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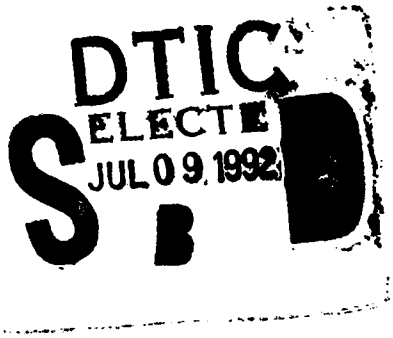

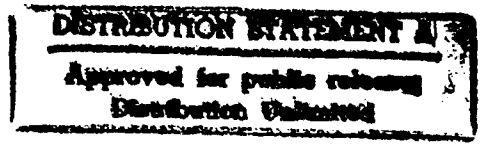

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Cardiovascular Risk Factors

in

Primary Relatives of Sudden Cardiac Death Victims

by

Abigail Palmer

A research proposal submitted in partial fulfillment
of the requirements for the degree of

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Chapter I

Problem Statement

Cardiovascular disease holds the deadly distinction of being the number one killer in America. Almost one in two Americans dies of cardiovascular disease (American Heart Association 1991). Of the current U.S. population, nearly 68 million people - more than one in four Americans -suffer some form of cardiovascular disease (American Heart Association 1991).

The major clinical manifestations of atherosclerotic cardiovascular disease are angina, myocardial infarction, cerebral hemorrhage, heart failure, peripheral vascular disease and sudden cardiac death (SCD). Coronary heart disease, a major subset of cardiovascular disease, refers more specifically to disease of the blood vessels that supply the heart muscle. Coronary heart disease accounts for 75% of all deaths within the spectrum of cardiovascular disease (National Institutes of Health 1983). Sudden cardiac death is one of the clinical manifestations of coronary heart disease. Sudden death accounts for approximately 20% of all mortality in the United States every year; 300,000-450,000 people (Kremers, Black & Wells, 1989). One half of all deaths due to coronary heart disease are sudden (Kannel, Doyle, McNamara, Quickenton, & Gordon 1975).

Further study is needed to learn how sudden cardiac death evolves in people. Also, there is no reliable method

to identify those who may be at increased risk. However, the literature suggests a strong association between the atherosclerotic disease process of coronary heart disease and sudden death.

Cardiovascular risk factors are characteristics that impart an increased chance of developing coronary heart disease. Major risk factors for sudden cardiac death and coronary heart disease that have been identified as being atherogenic are hypercholesterolemia, smoking, and hypertension (Kannel et al., 1975). Other contributing factors are obesity, lipoprotein ratios, physical inactivity, gender, glucose intolerance, fibrinogen levels, certain personality traits and family history.

Many studies have shown a familial aggregation for coronary heart disease (Hamby, 1981; Slack & Evans 1966; Sholtz, Rosenman, & Brand 1975). Epidemiological studies have shown that relatives of people with coronary heart disease have an increased risk for developing coronary heart disease (ten Kate, Boman, Daiger, & Motulsky 1982). It is not known if relatives of persons with sudden cardiac death are at increased risk for sudden cardiac death or other manifestations of cardiovascular disease. Relatives of sudden cardiac death victims could be at risk for developing coronary heart disease. How much they are at risk would depend upon the presence or absence of certain cardiovascular risk factors; risk factors which this study

intends to measure.

The relatives of sudden cardiac death victims may constitute a high risk population. Identification of these relatives is paramount in the prevention of sudden cardiac death and other manifestations of cardiovascular disease. Subsequent identification of their cardiovascular risk factors is important in primary prevention of sudden cardiac death and possibly other manifestations of cardiovascular disease such as myocardial infarction. Appropriate preventive measures, such as risk reduction, could then be tailored to the individual's needs.

The information gained in this study will apply to many areas concerning nursing. It is specifically in the area of risk factor identification and behavior modification that nurses can make a vital contribution toward decreasing coronary heart disease and/or cardiovascular mortality. A primary preventive role for nurses is to identify those at high risk for sudden cardiac death, increase their awareness and knowledge about risk factors, and to provide the necessary skills or techniques in order to change risk-perpetuating behavior to new risk-reducing behavior. Nurses practicing in multiple settings can take the lead in investigating and developing strategies and techniques to educate patients and families to facilitate life-style changes; hopefully to prevent the catastrophic sudden death of a loved one. Thus the overall goal is to determine if the

relatives of sudden cardiac death victims are a high risk population for sudden cardiac death; and a potential population for risk factor reduction.

This study is being conducted as a joint venture with Nichole Pashek, a fellow graduate student. Her study will also examine cardiovascular risk factors in relatives of sudden cardiac death victims, but will focus on upper body obesity, glucose intolerance, hypertriglyceridemia and hypertension as risk factors in relatives of sudden death victims. The sample for both studies will be the same.

Chapter II

The Conceptual Framework

Problem of Coronary Heart Disease and Sudden Death

In 1987 cardiovascular diseases killed nearly one million Americans , 45.9% of all deaths (American Heart Association 1991). The cost of cardiovascular disease is estimated by the American Heart Association at $ 94.5 billion annually. Cardiovascular disease demands our attention.

The chief form of cardiovascular disease is coronary heart disease. Coronary heart disease is the major cause of death and disability in the United States (Lipid Research Clinics Program 1984). Sudden cardiac death (SCD) is a striking feature of coronary heart disease. There are more than 450,000 sudden cardiac deaths each year, representing one-half of the annual toll of coronary mortality and approximately 20% of all mortality in the U.S. every year (Kremers, Black, & Wells, 1989).

Definition of Sudden Cardiac Death

Describing the phenomenon of SCD can be a difficult task. Death from natural causes may be sudden or unexpected, and unrelated to cardiac etiologies. Because few other pathologic conditions can cause death as rapidly as SCD, the amount of time from onset of symptoms to death is a major factor in differentiating SCD from other forms of sudden death.

The World Health Organization defines sudden death as one that occurs within 24 hours of the onset of symptoms (Lown, 1979). However, a number of noncardiac deaths, such as stroke, can occur within this time frame. For the purpose of this paper, sudden death is defined as "natural death from cardiac causes, heralded by abrupt loss of consciousness within one hour of the onset of acute symptoms, in an individual with or without known prexisting heart disease but in whom the time and mode of death are unexpected" (Dunn 1990). This definition was chosen because of the more realistic time parameter, and, investigators of the Framingham Heart Studies use the 1-hour time parameter. The Framingham Heart Studies have provided most of the research information on SCD.

