . ☐☐
 1 2 3   4 5 6 7   8   9 10
 Cluster   Sample   Group Interviewer

Variable No.		Column No.
5	First Blood Pressure Reading Systolic	$\overline{11}\ \overline{12}\ \overline{13}$
	Diastolic	$\overline{14}\ \overline{15}\ \overline{16}$

SECTION 1: BLOOD PRESSURE

To begin with I'd like to ask you a few questions about blood pressure.

Q1. Before this interview, have you ever had your blood pressure checked?

1. Yes
2. No - **Go to Q6**

Variable No. 6 — Column No. $\overline{17}$

Q2. When did you last have your blood pressure checked?

1. Last 6 months
2. 6 - 12 months
3. 1 to 2 years
4. More than 2 years
8. Don't know

Variable No. 7 — Column No. $\overline{18}$

Q3. Who checked your blood pressure at that time?

1. doctor
2. nurse
3. family member or friend
4. coin operated machine
5. self
6. other (specify)_____
8. not sure

Variable No. 8 — Column No. $\overline{19}$

Q4. Which of the following describes the information you were given? Was it:
[**read list**]

1. described in numbers
2. described in numbers <u>and</u> in words like high/low/normal
3. described in words only ⌐
4. not described   │ **Go to Q6**
8. not sure   ⌐

Variable No. 9 — Column No. $\overline{20}$

Q5. What was your blood pressure reading in numbers when it was last taken?

Leave blank if can't remember

		Column No.
	Systolic	$\overline{21}\ \overline{22}\ \overline{23}$
	Diastolic	$\overline{24}\ \overline{25}\ \overline{26}$

Variable No. 10

Q6. Have you ever been told by a doctor or nurse that you had high blood pressure?

1. Yes
2. No   ⌐ **Go to Q10**
8. Can't remember⌐

Variable No. 11 — Column No. ___ 27

Q7. Was any treatment or program prescribed for your high blood pressure?

1. Yes
2. No   ⌐ **Go to Q10**
8. Not sure ⌐

Variable No. 12 — Column No. ___ 28

Q8. What were you told to do?
Do <u>not</u> read list.

Variable No.			Column No.
13	a. take medicine	a	___ 29
14	b. take medicine and some other treatment	b	___ 30
15	c. go on salt-free diet (salt reduced, low salt)	c	___ 31
16	d. watch weight	d	___ 32
17	e. avoid stress, slow down and relax	e	___ 33
18	f. cut down or stop smoking	f	___ 34
19	g. cut down alcohol intake	g	___ 35
20	h. start exercise program	h	___ 36
21	i. use biofeedback	i	___ 37
22	j. other treatment (specify) _____	j	___ 38
23	k. not sure	k	___ 39

Q9. What treatment or program are you <u>now</u> following?
Do <u>not</u> read list.

Variable No.			Column No.
24	a. take medicine	a	___ 40
25	b. on salt-free diet (salt reduced, low salt)	b	___ 41
26	c. watch weight	c	___ 42
27	d. other diet change	d	___ 43
28	e. avoid stress, slow down and relax	e	___ 44
29	f. cut down or stop smoking	f	___ 45
30	g. cut down alcohol intake	g	___ 46
31	h. exercise program	h	___ 47
32	i. use biofeedback	i	___ 48
33	j. other treatment (specify) _____	j	___ 49
34	k. not sure	k	___ 50

Variable No.		Column No.

35 Q10. As far as you know, is your blood
pressure high now?

    1. Yes
    2. No
    8. Don't know/not sure   ___ 51

Q11. How do you think high blood pressure
can affect your health?
**Do not read list, but if respondent is
hesitant then probe.**

36	a. Stroke	a	___ 52
37	b. Kidney trouble	b	___ 53
38	c. Heart attack/problems	c	___ 54
39	d. Hardening of the arteries	d	___ 55
40	e. Eye problems	e	___ 56
41	f. Nose bleed	f	___ 57
42	g. Headache	g	___ 58
43	h. Dizziness	h	___ 59
44	i. Swelling	i	___ 60
45	j. Other	j	___ 61
46	k. Not sure	k	___ 62

Q12. What things do you think can cause high
blood pressure?
**Do not read list, but if respondent is
hesitant then probe.**

