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THE UNIVERSITY OF ALBERTA

RISK FACTORS AND MYOCARDIAL INFARCTION IN FEMALES: A COMPARISON WITH MALES

BY

JOHN H. PARKER

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH IN PARTIAL FULLFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF NURSING.

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John H. Parker
(Student's Signature)

6444 172 STREET
(Student's Permanent Address)

EDMONTON, ALBERTA

Date: October 14, 1988

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled Risk Factors and Myocardial Infarction in Females: A Comparison with Males submitted by John H. Parker in partial fulfilment of the requirements for the degree of Master of Nursing.

Jesse F. Kikuchi
(Supervisor)
Rene A. Day
Stephen Newman

Date: October 7, 1988

Dedication

This effort is dedicated to the memories of Matthew Johnston Parker and Micheal Harrison Parker. Even now, I feel your tiny hands slip into mine.

Abstract

The purpose of this study was to gain knowledge of some of the possible differences between males and females with myocardial infarction (MI) with respect to those factors which are associated with changes in the probability of occurrence of this disease. An answer to the following question was sought: Do males and females differ in terms of certain factors which have been found to be associated with changes in the probability of occurrence of MI? If so, how?

Using a cross sectional study design with a prospective component, a sample of 153 male and 147 female MI patients was identified through the hospital records. Using data from the hospital records of this sample, males and females were compared in terms of certain risk factors identified within the literature as being associated with changes in the probability of occurrence of MI. For heuristic purposes, they were also compared in terms of mortality and of some factors which may influence prognosis following the occurrence of MI. Both data which were nonstratified and stratified by age were analyzed, using nonparametric and parametric statistics.

For several factors, statistically significant differences between males and females were observed. These factors included age, marital status, employment status, history of cardiovascular disease, cigarette smoking,

diabetes, hypertension, complications following MI, mortality, and certain serum enzyme levels. When the data were stratified into age groups and analyzed, these statistically significant differences were, for the most part, no longer evident. This lack of statistical significance may have been a result of insufficient numbers of subjects in the designated age groups. The findings of this study indicated that males and females may, indeed, differ with respect to certain factors which have been found to be associated with changes in the probability of occurrence of MI. From the study findings, several recommendations for further research were generated.

Acknowledgement

I wish to thank Dr. Stephen Newman and Dr. Rene Day for their generous and gracious assistance. I am deeply indebted to Dr. June Kikuchi who, with great patience and skill, made me realize that I was capable of completing this project.

I also wish to thank my friends and family for their kind and generous help. These friends and family include me ol' mum and dad, Jim, Al and Leanne, and my son, Andrew.

Finally, the greatest debt I owe is to my wife, Bev. Without her love, understanding, and hard work, none of this would have been possible. We made it kiddo!

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CHAPTER I

Introduction

Background of the Problem

The overall incidence and prevalence of myocardial infarction (MI) in females is far lower than in males (Patrick, Palesch, Feinleib, & Brody, 1982; Weinblatt, Shapiro, & Frank, 1973; Wingard, 1982) and appears to be consistently so over various geographical locations (Pisa & Uemura, 1982, United Nations, 1986). The mean age of incidence of MI is greater in females than in males and the average number of years of life lost due to MI is lower in females than in males (Marmot, 1985). These observations also hold true for Canadian males and females (Parker, 1986; Statistics Canada, 1982; Statistics Canada, 1986). These statistics indicate that the occurrence of MI in females is less frequent than that in males and that, on average, females die at an older age than males as a result of MI.

If one assumes that the probability of occurrence of a disease is increased in the presence of a factor or factors, commonly referred to as risk factors (Mausner & Kramer, 1985), is it possible that the differences in incidence and prevalence of MI between males and females are the result of differences in risk factors unique to each sex? Or, more concisely, do males and females differ in terms of those factors which are associated with changes in the probability of occurrence of MI?

A large number of studies, in which researchers have investigated the effect on risk of MI of various factors, have appeared over the past ten years. The majority of these studies involved samples restricted to males only or homogeneous samples of males and females. If the factors which influence the probability of occurrence of MI are not the same for males and females, the observations reported in these studies do not allow relevant conclusions to be drawn concerning the possibility of females being unique from males with respect to these factors. (Johansson, Vedin, & Wilhelmsson, 1983; Johnson, 1977).

An implicit or explicit assumption of the researchers of many of the studies that have been conducted was that males and females are identical in terms of those factors which influence the probability of occurrence of MI. As a consequence of this assumption, females have been, in many instances, regarded as identical to males in terms of the primary, secondary, and tertiary prevention measures necessitated by MI. If males and females are found to differ in terms of those factors which influence the probability of occurrence of MI, then the assumption being held and the consequences which flow from it need to be reexamined. (Johnson, 1977; Szklo, 1984).

Statement of the Problem

The problem may be stated in terms of what is known and unknown concerning females being dissimilar from males with

regard to MI and those factors which influence the probability of occurrence of MI. What is known is that males and females differ with respect to the incidence and prevalence of MI. What is unknown is the mechanism or mechanisms which are responsible for such differences. One such possible mechanism may be patterns of factors, unique to males and females, which influence the probability of occurrence of MI.

Purpose of the Study

The purpose of this study was to gain knowledge of some of the possible differences that exist, if any, between males and females with respect to those factors which influence the probability of the occurrence of MI. If identified, such differences may provide insight into the mechanisms underlying the dissimilarities of incidence and prevalence of MI observed for males and females.

Objective of the Study

The research objective was to compare samples of males and females, with the diagnosis of MI, in terms of certain factors which have been observed in the literature to influence the probability of occurrence of MI.

Research Question

An answer to the following question was sought: Do males and females differ in terms of certain factors which have been found to be associated with changes in the probability of occurrence of MI? If so, how?

Definitions

Risk Factor. A risk factor is a factor "whose presence is associated with an increased probability that disease will develop" (Mausner & Kramer, 1985, p. 6).

Acute Myocardial Infarction. An acute myocardial infarction is an area of necrosis of the myocardium which results from complete oxygen deprivation. Diagnosis is based on unequivocal electrocardiographic (ECG) changes and/or unequivocal ECG changes with abnormal cardiac enzymes and/or prolonged cardiac pain with abnormal cardiac enzymes (Gillum, Fortmann, Prineas, & Kottke, 1984).

Conceptual Framework

The conceptual framework for this study is adapted from the diamond model of health and illness causation. Figure 1 depicts the components and relationships of this model.

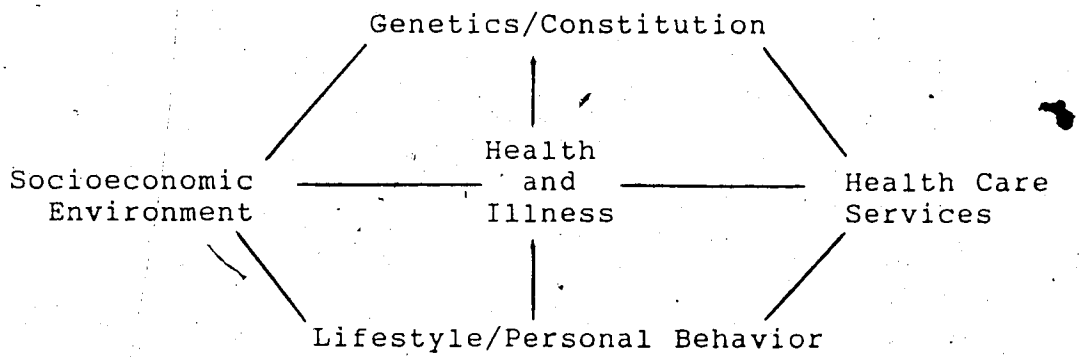

Figure 1. Diamond Model of Health and Illness Causation (Adapted from Long, 1985, p. 4)

Within this model, health and illness are viewed as

being determined by both independent and interactive effects of the host's constitution, the host's lifestyle and personal behavior, the socioeconomic environment, and the efficacy of available health care services. Thus, an individual's levels of health or illness result from the effects of the highly sophisticated interplay of these four variables.

In this model, the health and illness component is not fixed and may vary with the perspective of the researcher (Long, 1985, p. 37). The model may, therefore, be used as a framework for a variety of perspectives. For the purpose of this study, Fanshel's (1972) conceptualization of wellness is used to replace the health and illness component within the diamond. According to Fanshel, "a person is well if he is able to carry out his usual daily activities. To the extent that he cannot, he is in a state of dysfunction, or deviation from well being" (p. 319).

An individual's wellness is characterized by his functional ability rather than by the mere presence or absence of disease. Fanshel (1972) identified 11 states of functional ability, each with an operational definition, and placed these states along a continuum of wellness. This continuum ranges from well being (optimum functioning) to death (absence of functioning).

Within this perspective of wellness, "the rehabilitation of the total individual rather than the cure

of the disease is the object of the health system" (Fanshel, 1972, p. 320). Further, disease is only one determinant by which an individual is located along the continuum. In other words, an individual's functional ability, or wellness, is determined by the independent and interactive effects of the four variables and the factors within each variable; a disease, if present, being a factor in the variable of genetics/constitution.

The four variables or dimensions and the factors within each which determine an individual's wellness may be described as follows:

Health Care Services. This dimension includes the primary, secondary, and tertiary prevention and palliative services available from the health care system. The dimension also includes such factors as health promotion and education services, screening services, and the crucial factor of ease of access to such services.

Lifestyle/Personal Behavior. This dimension includes those factors which influence wellness that are determined and controlled by the individual. Such factors include smoking, diet, alcohol consumption, and exercise. Similarly, an individual's own perceptions of the meaning and value of wellness, his or her perceptions of the value of potential health care services, and his or her tolerance of compromised functional ability are also included in this dimension.

Socioeconomic Environment. This dimension includes the effect on wellness of such factors as place of residence, air pollution, occupation and occupational hazards, social background, education, the general societal structure, and the value of wellness the society holds. This dimension is distinguished from the dimension of lifestyle and personal behavior in that the individual has little control over the socioeconomic factors which influence wellness.

Genetics/Constitution. This dimension includes both genetic structure acquired at birth, an important determinant of both physical and mental growth potential, as well as those constitutional factors acquired through life such as the consequences of nutritional intake or compromised immunity. Both genetic structure and constitutional factors acquired through life determine an individual's ability to cope with tension (Antonovsky cited in Long, 1985, p. 4).

When a disease occurs, the presence or absence of factors which originate from within the four dimensions of the model can be measured. A factor which is consistently present with the occurrence of a disease may be said to be associated with an increased probability of the occurrence of that disease. Given the nature of the dimensions of the model, these factors may be either amenable or immutable to change. MI and the factors which influence the probability of its occurrence are only a part of what determines an

individual's functional ability at the time of onset of the disease. An individual's functional ability is also determined by the total effect of all other factors which originate within the model's four dimensions.

Assumptions

This study was based on the following assumptions:

1. The probability of occurrence of MI is influenced by the presence of a factor or factors.
2. Such factors are either amenable or immutable to change.
3. Following a MI, an individual's functional ability or wellness is influenced, in part, by the occurrence of MI as well as all of the other factors which originate from within the four dimensions of the diamond model of wellness.

Significance of the Study

The significance of the study may be considered in terms of the implications for primary, secondary, and tertiary prevention of MI, if females are found to differ from males in terms of those factors which influence the probability of occurrence of MI. Primary prevention consists of reducing or eliminating those factors which increase the probability of illness and enhance the probability of maximizing health potential. Secondary prevention consists of early detection and prompt treatment of illness. Tertiary prevention consists of limiting disability and enhancing rehabilitation once illness occurs (Mausner & Kramer, 1985, pp. 10-11).

If females do, indeed, differ from males in terms of those factors which influence the probability of occurrence of MI, then the current strategies of primary prevention used to reduce this probability may need to be modified in terms of this difference. Similarly, the present strategies of detection and treatment and limiting disability and enhancing rehabilitation may also require modification to accommodate the possible uniqueness of females with MI.

Such modifications have specific implications for nursing if one accepts that nursing is committed to the "promotion, maintenance, and/or the recovery of health and the prevention of disease" (Greene, 1979, p. 63) and that nursing interventions are "designed to enhance each individual's health seeking behavior, to stimulate his avoidance of disease and disability and to promote productive use of his own inherent capacities to be restored to health and maximum capability in circumstances of genetic inadequacy, trauma, disease or deprivation of several kinds" (Schlotfeldt, 1971, p. 141).

CHAPTER II

Review of the Literature

The following is an integrative review of the literature on the topic of risk factors and myocardial infarction (MI) in females. The review was guided by the following question: What is known concerning risk factors which predispose females to suffer a MI?

For the purpose of this review, a risk factor was defined as a factor "whose presence is associated with an increased probability that disease will develop" (Mausner & Kramer, 1985, p. 6). The risk factors discussed in this review have been investigated by researchers to determine if those risk factors influence the probability that MI will develop in females.

The review covered the time span from 1976 to 1987 and drew primarily from the disciplines of medicine and epidemiology. The majority of the literature integrated into the review consisted of reports of demographic, retrospective, and prospective studies. All study reports were either written in English or presented in an English abstract.

The review begins with the literature relevant to the etiology of MI in females. This is followed by the literature relevant to various factors associated with changes in the probability of occurrence of MI in females. The review concludes with the literature relevant to

mortality due to MI in females. Although etiology and mortality are not considered risk factors of MI, they are included in the review to provide a more complete picture of this disease process in females.

In almost all instances, operational definitions of the terms, MI and coronary artery disease, were not published in the reports of the studies which were reviewed. Across many of these reports, these two terms were used synonymously by researchers, particularly in relation to discussions of mortality. Every attempt has been made to clarify the meanings of these terms as they apply to the individual studies.

Etiology

The literature related to two aspects of the etiology of MI in females is reviewed in the following section. These two aspects are coronary artery disease and angina.

Coronary Artery Disease

The prevalence and severity of coronary artery disease in females have been the foci of interest of many researchers. Morris, Hurst, and Logue (1976) investigated the prevalence of coronary atherosclerotic/thrombotic events as the cause of death in 24 females less than 40 years of age. Following post-mortem analyses, MI due to these events could be traced in only 25% of the females. The remaining females showed little, if any, coronary occlusion. The authors concluded that the incidence of death due to

these events is significantly less in young females than in elderly females or young males. Similar findings were reported by Silver, Baroldi, and Mariani (1980) who conducted post-mortem analyses on 72 males and 28 females all with MI determined as the cause of death. Twenty-five per cent of the females were found to have acute coronary thrombi compared with 57% of the males. Such findings were also reported by Walters, Halphen, Theroux, David, and Mizgal (1978). Of 239 Canadian females referred for angiography for suspicion of coronary artery disease, 47% were diagnosed as having normal coronary arteries.

Sketch, Mohiuddin, Lynch, Zencka, and Runco (1976) performed stress tests and angiography on 56 females and 195 males suspected of having coronary heart disease. Eighteen per cent of the females had coronary angiograms indicative of significant coronary artery stenosis. Only 30% of these females were accurately diagnosed by stress testing as having such stenosis. In contrast, 53% of the males in the sample were diagnosed by coronary angiograms as having significant stenosis with 89% of these males having such stenosis accurately diagnosed by stress testing.

Wei and Bulkey (1982) compared the angiographic profiles of 20 females and 20 males, all with the diagnosis of MI, and found significant coronary stenosis in only 23% of the females while significant stenosis was found in 92% of the males. Similarly, Fried and Pearson (1987) reported

that, of 266 females and 645 males referred for arteriography to assess the presence and degree of coronary artery disease, 48% of the females and 83% of the males were diagnosed as having coronary stenosis of 50% or greater.

Coronary artery disease and its relationship to various risk factors was investigated by Handler, Warren, and Vieweg (1981). These researchers studied the angiographic records of 305 males and females with either one vessel or two to three vessel disease. While the number of males was greater than females for each group, the researchers found that the incidence ratios of hypertension and diabetes were disproportionately high for the females of both groups.