Pathology of Sudden Cardiac Death

There appear to be several different pathologic scenarios which render a heart vulnerable to sudden death. One common explanation for sudden cardiac death is that of an electrical "accident" superimposed on a vulnerable myocardium (Hallstrom, Cobb, & Ray 1986). This arrhythmic classification usually refers to an event of ventricular fibrillation, the precursor to death. In a Seattle study of 5,199 cardiac arrest victims, 3,893 or 75% had ventricular fibrillation recorded as their initial cardiac rhythm (Greene 1990). Kremers et al.(1989) describe a premature ventricular complex (PVC) theory of sudden

death. This theory holds that PVC's are markers of cardiac electrical instability, and can develop into life threatening arrythmias, usually ventricular fibrillation. Some feel that the sequence of events begins with a thrombus, which leads to coronary spasm, occlusion, myocardial ischemia, myocardial infarction and then sudden death.

Myocardial infarction (MI) is often a precursor to sudden death. In one ten year study, Newman, Tracey, Strong, Johnson & Olman (1982) reported autopsy findings on specimens of 1,292 men; both black and white. They found that 72% of the men dying suddenly had evidence of previous myocardial infarction, with necrotic or scarred myocardium. This study should be acknowledged for using a standardized protocol which required objective evaluation of findings and uniform application of definitions. However, the generalizability of the study is limited in that only a very small, well delineated geographic area was surveyed. All the subjects resided in the same Louisaiana community, were men only, aged 25-44 years.

Research has shown a repeating prevalence of occurence of an MI in families; supporting the hereditary aspect of coronary heart disease (tenKate, Boman, Daiger, & Motulsky, 1982 ; Nora, Lortscher, Spangler, & Kimberling, 1980; Hamby, 1981; Forde & Thele, 1977). In their case-control study, tenKate et al, reported a 2.14 increased risk

for MI among relatives of patients who had experienced an MI ($p < .001$). The same study showed, that among relatives of survivors of an MI, 16% had had an MI, compared with 8.9% of relatives of control subjects. However, the cohort consisted of only white, male subjects. In the Tromso Heart study, Forde & Thele (1977) investigated the occurence of MI in first degree relatives with those who had a positive family history of MI. They found subjects who had evidence of a previous MI, had a significantly higher frequency (78%) of relatives with MI. This study is commendable for its large size of 6,595 subjects. However, family history for an MI was obtained by interview. In instances where the interviewee was uncertain as to the meaning of infarction, or the actual occurence of a "heart attack", these relatives were classified as negatives. The negative answers were not verified, so the number of false negatives was unknown. Furthermore, the positive diagnoses, from hospital records, were always accepted and no attempt was made to standardize the basis for diagnosis.

Although further research is needed to describe the exact mechanisms of sudden death, one fact remains evident throughout the literature. An underlying abnormality in most cases of sudden cardiac death is the presence of an atherosclerotic plaque. The most frequent underlying pathology in patients with SCD is atherosclerosis of one or more of the four major arteries. Kremers et al., (1989)

stated that about 90% of victims of sudden death have atherosclerotic heart disease. Davies (1981) reported that 84% of athrosclerotic coronary heart disease patients, men only, who died suddenly, had severe two or three vessel disease. By comparison, 100 age matched controls who died of other causes, had a combined 27% incidence of two and three vessel disease. Reichenbach, Moss & Meyer (1977) examined pathologic coronary changes in 87 patients who died from SCD. They found complete atherosclerotic occlusion in one or more coronary vessels in 59% of the 87 autopsies done. Of the control group of persons who died suddenly of noncardiac causes (accidents, suicide) only 13% showed evidence of stenosis in one of the three major coronary vessels. T postmortem autopsies in this study were thorough, and included an x-ray angiogram examination of all the subjects myocardiums. Also, control groups were well matched for age. However, only 14% of the patients were female.

The association between coronary artery disease and sudden death is inescapable. The Framingham data (in a 20-year follow-up of men ages 45-74) suggest that since risk factors for sudden cardiac death are similar to those for coronary heart disease in general, there may be a positive relationship between the atherosclerotic process and sudden cardiac death. It is within this context that risk factors for sudden death are discussed.

Sudden Cardiac Death Risk Factors

Age and Gender

Like coronary artery disease, SCD is related to gender and age, occuring more frequently in men and in older age groups. In 1985, Kannel and McGee reported on the incidence of SCD among the 5,252 participants in the ongoing Framingham Heart Study. Subjects had been evaluated every other year for 26 years. The incidence of SCD increased with age and actually doubled for every decade after age 45. Among subjects 45 to 54 years old, the rate was 2.0 per 1,000 and 0.5 per 1,000 for men and women respectively. The incidence for those older than 75 was 8.2 per 1,000 for men and 6.1 per 1,000 for women. Hamby (1981), in his study of 411 patients using cardiac catheterization to examine vessel stenosis, found the mean age of coronary artery disease onset to be 46.7 years. However, it is not clear what endpoint was used for a diagnosis of coronary artery disease. Also, the investigation was confined to an all Caucasian, male only population, and all residents of the same Long Island Jewish community. The risk of sudden death is much higher among men than women. Women in the Framingham study (26 year follow-up of 5,128 men and women, 30-62 years of age) had only a third of the sudden death incidence found in men and women reached the male level of sudden death incidence 20 years later in life. In other words, women in their 60s had the same rate of SCD as men in their 40s.

(Kannel & Schatzkin 1985).

In men and women the "classical" risk factors for coronary heart disease also appear to be the risk factors for sudden death. However, the SCD risk factor profile is somewhat different for men and women. In men, the presence of the standard coronary risk factors of hypertension, hypercholesterolemia, cigarette smoking, obesity, and left vetricular hypertrophy was associated with an increased incidence of SCD (Schatzkin, Cupples, Heeren, Morelock, Mucatel, and Kannel 1984). In women, vital capacity, hematocrit, and blood glucose were the strongest risk factors. Age was a strong predictor in both sexes.

Cigarette Smoking

It is now acknowledged that cigarette smoking predisposes one to coronary heart disease. Cigarette smoking has been shown to enhance the adhesiveness of platelets, accelerate the heart rate, increase susceptibility to ventricular arrhthymias, reduce the oxygen carrying capacity of the blood, and transiently elevate blood pressure (Kannel, 1981).