47	a. Being overweight	a	___ 63
48	b. Smoking	b	___ 64
49	c. Eating too much salt	c	___ 65
50	d. Race or Ethnic group	d	___ 66
51	e. Worrying, tension, stress	e	___ 67
52	f. Eating fatty foods	f	___ 68
53	g. Drinking coffee	g	___ 69
54	h. Regular hard exercise	h	___ 70
55	i. Being pregnant	i	___ 71
56	j. Heredity (runs in family)	j	___ 72
57	k. Drinking too much alcohol	k	___ 73
58	l. Using birth control pills	l	___ 74
59	m. Being underweight	m	___ 75
60	n. Low income, low education	n	___ 76
61	o. Too much blood in system	o	___ 77
62	p. Getting little exercise	p	___ 78
63	q. Old age	q	___ 79
64	r. Other	r	___ 80
65	s. Not sure	s	___ 81

66 Q13. Do you think that high blood pressure
is related to things people eat or drink?

    1. Yes
    2. No   ┐**Go to Diabetes Q15**
    8. Not sure┘   ___ 82

Q14. What things that people eat and drink,
do you think are related to high blood
pressure?
Do not read list.

67	a. salt/salty foods	a	___ 83
68	b. sodium	b	___ 84
69	c. alcohol	c	___ 85
70	d. fats	d	___ 86
71	e. saturated fats	e	___ 87
72	f. cholesterol	f	___ 88
73	g. calories/eating too much	g	___ 89
74	h. additives/preservatives/food coloring	h	___ 90
75	i. caffeine/coffee	i	___ 91
76	j. sugar/sweet foods	j	___ 92
77	k. starch/starchy foods	k	___ 93
78	l. pork	l	___ 94
79	m. specific meat other than pork	m	___ 95
80	n. meats generally	n	___ 96
81	o. fried foods/greasy foods/oily foods	o	___ 97
82	p. calcium	p	___ 98
83	q. red meats	q	___ 99
84	r. fast foods	r	___100
85	s. other	s	___101
86	t. not sure	t	___102

    *   *   *   *   *

SECTION 2: DIABETES

The next few questions are about diabetes.

87 Q15. Have you ever been told by a doctor
that you have diabetes?

    1. Yes
    2. No   ┐**Go to "ALCOHOL" Q18**
    8. Not sure┘   ___103

88 Q16. How old were you when you were first
told you had diabetes?

    **Enter age
leave blank if not sure**   ___ ___ 104 105

Q17. Are you now on any treatment for your
diabetes?
Do not read list.

89	a. no current treatment	a	___106
90	b. insulin	b	___107
91	c. pills to control blood sugar	c	___108
92	d. diet	d	___109
93	e. weight loss	e	___110
94	f. other	f	___111
95	g. not sure	g	___112

    *   *   *   *   *

Variable No.		Column No.

SECTION 3: ALCOHOL

Now I would like to ask some questions about alcohol consumption.

96   Q18. Have you ever taken a drink of beer, wine, liquor or other alcoholic drink?

     1. Yes
     2. No
     9. Refused   ___113

97   Q19. In the past 12 months, have you taken a drink of beer, wine, liquor or other alcoholic drink?

     1. Yes
     2. No   ⌐ Go to "WEIGHT" Q22
     9. Refused⌐   ___114

98   Q20. How often, on average, did you have an alcoholic drink in the past 12 months? By that I mean

     Number of times per week   or
     Number of times per month
     88. Don't know
     99. Refused - Go to "WEIGHT" Q22

     $\frac{1}{2}$ __ __ wk
     __ __ mo
     115 - 117

99   Q21. On a day when you drink alcohol, how many drinks on average do you have throughout the day?

When we use the word "drink" it means:

     One bottle of beer or glass of draft   or
     One small glass of wine   or
     One shot or mixed drink with hard liquor

Enter number of drinks
Leave blank if refused   118 119

     *   *   *   *   *

Variable No.		Column No.

118   Q27. **Ask females only:**
To be sure we use the information correctly, I just need to ask if you are pregnant
     1. Yes
     2. No } Go to Q29   ___142

119   Q28. How many months?   ___143

     *   *   *   *   *

SECTION 5: EATING HABITS

The next section deals with eating habits.

119   Q29. As far as your health is concerned, do you think you eat too much, too little, or about the right amount of the following foods?
Read list and enter number for each item.