Angina

Angina and electrocardiographic (ECG) changes and their relationships to MI in females have been investigated by several researchers. Campbell, Elwood, Abbas, and Waters (1984) observed that the risk of mortality due to acute MI was 50% higher for females who had experienced pre-event angina than for females who had not. This increased mortality risk was greatest for the 45-54 year age group.

Lapidus, Bengtsson, Lindqvist, Sigurdsson, and Rafnsson (1985) found further evidence of varying prognosis related to prevalence of angina, in their 12 year prospective study of 1462 females. The relative risk of mortality from MI in females with pre-event angina in comparison with females without pre-event angina was found to be 6.1. The relative

risk of mortality from angina alone was calculated to be only 1.6 for this cohort. In their report, the researchers discussed the findings of studies conducted by other researchers which demonstrated a significant increase in mortality risk due to angina alone in males. Lapidus et al. (1985) suggested that, because mortality risk is not significantly increased in the presence of angina alone in females, there may be an unique etiology of MI found in females.

In an ongoing prospective study of 5127 males and females, Kannel and Abbott (1984) and Kannel, Dannenberg, and Abbott (1985) found that the overall percentage of unrecognized MI in females was 35% compared with 28% for males. In the 45-54 year age group, these percentages were 41% for females and only 18% for males. The researchers found the values to be independent of angina and that, with unrecognized MI, the prognosis for females was more favorable than for males. The researchers of both studies concluded that ECG changes which lead to the suspicion or diagnosis of MI are more nonspecific in females and that angina, which motivates investigation for coronary artery disease in many instances, is not related to the incidence of unrecognized MI to a greater degree in females than males. In contrast to these results, Klaeboe, Otterstad, Wisnes, and Espeland (1987) and Logan, Wong, and Barclay (1986) observed that pre-MI angina was more prevalent in

females and of greater predictive value for the occurrence of MI in females than in males.

In summary, with respect to coronary artery disease and angina, the unique angiographic profiles, stress test results, prevalence of unrecognized MI, and ECG results which have been observed in the case of females with MI suggest that females may differ from males in terms of these aspects of the etiology of MI. However, in order to confirm this hypothesis, further investigation of these aspects is warranted.

Risk Factors

The literature related to specific risk factors which have been found to be associated with changes in the probability of occurrence of MI in females is reviewed in the following section of the literature review.

Geographical Location

An often overlooked factor which may influence the risk of MI in females is geographical location. The majority of the findings of the studies reported in this section of the review are the results of researchers' analyses of census and demographic data and, as such, consist of various measures of mortality. A review of several statistics calculated from Canadian census data relevant to MI mortality is presented first, followed by a review of reports in which MI mortality in other countries was investigated.

In 1985, 10448 females and 16597 males died, in Canada, as a result of MI in Canada (Statistics Canada, 1986). In a study, Parker (1986) transformed these mortality totals into standardized death rates for the years 1971 and 1981 in order to determine how males and females compared with respect to mortality following MI. These years were chosen as standard years since they represent complete census years in which the entire population of Canada was assessed. For the 1981 population, these rates were found to be 174/100,000 and 75/100,000 for males and females respectively. For the 1971 population, these standardized death rates were found to be 238/100,000 and 96/100,000 for males and females respectively. This represents a reduction in the standardized death rate of 22% for females and 27% for males from 1971 to 1981. These figures indicate that, in Canada, during this time period, the death rates due to MI for females have been significantly lower than those for males.

Other differences between males and females with respect to mean age of mortality following MI and potential years of life lost were observed when the Canadian census data (Statistics Canada, 1982) were further examined by Parker (1986). The mean ages of MI mortality, in 1981, for males and females, in Canada, were found to be 68.0 and 73.0 years respectively. The potential years of life lost per death due to MI, in 1981, were found to be 5.5 and 2.5 for

males and females respectively. These figures indicate that, on average, in Canada, males may be dying at an earlier age as a result of MI than females.

The trends observed in Canada have also been observed in the United States where the overall incidence and prevalence of MI in females have been consistently lower than those in males (Folsom et al., 1987; Patrick, Palesch, Feinleib, & Brody, 1982; Szklo, 1984; Wingard, 1982). Declines in per 100,000 female fatality rates and in hospital female mortality rates, due to MI, have been observed by various researchers from as early as 1950 (Feinleib, 1984; Gillum, 1987; Gillum, Folsom, & Blackburn, 1984). Similar reductions in female mortality due to ischemic heart disease have been observed in Brazil, Japan, Belgium, Finland, France, Italy, Norway, Australia, and New Zealand as of 1968 (Lessa et al., 1987; Martin, Hobbs, & Armstrong, 1987; Pisa & Uemura, 1982; Shibata, Tanazawa, Hirasawa, Tuteda, & Kanda, 1987).

Reductions in female mortality due to ischemic heart disease have not been experienced in all countries, as of 1968. In fact, significant increases in female mortality due to ischemic heart disease have been observed in Austria, Bulgaria, Denmark, the Federal Republic of Germany, Hungary, Netherlands, Poland, Romania, Sweden, Scotland, and Yugoslavia (Hammer & Ahlbom, 1987; Pisa & Uemura, 1982).

Males and females, in various countries, have also been

observed to differ with respect to mean age of incidence and prevalence of nonfatal MI. Estimations of mean age of incidence and prevalence of nonfatal MI in Israel, Sweden, United States, and Australia, indicate that females with nonfatal MI are significantly older than their male counterparts (Eisenberg, Ehrenfeld, Friedman, & Friedlander, 1981; Gillum, 1986; Johansson et al., 1984; Johansson, Vedin, & Wilhelmsson, 1983).

The validity of the aforementioned observations needs to be assessed since many different sources of demographic and census data were used in these studies, all with varying degrees of accuracy and completeness. The reported changes in mortality may well be the result of increased or decreased reporting of mortality. (Shryock, Siegal, & Associates, 1976). However, many researchers attribute these mortality changes to changes in medical and surgical therapy, diagnostic technology, cholesterol and cigarette consumption, and physical activity (Dwyer & Hetzel, 1980; Feinleib, 1984; Gillum, 1987; Gillum, Folsom, & Blackburn, 1984).

In summary, geographical location may influence the risk of mortality due to MI in females. Trends in mortality do not appear to be similar on an international level. Further research is indicated to verify these differences on both an international and national level. Research into the effect of geographical variation and the risk of MI in

females in countries of high geographic diversity, such as Canada and the United States, is also warranted.

Oral Contraceptives

In all, 13 studies were located in which the relationship between oral contraceptive (OC) use and MI in females had been examined. The researchers of four studies (Krueger et al., 1980; Mann, Inman, & Thorogood, 1976; Slone et al., 1981; Stern et al., 1976) concluded from their findings that current OC use significantly increases the risk of MI in females. In these studies, relative risk of MI for current OC users ranged from 2.8 to 4.2. On the basis of this finding, these researchers recommended that physicians and health care personnel advise current OC users of the inherent risk of MI associated with these drugs. This recommendation may have been erroneous for the following reasons. First, in many of the above cited studies, the data collected concerning OC use were obtained from physicians, next of kin, and medical records. Data collected in such a manner may not have accurately represented the actual OC habits of the females studied. Second, the researchers of all four studies did not consider other potential risk factors which may have interacted synergistically with OC use. If such an interaction did exist, the conclusion that OC use significantly increases the risk of MI in females may have been based on a spurious association between OC use and MI (Goldzieher, 1987).

The possibility of an interaction between OC use and other factors led many researchers to investigate the relationship between other factors, OC use, and MI in females. Arthes and Masi (1976) and Pettiti, Wingard, Pelligrin, and Ramcharan (1979) investigated the effect of OC use and cigarette smoking on the risk of MI in females. The researchers of both studies calculated the relative risk of MI to be 2.8 in the presence of both OC use and smoking. These results appear to indicate that smoking does not interact with OC use to influence the relative risk of MI in females. Again, data on OC use and smoking were obtained from medical charts and, thus, may not have been an accurate representation of the smoking habits of these females.

Evidence of a synergistic effect between OC use and smoking was reported by Shapiro et al. (1979). The relative risk of MI was found by these researchers to be 7.0 with smoking alone, 4.5 with OC use alone, and 39 with both of these factors present. Because both the nurse interviewer and respondents knew of the hypothesis to be tested, the authors cautioned that these results may have been accentuated.

Evidence of a synergistic effect between smoking and OC use in relation to risk of MI in females was also uncovered in three separate studies (Goldbaum, Kendrick, Hogelin, Gentry, & Behavioral Risk Factor Survey Group, 1987; Rosenberg, Hennekens, et al., 1980; Salonen, 1982). The

researchers of these studies found that the relative risk of MI in females who smoked and were current OC users was greater than the sum of the individual risk scores for smoking alone and OC use alone.

Finally, Jain (1976) reevaluated the results of two previously reported studies (Mann & Inman, 1975; Mann, Vessey, Thorogood, & Doll, 1975) in which the possibility of interaction between smoking and OC use and their combined effect on the risk of MI in females was not investigated. By recalculating the published data of these two studies, Jain found the relative risk of MI due to OC use alone to be insignificant. When he included the data given for cigarette smoking, the relative risk of MI in combination with OC use increased to 5.4 and 15 for light and heavy smokers respectively.

Researchers who have observed a synergistic effect between smoking and OC use have recommended that OC users who smoke should be advised to stop smoking rather than discontinue OC use. This recommendation appears to be more appropriate presently than that of recommending that females discontinue OC use, as OC use alone may increase the risk of MI in females only minimally.

Studies in which the interaction between OC use and factors other than smoking, such as diet and physical activity, was studied, could not be found in the literature.

In summary, in combination with smoking, a synergistic

effect appears to occur in females who are current OC users which may increase the risk of MI in females several fold. Because of the strong influence of OC on normal female hormonal cycles, further research in which the possibility of interactions is investigated appears to be warranted. In many of the reported studies, data concerning OC use were obtained from sources other than the females themselves and may have served to confound the findings of these studies.

Noncontraceptive Estrogens

Seven studies were located in which researchers had investigated the possible effects of noncontraceptive estrogens (NCE) on the risk of MI in females. The findings of three of these studies led researchers to conclude that NCE use offered a slight protective effect against the risk of MI in females. In a retrospective study of 336 females with MI and 6730 age matched controls, Rosenberg, Armstrong, Phil, and Jick (1976) observed the relative risk of MI in females who were current NCE users to be .97. A relative risk of .9 for females with MI who were current NCE users was calculated by Bain et al. (1981). Finally, Szklo, Tonascia, Gordis, and Bloom (1984) observed the relative risk of MI for current NCE users to be .61.

In three studies, NCE use was found to have no effect on the risk of MI in females. Pfeffer, Whipple, Kurosaki, and Chapman (1978) observed, in a retrospective study of 220 female cases with MI and an unreported number of controls,

no association between MI and current NCE use. LaVecchia, Franceschi, Decarli, Pampallona, and Tognoni (1987) observed no effect on relative risk of MI in females due to NCE use, in a retrospective study of 168 female cases with MI and 251 female hospital controls. Similarly, Rosenberg et al. (1980a) found the relative risk of MI with current OC use to be 1.0. These researchers also observed evidence of a slight interaction between NCE use and smoking.

Only one study was located in which current NCE use was found to increase the relative risk of MI in females (Jick, Dinan, Herman, & Rothman, 1978). The relative risk of MI with current NCE use was found to be 9.6 in this study of 83 females with MI and 166 age matched controls.

In summary, the results of several studies suggest that NCE use may have no effect on or exert a slight protective effect against the risk of MI in females. However, such studies are few in number and the results are conflicting. Based on the available evidence, a conclusion concerning the effect of current NCE use on the risk of MI in females may be premature.

Menopause

Six studies were located in which the effect of menopause on the risk of MI in females had been investigated. In two of these studies, the effects of surgical and natural menopause on the risk of MI were investigated by researchers.

Gordon, Kannel, Hjortland, and McNamara (1978) found the risk of MI was almost identical with both natural and surgical menopausal females. Also, the risk of MI for both groups was found to be significantly higher than that for premenopausal females. The researchers cautioned that they did not investigate the role of NCE in this cohort and suggested that it be investigated in future research. This may, in fact, have been a reasonable suggestion as NCE use may exert a slight protective effect against the risk of MI in females. The identical increased risk of MI, which was observed in this study with both surgical and natural menopause, may not be correct if the risk of MI was attenuated by the effect of NCE use.

Rosenberg, Hennekens et al. (1981) came to a different conclusion regarding the effect of natural and surgical menopause on risk of MI in females. In their retrospective study, these researchers observed that the relative risk of MI with natural menopause was .9 while the relative risk of MI with surgical menopause, as a result of a hysterectomy with and without bilateral oophorectomy, was 1.6 and 2.9 respectively. When the cases and controls were stratified by age, relative risk of MI increased as age decreased for natural menopause and for surgical menopause as a result of a hysterectomy with bilateral oophorectomy. In women less than 35 years of age, these relative risk scores were 2.8 and 7.7 respectively. Further replication of this research

was recommended by these researchers in light of the fact that, at the time of publication, 12/1000 American females were undergoing hysterectomy with bilateral oophorectomy surgery.

The effect of age of onset of menopause on the risk of MI in females has been investigated by many researchers. From additional analysis of data of their prospective study (Kannel, Hjortland, McNamara, & Gordon, 1976), Gordon, Kannel, Hjortland, and McNamara (1978) found that the incidence of coronary heart disease was greater in postmenopausal females than in premenopausal females. This increase in incidence was greatest with menopausal onset at less than 44 years of age.

In contrast, Lapidus, Bengtsson, and Lindqvist (1985), in a prospective study of 1462 females, and LaVecchia, Decarli et al. (1987), in a retrospective study of 202 female cases with MI and 374 female controls, observed that the age of menopausal onset did not affect the risk of MI. Similarly, Blanc, Boschat, Morin, Clavier, and Penther (1977), who conducted a retrospective study of 146 females with MI, found that the age of menopausal onset did not significantly correlate with risk of MI. However, data were complete for only 71% of these females and the details of menopause were, in many instances, obtained from relatives. Data collected in such a manner may not provide accurate details and thus may not be trustworthy.

In summary, type of menopause (i.e., surgical and natural) as well as age of menopausal onset may or may not affect the risk of MI in females. Many researchers have suggested that further research is warranted in this area due to the presence of conflicting results.

Cholesterol

The possible protective effect of high density lipoprotein (HDL) cholesterol against the risk of MI has been under considerable investigation, since 1976. This protective effect appears to be more prevalent in females.

Evidence of the protective effect of HDL cholesterol against MI in females was presented in four reports of the same prospective study (Garrison et al., 1980; Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977a; Kannel, 1987; Lerner & Kannel, 1986). In this study, an eight-fold increase in the incidence of coronary heart disease in females was observed when HDL cholesterol levels decreased from 75 mg/dl to 25 mg/dl. HDL cholesterol levels were significantly higher in the females of this study than the males. Higher levels of HDL cholesterol in females, when compared with males, were also observed in three other separate studies conducted during the same time period (Eisenberg, Ehrenfeld, Friedman, & Friedlander, 1981; Johansson et al., 1984; Yano et al., 1987).

Four studies were located in which researchers had investigated familial patterns of HDL cholesterol with

particular emphasis on the female offsprings of patients with MI. Pometta, Micheli, Suenram, and Jarnot (1979) conducted a retrospective study of 231 cases consisting of first degree relatives of MI patients and 132 controls. Female first degree relatives had significantly lower HDL cholesterol levels than the male first degree relatives. The researchers suggested that a genetic influence which may result in low HDL cholesterol levels may be stronger for female first degree offspring than male first degree offspring.

Pometta, Suenram, Sheybani, Grab, and James (1986) replicated the previously described study by Pommetta, Micheli, Suenram, and Jarnot (1979) using 375 first degree relatives (200 females and 175 males) of MI patients and 557 controls. When they were stratified by age, females who were less than 20 years of age were found to have HDL cholesterol levels which correlated more strongly with those of the MI patients than was the case with females who were older than 20 years of age. Because all of the first degree female relatives less than 20 years of age were still living at home, the researchers suggested that HDL cholesterol levels in female offspring of MI patients may be influenced by both genetic and environmental factors.