All these factors combined could contribute to sudden cardiac death (Kannel & Schatzkin 1985). Data from the Framingham study (a 16 year follow-up) showed that cigarette smokers were at a significantly higher risk for heart disease and sudden death. In the entire cohort of 2,282 residing in Framingham, Massachusetts, smokers had a

threefold higher rate of sudden death than nonsmokers (Kannel, Doyle, & McNamara 1975). The Framingham study further demonstrated an annual SCD mortality rate of 22.4 per 1,000 in men, ages 45 to 74 years, who smoked >20 cigarettes per day compared to a mortality rate of 12 per 1,000 in non-smokers (Kannel, 1981). The incidence of SCD also increases with the number of cigarettes smoked.

It is interesting to note that the deleterious effects of smoking may be reversible. Hallstrom, Cobb, and Ray (1986), obtained information about smoking cessation in 310 SCD survivors who had been habitual smokers at the time of their event. Using recurrent cardiac arrest as an endpoint, they found long-term survival was improved in reformed smokers as compared with patients who continued to smoke (19 vs 27 percent at three years; p=0.038). Although these results are impressive, the study did not quantify the number of cigarettes smoked, did not consider the effects of other risk factors, or other life-style changes the subjects might have made which would impact survival rate.

Cholesterol

Research suggests a causal relationship between elevated serum cholesterol levels, atherogenesis and coronary heart disease. (Lipid Research, 1984). Evidence incriminating cholesterol and the atherosclerotic process as a risk factor in sudden death was found in a study in 208 subjects (182 males, 26 females) by Baroldi, Falzi, and

Mariani (1979). They found severe lumen reduction >70% due to atherosclerotic changes of at least one of the major coronary arteries in 75% of subjects who died because of sudden cardiac death, in comparison to the accidental death control group who had only 39% stenosis. However, because the population of females was so small, data were considered without regard to sex difference. Also, because the study was conducted in Italy, where culture and diet differ, one should be cautious about generalizing findings to the American population.

The Multiple Risk Factor Intervention Trial confirmed an independent effect of serum cholesterol as a risk factor for coronary heart disease (Kannel, Neaton, Wentworth, Thomas, Stamler, Hulley, & Kjeslberg 1986). The trial revealed that a 10% (15 mg/dl) reduction in cholesterol was associated with an 11.3% reduction in coronary heart disease mortality. Although this study examined a large cohort of people (325,384) only white, middle aged men were included.

In 1981, Hjermann, Home, Byre and Leren, conducted a five year intervention trial of 1,232 subjects who were considered to be at high risk for CHD, with a mean cholesterol level of 329mg/dl. The intervention group received nutritional counselling on diet modification for reduced cholesterol intake. Results showed a 47% lower MI and SCD incidence in the intervention group compared to the control group. The intervention group had a final reduced

plasma cholesterol level 13% lower than the control group. However, the study design included only male Norwegians, aged 40-49 years; and the intervention group was seen every 6 months as compared to 12 every months for the control group, which may have an effect on the intervention group's improved outcome.

High density lipoprotein cholesterol (HDL-C) is one of the cholesterol carriers in the blood stream. It is thought that HDL clears cholesterol from the blood stream, and high levels of it are associated with a decreased risk of heart disease (American Heart Association 1990). An inverse relationship between HDL levels and the incidence of coronary heart disease was seen in the Framingham study (Kannel 1987). Currently there is no data on the association of SCD and HDL cholesterol levels.

Obesity

Because of its great prevalence in the United States and its atherogenic metabolic and physiologic concomitants, obesity is a powerful contributor to coronary atherogenesis. The second National Health and Nutrition Examination Survey found that 26% of U.S. adults, or about 34 million people aged 20- 75 years, were overweight (U.S. Public Health Services 1983).

Hippocrates pointed out some 2400 years ago that sudden death is more common in those who are naturally fat than in lean persons (Kannel, & Thomas 1982). Risk of sudden death

in the Framingham study (26 year follow-up, subjects 35-84 years old) increased with the degree of adiposity in both sexes (Hubert, Feinleib, McNamara, and Castelli 1983). For men under 50 years who were <110% of their desirable weight, the incidence of SCD was 26 per 1000 compared to 80 per 1000 for men who were 130% of their desired weight. After a 26 year observation, the Manitoba study found increased body mass index to be a significant risk factor for the development of heart disease (Rabkin, Mathewson, & Hsu 1976).

Both studies screened their samples initially to establish that they were free from cardiovascular disease. The Framingham study used multivariate analysis to isolate the effects of obesity from the effects of other cardiac risk factors. A limitation of the Manitoba study was that it analyzed only age and blood pressure in relation to the development of CVD in obese subjects. In addition, the Manitoba study used only Royal Air Force pilots in their youth. Due to the fitness level that these men once had, this sample would not necessarily represent the general population. A limitation of the Framingham study was that it used the Metropolitan Relative Weight Table, which may not accurately reflect the weight of a person's lean body mass. The Framingham study also relied on self-reported weights. The accuracy of these measurements can be questioned.

The exact mechanism for the association of obesity and

SCD is unclear, but it may be related to fatty infiltration of the myocardium and the electrical conduction system of the heart; which might make the heart more prone to lethal ventricular arrhythmia's. (Dunn 1990). Obesity is also associated with elevated blood pressure (Dustan, 1989) and triglycerides (Wolf & Grundy 1983), diabetes and serum lipids (Kannel, Gordon & Castelli 1979); all contributors to cardiovascular disease.

Diabetes

In 1979, after a twenty year follow-up of the Framingham Study, Kannel & McGee reported on the relationship between diabetes and the incidence of cardiovascular disease, as well as the levels of cardiovascular risk factors. The incidence of cardiovascular disease among diabetic men was 2.1 that among nondiabetic men. Among diabetic women, the incidence was 2.7 that of nondiabetic women. The relative impact of diabetes was substantially greater for women than men. Diabetic women had a relative risk of cardiovascular mortality of 3.3 versus mens' relative risk of 1.7. The reason that women who have diabetes appear to eliminate their "immunity" to cardiovascular disease and are more susceptible to morbidity and mortality than men is not clear.

In this same study, diabetic individuals had higher levels of cardiovascular risk factors than nondiabetic individuals. Diabetic men had higher mean systolic blood

pressures than nondiabetic men (148 mmHg versus 139mmHg; p<.001), and were more obese (125 % relative weight versus 121 % ; p<0.05). Diabetic women in this cohort were more hypertensive than nondiabetic women, (150mmHg versus 138 mmHg; p<0.001), and more obese. In addition, diabetic women had lower HDL cholesterol levels (53.5) than their nondiabetic counterparts (57.8). Diabetic individuals of both sexes had a higher prevalence left ventricular hypertrophy (LVH). Men with diabetes had a 2.7 relative risk for LVH and women of 3.5 for LVH. Unfortunately data relating diabetes and sudden death is not well documented.