		Too much	Too little	About right	Not sure	
120	a) Lean meat, fish, poultry(chicken, turkey)	1	2	3	8	___144
121	b) Processed meats (wieners, salami, luncheon meats)	1	2	3	8	___145
122	c) Baked foods (cookies, muffins, cakes, pies)	1	2	3	8	___146
123	d) Fried foods (french fries, doughnuts)	1	2	3	8	___147
124	e) High-fibre foods (cereals, veg., wholegrain bread, fruit, beans)	1	2	3	8	___148
125	f) Salt or salty foods (pretzels, chips, salted nuts, pickled foods)	1	2	3	8	___149

126   Q30. How would you rate your diet compared to this time last year?
Read list.

     1. Definitely different
     2. Small change only
     3. No change —┐ Go to Q33
     8. Not sure —┘   ___150

Q31. Compared to last year, would you say you are eating more, less or about the same of:
Read list and enter number for each item

		More	About the same	Less	Not sure	
127	a) Lean meat, fish, poultry (chicken, turkey)	1	2	3	8	___151
128	b) Processed meat (wieners, salami, luncheon meats)	1	2	3	8	___152
129	c) Baked foods (cookies, muffins, cakes, pies)	1	2	3	8	___153
130	d) Fried foods (french fries, doughnuts)	1	2	3	8	
131	e) High-fibre foods (cereals, veg., wholegrain bread fruit, beans, etc.)	1	2	3	8	___155
132	f) Salt or salty foods (pretzels, chips, salted nuts, pickled foods)	1	2	3	8	___156

133   Q32. What was the main reason for changing your diet?
Do _not_ read list.

     1. Mainly to improve appearance (e.g., to improve figure)

     2. Mainly for medical reasons (e.g., on the doctor's advice)

     3. Mainly for health reasons (e.g., to feel fitter or to eat "healthy" foods)

     4. Mainly for economic reasons (e.g., a change in income)

     5. Availability of foodstuffs

     6. Other _____   ___157

     *   *   *   *   *

SECTION 6: FATS & CHOLESTEROL

I would like to ask you some specific questions now, about fats and cholesterol.

Q33. What health problems do you think might be related to the amount of _fat_ that people eat?
Do _not_ read list.

134	a. overweight/obesity	___158
135	b. heart disease/coronary disease/ heart problems/heart attack	___159
136	c. high blood cholesterol	___160
137	d. high blood pressure	___161
138	e. arteriosclerosis/hardening of the arteries/fat build up in the arteries	___162
139	f. other _____	___163
140	g. not sure	___164

141

Q 34. Do you think that <u>cholesterol</u> is found in:
Read list (1-4)

1. Foods   **Go to Q35**
2. Your blood   **Go to Q36**
3. Both   **Go to Q35**
4. Neither
8. Don't know──┘ **Go to Q37**

___165

Q35. How do you think foods which are rich in <u>cholesterol</u> can affect your <u>health?</u>
Do <u>not</u> read list.

142	a. hardening or clogging of the arteries	a	___166
143	b. increase blood pressure	b	___167
144	c. heart attack	c	___168
145	d. stroke	d	___169
146	e. angina (pain in the chest)	e	___170
147	f. increase blood cholesterol	f	___171
148	g. other _____	g	___172
149	h. not sure	h	___173

Q 36. How do you think that high levels of <u>cholesterol</u> in your <u>blood</u> can affect your health?
Do not read list.

150	a. hardening or clogging of the arteries	a	___174
151	b. increase blood pressure	b	___175
152	c. heart attack/heart disease	c	___176
153	d. stroke	d	___177
154	e. angina (pain in the chest)	e	___178
155	f. other _____	f	___179
156	g. not sure	g	___180

157

Q37. Have you ever had your blood cholesterol measured?

1. Yes
2. No
8. Not sure──┐ **Go to Q39**

___181

158

Q38. Were you told what your blood cholesterol level was?

1. Yes
2. No
8. Not sure

___182

159

Q39. Were you ever told by a doctor, nurse or other health professional that your blood cholesterol was high?

1. Yes
2. No
8. Not sure──┐ Go to Q41

___183

160

Q40. Did the doctor prescribe any treatment or tell you what to do to lower your blood cholesterol?

1. Yes
2. No
8. Not sure

___184

161

Q41. Are you presently on a diet, which was recommended by a doctor or other health professional, to lower your blood cholesterol?