Finally, Garrison et al. (1979) reported additional observations of a previously described study conducted by Gordon, Castelli, Hjortland, Kannel, and Dawber (1977a).

These researchers observed that HDL cholesterol levels in both male and female offspring correlated more strongly with the HDL cholesterol levels of female MI patients than male MI patients. On the basis of these observations, the researchers suggested the possibility that the X chromosome may not be a contributing factor in determining HDL cholesterol levels in offspring, and that, in fact, HDL cholesterol levels in offspring may be related to intrauterine environment and subsequent environmental influence.

In summary, HDL cholesterol levels appear to be higher in females than in males and may offer a protective effect against the risk of MI in females. This may well be a reason for the low incidence and prevalence of MI in females. Also, HDL cholesterol levels appear to be influenced by both environmental and genetic factors.

Uric Acid

Recently, several researchers have investigated the characteristics of serum uric acid levels in females and males with MI, and the effect of these levels on risk of MI in females. Serum uric acid levels were observed to be significantly lower in females than in males with MI, in two cross sectional studies (Eisenberg, Ehrenfeld, Friedman, & Friedlander, 1981; Moulopoulos et al., 1987).

In an ongoing prospective study (Kannel, 1987), when data were analyzed using univariate analysis, serum uric

acid level was observed to b associated with a greater risk of MI in females than in males. Most of this association was explained, following multivariate analysis, by the high correlation of uric acid with other factors associated with this disease such as cholesterol, body weight, diuretic therapy, and blood pressure. Similarly, Cullen, Stenhouse, Wearne and Welborne (1983) found, in their prospective study of 840 males d 724 females, that uric acid was not a significant predictor of mortality from coronary heart disease when multivariate analysis was used to analyze several risk factors.

A strong correlation between serum uric acid level and diuretic therapy, and a possible correlation between disturbed purine metabolism and atherogenesis have been reported by Kannel (1987). This researcher suggested that these relationships be further investigated to clarify the role that uric acid may play in the risk of MI in females.

In summary, how serum uric acid may influence the risk of MI in females is not clear. It appears that the effect of serum uric acid on the risk of MI in females may be mediated through other factors associated with this disease. Further investigation of the role of such factors is warranted.

Cigarette Smoking

Twelve studies were located in which researchers had investigated the prevalence of smoking among males and

females with MI, and the effect of cigarette smoking on the risk of MI in females. The differences in prevalence of smoking and in the number of cigarettes smoked, between males and females with MI, were investigated in three of these studies (Carlson & Bottiger, 1985; Dick & Stone, 1978; Moulopoulos et al., 1987). The researchers of these three studies observed a significantly lower prevalence of smoking and a lower number of cigarettes smoked, among females with MI, when compared with males with MI. In contrast, in their cross-sectional study of 39 males and 12 females with MI, Wyndham, Seftal, Pilcher, and Baker (1987) observed the prevalence of cigarette smoking and the number of cigarettes smoked not to be significantly different among males and females with MI .

In three separate studies (Mann, Doll, Thorogood, Vessey, & Waters, 1976; Rosenberg et al., 1983; Willett, Hennekens, Bain, Rosner, & Speizer, 1981), relative risk of MI was observed to increase with the number of cigarettes smoked. The effect of smoking, in combination with other risk factors, was not addressed by the researchers of these studies. The observed risk scores of MI in females may, thus, be an inaccurate representation of the actual risk attributable to smoking.

The possibility of interaction between smoking and other risk factors and their effect on risk of MI in females has been the subject of numerous investigations. Slone et

al. (1978) examined the effect of the number of cigarettes smoked and of age on the risk of MI, in 55 females with MI and 843 female age matched controls. For females less than 45 years of age, who smoked more than 25 cigarettes/day, the relative risk of MI was calculated to be 25. For females 45 years or older, who smoked the same amount, the relative risk of MI was found to be 11.

The preceding study was extended by Rosenberg, Shapiro et al. (1980) and involved 318 female cases with MI and 1272 female age matched controls. Relative risk of nonfatal MI in females was measured for smoking alone and in combination with other factors such as obesity, diabetes, hyperlipidemia, hypertension, and angina. The relative risk of MI for smoking alone was calculated to be 9.7. In combination with one or more other factors, the relative risk was calculated to be 24. The relative risk score calculated for smoking alone was considerably less than the relative risk score calculated, in the preceding study, for females less than 45 years of age who smoked more than 25 cigarettes/day. This suggests that, while smoking alone appears to significantly increase the risk of MI in females, smoking may also interact with several other factors to further increase this risk.

The studies by Slone et al. (1978) and Rosenberg, Shapiro, Kaufman, Slone, Miettinen, and Stolley (1980) were further extended by Rosenberg, Kaufman, Helmrich, Miller,

Stolley, and Shapiro (1985) who investigated the interaction between smoking and age, cholesterol, hypertension, diabetes, OC use, and type A behavior in 555 females cases with nonfatal MI. Again, relative risk of MI was found to increase with the number of cigarettes smoked. Females less than 39 years of age, who smoked more than 25 cigarettes/day, with none or few of the other factors present, had the greatest relative risk of MI. A synergistic effect between smoking and OC use was observed. The smoking of more than 25 cigarettes/day was shown to be significantly interactive with higher total serum cholesterol and lower HDL cholesterol levels. No significant interaction was found between smoking and hypertension, diabetes, or type A behavior. Similar findings were also reported by Willett et al. (1987), in a 6 year prospective study of 119,404 females. Relative risk of MI was observed to increase with the number of cigarettes smoked and smoking was observed to interact additively with age, parental history of MI, and obesity, but not with hypertension and diabetes.

Bush and Comstock (1983) also observed a greater risk of MI in young females who smoked, in their 12 year prospective study of 23,572 females. In this study, the relative risk of MI was highest in the 25-44 year age group with a value of 3.6.

In summary, smoking appears to significantly increase

the risk of MI in females. Smoking appears to interact with other factors such as OC use, total cholesterol, and HDL cholesterol. Finally, this increased risk of MI in females who smoke appears to be most significant for younger females.

Alcohol

Four studies were uncovered in which researchers had investigated the effect of alcohol consumption on the risk of MI in females. Klatsky, Friedman, and Siegelaub (1979) investigated the interaction between alcohol, smoking, hypertension, and MI in 76 females and 332 males. The majority of the reported findings had only to do with the male sample. The only finding which was reported with regard to the female sample was that the prevalence of hypertension in the females increased significantly with amount of alcohol consumed. Figures for consumption of alcohol by the females were not provided by the researchers. This would be essential information for assessing the relationship, in females, between hypertension and alcohol consumption as well as demonstrating the distribution of the females by age in terms of the amount of alcohol consumed.

Rosenberg, Slone et al. (1981), who conducted a retrospective study of 513 female cases with MI and 918 female age matched controls, calculated the relative risk of MI with alcohol consumption, in females, to be .7. The researchers suggested that this apparent protective effect

of alcohol may be related to its effect of elevating HDL cholesterol levels. There were two strategies chosen by the researchers which may have jeopardized the validity of these results. First, data concerning alcohol consumption were gathered by a verbally administered questionnaire. Underreporting and underestimation of alcohol consumption may have occurred. If so, the resulting inaccurate measurement of alcohol consumption may have resulted in an erroneous estimation of relative risk. Second, HDL cholesterol levels were not measured in this study. Thus, the researchers' suggestion concerning the effect of alcohol on serum HDL cholesterol levels cannot be substantiated by the data collected.

A protective effect of alcohol consumption against the risk of MI in females has also been reported by Marmot (1984) and Scragg, Stewart, Jackson, and Beaglehole (1987). In the retrospective studies of these researchers, the relative risk of MI in females due to moderate alcohol consumption ranged from .6 to .9.

In summary, conclusions concerning the effect of alcohol on the risk of MI in females may be premature in light of the available evidence. However, it seems that moderate alcohol consumption may reduce the risk of MI in females. Future research in this area, with particular emphasis on alcohol consumption and possible interactions with other factors, is warranted.

Diabetes

Ten studies were located in which researchers had investigated the prevalence of diabetes in females and males with MI, and the effect of diabetes on the risk of MI in females. In three of these studies (Bjarveit, Foss, & Gjervig, 1983; Leitersdorf et al., 1986; Wilhelmsen et al., 1977), prevalence of diabetes in female MI patients was observed to be significantly higher than in male MI patients. In contrast, among nondiabetic patients, males were observed to have significantly higher fasting blood glucose levels than females, in a prospective study of 87 males and 83 females with MI (Donahue, Orchard, Becker, Kuller, & Drash, 1987).

Evidence of a greater effect of diabetes on risk of MI mortality in females, as compared with males, has been observed by several researchers. Kannel and McGee (1979) found, when controlling for blood pressure, age, cigarette smoking, and cholesterol levels, the relative risk of MI mortality in female diabetics was 3.3 compared with 1.7 for male diabetics, at the 20 year observation point of an ongoing prospective study. Further evidence, from the same prospective study, which supports these observations, was reported by Gordon, Castelli, Hjortland, Kannel, and Dawber (1977b).

In their study, Heyden, Heiss, Bartel, and Humes (1980) observed that the standardized coronary heart disease

mortality ratio for female diabetics was 2.8 compared with 1.3 for male diabetics, while controlling for cholesterol levels, triglycerides, blood pressure, weight, and smoking. Tansey, Opie, and Kennelly (1977) also observed significantly higher MI mortality rates among females diabetics than male diabetics. In the presence of obesity, the female MI mortality rate was eight times greater that of obese male diabetics.

In their seven year prospective study of 1358 females and 1089 males, Barrett-Conner and Wingard (1983) observed that the relative risk of MI for female diabetics was 3.4 when compared with female nondiabetics. The female diabetics had significantly higher levels of serum cholesterol and number of cigarettes smoked than the female nondiabetics.

Finally, Lapidus, Bengtsson, Blohme, Lindqvist and Nystrom (1985) examined the effect of borderline diabetes (mildly elevated serum glucose) and true diabetes (requiring oral hypoglycemic or insulin therapy) on the risk of MI, in a 12 year prospective study of 1352 females. The relative risk of MI in borderline diabetics was 8.1, while the relative risk of MI in true diabetics was 9.4. It is possible, the researchers suggested, that even mildly elevated serum glucose may significantly increase the risk of MI in females.

In summary, when compared with males with MI, the

prevalence of diabetes, risk of MI mortality, and risk of MI, appear to be higher in females with MI. This increased risk may also be applicable to borderline female diabetics not requiring oral hypoglycemic or insulin therapy.

Hypertension

The prevalence of hypertension and the effect of hypertension on relative risk of MI in females has been under considerable investigation. The prevalence of hypertension has been repeatedly observed to be greater in females with MI than in males with MI and in females free of this disease. A greater prevalence of hypertension in females than in males prior to MI occurrence was observed in two cross sectional studies (Eisenberg, Ehrenfeld, Friedman, & Friedlander, 1981; Johansson et al., 1984).

A possible explanation for this greater prevalence of hypertension in females was put forward by Bjarveit, Foss, and Gjervig (1983). In their prospective study of 57,859 males and females, these researchers observed that significantly more females than males sought and were under routine medical management prior to the onset of cardiovascular disease. Consequently, diagnosis and treatment of hypertension were more readily available to females. The researchers suggested that the prevalence of hypertension in males may be underestimated due to lesser exposure to routine medical management. However, males and females with cardiovascular disease have been observed, by

other researchers, to be similar with respect to prevalence of hypertension (Yano et al., 1987). In contrast to these observations, in the Canadian population, Millar (1985) estimated the prevalence of hypertension to be greater in males (21.8%) than in females (12.6%). Also, among both males and females, the prevalence of hypertension was reported by Millar to increase with age.

In comparison with females free of cardiovascular disease, females with MI were observed to have a greater prevalence of hypertension in two retrospective studies (Dick & Stone, 1978; Krueger et al., 1981) and one cross sectional study (Arthes & Masi, 1976).

Conflicting results have been published with regard to hypertension and the risk of MI in females. While controlling for age and cigarette smoking, a relative risk of 2.0 was observed for hypertensive females, in a retrospective study of 255 female cases with MI and 802 controls (Rosenberg et al., 1983). A relative risk of 4.7 (with age adjusted analysis) and 6.5 (with multivariate analysis) was observed for hypertensive females, in a retrospective study of 168 female cases with MI and 251 female controls (LaVecchia, Franceschi, Decarli, Pampallona, & Tognoni, 1987). Also, untreated hypertension was observed to be a significant predictor of MI in females, in two prospective studies (Carlson & Bottiger, 1985; Lapidus, 1985).

In contrast to the results of these studies, hypertension was not observed to be a significant predictor of MI in females, in a prospective study of 840 males and 724 females (Cullen, Stenhouse, Wearne, & Welborne, 1983) and in a cross sectional study of 159 males and 80 females (Yano et al., 1987), when multivariate analysis was used to assess the effect of several risk factors.

In summary, reports concerning the prevalence of hypertension and the effect of hypertension on the risk of MI in females are conflicting. The prevalence of hypertension in males and females and the effect of hypertension on the risk of MI in females require further investigation.

Family History of MI

Five studies were located in which researchers had investigated the prevalence of a positive family history of MI among males and females with MI, and the effect of a positive family history of MI on the risk of MI in females. The prevalence of a positive family history of MI was observed to be similar among males and females with MI, in a cross sectional study of 151 males and 49 females (Eisenberg, Ehrenfeld, Friedman, & Friedlander, 1981). In contrast, the prevalence of a positive family history of MI was observed to be significantly greater among females than among males with MI, in a prospective study of 1774 males and 2240 females (Khaw & Barrett-Conner, 1986).

Findings concerning the risk of MI in female relatives of MI patients are conflicting. Colditz et al., (1986) observed a moderate increase in the relative risk of MI in females whose parents' first MI occurred before 60 years of age. This increased risk remained, on controlling for hypertension, diabetes, cholesterol, smoking, obesity, and OC use.

In a nine year prospective study of 2240 females and 1774 males, Khaw and Barrett-Conner (1985) did not observe any appreciable increase in the risk of MI in females with a positive family history of MI. A positive family history added slightly to the risk of MI if one or more other factors, such as diabetes or smoking, were present. Conversely, a positive family history of MI offered a slight protective effect against the risk of MI in those females who were current NCE users. Similarly, LaVecchia, Franceschi, Decarli, Pampallona and Tognoni (1987) did not observe a significant effect, of positive first degree relative history of MI, on the age adjusted risk of MI in females. However, a positive history, of more than one first degree relative with MI, resulted in an age adjusted relative risk of 3.2.

Finally, ten-Kate, Boman, Daiger and Motulsky (1984) conducted a retrospective study of 126 females, all wives of MI patients, and 126 age matched controls. The incidence of MI in the first degree relatives of both the MI patients and

their wives was significantly greater than in the first degree relatives of the controls. Since the offspring of the MI patients and their wives were not included in the analysis, the first degree relatives included in the analysis were not consanguineous. The researchers suggested that the observed increased risk of MI in nonconsanguine females may be related to assortive mating in which marriage partners choose mates with similar risk factor patterns and lifestyles. How this assortive mating contributed to the risk of MI in the offspring was not investigated. If it had, it would have allowed comparison of risk of MI between the consanguine and nonconsanguine relatives.

In summary, how a positive family history of MI affects the risk of MI in female offspring is not clear. The study results in this regard are conflicting, making an assessment of what is known in this area premature. Further research which investigates the effect of a positive family history on the risk of MI in female offspring is warranted.

Obesity

Ten studies were located in which researchers had investigated the prevalence of obesity or the effect of obesity on risk of MI in females. Canadian population estimates of obesity, based on data from the Canada Fitness Survey (Canada Fitness Survey Fitness and Amateur Sport - Women's Program, & Fitness Canada 1984; Millar, 1986), revealed the prevalence of obesity, among Canadians, to be

greater in females (12%) than in males (9%); the highest prevalence occurring in females, age 60-69 years (22.0%). Prevalence of obesity was also estimated to increase with age.