Physical Activity

Epidemiologic studies have shown the existence of a strong inverse relationship between physical exercise, mortality and coronary heart disease (Kannel and Thomas, 1982; Kannel, Doyle, and McNamara, 1975; Morris, Pollard, and Everett, 1980). It has been suggested that those who are inactive are at greater risk for coronary heart disease. San Francisco longshoremen were followed for 22 years to assess their work activity and this relationship. Among 3,686, men only, those who expended $\geq$ 8,500 kcal/week, had only about half (0.56) the number of fatalities from coronary heart disease (p<0.001) than the rate for men with less energetic duties (Paffenbarger & Hyde 1984). An "activity index" was computed for each indivudual in the Framingham study. The most sedentary men appeared to have more than double the

risk of sudden death compared with the more active (Kannel, Doyle, & Mcnamara 1975).

Hypertension

Hypertension (described as a systolic blood pressure above 140 mmHg and/or a diastolic blood pressure greater than 90 mmHg [Joint National Committee, 1988]) is a powerful contributor to coronary disease in general (American Heart Association, 1991). It is presumed that hypertension creates conditions in the arterial endothelium that encourages the development of atheromas (the lipid containing plaques in athersclerosis) , (Guyton, 1981). Hypertension is a significant contributer to sudden death. In the Framingham study, when hypertension was classified as low (<140/85), middle (140-159/85-94), or high (>160/95), those subjects with systolic pressures greater than 160mmHg had an incidence of SCD 3 times that of subjects in the low category (67.6% compared to 25.4%), (Kannel & Thomas, 1982). Thomas and Hirschorn (1955) noted that siblings of a parent with coronary heart disease had twice the prevalence of hypertension compared to siblings whose parents did not have coronary heart disease.

Left Ventricular Hypertrophy

The Framingham study has shown that left ventricular hypertrophy (LVH) as assessed by electrocardiogram is a risk factor for sudden cardiac death (Kannel et al 1975). Sudden death occured significantly more frequently in each age

group when there was evidence of LVH on an electrocardiogram. Men with LVH had a five fold increase in risk of sudden death. Age adjusted annual rates were 14.0 and 2.7 per 1,000 respectively, for those with and without LVH. The LVH pattern that emerges for SCD applies to both men and women. However, in women with CHD already evident, LVH was not associated with sudden death, only with a previous myocardial infarction (Kreger, Kannel & Cupples 1987). Levi, Garrison, Savage, Kannel & Castelli (1990) in a multivariate analysis of a Framingham study, found LVH was significantly associated with the risk of cardiovascular disease death from cardiovascular disease and death from all causes in both sexes even after adjustment for age, blood pressure, cigarette smoking, diabetes and body mass index. The age adjusted rates for cardiovascular disease, mortality, and death from all causes in men with LVH, as compared with those without LVH were 12.1 versus 6.8 percent ($p<0.001$), 4.8 versus 1.0 percent ($p<0.0001$), and 9.1 versus 4.0 percent ($p<0.05$) respectively. A strength of this study was that echocardiography (versus electrocardiography) was used to determine LVH; echocardiography allows a clearer, three dimensional picture of the heart for diagnostic purposes. However, this study was done on a predominanlty white population, thus generalization to other racial groups may not be warranted. Also, standardized methods for evaluating echocardiograms may not always be followed.

Hence, the values used to define LVH may differ in various laboratories.

The mechanisms by which cardiac hypertrophy may promote cardiovascular morbidity and mortality are not completely understood. LVH increases myocardial oxygen consumption, while reducing coronary blood flow reserve (Levy et al 1990). This supply-demand mismatch may predispose the patient to ischemia, myocardial infarction, ventricular arrhythmias, and sudden death. One study which used twenty-four hour ambulatory electrocardiogram monitoring to quantify cardiac arrhythmias found, more premature ventricular contractions (PVCs) in hypertensive patients with LVH, 475 $\pm$ 852 PVCs per 24 hours, than in hypertensive patients with no hypertrophy, 10 $\pm$ 22 PVCs per 24 hours, or in normtensive patients, 8 $\pm$ 20 PVCs per 24 hours (Messereli, Ventura, Elizardi, Dunn, & Frohlich 1984).

Familial Tendency

No studies were found that examined the specific incidence of SCD among family members. However, numerous studies have revealed an increased incidence of CHD among patients with a family history of CHD (Slack and Evans, 1966; Sholtz, Rosenman, and Brand, 1975; Forde and Thele,1977; Nora, Lortscher, Spangler, and Kimberling,1980; Snowden, McNamara, Feinleib, Kannel and Epstein, 1982; tenKate, Boman, Daiger and Motulsky, 1982). In their study, tenKate et al., (1982) found the frequency of coronary heart

disease among first degree relatives of people who had survived an MI was 20.5%, compared to 13.7% for control patients. In the same study, MI was reported in 16.3% of the relatives of patients, compared to 8.9% of the control patients. Slack and Evans conducted a case control study of 217 documented CHD patients and age-sex matched controls without CHD. A relative risk of death from CHD in male relatives of male patients with CHD was 5 fold that of the general population; a 7 fold risk was found for women. Although this study was very systematic, and included women, a seemingly arbitrary age for onset of CHD of 55 years for men and 65 years for women was used. Also, the general population is specific to England and Wales only. An analysis of brothers in the Framingham study shows that the incidence of MI in older brothers is significantly related ($p<0.001$) to MI in younger brothers (Snowden, et al., 1982). Although a multivariate analysis was used to control for the possible confounding effects of other risk factors; there is no mention of specific paramaters for hypertension or hypercholesterolemia in the article.