1. Yes
2. No
8. Not sure

___185

162

Q42. Are you presently on any medication, which was recommended by a doctor or other health professional, to lower your blood cholesterol?

1. Yes
2. No
8. Not sure

___186

Q43. What do you think a person can do to lower his/her blood cholesterol level?
Do <u>not</u> read list.

163	a. exercise regularly (be more active)	a	___187
164	b. control stress	b	___188
165	c. control fatigue	c	___189
166	d. take prescribed medication	d	___190
167	e. eat food with less cholesterol	e	___191
168	f. eat less fatty food	f	___192
169	g. lose weight	g	___193
170	h. use skim milk/ low fat dairy products	h	___194
171	i. other _____	i	___195
172	j. not sure	j	___196

*   *   *   *   *

Variable No.		Column No.

SECTION 7: SMOKING

I would now like to ask you some questions about smoking.

173 Q44. Have you ever smoked cigarettes, cigars, or a pipe?

    1. Yes
    2. No } Go to Q54   ___197

174 Q45. At the present time do you smoke a pipe?

    1. Yes
    2. No } Go to Q47   ___198

175 Q46. At the present time do you smoke a pipe regularly (usually every day) or occasionally (not every day)?

    1. Regularly
    2. Occasionally   ___199

176 Q47. At the present time do you smoke cigars?

    1. Yes
    2. No } Go to Q49   ___200

177 Q48. At the present time do you smoke cigars regularly (usually every day) or occasionally (not every day)?

    1. Regularly
    2. Occasionally   ___201

178 Q49. At the present time do you smoke cigarettes?

    1. Yes
    2. No } Go to Q54   ___202

Variable No.		Column No.

179 Q50. At the present time do you smoke cigarettes regularly (usually every day) or occasionally (not every day)?

    1. Regularly
    2. Occasionally } Go to Q52   ___203

180 Q51. How many cigarettes do you usually smoke per day?

        number of cigarettes   ‾204‾ ‾205‾

Q52. Of the following reasons for giving up smoking, which do you think are very important, somewhat important, or not important?
Read list and for each item enter:

                1= Very Important
                2= Somewhat Important
                3= Not Important
                8= Not sure

181   a) To improve fitness   ___206

182   b) To prevent disease and ill-health   ___207

183   c) To set a good example to the family   ___208

184   d) To save money   ___209

185   e) To demonstrate self-control   ___210

186   f) To respect the wishes of non-smokers   ___211

187   g) To be sociable   ___212

188   h) To be more attractive   ___213

189 Q53. Would you yourself like to give up smoking?

    1. Yes
    2. No
    8. Not sure   ___214

Variable
No.

Column
No.

Variable
No.

Column
No.

190 Q54. How often are you exposed to·other people
smoking?
Read 1 - 4

1. Frequently
2. Occasionally
3. Rarely
4. Never
8. Not sure ⌐Go to Q56   ___215
9. No response⌐

191 Q55. What do you usually do if you're
bothered by other people smoking?
Do **not** read list.

1. Leave the area
2. Ask them to stop or move
3. Other _____
4. Nothing
5. I'm not bothered by it
8. Not sure   ___216

*   *   *   *   *

SECTION 8: PHYSICAL ACTIVITY

**The next few questions are about your
current physical exercise.**

192 Q56. Do you regularly engage in physical
exercise during your leisure time?
By regularly we mean at least once a
week during the past two months.

1. Yes
2. No   } Go to Q60
3. Just started   ___217

193 Q57. How much of this exercise is strenuous
enough to cause sweating or breathing
heavily? **Read list**

1. Most of it
2. Some of it
3. None of it   ___218

194 Q58. How long do you usually exercise?
Read list.

1. less than 15 minutes
2. 15 - 30 minutes
3. half an hour - an hour
4. more than an hour
8. not sure   ___219

195 Q59. How many times per week do you exercise
at least 15 minutes? **Do not read list.**

1. daily
2. 5-6 times/week
3. 3-4 times/week
4. 1-2 times/week
5. less than once/week
6. never
8. not sure   ___220

196 Q60. Which of the following choices best
describes the work or other daytime
activity you usually do?
Read list.

1. I am usually sitting down during the
day and do not walk around very much.

2. I stand or walk around quite a lot
during my day, but I do not have
to carry or lift things very often.

3. I usually lift or carry light loads
or I have to climb stairs or hills
often.

4. I do heavy work or carry very
heavy loads.

8. Not sure   ___221

197 Q61. Do you feel you get as much exercise as
you need or less than you need?

1. Less than needed
2. As much as needed⌐Go to Q63
8. Not sure   ⌐   ___222

Q62. Do any of the following reasons prevent you from doing more exercise or being more active?
Read list.