With respect to prevalence of obesity among males and females with cardiovascular disease and MI, no significant differences, between the genders, were detected in several studies conducted recently (Hubert, Feinleib, McNamara, & Castelli, 1983; Johansson et al., 1984; Krueger et al., 1981; Yano et al., 1987). Also, in these studies, prevalence of obesity, among males and females, was observed to be greater than that revealed by the population estimates of the Canada Fitness Survey.

With respect to the effect of obesity on the risk of MI in females, no direct relationships between weight index, body weight change, or sum of subscapular skinfolds and MI in females were observed, in a prospective study of 1462 females (Noppa, 1980; Noppa, Bengtsson, Wedel, & Wilhelmsen, 1980). Multivariate analyses revealed no direct relationship between body mass index and MI in females, in a retrospective study of 168 females with MI and 251 female controls (LaVecchia, Franceschi, Decarli, & Tognoni, 1987) and in a prospective study of 3786 males and 4120 females (Tuomilehto et al., 1987).

In contrast to these results, distribution of adipose tissue and body mass index were observed to be significantly

and independently associated with an increased risk of MI in females, in a 12 year prospective study of 1462 females (Lapidus et al., 1984). Similarly, Metropolitan Relative Weight was observed to be an independent predictor of MI in females in the absence of hypertension, hypercholesterolemia, smoking, glucose intolerance, and left ventricular hypertrophy in the ongoing Framingham cohort study (Hubert, Feinleib, McNamara, & Castelli, 1983).

In summary, the results of the studies which were reviewed are conflicting. Several different measures of obesity and methods of analysis were employed by the researchers. This makes comparisons across studies difficult. It seems that adipose distribution, body mass index, and Metropolitan Relative Weight may influence the risk of MI in females. The question of which measure of obesity most strongly correlates with MI in females requires further investigation.

Physical Fitness

Three studies were located in which researchers had investigated the effect of varying levels of physical fitness on risk of MI in females.

Salonen, Puska, and Tuomilehto (1982) observed the relative risk of MI in females with 'low' physical acti it at work to be 2.4. No significant increase in risk was observed for females with low physical activity during leisure. Data concerning physical activity were collected

through the use of only one item on a questionnaire which did not address type of occupation, regularity of working hours, amount of leisure time, and activity during leisure time. Such factors may have been significant to assess with regard to their effect on risk of MI.

In a retrospective study of 439 males with MI and 1017 male controls and of 111 females with MI and 569 female controls, regular exercise (i.e., at least once weekly) was observed to provide a protective effect against the risk of MI in both males and females (Scragg, Stewart, Jackson, & Beaglehole, 1987). A dose-response relationship was also detected by the researchers between duration of regular exercise and risk of MI in males and females.

In their descriptive study of 1700 females who referred themselves for physical fitness evaluation, Gibbons, Blair, Cooper, and Smith (1983) observed that level of physical fitness affected the prevalence of other factors which contribute to the risk of MI in females. Females whose physical fitness was classified as 'very poor' had significantly higher levels of total serum cholesterol, triglycerides, blood pressure, and cigarette smoking and significantly lower HDL cholesterol levels than those females whose physical fitness was classified as 'very good'.

In summary, it seems that level of physical fitness and duration of regular exercise may influence the risk of MI in

females as well as the prevalence of other risk factors. Further research in which the effects of type, duration, and frequency of physical activity during work and leisure hours are investigated is warranted.

Psychosocial

The effect of various psychosocial factors, such as situational stress, behavior type, and previous psychiatric illness, on the risk of MI in females has been under considerable investigation. Four separate reports (Haynes & Feinleib, 1980; Haynes, Feinleib, & Kannel, 1980; Haynes, Feinleib, Levine, Scotch, & Kannel, 1978; Haynes, Levine, Scotch, Feinleib, & Kannel, 1978) contain observations of a single prospective study in which researchers investigated the relationships among smoking, cholesterol, hypertension, behavior type, situational stress, somatic strain, and MI, in 1006 females and 8806 males. The following is a summary of the study results which pertain to the effect of various psychosocial factors on the risk of MI in females. The researchers observed that the females with MI were more likely than their male counterparts to be emotionally labile and experience tension and symptoms of anger. When smoking, cholesterol, and hypertension were controlled for, these behaviors were found to be significantly correlated with coronary heart disease in females. Also, females with these emotional characteristics were observed to have significantly higher levels of serum cholesterol, diastolic

blood pressure, and cigarette smoking than their male counterparts.

Three reports (Hallstrom, Lapidus, Bengtsson, & Edstrom, 1986; Lapidus, 1985; Lapidus & Bengtsson, 1986) contain observations of a 12 year prospective study of 1462 females. Significant inverse correlations were observed between the risk of MI in females and the presence and severity of guilt feelings and 'neurotic self-assertiveness'. The researchers observed no significant influence of type A behavior on the risk of MI in these females.

Talbott, Kuller, Detre, and Perper (1977) and Talbott, Kuller, Perper, and Murphy (1981) conducted two similar retrospective studies in which they investigated the effect of previous psychiatric illness on the risk of MI in females. In both studies, the risk of MI was observed to increase significantly in the presence of previous psychiatric illness and treatment. In the first study, the prevalence of hypertension, smoking, and alcohol abuse was found to be significantly higher among the female cases than among the female controls. No such difference was observed by the researchers of the second study.

Finally, Abramov (1976) investigated the effect of sexual satisfaction on risk of MI in 100 female cases with MI and 100 age matched controls. Sexual frigidity and dissatisfaction were described by 65% of the cases compared

with 24% of the controls. Only 8% of the women with MI attributed their dissatisfaction to their underlying pathology and symptomatology. The cases and controls did not differ with respect to marital status: married, widowed, or divorced,

In summary, the risk of MI in females appears to increase inversely with certain emotional characteristics such as guilt feelings and 'neurotic self assertiveness'. It seems that risk of MI may also increase as emotional lability, tension, anger, and sexual dissatisfaction increase.

In many of these studies, serious design flaws are evident which may have jeopardized the validity of their findings. Many of the psychosocial factors investigated in these studies are complex constructs which require instruments of proven reliability and validity for accurate measurement. In only one study (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978) was the reliability coefficients of the instruments used for data collection published. The reliability coefficients of the instruments used for data collection in the remaining studies were either not published or unknown. In none of the studies was the operational definitions of these complex constructs published. Both these factors, in combination, increase the risk of the researchers having measured something other than the factors of interest.

In many of the studies, several instruments were used simultaneously or with considerable revision without subsequent testing. The use of several instruments simultaneously may have resulted in significant interaction between the instruments. The use of revised instruments without subsequent testing makes it impossible to assess what the instruments were measuring.

Certain psychosocial factors may contribute significantly to the risk of MI in females and warrant further research. Because of the complexity of these various constructs, future research should involve instruments of proven reliability and validity or the testing of instruments for their reliability and validity. Furthermore, operational definitions should be clearly worded, and instrument interaction should be kept to a minimum or eliminated.

Socioeconomic

The effects of type of occupation, educational level, and marital status on the risk of MI in females have been investigated by several researchers.

The following is a summary of the observations, reported by the researchers of four previously described studies (Haynes & Feinleib, 1980; Haynes et al., 1980; Haynes, Feinleib, Levine, Scotch, & Kannel, 1978; Haynes, Levine, Scotch, Feinleib, & Kannel, 1978) which pertain to occupation and risk of MI in females. Females who were blue

collar workers and who experienced little job mobility, substantial daily tension, and a nonsupportive 'boss', were found to have significantly higher total serum cholesterol levels and mean diastolic blood pressure than males with these same characteristics. Females employed in clerical related occupations were found to have the highest incidence of coronary heart disease among all occupations and this incidence was more than twice that of male clerical workers. No significant differences in the prevalence of hypertension, cigarette smoking and glucose intolerance were observed between working and nonworking females.

In a cross sectional study of 1041 females (Hazuda et al., 1986), nonemployed females were observed to have a greater risk of MI than their employed counterparts. While the number of cigarettes smoked was observed to be significantly higher in employed females, HDL cholesterol levels and alcohol consumption were observed to be significantly lower in nonemployed females. Both HDL cholesterol and alcohol consumption, discussed previously in this review, have been observed to offer a protective effect against the risk of MI in females.

The effect of occupation on risk of MI in females was also investigated by Alfredsson, Spetz, and Theorell (1985) and Theorell (1986). The females in these studies who described their work as being hectic, monotonous, and consisting of irregular working hours were found to have a

significantly greater risk of having a MI than those females who did not describe their work in terms of these characteristics. Similarly, Orth-Gomer, Hamsten, Perski, Theorell, and DeFaire (1986) reported that females with low education who described their work as hectic, demanding, and monotonous were at greater risk than those females with low education, who did not describe their work using these terms, or females with high education.

The researchers of three previously described studies (Hallstrom et al., 1986; Lapidus, 1985; Lapidus et al., 1986) also investigated the effects of various socioeconomic factors on the risk of MI in females in their studies. Significant inverse correlations were observed between the risk of MI in females and the socioeconomic status of the husband's profession and the level of education of the females themselves. No significant correlations were observed between risk of MI and marital status, number of children, and the socioeconomic status of the females' professions. Other researchers have reported similar findings. Using population estimates derived from the Canada Health Survey and Canada Fitness Survey, level of education in females was observed by Millar and Wigle (1986) to be inversely associated with the prevalence of several risk factors associated with MI: number of cigarettes smoked, obesity, hypertension, and diabetes.

Additional evidence which supports the above mentioned

findings related to an inverse relationship between level of education and risk of MI in females was reported by Szklo, Tonascia, and Gordis (1976). The relative risk of MI in females with significantly less education than that of their husbands was found to be 9.7, when hypertension, angina, and diabetes were controlled. No significant increased risk of MI was observed for females whose education was comparable with that of their husbands.

In summary, the risk of MI in females appears to increase in the presence of certain work related characteristics such as lack of job mobility, monotony, irregular working hours, and clerical work. Risk of MI also appears to increase inversely with the husbands' work status and the educational level of the females themselves.

Other

Five studies were located in which researchers investigated the effects of coffee consumption, aspirin use, erythrocyte sedimentation rate, days of the week, and seasonal variation on the risk of MI in females.

Rosenberg, Slone, Shapiro, Kaufman, Stolley, and Miettinen (1980b) observed no significant contribution of coffee consumption to the risk of MI in females. The amount of coffee consumed was actually found to be higher in the control group. Rosenberg, Slone, Shapiro, Kaufman, Miettinen, and Stolley (1982) did not observe any effect of aspirin use on the risk of MI, protective or otherwise, in a

retrospective study of 554 females with MI and 909 female controls. Rafnsson and Bengtsson (1982) observed that erythrocyte sedimentation rate did not influence the risk of MI, in their 12 year prospective study of 1295 females.

Massing and Angermeyer (1985) summarized mortality statistics for the years 1968 - 1977 and found no association between the days of the week and MI mortality in females. The researchers were testing a hypothesis put forth by Holmes and Rahe's stress model which suggests that Saturdays and Mondays may be related to the occurrence of MI.

Finally, Yosuf, Kolar, Bhatnager, Huduk, and Smid (1986) did not observe any association between seasonal variation (i.e., winter or spring) and incidence of MI in females. An association was observed between seasonal variation and incidence of MI in males with male MI incidence being significantly higher in the summer months.

Mortality

Mortality rate in females following MI has been investigated by many researchers. In summarizing the results of the National Health Discharge Survey which pertain to mortality following MI, Roig et al. (1987) reported that MI mortality rates were consistently higher in females than in males for all age groups below 70 years of age, during 1973 to 1984.

Puletti, Sunseri, Curione, Erba, and Borgia (1984)

found that, in the presence of hypertension, diabetes, and previous MI, the mortality rate for the 106 females in their study was consistently more than twice that for the 575 males: 38% for the females compared with 18% for the males. Similar findings were reported by Kannel, Sorlie, and McNamara (1979). In their prospective study of 81 females and 231 males diagnosed with MI, the incidence of mortality within 30 days following a MI was found to be 28% for the females compared with 16% for the males. The percentage of each sex suffering a second MI within 5 years following a first MI was 39% for the females and 13% for the males. Within 10 years following the first MI, the percentage of those suffering a second MI was 10% and 3% for females and males respectively. Similarly, Madsen, Thomsen, Sorensen, Kjeldgaard, and Kromann-Andersen (1987) observed the risk of future cardiac complications following an initial MI to be 2.2 times greater in females than in males, in a prospective study of 59 females and 216 males with MI. This increased risk remained, on controlling for age, cardiac history, drug therapy, and diabetes.

In their study, Tofler et al. (1987) found the four year cumulative mortality rate following a first MI to be 36% for females and 21% for males. The females in this study had significantly more 'preinfarction characteristics' than their male counterparts. These preinfarction characteristics included hypertension, diabetes, family

history of MI, and congestive heart failure.

In contrast to the results of these studies, Olmsted, Groden, and Silverman (1987) observed no significant difference between female and male mortality rate following MI, in a prospective study of 57 males and 56 females with MI. Similarly, Saito et al. (1987) observed males and females to be similar with respect to the incidence of cardiac death, recurrent MI, and other cardiac events following MI, in a prospective study of 447 males and 116 females with MI.

Only one study, conducted by Pohjola, Siltanen, and Romo (1980), was uncovered in which female mortality risk was found to be less than that for males following MI. The five year incidence of mortality for females was 21% compared with 32% for the males of this study.

Finally, in their retrospective study of 338 females diagnosed with MI, Krueger et al. (1981) found that the prevalence of hyperlipidemia, diabetes, and hypertension was higher in those females with a previous history of MI than in those females whose first MI was fatal.

A factor which has been reported to be strongly predictive of mortality following MI is infarct size. In several prospective studies which involved homogeneous samples of males and females, mortality rate was found to increase as the size of myocardial necrosis increased (Bleifield, Mathey, Hanrath, Buss, & Effert, 1976; Geltman

et al., 1979; Lee & Goldman, 1986; Pitt, 1981; Shell & Sobel, 1976; Sobel, Bresnahan, Shell, & Yoder, 1972). Further, infarct size has been observed to vary with the location of infarct. In a prospective study of 33 males and females with MI (Sobel et al., 1972), anterior infarcts were found to be significantly greater in size than inferior infarcts. No studies were located in which males and females were compared with respect to infarct size or location.

Recently, many researchers have attempted to identify factors which may be useful in predicting infarct size. Creatine phosphokinase - multiband (CPK-MB), lactic dehydrogenase (LDH) and its first subfraction (LD1) are three such possible factors. In several prospective studies of homogeneous samples of males and females with MI, strong positive correlations were observed between serum CPK-MB, LDH, and LD1 levels and infarct size (Grande, Hansen, Christiansen, & Naestoft, 1982; Ryan et al., 1981; Shell & Sobel, 1976; Shibata et al., 1985; Smith et al., 1983). However, these enzyme serum levels were influenced by more than the extent of myocardial necrosis. Coronary artery reperfusion, infarct extension, streptokinase and tissue plasminogen activator therapy, and direct current countershock were reported to have influenced the CPK-MB, LDH, and LD1 levels in serum. These enzymes have, therefore, been recommended as qualitative estimates of

infarct size (Lee & Goldman, 1986). No studies were located in which males and females were compared in this regard.

Because serum levels of these enzymes appear to be influenced by more than infarct size, their reliability as predictors of mortality following MI is compromised. Results are conflicting with respect to CPK-MB, LDH, and LDH enzymes as predictors of mortality following MI. In a prospective study of a homogeneous sample of 173 males and females with MI, peak CPK level was found to be a significant predictor of MI mortality (Geltman et al., 1979). In contrast, CPK level was found not to be a significant predictor of either short term or long term mortality, in two prospective studies of homogeneous samples of males and females with MI (Madsen, Gilpin, & Henning, 1984; Madsen, Gilpin, Henning, Ahnve et al., 1984). No studies were located in which males and females with MI were compared in this regard.

In summary, females may be at greater risk of mortality following MI than males, although evidence that this risk may be equivalent between males and females has been reported. It seems that risk of female mortality may increase as the number of prevalent risk factors prior to MI decreases. Also, it appears that infarct size and serum levels of certain enzymes may be significant predictors of mortality following MI. How males and females compare in this regard is not known. Further research is warranted in

which gender differences with respect to indicators of mortality following MI, such as infarct size, serum enzyme levels, prevalence of risk factors, and treatment strategies, are investigated.