Other studies have shown increased familial tendencies of specific cardiovascular risk factors that have been shown to contribute to the risk of developing CHD. Levine, Hennekens, Rosner, Gourley, Gelband and Jesse (1981), examined cholesterol levels among children of men with premature MI and children of healthy men. The mean

cholersterol level in the children of the affected men, 200+/-52mg/100ml was higher than the level of 176+/-27mg/100ml among the children of the controls (p=.001). Hamby (1981), compared parental history of two patient groups; those with CHD and those without. He found a significant prevalence (65%) of hypertension in fathers of patients with CHD, compared with control group fathers (43%). Comparisons for cigarette smoking revealed a higher incidence (50%) among both parents, compared to parents of the non-CHD group (32%, p<.025). Becker, Pearson and Kwiterovich (1988) studied 86 siblings of people who had documented CHD. Although 99% of the sibling subjects were white, accuracy was increased in two ways. Self-report of family history was not used (a method that can be questioned), and blood pressure/cholesterol cut off points were matched to specific nationally established guidelines (Joint National Commission on High Blood Pressure; Lipids Research Clinic percentile distributions). They found of the total siblings studied, 78% had one or more risk factors (smoking, hypertension, elevated cholesterol). The overall prevalence of risk factors was higher in brothers (83%), than in sisters (73%). Only 17% of brothers were free of risk factors, whereas 27% of sisters were without risk factors.

The literature supports a familial pattern of heart disease and cardiovascular risk factors that may preclude

disease. However, such familial aggregation could be caused by yet undefined genetic factors, environmental factors common to family members or the interaction of both agents. Further research is needed in this area.

Prevention

Victims of sudden death share most of the major risk factors for coronary heart disease. The best prospect for prevention would appear to entail a reduction in overall coronary heart disease. Thus far it has not been conclusively demonstrated that sudden death can be avoided by controlling risk factors. However, it would seem logical that the same measures that succeed in reducing coronary heart disease would reduce the incidence of sudden death.

Many studies have been done to test the efficacy of modifying certain risk factors to reduce the incidence of coronary heart disease. The Lipid Research Center's Coronary Primary Prevention Trial showed that a 1% decrease in serum cholesterol was assiciated with a 2% decrease in mortality (1984). The Hypertension Detection and Follow-up Program compared the effects of a systematic antihypertensive treatment program to that of a control referral group which received no intervention. Overall mortality was 17% lower for the group that received antihypertensive treatment (1979). The Multiple Risk Factor Intervention Trial (1982) confirmed the independent contributions of serum cholesterol, hypertension, and cigarette smoking as coronary

risk factors. The Life Style Heart Trial (Ornish, Brown, Scherwitz, Billings, Armstrong & Ports, 1990) showed a significant overall regression of coronary atherosclerosis brought about by comprehensive life-style changes. As research continues to discover causal relationship between coronary heart disease, sudden death and predisposing risk factors, we as health professionals have a resposibility to utilize this knowledge in nursing practice.

Purpose

The three purposes of this descriptive study are to; (1) describe the level of cardiovascular risk in fifteen male and/or female siblings and/or offspring of sudden cardiac death victims by measuring their cardiovascular risk factors, including age, total serum cholesterol, HDL cholesterol, systolic blood pressure, cigarette smoking, diabetes, and EKG for left ventricular hypertrophy ; (2) calculate the likelihood of developing coronary heart disease and the possibility of having a cardiac event (a heart attack and/or sudden cardiac death) using the 1990 American Heart Association coronary heart disease risk factor prediction chart (Appendix B); and (3) to compare the likelihood of SCD relatives developing coronary heart disease within 10 years, to risk of an average man or woman in the same age categories. The University of Washington Medical Center will be the setting for this study.

Chapter III

Methodology

Research Design

A descriptive survey design will be used. This type of design was chosen; 1) in order to describe the prevalence of certain cardiovascular risk factors in the primary relatives (siblings and offspring) of persons experiencing sudden cardiac death and, 2) to describe the likelihood of SCD relatives developing coronary heart disease and compare their risk with the risk in age and gender matched data for the general population.

This study is part of a collaborative effort with another graduate nursing student, Nicole Pashek, who will examine upper body obesity, glucose intolerance, hypertriglyceridemia, and hypertension as cardiovascular risk factors in relatives of sudden cardiac death victims. Data collection for both studies will be done simultaneously.

The Sample

The names of individuals who have experienced a sudden cardiac death event will be obtained from a Seattle support group list. Permission for access to this population has been obtained from the patients' physician. These individuals are the fortunate survivors (having responded to cardiac resusitation) of sudden cardiac death. The criteria for inclusion for the index cases is as follows: 1) a

confirmed diagnosis of sudden cardiac death; 2) having at least one sibling or offspring; 3) an age of 30-60 years for men, an age of 30-74 years for women. This age range is chosen based on a study by tenKate, et.al (1982) which states that people whose family members have had a myocardial infarction before age 60 are at an increased risk for developing coronary heart disease. The age range was extended an additional 14 years to account for women subjects who reach the male level of SCD incidence 20 years later in life (Kannel & Schatzkin 1985). The specific age range of 30-74 years was chosen in accordance with the age range used in the risk evaluation tool which is based on data from the Framingham Study (Appendix B). Index cases and primary relatives from any geographic location will be included. The criteria for inclusion for the primary relatives is that they are 30-74 years in age, in accordance with the age range used in our evaluation tool. Any index case or primary relative will be excluded if: 1) they do not speak English ; 2) are mentally incapable of informed consent; 3) are unable to come to the University of Washington to have their risk factors measured. Every index case that meets the inclusion/exclusion criteria will be contacted until an N of 15 primary relatives (siblings and offspring) is obtained. This number was chosen considering the preliminary nature of the study, limitations of time, and cost.

Data Producing Instruments

There will be four instruments that will be used for measurement of cardiovascular risk factors and demographic data. A Hawksley Random zero sphygmomanometer will be used to determine blood pressure. This scientific instrument was chosen to increase validity by eliminating observer bias and digit preference when taking blood pressures. The random zero sphygmomanometer incorporates a shifting zero of mercury column and a constant discharge valve. The zero displacement feature of the device blinds the observer to the true value of the measurement until after the blood pressure is taken (Wright and Dore,1970).

Blood pressures will be taken after the subjects have been seated for five minutes. All measures will use the right arm for consistency. Once the blood pressure has been taken, the "zero" offset digit provided by the sphygmomanometer will be subtracted from the blood pressure readings to give a true value. The average of two seperate readings will be calculated, with three minute intervals between readings. The true blood pressure value, and corresponding points, will be recorded on the Data Collection Worksheet (Appendix A).

Blood samples will be drawn at the University's laboratory. The lipid glucose profile will include total serum cholesterol, HDL cholesterol, and triglyceride levels. The blood sample will consist of 10cc of blood. The blood

samples will then be analyzed using the Abell-Kendall procedure; a national reference method used by the Center for Disease Control (National Cholesterol Education Program, 1988). The total cholesterol and HDL cholesterol values, and corresponding points will be recorded on the Data Collection Sheet (Appendix A).