198   a. Lack of time   ___223

199   b. Lack of transport   ___224

200   c. Lack of money   ___225

201   d. Lack of easily available facilities in the community   ___226

202   e. Lack of interesting or relevant activities   ___227

203   f. Illness or disability   ___228

204   g. Lack of incentive   ___229

205   h. No one to exercise with   ___230

206   i. Any other reasons _____   ___231

207   Q63. Overall, would you say you were physically more active, about the same, or less active than others your age?

1. More active
2. About the same
3. Less active
8. Not sure   ___232

*　　*　　*　　*　　*

SECTION 9: HEART DISEASE

Now I would like to ask you a few questions about cardiovascular disease.

Q64. What do you think are the major causes of heart disease or heart problems?
Do not read list.

Variable No.			Column No.
208	a. poor diet	a	___233
209	b. overweight	b	___234
210	c. excess fat	c	___235
211	d. excess salt	d	___236
212	e. high blood cholesterol level	e	___237
213	f. foods with high cholesterol	f	___238
214	g. excess stress, worry or tension	g	___239
215	h. overwork or fatigue	h	___240
216	i. lack of exercise	i	___241
217	j. smoking	j	___242
218	k. heredity	k	___243
219	l. high blood pressure/hypertension	l	___244
220	m. arteriosclerosis/hardening of the arteries	m	
221	n. Other _____	n	___245 / ___246
222	o. Not sure	o	___247

223   Q65. Based upon what you have heard or read, do you believe that heart disease can be prevented?

1. Yes
2. No
3. Sometimes
8. Not sure   ___248

224   Q66. Have you ever had a heart attack?
(If necessary, explain what a heart attack is).

1. Yes
2. No
8. Not sure   ___249

225   Q67. Do you suffer from any other kind of heart disease?

1. Yes. What is it? _____
2. No   ___250

226   Q68. Are you presently taking any medicine that your doctor prescribed for your heart?

1. Yes
2. No
8. Not sure   ___251

Variable No.		Column No.

Q69. What do you think are the major causes of a stroke? (If necessary explain what a stroke is.)
Do <u>not</u> read list.

227	a. poor diet	a	__252
228	b. overweight	b	__253
229	c. excess fats	c	__254
230	d. excess salt	d	__255
231	e. high blood cholesterol level	e	__256
232	f. foods with high cholesterol	f	__257
233	g. excess stress, worry or tension	g	__258
234	h. overwork or fatigue	h	__259
235	i. lack of exercise	i	__260
236	j. smoking	j	__261
237	k. heredity	k	__262
238	l. high blood pressure/hypertension	l	__263
239	m. arteriosclerosis/hardening of the arteries	m	
			__264
240	n. other _____	n	__265
241	o. Not sure	o	__266

Q70. Have you ever had a stroke?

242

1. Yes
2. No
8. Not sure   __267

Q71. Based upon what you have heard or read, do you believe that strokes can be prevented?

243

1. Yes
2. No
3. Sometimes
8. Not sure   __268

Q72. Has anyone in your immediate family (parents, siblings, children) ever had any of the following health problems? (Grandparents are not immediate family.)
Read list.

244	a. Heart disease	a	__269
245	b. High blood pressure	b	__270
246	c. Stroke	c	__271
247	d. Diabetes	d	__272
248	e. High cholesterol	e	__273

Q73. ASK WOMEN ONLY: Are you presently taking..

249	Oral contraceptives	__274
250	Other hormonal pill	__275

    *   *   *   *   *

Variable No.		Column No.

SECTION 10: GENERAL

Finally, I'd like to ask you a few general questions about health.

Q74. In the past year, have you seen the following or received any information about health topics at your place of work? (e.g., posters, bulletin boards, pamphlets)
Read list.