Summary

The following is a brief summary of what seems to be the case concerning the risk factors which predispose females to suffer a MI:

1) Females appear to demonstrate an unique angiographic profile evidenced by an apparent low prevalence of atherosclerotic/thrombotic events and a relatively high prevalence of unrecognized MI in comparison with males.

2) Variations in prognosis for different symptomatology suggests an unique etiology of actual infarction in females.

3) Incidence and prevalence of MI and secular trends in rates appear to vary with geographical location.

4) The effect of OC use on relative risk of MI in females appears to be minimal. OC use and cigarette smoking appear to interact synergistically, particularly in females over the age of 35 years.

5) The effect of NCE use on risk of MI in females appears to be minimal.

6) There is conflicting evidence concerning the effects of age of onset and type of menopause on risk of MI in females.

7) HDL cholesterol appears to exert a protective effect against the risk of MI in females and HDL cholesterol levels may be influenced by both genetic and environmental factors.

8) Female diabetics appear to have a significantly higher risk of MI than male diabetics. This increased risk may hold true for borderline diabetics as well.

9) The risk of MI in females may increase in the presence of certain employment related characteristics such as lack of job mobility and irregular working hours.

10) The risk of MI in females may vary inversely with feelings of guilt, and 'neurotic self assertiveness', and the husband's socioeconomic status.

11) Generalizations concerning what is known about the relationships between MI in females and alcohol, serum uric acid level, hypertension, family history of MI, obesity, and physical activity may be premature in light of the available evidence.

The above summary of what is indicated by the literature is replete with opportunities for future research, particularly in light of some of the serious design weaknesses of many of the studies which were reviewed. A recent publication entitled, "Report of the Workshop on the Directions for Research in the Epidemiology of Ischemic Heart Disease in Canada" (1985), lists several research priorities: fatal versus nonfatal MI; regional

differences; sex, ethnic, and minority variations; psychosocial variables; occupational and environmental factors; and primary and secondary prevention. Many of the research priorities suggested throughout this review concur with those of this publication.

CHAPTER III

Method

Using a cross sectional study design with a prospective component, a sample of male and female MI patients was identified through the hospital records. Using the hospital records of this sample, males and females were compared in terms of certain risk factors identified within the literature as being associated with changes in the probability of occurrence of MI. For heuristic purposes, they were also compared in terms of mortality and some factors which may influence prognosis following the occurrence of MI. In such comparisons, nonparametric and parametric statistics were used to determine if significant differences exist between the genders. Data which were both nonstratified and stratified by age were analyzed.

Design

A cross sectional design with a prospective component was used in this study. In a cross sectional study in epidemiology, both exposure and disease are measured at a point in time (Kelsey, Thompson, & Evans, 1986, p. 187). With such a study design, one usually cannot determine if exposure is antecedent to or a consequence of a disease. This difficulty was minimized in this study, by limiting the risk factors investigated to those which have been identified in the literature as being antecedent to the occurrence of MI.

Sample

Sample Identification

Subjects in the study sample were identified from the 1986 and 1987 hospital records of patients who had been discharged from the coronary care unit (CCU) of a 1300 bed urban teaching hospital in western Canada, with the diagnosis of acute MI. A preliminary investigation revealed that the coding procedure for hospital records employed by this hospital followed the International Classification of Diseases - Ninth Revision criteria for coding of diagnoses. In this classification scheme, acute MI is coded as number 410. All cases with the discharge diagnosis coded as 410 were identified by computer and, subsequently, the hospital records of these cases were retrieved and examined to determine if the criteria for selection were met.

Criteria for Selection of Sample

A random sample was drawn from the population of all patients discharged from the CCU with the diagnosis of acute MI during the years 1986 and 1987. Patients were considered suitable for the study if they met the following criteria:

1. Discharged from the CCU with the diagnosis of acute MI.
2. over 18 years of age.

Sample Size and Sampling

The method described by Schlesselman (1982) for determining sample size was used in this study. On page 62,

Table 1 contains examples of the necessary female sample sizes (n) for various risk factors, when alpha=.05, beta=.20, and power=.80. The final column of Table 1 contains the actual power (calculated following the method described by Schlesselman (1982)) with respect to the sample size of females (i. e., 147 females) of this study. With a sample size of 147 females, the actual study power often exceeds a power of .80 for those factors contained within Table 1.

To derive these sample size computations, Po, or the proportion of females exposed to a given risk factor among female controls in the target population, and the relative risk (R) associated with a given risk factor must be known. Not all risk factors are included in this Table as these values are either unknown or could not be located within the literature.

Table 1

Calculation of Sample Size

Factor	Data Source	Po	R	n	Study Power
smoking	Rosenberg et al. (1985)	.54	2.7	74	.97
diabetes	Lapidus, Bengtsson, Blohme, et al. (1985)	.01	9.4	116	.89
obesity	Hubert et al. (1983)	.66	2.0	172	.78
family Hx	Colditz et al (1986)	.26	2.1	137	.82

A preliminary investigation revealed that, between July 1, 1986 and June 30, 1987, 79 females and 246 males were discharged from the CCU with the diagnosis of acute MI. In assuming that the numbers of male and female admissions to the CCU had been relatively constant over the past few years, it was thought that a time span of two years would likely yield approximately 160 female cases and 500 male cases of acute MI. Since it was calculated that a sample size of approximately 160 females and 160 males would satisfy the number of candidates required for the risk factors outlined in Table 1 (for the study to have a power of .80), a decision was made to use the entire population of female cases for the years 1986 and 1987, and to obtain an equal number of male cases by applying simple random sampling techniques to the available population of males, for the same time period.

It was found that, for this two year period, 161 females and 477 males were discharged from the CCU with the diagnosis of MI. All the females and a random sample of 160 males were considered for inclusion in the study. The hospital records of 147 of the 161 females and 153 of the 160 males were located within the medical records. The records of the remaining cases were missing for various reasons: no written records were available for some cases and, in other cases, the records were being used for legal, research, and treatment purposes.

Using the male sample size of this study, the smallest detectable relative risk was calculated for various values of Po following the method described by Schlesselman (1982). For these computations, alpha=.05, beta=.20, and n=153. On page 64, Table 2 contains the smallest detectable relative risk for various values of Po for the male sample of this study.

Table 2

Smallest Detectable Relative Risk for Male Sample

Po	R
.05	3.18
.10	2.47
.20	2.06
.30	1.93
.40	1.89
.50	1.91
.60	1.96

The computations contained in Table 2 should be interpretted in the following manner. With a sample size of 153 males, the column labelled R (relative risk) represents the smallest relative risk which could be detected for a given value of Po. If the actual relative risk is less than the value calculated for a given value of Po, the male sample is of insufficient size to detect that relative risk.

Setting

The study was conducted in a 1300 bed urban teaching

hospital in western Canada. The hospital is an acute care facility which serves as a major referral centre for this region of Canada. In this hospital, when patients are admitted with a preliminary diagnosis of acute MI, they are treated in the CCU and, barring any complications, are typically transferred to a coronary care step down-uni within 4 to 5 days post admission.

Data Collection Procedures

Data pertaining to the following factors, which have been identified in the literature as influencing the probability of occurrence of MI, were collected: age, marital status, employment status, family history of MI, smoking, obesity, diabetes, history of cardiovascular disease, hypertension, and serum uric acid level.

For the purpose of enhancing the comparison between males and females, data pertaining to in hospital mortality and to the following factors, which do not influence the probability of occurrence of MI but may be significant in influencing prognosis, were also collected: type of MI, post MI complications, creatine phosphokinase - multi band (CPK-MB), lactic dehydrogenase (LDH), and lactic dehydrogenase subfraction one (LD1) serum levels.

Once potential cases were identified within the hospital records, their individual records were examined. If the selection criteria were met, pertinent data from the cases' records were recorded. Data were organized by gender

to allow cross gender comparisons during data analysis.

The data were recorded relative to the following routine diagnostic procedure which is carried out in the study setting, whenever any patient is admitted with a preliminary diagnosis of acute MI. First, the patient is assessed by the physician in the emergency department. The physician records the patient's medical history as well as the history of events leading up to the admission of the patient. The physician then conducts a general systems physical assessment and records the observations made. Urgent treatment, such as oxygen and medication administration, is initiated within the emergency department. If acute MI is the preliminary diagnosis, the patient is then transferred to the CCU for further care. Upon admission to the CCU, additional histories and general systems physical assessments are performed by the admitting nurse and the cardiology resident or consultant. Data for the following variables were obtained from the observations made and recorded up to this point: age, marital status, employment status, family history of MI, smoking, obesity, hypertension, history of cardiovascular disease, and diabetes.

Once admitted to the CCU, each patient undergoes a series of diagnostic tests. These tests are conducted at specified times and are identical for each patient. These tests include: ECG done once a day for three days starti

with the day of admission; serum CPK enzyme levels drawn at time of admission and every 8 hours thereafter, for a total of 6 levels; LDH and LD1 serum levels drawn at time of admission and every 24 hours thereafter, for a total of three levels; electrolytes, urea, creatinine, serum osmolality, liver function profile, uric acid level, and complete blood count drawn at time of admission; portable chest x-ray and urinalysis done following admission. Data for the following variables were collected from the recorded results of these tests: LDH, LD1, and CPK-MB serum levels. For the purpose of data analysis, serum levels were grouped according to the sequence in which the levels were drawn following admission to the CCU.

While in the CCU, the patient is closely monitored by the nursing and medical staff until he or she is considered stable enough to be transferred to the step down unit. The nursing and medical staff regularly record observations in the patient's chart which pertain to the patient's progress, response to treatment, and development of complications. Data for the following variables were obtained from these recorded observations: type of MI, post-event complications, and in-hospital mortality.

Data regarding total serum triglycerides and cholesterol levels and HDL and LDL serum levels were also intended to be collected in this study. However, they were not collected, since an examination of the hospital records

indicated that only 16.3% of the males and 8.8% of the females in the study sample had had these blood tests performed. These percentages represented 25 males and 13 females of the total sample. A decision was made not to collect such data since so few cases made statistical analysis both unreliable and impractical.

Data Analysis

The following factors were measured at the nominal (categorical) level and analyzed for equivalence between males and females using chi-square analysis: marital status, employment status, family history of MI, smoking, obesity, hypertension, history of cardiovascular disease, diabetes, type of MI, post [illegible]mplications, and in-hospital mortality.

The following factors were measured at the ratio (continuous) level and analyzed for differences between males and females using the t test for independent groups: age and uric acid, LDH, LD1, and CPK-MB serum levels. Pearson's product-moment correlation and linear regression were calculated for uric acid, LDH, LD1, and CPK-MB with respect to age within each gender.

Descriptive statistics including means, standard deviations, variances, and minimum and maximum values were calculated for the factors measured at the ratio level. Frequency counts and percentages were calculated for the factors measured at the nominal level.

The aforementioned statistical procedures were repeated after both groups had been stratified into ten year age groups (i.e., 20-29, 30-39, 40-49, 50-59, 60-69, 70-79, 80-89). Data were analyzed using the Statistical Package for the Social Sciences (SPSS Inc., 1983).

In the analysis, the following units of measure of each factor were used:

age: complete solar years from date of birth;

marital status: single, common law, married, divorced widowed;

employment status: employed/not employed;

positive history of MI: present if first degree relative had MI/not present if no history of first degree relative having had MI;

smoking: never smoked, past smoker, current smoker;

obesity: present if determined subjectively by admitting physician or nurse/not present if no subjective determination by admitting physician or nurse;

hypertension: present if being treated with antihypertensive medications at time of admission/not present if not being treated with antihypertensive medication at time of admission;

diabetes: present if being treated with diet, oral hypoglycemics, or insulin at time of admission/not

present if not being treated with diet, oral hypoglycemics, or insulin at time of admission;

history of cardiovascular disease: no history or history of cardiovascular disease: angina, previous MI, previous cardiac surgery, cerebral vascular accident, and congestive heart failure;

serum uric acid level: measured in Umol/l;

type of MI: based on ECG diagnosis and includes anterior, anterior-lateral, anterior-septal, inferior, posterior, and subendocardial MI;

complication following MI: no complication or complication following MI:intubation, cardiogenic shock, cardiac arrest, and anoxic brain insult;

in hospital mortality: vital status at discharge from hospital;

CPK-MB serum level: measured in IU/ml;

LDH serum level: measured in IU/ml; and

LD1 serum level: measured in IU/ml.

Ethical Considerations

Ethical clearance was obtained from the Faculty of Nursing, University of Alberta, and from the Research Committees of the hospital in which the study was conducted.

Confidentiality and anonymity during data collection and in the final report were maintained by the use of several measures. First, no identifying information of individual cases was recorded. Data extracted from each

chart were coded in such a way that it would be impossible to trace the data to individual cases. Second, the data were available only to the researcher and, when necessary, the researcher's thesis committee. Third, upon completion of the final report, the collected data were destroyed.

Limitations of the Study

The study findings presented in the next chapter must be considered in light of the limitations of the study. Six limitations have been identified.

First, since the hospital records examined in this study were not kept for the purposes of research, the data contained within them were unstandardized. Also, the validity and reliability of the data are questionable, since the instruments used for collecting the data recorded within the medical records have unknown validity and reliability.

Second, the hospital records were incomplete in that some of the data necessary for the study were missing, a recognized occurrence in cross sectional research (Levy & Lemeshow, 1980; Mausner & Kramer, 1985). There were some missing data for all of the factors measured in this study. However, the percentage of missing data was small, ranging from 0.0% to 5.4% of the total data collected for each factor.

Third, many of the factors investigated in this study were measured using crude categories of measurement. This reduced measurement of these factors, which could otherwise

have been measured at the interval or ratio level, using different research designs, to the nominal level of measurement.

Fourth, prevalence-incidence bias, also known as Neyman bias, may have distorted the statistical analysis of the gender comparisons of this study. The sample in this study was comprised of prevalent cases of MI. Incident cases who suffered unrecognized or fatal MI prior to or while in hospital were not included in this sample. The risk factors measured in these prevalent cases may have been indices of protective rather than putative exposures since data from fatal and unrecognized incident cases of MI were not included (Sackett, 1979; Schlesselman, 1982).

Fifty-six [illegible] information of the cases occurred at the stratified stage of analysis. Several age strata for most factors included only a few cases and statistical significance may have been influenced by the small samples of cases within these age strata (Schlesselman, 1982).

CHAPTER IV

Findings and Discussion

The findings and discussion of this study are presented in the following format. The findings related to each factor measured in this study are presented, in turn, and in terms of nonstratified and stratified findings. Each presentation of findings is followed by a discussion of the findings. Complete nonstratified and stratified descriptive statistics and the results of statistical analyses for each factor are contained within separate appendices and are referred to throughout the text. Appendix A contains descriptions of how the data are presented and statistical procedures used during analysis.

Only the findings which pertain to the age groups between 40 and 89 years (i. e., the 40-49, 50-59, 60-69, 70-79, 80-89 year age groups) are presented and discussed for the following reason. Stratification of the sample by age revealed that there was only 1 female in each of the 20-29, 30-39, 90-99 year age groups. The numbers of males in these age groups were 1, 11, and 3 respectively. However, descriptive statistics and the results of statistical analyses of these age groups are included in the appendices, for descriptive purposes.

For many of the factors measured, statistically significant differences, at the .05 level, between males and females were evident in nonstratified analyses. When the

data were stratified by age, statistically significant differences between the genders were no longer evident. This loss of statistical significance may have been the result of: 1) age being a confounder in the relationships between the various factors measured and gender, or 2) the sample size being insufficient to identify the presence of statistically significant differences between males and females. If the observed loss of statistical significance were due to insufficient sample size, both within and cross gender comparisons of stratified data may reveal differences between the genders which might have been significant had the sample been of sufficient size (Schlesselman, 1982). For this reason, the focus of the findings and discussion of this study is on the relationships and trends within both nonstratified and stratified data, which were observed, and how they relate to the findings observed in those studies discussed in the review of the literature.