A 12 lead electrocardiogram will be taken on each subject, by the same person each time. The EKG machine is manufactured by Hewlett-Packard and has been licensed by the FDA for clinical diagnostic use. The machine has been calibrated and tested by the University's bioengineering department. The machine has been internally calibrated to identify left ventricular hypertrophy (LVH) according to the 12 lead electrocardiogram. Corresponding points for LVH will be recorded on the Data Collection Sheet.

In addition, subjects will be asked to answer questions to complete the Data Collection Sheet (Appendix A), which addresses smoking habit, family history of heart disease and diabetes, and current medications. Corresponding points will be recorded on the Data Collection Sheet.

Methods of Procedure

An introductory letter describing the study (Appendix C) will be mailed to all sudden cardiac death victims (index cases who meet inclusion criteria) whose names appear on the support group listing. If the index case believes he/she has a primary relative interested in participating in the study,

they are informed that they will be contacted by telephone in approximately two weeks. If they do not wish to participate, instructions to return the enclosed refusal post card will be included (Appendix D). If a refusal postcard is not received within two weeks, each index case will be contacted by telephone. They will be asked if they would provide the investigator with the name and address of a primary relative(s), who meet the study criteria, and whom we can contact by mail.

An explanatory letter describing the study will then be sent to the primary relative (Appendix E). A refusal post card will also be included with the letter. If a refusal post card is not received, contact by telephone will be made with the primary relatives two weeks after mailing the letter. At this time the subjects will be asked if they wish to participate in the study, and if they would be willing to come to the University to have their risk factors measured and answer some questions concerning age, family history of heart disease/diabetes, smoking, and current medications. The entire proceedure will be expected to take approximately two hours. Also at this time, subjects will be informed of their right not to participate in the study, and asked for verbal consent to participate. They will also be informed of the need for written consent as well (Appendix F). Subjects who agree to participate will also be instructed to fast for 10 hours before having their blood drawn. Primary relatives

will continue to be contacted until a N of 15 is obtained.

Written consent will be obtained at the University prior to any data collection. Then data collection will proceed at the University of Washington laboratory, where their blood will be drawn. Previous arrangements have been made with the laboratory for this patient population. After completing the blood draw, subjects will be escorted to the University of Washington nursing laboratory where a breakfast of muffins and juice will be offered. They will then be asked questions to complete the Data Collection Sheet. Then blood pressure, waist/hip measurement and an EKG will be obtained. Permission has been obtained for entry to the data collection setting, and for use of the equipment. Each participant will be given their own client information sheet (Appendix G) on which to record their blood pressure and the results of their cholesterol profile. Any participant that has a systolic blood pressure greater than 200mmHg will be referred to the emergency room at the University. Participants that have questions which may require additional counselling will be referred to their personal physician. Participants will be contacted by telephone in approximately one week and told the results of their laboratory tests. American Heart Association literature on cardiovascular risk factors will be available for participants to take home. Participants will be reimbursed for parking costs.

Protection of Human Rights

Approval for the study will be obtained from the University of Washington Human Subjects Review Committee. All potential participants will have been sent a refusal post card and an explanatory letter which contains information similiar to a consent form. At the time of telephone contact, subjects will be given an opportunity not to participate in the study. Written consent will be obtained prior to data collection.

The potential risks to the participants are minimal. Standard sterile venipuncture procedure will be followed. They are told about the possibilty of a bruised area and that pressure will be applied over the vein to minimize this risk. The minimal risk involved does not outway the potential benefit to the individual who may discover that he/she has a cardiovascular risk factor that they were previously unaware of; and that there are community resources avaliable to them.

Data Analysis

The data will be analyzed using descriptive statistics. The shape (using frequency distribution), location (using measures of central tendency), and spread (using a standard deviation) of the various risk factors will be described.

To calculate the likelihood of developing coronary heart disease, the Coronary Heart Disease Risk Factor Prediction Chart will be used (Appendix A). This American

Heart Association tool is based on a recent analysis of the ongoing Framingham Heart study by Anderson & Kannel (1990), recognized experts in their field. This tool is the most complete, current data base for identifying persons at high risk for developing heart disease.

Among the risk factors identified are: gender, age, cigarette smoking, elevated blood pressure, high levels of serum cholesterol, low HDL-cholesterol, diabetes, and EKG abnormalities. These factors are not the only risk factors which might be considered in assessing risk of coronary heart disease, but they are a set of proven merit which can readily be measured. Also, analyzing a single risk factor is neither a logical or an effective means of detecting persons at high risk. The most "efficient and sensible" method of evaluating risk is to synthesize the major contributors to coronary heart disease quantitatively into a composite score.

The chart provides a synthesis of the information expressed as the probability of a coronary event within five or ten years in chances per 100. To determine a risk level, the investigator will assign the appropriate number of points for each risk factor that has been asterisked on the Data Collection Sheet (Appendix A). These point values will be obtained from the Risk Factor Prediction Chart (Appendix B). Then all the points for each risk factor will be summed. This point total, (age + HDL-C + total-C + systolic blood

pressure + smoking + diabetes + EKG-LVH) will be used to calculate the probability/risk of developing coronary heart disease in a 10 year period, according to the Risk Factor Prediction Chart (Appendix B). The investigator will then compare the probabilty of SCD relatives developing coronary heart disease to the average 10 year risk given for a man or woman in the same age category, using the Risk Factor Prediction Chart. An unpaired t-test will be used to determine if there are any significant risks, and to test the hypothesis that there will be increased risk in primary relatives.

Appendix A

DATA COLLECTION SHEET

Date: _____

1. Code number _____ 1-2

2. What is your age? _____ 3-4

3. What is your birthday? ____/____/____ 5-6 (MT) 7-8 (Day) 9-10 (YR)

4. Gender: 1) Female 2) Male _____ 11

5. Age/ Gender/ Points * _____ 12-13-14

Has any of your family had chest pain, heart attack or heart disease?

6. Father 1) yes 2) no 3) Don't know _____ 15

7. Mother 1) yes 2) no 3) Don't know _____ 16

8. Siblings 1) yes 2) no 3) Don't know _____ 17

9. Grandparents 1) yes 2) no 3) Don't know _____ 18

Has any of your family had high blood sugar/diabetes?

10. Father 1) yes 2) no 3) Don't know _____ 19

11. Mother 1) yes 2) no 3) Don't know _____ 20

12. Siblings 1) yes 2) no 3) Don't know _____ 21

13. Grandparents 1) yes 2) no 3) Don't know _____ 22

14. Do you smoke cigarettes? 1) yes 2) no _____ 23
(Must have quit for over 12 months for no)
Other smoking: _____

15. Smoking / Points * _____ 24

If yes to #14, go to #16. If no to #14, go to #17.