251	a) Heart Health	a	__276
252	b) Smoking	b	__277
253	c) Exercise	c	__278
254	d) Stress	d	__279
255	e) Diet	e	__280
256	f) Safety	f	__281
257	g) Drugs/Alcohol	g	__282
258	h) Other _____	h	__283

259

Q75. To what extent have you found the information helpful?
Read 1-3.

1. Very helpful
2. Somewhat
3. Not at all
8. Not sure   __284

260

Q76. Do you think your place of work is an appropriate place to promote heart health?

1. Yes
2. No
8. Not sure   __285

Q77. Where else do you think heart health should be promoted?
Do <u>not</u> read list.

261	a. schools	a	__286
262	b. television	b	__287
263	c. radio	c	__288
264	d. newspapers	d	__289
265	e. church	e	__290
266	f. service clubs	f	__291
267	g. recreational facilities	g	__292
268	h. doctor's offices	h	__293
269	i. public health offices	i	__294
270	j. hospitals	j	__295
271	k. Other _____	k	__296
272	l. Not sure	l	__297

Variable No.		Column No.

Q78. At your workplace, is smoking permitted?
Read list.

1=Yes
2=No

273   a. In your immediate work area   a   ___298

274   b. In designated areas only   b   ___299

275   c. Not at all   c   ___300

276   Q79.We've been talking about health and health behaviors; what would you say is the single most important thing you have done in the past year to improve your health? **Do not read.**

1. Nothing
2. Increased exercise (become more active)
3. Lost weight
4. Improved eating habits
5. Quit/ reduced smoking
6. Reduced drugs/medication
7. Drank less alcohol
8. Had blood pressure checked
9. Attempted to control blood pressure
10. Learned to manage stress
11. Reduced stress level
12. Received medical treatment
13. Other ._____
88. Not sure

301 302

Q80. Is there anything you <u>intend</u> to do to improve your health in the next year? **Do not read.**

277	a.	Nothing	a	___303
278	b.	Increase exercise	b	___304
279	c.	Lose weight	c	___305
280	d.	Improve eating habits	d	___306
281	e.	Quit/ reduced smoking	e	___307
282	f.	Reduce drugs/medication	f	___308
283	g.	Drink less alcohol	g	___309
284	h.	Have blood pressure checked	h	___310
285	i.	Attempt to control blood pressure	i	___311
286	j.	Learn to manage stress	j	___312
287	k.	Reduce stress level	k	___313
288	l.	Receive medical treatment	l	___314
289	m.	Other _____	m	___315
290	n.	Not sure	n	___316

Variable No.		Column No.

DEMOGRAPHIC INFORMATION

The last few questions let us look at health factors by different groups like age, sex, income and occupation.

291   Q81. **Enter respondent's sex:**

1. Male
2. Female

___317

292   Q82. What is your date of birth?

Day   318 319

Month   320 321

Year   322 323

293   Q83. What is your current employment status? **Read this list.**

1. full time (35 hours or more a week)
2. part time (less than 35 hours a week)
3. unemployed
4. laid off
5. retired
6. other _____
7. homemaker
8. student   Go to Q85

___324

294   Q84. What is your occupation?
_____. **Do not read**

1. Professional
2. Management
3. Office/Clerical/Sales
4. Foreman
5. Semi-skilled
6. Unskilled
7. Other _____
8. No response

___325

295   Q85. What is your current marital status? **Do not read list.**

1. single
2. married/common law
3. widowed/widower

___326

Variable No.		Column No.

296

Q86. What is the highest grade or year of
education you have completed?

1. No schooling
2. Elementary (Grade 6)
3. Secondary (Grade 12)
4. Post secondary
5. Not sure/no response

____327

297

Q87. What language did you first speak
in childhood?

1. English
2. French
3. Other _____
8. Not sure

____328

298

Q88. How many people live in this household?

$\overline{329}$ $\overline{330}$

299

Q89. For statistical purposes only, we need
to know the range of your total, gross
household income last year. Could you
please indicate from the following list the
income range for your household?
Read list.

1. under $12,000
2. $12,000 to $24,999
3. $25,000 to $49,999
4. $50,000 to $74,999
5. $75,000 and over
8. respondent refused to answer

____331

**Thank you for taking time to
answer these questions.**

    ★   ★   ★   ★   ★

300

Second Blood Pressure Reading

Systolic

$\overline{332}$ - $\overline{334}$

Diastolic

$\overline{335}$ - $\overline{337}$