Age

Nonstratified Analysis

The mean ages of males and females were 58.3 and 69.3 years respectively. Median and mode values were 59.0 and 59.0 years respectively for males, and 71.0 and 72.0 years respectively for females. Nonstratified age variance was similar for both genders with an F value two tail probability of .281. The t test result, using the pooled variance estimate, was significant with respect to age at

the .001 level in nonstratified analysis. These calculations indicate that the mean ages of males and females were significantly different, with females being significantly older than males. Appendix B contains descriptive statistics and t test analysis results for nonstratified age data.

Stratified Analysis

Distribution of cases by age group revealed further differences between the genders with respect to age. Approximately 52% of the females were 70 years of age or older compared with 16% of the males. Within each age group, age variance was statistically similar between the genders indicating that the age distribution of males and females within each 10 year age range were similar.

Discussion

Females have been reported in other studies to be significantly older than males with respect to mean age of prevalence of MI (Eisenberg et al., 1981; Gillum, 1986, Johansson et al, 1984; Johansson et al., 1983), a finding which is supported by this study. Also, while the cases in this study were prevalent cases comprised of both fatal and nonfatal MI, the observed gender differences with respect to age are consistent with the age of MI mortality and potential years of lost life statistics reported in the literature (Parker, 1986; Statistics Canada, 1982).

In summary, the females of this study were found to be

significantly older than their male counterparts. This finding is consistent with observations reported in other studies. This observed gender difference with respect to age is an important consideration in the findings and discussion of other demographic factors measured in this study, such as marital status and employment status.

Marital Status

Nonstratified Analysis

Males and females were found to be significantly different with respect to marital status, at the .001 level in nonstratified chi square analysis. Of the females, 45.9% were widowed compared with 7.2 % of the males. Appendix C contains descriptive statistics and chi square analysis results for nonstratified marital status data.

Stratified Analysis

Significant differences with respect to marital status, at the .001 and .05 levels, existed in the 60-69 and 70-79 year age groups respectively. In the 60-69 year age group, 37% of the females and 2.5% of the males were widowed compared to 61.7% of the females and 28.6% of the males in the 70-79 year age group.

The percentage of those who were widowed increased with age, ranging from 0.0% in the 40-49 year age group for both genders to 65.5% for females and 25.5% for males in the 80-89 year age group indicating that amount of increase was greater in females than in males. Appendix C contains

descriptive statistics and chi square analysis results for age stratified marital status data.

Discussion

The findings with respect to marital status may be explained, in part, by gender differences with respect to mean age of prevalence and mortality of MI. Mean age of prevalence of MI has been reported to be lower in males than in females (Eisenberg et al. 1981; Gillum, 1986; Johansson et al., 1984; Johansson, 1983). Similarly, mean age of MI mortality has been reported to be significantly lower in males than in females (Parker, 1986; Statistics Canada, 1982). These reported observations indicate that males die at an earlier age than females as a result of this disease. Therefore, the likelihood that females will outlive their husbands is greater than the likelihood that males will outlive their wives with respect to MI mortality. Also, in Canada, the mean age of death from all known causes has been reported to be lower in males than in females (Statistics Canada, 1982). These findings may explain the greater percentage of widows than widowers observed in this study. Significant differences, with respect to marital status, consistent with those observed in this study, have been reported elsewhere (Johansson et al., 1984).

In summary, the percentage of males and females who were widowed increased with age, with the percentage and rate of increase being greater in females than males. This

difference may be explained, in part, by gender differences with respect to MI mortality. The findings of this study are consistent with those reported by other researchers.

Employment Status

Only the findings regarding employment status which pertain to the 40-49, 50-59, and 60-69 year age groups are presented and discussed, as the older age groups are beyond the mandatory age of retirement. In both the 70-79 and 80-89 year age groups, only 1 female and no males were currently employed at the time of their MI.

Nonstratified Analysis

Males and females were found to be significantly different with respect to employment status, at the .001 level in nonstratified chi square analysis. Of the females, 11.6% were employed at the time of their MI compared with 57.2% of the males. Appendix D contains descriptive statistics and chi square analysis results for nonstratified employment status data.

Stratified Analysis

In the 40-49 year age group, males and females were found to be statistically similar with respect to employment status, at the .05 level in stratified chi square analysis. Eighty per cent of the females and 88.5% of the males were employed at the time of their MI. Significant differences at the .001 and .01 levels, between the genders, with respect to employment status were evident in the 50-59 and

60-69 year age groups respectively. The percentages employed, at the time of their MI, in the 50-59 year age group were 36.8% and 82.6% for females and males respectively, and 9.1% and 35%.0% for females and males respectively in the 60-69 year age group. The percentage who were employed declined with age in the case of females, while it remained relatively constant with age for males. Appendix D contains descriptive statistics and chi square analysis results for age stratified employment status data.

Discussion

The differences which were found with respect to employment status may be explained, in part, by gender differences with respect to the age distributions of males and females in this study. The mean ages for males and females in this study were 58.3 and 69.3 years respectively. Of the males, 71% were of employable age compared with 31% of the females. A greater percentage of males than females were within the employable age range, a fact which may account for the differences noted.

Of interest is the decline, with age, in the percentage of females who were employed. In the 40-49 year age group, the percentage of males and females who were employed were very similar and, thereafter, declined sharply with age in females. While the incidence of MI in females has been observed to increase as the number of working women increases (Alfredsson et al., 1985, Haynes & Feinleib, 1980;

Haynes, Feinleib et al., 1978; Haynes, Levine et al., 1978), demographic descriptions of employment status and age in females with MI could not be found in the literature. Therefore, how the women of this study compare with other females cannot be determined. However, the finding of the greatest percentage of employed females in the 40-49 year age group is consistent with the hypothesis that more females have recently entered the work force and that employment may influence the risk of MI in females of this age.

In summary, males and females differed significantly with respect to employment status. Employment status at the time of MI was similar among males and females in the 40-49 year age group and declined sharply with age in females, while remaining relatively constant with age in males. This difference may be explained, in part, by the fact that there were more males than females in the sample who were of employable age.

Family History Of MI

Nonstratified Analysis

Males and females were found to be statistically similar with respect to family history of MI, at the .05 level in nonstratified chi square analysis. Of the females, 55.2% had a positive family history of MI compared with 44.4% of the males. Appendix E contains descriptive statistics and chi square analysis results for nonstratified

family history of MI data.

Stratified Analysis

In all but the 40-49 year age group, males and females were found to be statistically similar with respect to family history of MI, at the .05 level in stratified chi square analysis. Also, in all but the 50-59 year age group, the percentage of females with a positive family history of MI was consistently greater than that of males.

The percentage of males with a positive family history of MI was 46.2% in the 40-49 year age group, increased to and was greatest at 55.0% in the 50-59 year age group, and declined steadily, thereafter, to 0.0% in the 80-89 year age group. In contrast, the percentage for females was greatest in the 40-49 year age group at 100.0% and then remained relatively constant, with age, ranging between 40.9% and 68.0% in the remaining age groups. Appendix E contains descriptive statistics and chi square analysis results for age stratified family history of MI data.

Discussion

As the percentage of males with a positive family history declined with age and the percentage of females with a positive family history of MI remained relatively constant with age, the differences between the genders became greater with advancing age. This trend is consistent with the findings reported by Khaw et al. (1986) and supports the hypothesis that a positive family history of MI becomes of

lesser importance, with respect to risk of MI, with advancing age in males and remains of equal importance in females throughout all ages.

In summary, males and females were found to be statistically similar with respect to positive family history of MI. In all but one age group, the percentage of females with a positive family of MI was consistently higher than the corresponding percentage for males. This finding is consistent with observations reported by other researchers.

History of Cardiovascular Disease

Nonstratified Analysis

Males and females were found to be significantly [illegible]ferent with respect to history of cardiovascular disease, the .001 level in nonstratified chi square analysis. Of [illegible]females, 39.5% had no history of cardiovascular disease to their MI compared with 59.5% of the males. For [illegible]who had a history of cardiovascular disease, the percentages for females and males respectively, for the various types of cardiovascular disease, were as follows: i) angina - 19.7% and 17%; ii) cerebral vascular accident - 4.8% and 1.3%; iii) previous cardiac surgery - 3.4% and 1.3%; iv) previous MI 25.2% and 20.9%; and v) congestive heart failure - 7.5% and 0.0%. Appendix F contains descriptive statistics and chi square analysis results for nonstratified history of cardiovascular disease data.

Stratified Analysis

In all but the 40-49 year age group, males and females were found to be statistically similar with respect to history of cardiovascular disease, at the .05 level in stratified chi square analysis. In the 40-49 year age group, 73.4% of the males had no history of cardiovascular disease compared with 40.0% of the females. In all but the 50-59 year age group, the percentage of females with no history of cardiovascular disease was less than that of the males.

With respect to angina, the percentage of males with a history of angina prior to MI was greater than that of females in the 40-49, 50-59, 60-69, and 70-79 year age groups. In contrast, the percentage of females with a history of cerebral vascular accident prior to MI was greater than that of males in the 60-69, 70-79, and 80-89 year age groups. No males or females had a history of cerebral vascular accident in the 40-49 and 50-59 year age groups.

The percentage of females with a history of previous cardiac surgery prior to MI was greater than that of males in the 40-49, 50-59, and 60-69 year age groups. No males or females had a history of cardiac surgery in the 80-89 year age group.

The percentage of females with a previous MI was greater than that of males in the 40-49 and 70-79 year age

groups. The percentages were similar between the genders in the remaining age groups.

No males and 7.5% of the females had a history of congestive heart failure. For the females, congestive heart failure occurred in the 60-69, 70-79, and 80-89 year age groups.

Appendix F contains descriptive statistics and chi square analysis results for age stratified history of cardiovascular disease data.

Discussion

The finding that females had a greater history of cardiovascular disease is consistent with those reported in other studies (Madsen et al., 1987; Tofler et al., 1987). With respect to the prevalence of angina, conflicting observations have been reported. In this study, males had a greater age specific history of angina prior to MI than females, a finding consistent with those of Kannel et al. (1984) and Kannel et al. (1985). In contrast to these reports and the findings of this study, Klaeboe et al. (1987) and Logan et al. (1986) reported a greater prevalence of angina in females than in males prior to MI.

Descriptions of the prevalence of cerebral vascular accident and previous cardiac surgery in males and females prior to MI could not be found in the literature. In the context of this study, previous cardiac surgery included coronary bypass surgery only. The finding that more females

than males in this study underwent coronary artery bypass surgery prior to MI is interesting. In many studies, females suspected of having coronary artery disease have been observed to have significantly less coronary artery stenosis than their male counterparts (Fried et al., 1987; Morris et al., 1976; Silver et al., 1980; Sketch et al., 1976; Walters et al., 1978; Wei et al., 1982). The findings of these studies indicate that more males than females may require coronary artery bypass surgery since males appear to have significantly greater coronary artery stenosis.

If more males than females do undergo coronary artery bypass surgery, more males than females should have had a history of coronary artery bypass surgery in this study. Because this study was restricted to prevalent cases of MI, incident cases of males with a history of coronary artery bypass surgery, who died following this surgical procedure as a result of MI, were not included in the study. These possibly missing male incident cases of MI and the greater number of females than males, with a history of coronary artery bypass surgery observed in this study, may be indicative of the fact that survival following this surgical procedure is greater in females than in males. Testing this hypothesis would provide further insight into differences between males and females with MI.

The observation that there was a higher percentage of females than males with a history of previous MI, in the 40-

49 and 70-79 year age groups, is consistent with that of Pohjola et al. (1980) who reported a greater mortality rate following the first MI in males than females. Because the present study was restricted to prevalent cases of MI, incident cases of males who died following their first MI were not included, a fact which could account for the reduction in the percentage of males, who had had a previous MI, observed in this study.

The observation that there were almost identical percentages of males and females with a history of a previous MI, in the 50-59, 60-69, and 80-89 year age groups, is consistent with that of Olmsted et al. (1987) and Saito et al. (1987) who reported no difference between males and females with respect to mortality rate following the first MI. Because the present study was restricted to prevalent cases of MI, and, if what Olmsted and Saito found is true, equal numbers of male and female incident cases who died following their first MI may not have been included in the study. This could then account for the almost identical percentages of males and females with a history of previous MI observed in the 50-59, 60-69, and 80-89 year age groups in the study.

Finally, in light of what has just been discussed, the fact that many studies seem to indicate that females may have a significantly greater mortality rate following their first MI than males, in all age groups, warrants discussion

(Kannel et al., 1979; Madsen et al., 1987; Puletti et al., 1984; Roig et al., 1987; Tofler et al., 1987). In terms of the present study, if this were so, a greater number of female than male incident cases of initial fatal MI may have been excluded from the sample and the percentage of males who had had a previous MI would then have exceeded that of females. As this trend was not consistently evident in the stratified data of the study, mortality rate following initial MI may be similar between males and females or slightly greater in males than in females.

The observation that 0.0% of the males and 7.5% of the females were diagnosed with congestive heart failure prior to their MI is consistent with that of Tofler et al. (1987) who reported a greater prevalence of congestive heart failure in females than in males with MI.

In summary, the females of this study had a greater history of cardiovascular disease prior to their MI than males. This was evident for all but one age group. The percentage of males with a history of angina prior to their MI was greater than that of females, while the reverse was true for females with respect to history of cerebral vascular accident, previous cardiac surgery, and congestive heart failure prior to MI. Several of these observations may have been influenced by the prevalence bias of this study.

Cigarette Smoking

Nonstratified Analysis

Males and females were found to be significantly different with respect to cigarette smoking, at the .001 level in nonstratified chi square analysis. Of the females, 52.4% had never smoked compared with 18.5% of the males. For past smokers, the percentages were 21.1% and 34.4% for females and males respectively. For current smokers, the percentages were 26.5% for females and 47.0% for males. Appendix G contains descriptive statistics and chi square analysis results for nonstratified cigarette smoking data.

Stratified Analysis

Males and females were found to be significantly different, in the 60-69 and 70-79 year age groups, with respect to cigarette smoking, at the .001 and .05 levels respectively in stratified chi square analysis. In the 60-69 year age group, the percentages of never, past, and current smokers for males were 15.8%, 57.9%, and 26.3% respectively. The corresponding percentages for females were 43.2%, 20.5%, and 36.4%. In the 70-79 year age group, the percentages of never, past, and current smokers for males were 28.6%, 42.9%, and 28.6% respectively. The corresponding percentages for females were 61.7%, 23.4%, and 16.9%. Appendix G contains descriptive statistics and chi square analysis results for age stratified cigarette smoking data.

For both genders, the percentage of those who had never smoked increased with age. This percentage was consistently higher in the case of females than males in all age groups.

For males, the percentage of those who were past smokers was 26.9% in the 40-49 year age group, increased to 57.9% in the 60-69 year age groups, and declined steadily, thereafter, to 25.0% in the 80-89 year age group. In contrast, the percentage of female past smokers was 0.0% in the 40-49 year age group, and increased to and remained relatively constant at approximately 20.0% throughout the remaining age groups.

For both genders, the percentage of current smokers peaked in the 40-49 year age group with 61.5% of the males and 80.0% of the females in this age group being classified as current smokers at the time of their MI. For both genders, these percentages declined, with age, with the percentage being consistently higher in males than females in all but the 60-69 year age group.

Discussion

The observation of a greater percentage of females than males who had never smoked and of a greater percentage of male than female current smokers is consistent with that of a lower prevalence of smoking and number of cigarettes smoked in females than in males observed in other studies (Carlson et al., 1985; Dick et al., 1978; Moulopoulos et al., 1987). Similarly, the finding of the greatest

percentage of female current smokers in the 40-49 year age group observed in this study is consistent with that of Bush et al. (1983), Rosenberg et al. (1985), Rosenberg et al. (1980), Shapiro et al. (1980), and Slone et al. (1978) who found that the greatest percentages of females with MI, who were current smokers, were in the younger age groups.