Date: _____
Code # _____

16. How many cigarettes do you smoke every day?
    1) Smoke < 1/2 pack per day
    2) Smoke 1/2 to 1 pack per day
    3) Smoke 1 pack to 2 packs per day
    4) Smoke > 2 packs per day
    _____ 25

17. Do you have Diabetes Mellitus (high blood sugar)?
    1) yes 2) no 3) Don't know _____ 26

Do you take medications for:

18. High blood pressure? 1) yes 2) no 3) Don't know _____ 27
    Meds if yes: _____

19. High blood cholesterol? 1) yes 2) no 3) Don't know _____ 28
    Meds if yes: _____

20. High blood sugar? 1) yes 2) yes 3) Don't know _____ 29
    Meds if yes: _____

21. First Systolic BP _____-_____=_____ _____ 30-31-32

22. First Diastolic BP _____-_____=_____ _____ 33-34-35

23. Second Systolic BP _____-_____=_____ _____ 36-37-38

24. Second Diastolic BP _____-_____=_____ _____ 39-40-41

25. Average $\frac{(21+23)}{2}$ Systolic BP _____ _____ 42-43-44

26. Systolic BP / Points * _____ 45-46

27. EKG/LVH 1) yes 2) no _____ 47

28. EKG/LVH / Points * _____ 48

29. Waist = _____ cm. _____ 49-50-51

30. Hip = _____ cm. _____ 52-53-54

31. Waist/Hip ratio _____ 55-56-57

32. HDL Cholesterol _____ 58-59-60

33. HDL Cholesterol / Points * _____ 61-62

34. Total Cholesterol _____ 63-64-65

35. Total Cholesterol / Points * _____ 66-67

Date: _____
Code #: _____

36. LDL Cholesterol _____ 68-69-70

37. Triglyceride _____ 71-72-73-74

38. Fasting Blood Glucose _____ 75-76-77

39. Blood Sugar / Points * _____ 78

    Yes if: Blood sugar >140mg/dl (#38)
        or: On insulin or oral agents (#20)
        or: Report history of diabetes (#17)

40. Total Point Score:
    (#5 + #15 + #26 + #28 + #33 + #35 + #39) _____ 79-80-81

41. Percent Risk (10 year) _____ 82-83

42. Age/Gender (Average 10 year risk) _____ 84-85

Coronary Heart Disease
Risk Factor Prediction Chart

American Heart Association

1. Find Points For Each Risk Factor

Age (If Female)				Age (If Male)				HDL-Cholesterol		Total-Cholesterol		Systolic Blood Pressure			
Age	Pts.	Age	Pts.	Age	Pts.	Age	Pts.	HDL-C	Pts.	Total-C	Pts.	SBP	Pts.	Other	Pts.
30	-12	47–48	5	30	-2	57–59	13	25–26	7	139–151	-3	98–104	-2	Cigarettes	4
31	-11	49–50	6	31	-1	60–61	14	27–29	6	152–166	-2	105–112	-1	Diabetic-male	3
32	-9	51–52	7	32–33	0	62–64	15	30–32	5	167–182	-1	113–120	0	Diabetic-female	6
33	-8	53–55	8	34	1	65–67	16	33–35	4	183–199	0	121–129	1	ECG-LVH	9
34	-6	56–60	9	35–36	2	68–70	17	36–38	3	200–219	1	130–139	2		
35	-5	61–67	10	37–38	3	71–73	18	39–42	2	220–239	2	140–149	3	0 pts for each NO	
36	-4	68–74	11	39	4	74	19	43–46	1	240–262	3	150–160	4		
37	-3			40–41	5			47–50	0	263–288	4	161–172	5		
38	-2			42–43	6			51–55	-1	289–315	5	173–185	6		
39	-1			44–45	7			56–60	-2	316–330	6				
40	0			46–47	8			61–66	-3						
41	1			48–49	9			67–73	-4						
42–43	2			50–51	10			74–80	-5						
44	3			52–54	11			81–87	-6						
45–46	4			55–56	12			88–96	-7						

2. Sum Points For All Risk Factors

_____ (Age) + _____ (HDL-C) + _____ (Total-C) + _____ (SBP) + _____ (Smoker) + _____ (Diabetes) + _____ (ECG-LVH) = _____ (Point Total)

NOTE: *Minus Points Subtract From Total.*

3. Look Up Risk Corresponding To Point Total

Probability			Probability			Probability			Probability		
Pts.	5 Yr.	10 Yr.	Pts.	5 Yr.	10 Yr.	Pts.	5 Yr.	10 Yr.	Pts.	5 Yr.	10 Yr.
≤1	<1%	<2%	10	2%	6%	19	8%	16%	28	19%	33%
2	1%	2%	11	3%	6%	20	8%	18%	29	20%	36%
3	1%	2%	12	3%	7%	21	9%	19%	30	22%	38%
4	1%	2%	13	3%	8%	22	11%	21%	31	24%	40%
5	1%	3%	14	4%	9%	23	12%	23%	32	25%	42%
6	1%	3%	15	5%	10%	24	13%	25%			
7	1%	4%	16	5%	12%	25	14%	27%			
8	2%	4%	17	6%	13%	26	16%	29%			
9	2%	5%	18	7%	14%	27	17%	31%			

4. Compare To Average 10 Year Risk

Probability		
Age	Women	Men
30–34	<1%	3%
35–39	<1%	5%
40–44	2%	6%
45–49	5%	10%
50–54	8%	14%
55–59	12%	16%
60–64	13%	21%
65–69	9%	30%
70–74	12%	24%

These charts were prepared with the help of William B. Kannel, M.D., Professor of Medicine and Public Health and Ralph D'Agostino, Ph.D., Head, Department of Mathematics, both at Boston University; Keaven Anderson, Ph.D., Statistician, NHLBI, Framingham Study; Daniel McGee, Ph.D., Associate Professor, University of Arizona.

Framingham Heart Study

APPENDIX C

Introductory Letter to Index Cases

Since you have had a sudden cardiac arrest, your family member(s) may be eligible for a free cardiac health screening. A study is being conducted by two graduate nursing students at the University of Washington who are studying cardiac risk factors of primary relatives (brothers, sisters, and children related by blood) of people who have had a sudden cardiac arrest. Through simple tests, we hope to determine the risk level for heart disease in your family members.

The cardiac risk assessment will be done free of cost for your relative. It will involve a blood sample for a complete cholesterol profile, a test for blood sugar, a tape measurement of the hip and waist, an electrocardiogram (a non-invasive recording of your heart beat) and a brief questionnaire. All information will be kept confidential. Results of the assessment and information on how to reduce risk factors will be made available for your relative. If you have a brother, sister or child that you believe would be interested in having their cardiac risk factors measured, they may be eligible to participate in this study. We will contact you by phone for the names of relatives you recommend. If you do not wish to participate, please return the enclosed stamped refusal post card.

We appreciate the time that you have taken to read

about our study. If you have any questions, we can be contacted at (206) 543-1047.

Sincerely,

Abigail Palmer, R.N.

Nicole Pashek, R.N.

Susanna Cunningham, R.N., Ph.D.

Associate Professor

APPENDIX D

REFUSAL POSTCARD

Dear Ms. Pashek and Ms. Palmer,

I do not wish to participate in your study. Please do not contact me.

Signed : _____

Subject # : _____

Appendix E

Introductory Letter to Primary Relatives

Address

Dear_____

We are writing to request your participation in a study to measure your risk for heart disease. You have been selected because you are a relative of a person who experienced sudden cardiac death. Your name was given to us by that relative.

This study is being conducted by Abigail Palmer and Nichole Pashek, graduate nursing students at the University of Washington. The study involves coming to the University of Washington to have certain risk factors for heart disease measured. These include; blood pressure, a complete blood cholesterol profile, a test for blood sugar, an electrocardiogram (a non-invasive heart test), a measurement of your hip and waist and a brief questionaire. It should take about two hours. <u>These simple tests will be provided at no cost to you.</u> You will receive the results of all the tests, an explanation and literature.

A member of the research team will contact you by telephone in about a week. At that time you may choose to (1) ask any questions or concerns; (2) schedule a convenient

time to participate, or (3) not participate. If you are sure you do not want to participate, please return the enclosed refusal post card.

The risks associated with this study are minimal. Your name will not be used in association with the information that you give. You are free to withdraw from this study at any time.

We hope you will participate in this study. Your contribution will assist us in describing the degree of risk for heart disease that may exist for family members like yourself.

If you have any questions, we can be contacted at (206) 527-6178 or 526-1820.

Sincerely,

Abigail Palmer, R.N. Graduate student

Nichole Pashek, R.N. Graduate student

These individuals are graduate students in the Department of Physiological Nursing.

Susanna Cunningham, PhD.

Associate Professor

Appendix F

UNIVERSITY OF WASHINGTON

Consent Form

Cardiovascular Risk Factors in Primary Relatives
of Sudden Cardiac Death Victims

Four Cardiovascular Risk Factors in Primary Relatives
of Sudden Cardiac Death Survivors

Investigators: Abigail Palmer, RN, Graduate Student,
Department of Physiological Nursing,
526-1820

Nicole Pashek, RN, Graduate Student,
Department of Physiological Nursing,
527-6178

Co-Investigator: Susanna Cunningham, RN, PhD,
Associate Professor,
Department of Nursing
543-1047

Investigator's Statement

PURPOSE AND BENEFITS

The purpose of this study is to measure certain cardiovascular risk factors in relatives of people who have had a sudden cardiac death event. Your contribution will assist us in describing the degree of risk for heart disease that may exist for family members like yourself. This study may be useful in the future to help identify a population potentially at risk for developing sudden death and other manifestations of cardiovascular disease. Subsequent prevention programs for risk factor reduction could then be implemented. This study is being done in partial fulfillment of the requirements of a Master's thesis.

PROCEDURES

The cardiac assessment that we will perform includes: taking your blood pressure, a measurement of your hip and waist, an electrocardiogram (a non-invasive test of your heart beat), having one blood sample that will be tested for blood suagar and cholesterol levels (drawn in the University's laboratory), and answering a brief questionare for background information. The proceedure should take about two hours.

RISKS, STRESS OR DISCOMFORT

The study will involve minimal discomfort to you during withdraw of blood from your vein. A total of 10cc or about two teaspoons of blood will be taken. There is a slight possibility of a hematoma (blood under the skin) from this procedure, although pressure will be applied over the vein to minimize this risk. You will also be required to fast for 10 hours before getting your blood drawn. Breakfast will be provided after your blood draw. The electrocardiogram will take about 10 minutes, during which you will need to lie flat. Ten patches are placed on your chest, and a recording of your heart is made. During all of the procedures, privacy will be maintained.

OTHER INFORMATION

These simple tests will be provided at no cost to you. You will receive the results of all the tests, an explanation and American Heart Association literature. You are free to refuse to participate and to withdraw from the study at any time. You will have an opportunity to ask any questions before consenting to participate in the study and anytime during the study.

The identity of all patients in this study will be kept confidential. Each patient will be assigned a code number and only the investigators will be able to identify a patient by name or data. Access to the data will be restricted to the investigators and their advisor. The information will be retained for one year following the study. The results will be interpreted in a Master's thesis with possible future publication.

_____ _____

Signature of the Investigator Date

_____ _____

Signature of the Investigator Date

Subject's Statement

The study described above has been explained to me, and I voluntarily consent to participate in the study. I have had an opportunity to ask questions. I understand that future questions I may have about the research or about subject's rights will be answered by one of the investigators listed above.

_____ _____

Signature of the Patient Date

Copies to: Subject
Investigators file

Appendix G

Client Information Sheet

1. Blood Pressure _____ systolic / _____ diastolic

   * Desired value: less than 140mmHg systolic / less than 90mmHg diastolic

2. Total Cholesterol _____

   + Desired value: less than 200mg/dl

3. HDL Cholesterol _____

   ++ Desired value: greater than 35mg/dl

4. LDL Cholesterol _____

   + Desired value: less than 130mg/dl

5. Triglyceride _____

   + Desired value: less than 500mg/dl

6. Plasma Glucose _____

   ++ Desired value: less than 140mg/dl

7. Waist/Hip ratio _____ waist cm. / hip cm.

   * Desired value: Men less than or equal to 1.0
   Women less than or equal to 0.8

Please remember, one abnormal value does not necessarily mean that you are at increased risk. However, we do suggest if any of your blood tests or blood pressures are not within the desired range you should go to your physician for a recheck. For more information about cardiovascular risk factors contact the American Heart Association, Washington Affiliate at 632-6881.

* American Heart Association, 1991
\+ National Institutes of Health, 1989
++ Grundy, S.M. et al., 1987

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