In summary, fewer females than males were current smokers, while the reverse was true with respect to those who had never smoked. For both genders, the greatest percentage of current smokers was found in the 40-49 year age group. Both within and between gender differences were evident with respect to those who had quit smoking prior to their MI.

Obesity

Nonstratified Analysis

Males and females were found to be statistically similar with respect to obesity, at the .05 level in nonstratified chi square analysis. Of the females, 40.3% were obese compared with 41.2% of the males. Appendix H contains descriptive statistics and chi square analysis results for nonstratified obesity data.

Stratified Analysis

In all but the 70-79 year age group, males and females were found to be statistically similar with respect to occurrence of obesity, at the .05 level in stratified chi square analysis. For both genders, the percentage of those

who were obese was greatest in the 50-59 year age group. In this age group, the percentages of males and females who were obese were 53.2% and 63.2% respectively. The percentage of those who were obese declined with age in both genders to 25.0% for males and females in the 80-89 year age group. In all but the 40-49 year age group, the percentage of obese females was greater than that of males. Appendix H contains descriptive statistics and chi square analysis results for age stratified obesity data.

Discussion

The validity of the findings, with respect to obesity, may have been jeopardized by the way in which obesity was measured in the study, that is, by subjective determinations of obesity made by admitting physicians. This method of measuring obesity may have been less reliable than other methods of measurement such as sum of subscapular skinfolds, hip to waist circumference ratio, and Metropolitan Relative Weight. For this reason, the findings of this study, with respect to obesity, should be interpreted with caution.

In all age groups and for both genders, the percentages of occurrence of obesity observed in the study exceeded the Canadian population estimates of the occurrence of obesity in males and females, reported in the Canada Fitness Survey (1984) and by Millar (1985). However, the greater percentage of females than males, who were found to be obese in the 50-59, 60-69, and 70-79 year age groups, is

consistent with the population estimates of greater prevalence of obesity in females than in males reported in the Canada Fitness Survey and by Millar.

The observation that males and females were statistically similar with respect to obesity concurs with the observations of other researchers (Hubert et al., 1983; Johansson et al., 1984; Kreuger et al., 1981; Yano et al., 1987). Finally, the findings of this study are also consistent with those of other researchers who observed that obesity increased the risk of MI in females (Hubert et al., 1983, Lapidus et al., 1984).

In summary, males and females were found to be similar with respect to obesity. The percentages of occurrence of obesity which were observed were greater than Canadian population based estimates of obesity. For both genders, obesity occurred to the greatest extent among those in the 50-59 year age group and declined, with age, thereafter.

Diabetes

Nonstratified Analysis

Males and females were found to be significantly different with respect to diabetes, at the .005 level in nonstratified chi square analysis. Of the females, 24.5% were being treated for diabetes at the time their MI compared with 11.8% of males. Appendix I contains descriptive statistics and chi square analysis results for nonstratified diabetes data.

Stratified Analysis

In all but the 40-49 year age group, males and females were found to be statistically similar with respect to diabetes, at the .05 level in stratified chi square analysis In the 40-49 year age group, 20.0% of the females were being treated for diabetes at the time of their MI compared with 0.0% of the males. Appendix I contains descriptive statistics and chi square analysis results for age stratified diabetes data.

In all age groups, the percentage of females who were being treated for diabetes at the time of their MI was greater than that of males. No consistent trend in the percentage of those who were diabetic was evident in either gender with respect to age. For both genders, the greatest percentage of those who were diabetic occurred in the 70-79 year age group. This percentage was 19.0% and 34.0% for males and females respectively. The percentage of females with diabetes was more than twice that of males in the 40-49, 50-59, and 80-89 year age groups.

Discussion

The findings of this study, with respect to diabetes, are consistent with the finding of a greater prevalence of female than male diabetics with MI reported in other studies (Bjarveit et al., 1983; Leitersdorf et al., 1986; Wilhelmsen et al., 1977). The findings of this study are also consistent with those of Gordon et al. (1977b) and Kannel et

al. (1978) who found that diabetes contributed to risk of MI to a greater degree in females than in males.

In summary, a greater percentage of females than males, in this study, were being treated for diabetes at time of their MI. The percentage of females with diabetes was consistently greater than that of males in all age groups. These findings are consistent with those reported in many studies.

Hypertension

Nonstratified Analysis

Males and females were found to be significantly different with respect to hypertension, at the .005 level in nonstratified chi square analysis. Of the females, 54.4% were being treated for hypertension at the time of their MI compared with 36.6% of the males. Appendix J contains descriptive statistics and chi square analysis results for nonstratified hypertension data.

Stratified Analysis

In all but the 70-79 year age group, males and females were found to be statistically similar with respect to hypertension, at the .05 level in stratified chi square analysis. In the 70-79 year age group, 63.8% of the females were being treated for hypertension at the time of their MI compared with 33.3% of the males. Appendix J contains descriptive statistics and chi square analysis results for age stratified hypertension data.

For females, the percentage of those who were being treated for hypertension increased with age from 40.0% in the 40-49 year age group to 58.6% in the 80-89 year age group. In contrast, the percentage of males who were being treated for hypertension remained relatively constant at approximately 40.0% in the 40-49, 50-59, and 60-69 year age groups and declined steadily, thereafter, to 25.0% in the 80-89 year age group. The percentage of females being treated for hypertension at the time of their MI exceeded that of males in all but the 40-49 year age group where these percentages were 40.0% and 42.3% for females and males respectively.

Discussion

In this study, for both genders, and in all age groups, the percentage of occurrence of hypertension exceeded the Canadian population based prevalence estimates of occurrence of hypertension reported by Millar (1985) as 21.8% for males and 12.6% for females. Furthermore, in contrast to these estimates, the percentage of females in this study who were hypertensive was greater than that of males in all but the 40-49 year age group. This finding is consistent with those of Eisenberg et al. (1981) and Johansson et al. (1983) who reported a greater prevalence of hypertension in females than in males with MI. In line with the hypothesis put forward by Bjarveit et al. (1983), the greater percentage observed in this study, of females, who were hypertensive,

may be related to the females having had greater exposure, than males, to medical diagnostic and therapeutic strategies prior to the onset of their cardiovascular disease and MI.

In summary, a greater percentage of females than males were being treated for hypertension at the time of their MI. The percentage of females who were being treated for hypertension was greater than that of males in all age groups but the 40-49 year age group. These findings are consistent with those reported in many studies.

Uric Acid

Nonstratified Analysis

Males and females were found to be statistically similar with respect to uric acid, at the .05 level in nonstratified t test analysis. The mean uric acid levels for males and females were 412.5 and 421.3 respectively. Median and mode values were 402.0 and 376.0 respectively for males, and 410.0 and 480.0 respectively for females. Range values for males and females were 695.0 and 731.0 respectively. Nonstratified uric acid level variances for both genders were significantly different with a F value two tail probability of .000. These figures indicate that, while the mean uric acid levels for males and females were statistically similar, the distribution of uric acid scores for each gender was statistically different. Appendix K contains descriptive statistics and t test analysis results for nonstratified uric acid data.

Stratified Analysis

In the 40-49, 50-59, and 80-89 year age groups, males and females were found to be statistically similar with respect to uric acid, at the .05 level in stratified t test analysis. The uric acid level variances within these age groups were statistically similar for males and females. These findings indicate that the mean uric acid levels and distribution of uric acid scores were similar for both males and females within these 10 year age ranges. Appendix K contains descriptive statistics and t test analysis, Pearson product-moment correlation coefficients, and linear regression results for age stratified uric acid data.

In the 60-69 and 70-79 year age groups, males and females were found to be statistically different with respect to uric acid, at the .05 level in stratified chi square analysis. In the 60-69 year age group, the mean uric acid levels for males and females were 429.1 and 366.8 respectively. The uric acid level variances for the genders were similar within this age group and the two tail probability of the t test statistic using the pooled variance estimate was .018. These figures indicate that the mean uric acid level in males was significantly greater than that in females in this age group.

In the 70-79 year age group, the mean uric acid levels for males and females were 407.2 and 477.1 respectively. The uric acid level variances for the genders were

statistically different in this age group with a F value two tail probability of .024. The two tail probability of the t test statistic using the separate variance estimate was .044. These figures indicate that the mean uric acid level in females was significantly greater than that in males and that the genders differed significantly with respect to variance.

For females, the mean uric acid level increased, with age, from 406.0 in the 40-49 year age group to 486.7 in the 80-89 year age group. For females, the Pearson product-moment correlation coefficient between uric acid level and age was .33. Explained variance of uric acid level due to age was 10.9% with a significance of .000.

For males, mean uric acid level increased with age from 407.8 in the 40-49 year age group to 440.5 in the 80-89 year age group. The Pearson product moment correlation coefficient between uric acid level and age in males was .149. Explained variance of uric acid level due to age in males was 2.20% with a significance of .035.

For the 40-49, 50-59, and 60-69 year age groups, the mean uric acid levels of males were consistently higher than those of females. This trend was reversed in the 70-79 and 80-89 year age groups where the mean uric acid levels of females were greater than those of males.

Discussion

Few studies were located in which males and females

with MI were compared on the basis of uric acid level. The finding of greater mean uric acid levels in males of the 40-49, 50-59, and 60-69 year age groups, is consistent with those of Eisenberg et al. (1981) and Moulopoulos et al. (1987) who reported mean uric acid levels to be significantly higher in males than in females with MI. However, in contrast to the observations reported in these studies, the mean uric acid levels of females in the 70-79 and 80-89 year age groups of this study were greater than those of the males.

In summary, nonstratified analysis revealed that the mean uric acid levels of males and females were similar. However, stratified analysis indicated that these levels were significantly different in the 60-69 and 70-79 year age groups. Mean uric acid levels increased, with age, in both genders and the correlation between age and uric acid level was considerably stronger in females than in males. In the younger age groups, the mean uric acid level was consistently greater in males than females, a finding consistent with those reported in other studies. However, in the older age groups, the reverse was found, a finding not consistent with those reported in other studies.

Type of MI

Nonstratified Analysis

Males and females were found to be statistically similar with respect to type of MI, at the .05 level in

nonstratified chi square analysis. For both genders, an inferior MI occurred most frequently. Of the males, 44.4% were diagnosed with an inferior MI compared with 39.0% of the females. For males, the frequency of occurrence of the other types of MI was as follows: anterior (19.6%), anterior-lateral (13.7%), anterior-septal (12.4%), subendocardial (5.9%), and posterior (3.9%). The following frequencies were observed for females: anterior (16.4%), subendocardial (15.8%), anterior-septal (15.8%), anterior-lateral (8.2%), and posterior (4.8%). Appendix L contains descriptive statistics and chi square analysis results for nonstratified type of MI data.

Stratified Analysis

In all but the 60-69 and 70-79 year age groups, males and females were found to be statistically similar with respect to type of MI, at the .05 level in stratified chi square analysis. In the 60-69 year age group, the frequency of occurrence of types of MI for males was as follows: inferior (45.0%), anterior-lateral (20.0%), anterior-septal (12.5%), anterior (12.5%), posterior (5.0%), and subendocardial (5.0%). The following frequencies were observed for females: inferior (38.6%), anterior (20.5%), anterior-septal (20.5%), subendocardial (13.6%), posterior (6.8%), and anterior lateral (0.0%). In the 70-79 year age group, the frequency of occurrence of types of MI for males was as follows: inferior (38.1%), anterior (23.8%),

anterior-septal (19.0%), anterior-lateral (14.3%), subendocardial (4.8%), and posterior (0.0%). The following frequencies were observed for females: inferior (38.3%), subendocardial (19.1%), anterior (12.8%), anterior-septal (10.6%), posterior (6.4%), and anterior-lateral (1.8%). These figures indicate considerable variation with respect to type of MI both within and between genders in these age groups. Appendix K contains descriptive statistics and chi square analysis results for age stratified type of MI data.

In all but the 70-79 year age group, the percentage of females with an anterior MI was consistently greater than that of males. For both genders, the frequency of occurrence of anterior MI declined, with age, from 40.0% to 7.1% in the 40-49 to 80-89 year age groups respectively for females and from 30.8% to 0.0% in the 40-49 to 80-89 year age groups respectively for males.

In all but the 80-89 year age group, the percentage of males with an anterior-lateral MI was consistently greater than that of females. The distribution of the frequency of occurrence of anterior-lateral MI, with respect to age in males, followed an inverted U shape, with the greatest percentage (20.0%) occurring in the 60-69 year age group. In contrast, the corresponding distribution in females was U shaped with the least percentage (0.0%) of anterior-lateral MI occurring in the 60-69 year age group.

Differences between males and females with respect to

the frequency of occurrence of an anterior-septal MI were also evident. For females, the percentage of those who suffered an anterior-septal MI increased linearly, with age, from 0.0% to 21.4% in the 40-49 to 80-89 year age groups respectively. In contrast, the distribution of the frequency of occurrence of an anterior-septal MI, with respect to age in males, was an inverted U shape with the greatest percentage (19.0%) occurring in the 70-79 year age group.

An inferior MI was the most commonly occurring infarct type for both genders in all age groups. For females, the frequency of occurrence of an inferior MI declined steadily, with age, from 60.0% to 32.1% in the 40-49 to 80-89 year age groups respectively. For males, the greatest frequency of occurrence of an inferior MI was observed in the 40-49 (57.7%) and 80-89 (50.0%) year age groups. This type of MI comprised approximately 38.0% of all infarcts, diagnosed in males and females who were in the 50-59, 60-69, and 70-79 year age groups.

Only 3.9% of males or 6 males and 4.8% of females or 7 females were diagnosed as having a posterior MI. So few cases made identification of trends difficult. For males, 83.0% of the posterior MI cases were in the 50-59 (3 cases) and 60-69 (2 cases) year age groups. For females, 86.0% of the posterior MI cases were in the 60-69 (3 cases) and 70-79 (3 cases) year age groups.

The percentage of males with a subendocardial MI was relatively constant throughout the age groups and ranged from 0.0% in the 40-49 year age group to 5.3% in the 70-79 year age group and increased to 25.0% in the 80-89 year age group. In contrast, the percentage of females with a subendocardial MI increased linearly with age from 0.0% to 25.0% in the 40-49 to 80-89 year age groups respectively. This consistent increase, with age, in the percentage of females with a subendocardial MI corresponded with the declines in percentage, with age, observed for both anterior and inferior MI in females. In all but the 80-89 year age group, the percentage of females with a subendocardial MI was consistently greater than that of males.

Discussion

No studies were located in the literature in which males and females were compared on the basis of type of MI. In one cross sectional study restricted to female cases of MI mortality, reported in the literature, 25.0% and 50.0% of the females had been diagnosed as having an anterior and an inferior MI respectively (Morris et al., 19[illegible]). These percentages are consistent with the percentages of anterior (16.4%) and inferior (39.0%) MI observed among females in this study.

In the present study, the percentage of females with an anterior MI was greater than that of males while the reverse was true in the case of an inferior MI. This finding may

have implications for differences between males and females with respect to mortality following MI for the following reasons. First, mortality rate following MI has been observed to increase as size of infarct increases (Bleifield et al., 1979; Lee et al., 1981; Pitt, 1981; Shell et al., 1976; Sobel et al., 1972). Second, Sobel et al. (1972) reported that anterior infarcts were significantly greater in size than inferior infarcts. Third, mortality rate following MI has been reported to be greater in patients with an anterior MI than in patients with an inferior MI(Kannel et al., 1979). If what these researchers found is true, the females of this study may have suffered infarcts of greater size and mortality risk than males. The greater female than male mortality rate observed in this study may have been the result of there being more females than males in the sample, who suffered anterior infarcts of greater size. Comparisons of males and females with respect to infarct location and mortality is possible with the data collected in this study and would require stratification of the sample by these variables. Such an analysis goes beyond the current objectives of this study but, if performed, may provide further insight into differences between males and females with respect to MI.

In summary, males and females were found to be statistically similar with respect to type of MI, in nonstratified analysis and in all but the 60-69 and 70-79

year age groups in age stratified analysis. Consistent differences between females and males with respect to type of MI were observed. No studies were located in the literature in which males and females were compared in this regard. Differences between mortality rates in males and females observed in this and other studies may be related to gender differences with respect to infarct location and size.

Complication Following MI

Nonstratified Analysis

Males and females were found to be significantly different with respect to the occurrence of complications following MI at the .001 level in nonstratified chi square analysis. Of the females, 59.2% experienced no complications following MI compared with 82.4% of the males. Appendix M contains descriptive statistics and chi square analysis results for nonstratified complication following MI data.

Respiratory dysfunction was the most frequent complication experienced by males. Of the males, 8.5% required intubation following MI. The percentages of males diagnosed with cardiogenic shock, cardiac arrest, and anoxic brain insult following MI were 5.2%, 3.9%, and 0.0% respectively.

In contrast, cardiac related complications occurred most frequently in females. The complications experienced

by females following MI were cardiogenic shock (14.3%), cardiac arrest (12.2%), intubation (9.5%), and anoxic brain insult (4.8%).

Stratified Analysis

Males and females were found to be statistically similar with respect to the occurrence of complications following MI, at the .05 level in stratified chi square analysis. Appendix M contains descriptive statistics and chi square analysis results for age stratified complication following MI data.

In a but the 40-49 year age group, the percentage of females who experienced no complications following MI was consistently less than that of males . The percentage of females who experienced no complications following MI declined steadily with age from 80.0% to 48.3% in the 40-49 to 80-89 year age groups respectively. The corresponding percentage of males who experienced no complications following MI remained relatively constant throughout the age groups, and ranged from 75.0% to 87.5% in all age groups.

The percentage of males who required intubation following MI declined steadily with age from 15.4% to 0.0% in the 40-49 to 80-89 year age groups respectively. In females, the percentage of those who required intubation was greater than that of males in all but the 50-59 year age groups. A trend in this percentage with respect to age was not discernible in females.

The percentage of females who suffered cardiogenic shock following MI increased steadily with age from 0.0% to 20.7% in the 40-49 to 80-89 year age groups respectively. In all but the 40-49 year age group, the percentage of females who experienced cardiogenic shock following MI was greater than that of males. For both genders, the greatest percentage of cardiogenic shock occurred in the 70-79 year age group.

The percentage of males who experienced cardiac arrest following MI was relatively constant throughout the age groups ranging from 0.0% to 5.0%. Cardiac arrest was experienced by females in the 60-69, 70-79, and 80-89 year age groups only and increased slightly with age. In all but the 80-89 year age group, the percentage of females diagnosed with cardiac arrest following MI was greater than that of males.

Only 7 females (4.8%) and no males experienced anoxic brain insult following MI. For females, this complication occurred most frequently in the older age groups. Eighty-five per cent of the cases occurred in the 70-79 (4 cases) and 80-89 (2 cases) age groups.

Discussion

The trends identified in this study with regard to complications following MI are consistent with those observed by Madsen et al. (1987) who reported a significantly greater percentage of females than males who

experienced post MI cardiac related complications. In contrast to the findings of the present study, Saito et al. (1987) reported no difference between males and females with respect to type and number of cardiac complications following MI.

The greater percentage of females than males who experienced cardiac related complications following MI may be related to the greater percentage of females than males who suffered an anterior MI in this study, since an anterior MI has been observed to be of greater mortality risk and infarct size than an inferior MI (Bleifield et al., 1979; Lee et al., 1981; Pitt, 1981, Shell et al., 1976; Sobel et al., 1972). Comparison of males and females with respect to type of MI and complication following MI is possible with the data collected in this study and would require stratification of the sample by these variables. Such an analysis goes beyond the current objectives of this study but, if carried out, may provide further insight into how males and females differ with respect to MI.

In summary, the percentage of males who experienced no complications following their MI was found to be greater than that of females, in both stratified and nonstratified analysis. In males, the greatest percentage of post MI complications were respiratory in nature while the greatest percentage of post MI complications were cardiac related in females. For all complications and in all age groups, the

percentage of females affected was almost consistently greater than that of males. Differences between males and females with respect to complications following MI may be related to differences between the genders with respect to type of MI observed in this study.

Mortality

Nonstratified Analysis

Males and females were found to be significantly different with respect to mortality following MI, at the .001 level in nonstratified chi square analysis. Of the females, 25.2% died following MI compared with 10.5% of the males. Appendix N contains descriptive statistics and chi square analysis results for nonstratified mortality data.

Stratified Analysis

Males and females were found to be statistically similar with respect to mortality following MI, at the .05 level in all age groups in stratified chi square analysis. Appendix N contains descriptive statistics and chi square analysis results for age stratified mortality data.

For males, the incidence of mortality increased steadily, with age, from 11.5% to 25.0% in the 40-49 to 80-89 year age groups respectively. The incidence of mortality also increased, with age, for females with the amount of increase being greater than that for males. The incidence of mortality for females increased from 0.0% to 34.5% in the 40-49 to 80-89 year age groups respectively.

In all but the 40-49 year age group, the incidence of mortality for females was consistently greater than that for males. In this age group, the incidence of mortality for males and females was 0.0% and 11.5% respectively. In the 70-79 year age group, the incidence of mortality for females (38.3%) was more than twice that of males (19.0%).

Discussion

The findings of this study are consistent with those reported by researchers of other studies in which a greater female than male mortality rate following MI was observed (Kannel et al., 1979; Madsen et al., 1987; Puletti et al., 1984; Roig et al., 1987; Tofler et al., 1987).

The greater female than male mortality rate which was observed in this study may be related to the greater percentage of females than males with an anterior MI and cardiac related complications following MI observed in this study. An anterior MI has been reported to be of greater size and mortality risk than an inferior MI, by several researchers (Bleifield et al, 1979; Lee et al., 1981; Pitt, 1981; Shell et al., 1976; Sobel et al., 1972;). An analysis the relationship between and among type of MI, complication following MI, and mortality following MI is possible with the data collected in this study and would require stratification of the sample by these variables. Such an analysis goes beyond the current objectives of this study but, if carried out, may provide further insight into

differences which may exist between males and females with MI.

In summary, with nonstratified and stratified analyses, a greater percentage of females than males were found to have died following their MI. For both males and females, the mortality percentage increased with age, with the amount of increase being greater in females than males. In all but one age group, the mortality percentage for females was greater than that for males. These findings are consistent with those reported by several researchers. The observed greater female mortality may be related to differences between the genders observed in this study, with respect to infarct location and complications following MI.

Lactic Dehydrogenase (LDH)

Nonstratified Analysis

Males and females were found to be statistically similar with respect to the results of all three LDH tests, at the .05 level in nonstratified t test analysis. The LDH level variances in all three tests were similar for the genders. The mean LDH level increased from the first to third tests in both genders, with the amount of increase being greater in males than in females. The mean LDH level was greater in males than in females in all but the first LDH test where the reverse was true. Appendix O contains descriptive statistics and t test analysis results for nonstratified LDH data.

Stratified Analysis

With the exception of the third LDH test results for the 50-59 year age group, males and females were found to be statistically similar with respect to the results of all three LDH tests, at the .05 level in stratified t test analysis. Appendix O contains descriptive statistics and t test analysis, Pearson product-moment correlation coefficients, and linear results for age stratified LDH data.

For the first LDH test, the LDH level variances were similar for both genders in all but the 40-49 and 60-69 year age groups. In the second LDH test, the LDH level variances were similar for both genders in all but the 60-69 and 70-79 year age groups. In the third LDH test, the LDH level variances were similar for both genders in all but the 70-79 year age group.

With each LDH test, from the first to third LDH tests, the mean LDH level in males increased within all age groups. The amount of increase was greatest in the 40-49 year age group in which the mean LDH level more than doubled from the first (294.5) to the third test (636.3).

Within each LDH test, the mean LDH level in males decreased slightly with age. The Pearson product-moment correlation coefficients between each LDH test and age in males indicated very weak negative correlations which increased in strength from the first (-.076) to the third

(-.121) test. Explained variance in LDH level due to age in males was small for each test and failed to reach the .05 level of significance for all three LDH tests. These calculations indicate that a linear relationship did not exist between LDH and age in males.

With each LDH test, from the first to third LDH tests, the mean LDH level increased in females within all but the 40-49 and 50-59 year age groups. In contrast to males, the greatest amount of increase in mean LDH level, from the first to third test, occurred in females in the 70-79 year age group in which the mean LDH level increased from 380.9 to 530.9 respectively.

Within each LDH test, the mean LDH level for females tended to increase slightly with age. The Pearson product-moment correlation coefficients between each LDH test and age in females indicated very weak positive correlations between these two variables. These correlation coefficients increased slightly in strength from the first (.030) to the third (.085) LDH tests. Explained variance in LDH level due to age in females was small and failed to reach the .05 level of significance in all three LDH tests. These calculations indicate that a linear relationship between LDH level and age did not exist in females.

When males and females were compared, the mean LDH level in females was greater than the mean LDH level in males in the 40-49, 60-69, 70-79, and 80-89 year age groups,

for all three LDH tests. In contrast, the mean LDH level in males was greater than the mean LDH level in females in the 50-59 year age group for all three LDH tests.

Discussion

No studies were located in the literature in which males and females were compared with respect to LDH levels following MI. LDH is an ubiquitous enzyme found in most tissues of the body (Sobel et al., 1972). As such, LDH levels may be determined by many factors other than infarct size (Lee et al., 1986). However, LDH level has been observed to be correlated with infarct size (Shibata et al., 1985). If the LDH levels observed in this study do correlate with infarct size in both males and females, the findings of this study are consistent with the hypothesis that, both in and between males and females, infarct size differences may exist with respect to age. Because infarct size has been observed to be correlated with both infarct location and mortality (Bleifield et al. 1976; Geltman et al., 1979; Kannel et al., 1979; Lee et al., 1986; Pitt, 1981; Shell et al., 1976; Sobel et al., 1972), testing this hypothesis may provide further insight into differences between males and females with respect to mortality and location of MI observed in this study and in other studies (Kannel et al., 1979; Madsen et al., 1987; Puletti et al., 1984; Roig et al., 1987; Tofler et al., 1987).

In summary, in nonstratified analysis, and in most age

groups, in stratified analysis, males and females were found to be statistically similar with respect LDH level. Both statistically significant and nonsignificant differences were observed between males and females with respect to LDH test sequence and age. These findings suggest that infarct size differences may exist both in and between males and females with respect to age. The testing of this hypothesis may provide further insight into differences which may exist between males and females with MI.

Lactic Dehydrogenase Subfraction One (LD1)

Nonstratified Analysis

In all but the third LD1 test results, males and females were found to be statistically similar with respect to the results of the three LD1 tests, at the .05 level in nonstratified t test analysis. The mean LD1 level for males and females, in the third test, was .38 and .35 respectively. The LD1 level variances in all three tests were similar for the genders. In males, the mean LD1 level increased from the first (.27) to the third (.38) test. In females, the greatest mean LD1 level occurred in the second test with a value of .37. In all but the second LD1 test, the mean LD1 level was greater in males than in females. Appendix P contains descriptive statistics and t test analysis results for nonstratified LD1 data.

Stratified Analysis

With the exception of the third LD1 test results for

the 50-59 year age group, males and females were found to be statistically similar with respect to the results of the three LD1 tests, at the .05 level in stratified t test analysis. In the 50-59 year age group, the mean LD1 level for males and females was .40 and .33 respectively. The LD1 level variances for all age groups in all three LD1 tests were similar for males and females. Appendix P contains descriptive statistics and t test analysis, Pearson product-moment correlations, and linear regression results for age stratified LD1 data.

With each LD1 test, from the first to third LD1 tests, the mean LD1 level in males increased within each age group. The amount of increase was greatest in the 40-49 year age group in which the mean LD1 level increased from .27 to .40 from the first to the third test respectively.

Within each LD1 test, the mean LD1 level in males decreased slightly with age. In males, the Pearson product-moment correlation coefficients between each LD1 test and age indicated very weak negative correlations between these variables. These correlations increased in strength from the first (-.009) to the third (-.140) test. Explained variance in LD1 level due to age in males was small but reached significance at the .05 level in the third LD1 test with a value of 2.0%. These calculations indicate that a weak linear relationship may have existed between LD1 and age in males.

With each LD1 test, from the first to third LD1 tests, the mean LD1 level increased in females within each age group but the 50-59 year age group. The amount of increase was greatest in the 40-49 year age group in which the mean LD1 level increased from .27 to .42 from the first to third tests respectively.

Within each LD1 test, the mean LD1 level in females increased slightly with age in the first LD1 test and tended to decrease with age in the second and third LD1 tests. In females, the Pearson product-moment correlation coefficients between each LD1 test and age indicated a weak positive correlation between these two variables in the first LD1 test, and weak negative correlations between these two variables in the second and third LD1 tests. Explained variance in LD1 level due to age in females was very small and failed to reach the .05 level of significance in all three tests. These calculations indicate that a linear relationship between LD1 and age did not exist in females.

When males and females were compared, the mean LD1 level in males was greater than the mean LD1 level in females in the 40-49 and 80-89 year age groups. In contrast, the mean LD1 level in females was greater than the mean LD1 level in males in the 70-79 year age group. No consistent pattern emerged when the genders, in the 50-59 and 60-69 age groups, were compared.

Discussion

No studies were located in the literature in which males and females were compared on the basis of LD1 following MI. LD1 is less ubiquitous than LDH, being found primarily in myocardial muscle and, to a lesser extent, in erythrocytes, kidneys, brain, stomach and pancreas (Lee et al., 1986). As such, LD1 may be a more reliable predictor of infarct size than LDH but may still be influenced by factors other than infarct size (Lee et al., 1986). If the LD1 levels observed in this study do correlate with infarct size in both males and females, as has been reported elsewhere (Shibata et al., 1985), the findings of this study are consistent with the hypothesis that differences in infarct size may exist both in and between males and females with respect to age. Because infarct size has been observed to be correlated with both infarct location and mortality (Bleifield et al., 1976; Geltman e. al., 1979; Kannel et al., 1979; Lee et al., 1986; Pitt, 1981; Shell et al., 1976; Sobel et al., 1972), testing this hypothesis may provide insight into differences which may exist between males and females with respect to mortality and location of MI observed in this study and in other studies (Kannel et al. 1979; Madsen et al., 1987; Puletti et al., 1984; Roig et al., 1987; Tofler et al., 1987).

In summary, in nonstratified analysis and, in most age groups, in stratified analysis, males and females were found

to be statistically similar with respect to LD1. Both statistically significant and nonsignificant differences were observed both in and between males and females with respect to LD1 test sequence and age. These differences suggest that differences in infarct size may exist both in and between males and females with respect to age. The testing of this hypothesis may provide further insight into differences between males and females with MI.

Creatine Phosphokinase Multiband (CPK-MB) Nonstratified Analysis

In all but the fourth and sixth CPK-MB test results, males and females were found to be statistically similar with respect to the results of the six CPK-MB tests, at the .05 level in nonstratified t test analysis. In the fourth CPK-MB test, the mean CPK-MB level for males and females was 94.3 and 72.6 respectively. In the sixth CPK-MB test, the mean CPK-MB level for males and females was 43.7 and 34.8 respectively.

In all but the third CPK-MB test, the CPK-MB level variances for males and females were significantly different and the CPK-MB values for males were consistently greater than those for females with respect to mean, range, standard deviation and variance. These figures indicate that mean CPK-MB level and distribution were greater in males than in females.

For both males and females, an inverted U shape

distribution of mean CPK-MB level was found with respect to CPK-MB test sequence. For males, the mean CPK-MB level was greatest in the second test and, for females, the mean CPK-MB level was greatest in the third test. Appendix Q contains descriptive statistics and t test analysis results for nonstratified CPK-MB data.

Stratified Analysis

With the exception of the first and third CPK-MB test results for the 50-59 year age group, males and females were found to be statistically similar with respect to the results of the six CPK-MB tests, at the .05 level in stratified t test analysis. In the first CPK-MB test, the mean CPK-MB level for males and females in the 50-59 year age group was 58.7 and 47.5 respectively. In the third CPK-MB test, the mean CPK-MB level for males and females in the 50-59 year age group was 116.4 and 93.6 respectively. In both tests, the CPK-MB variance was statistically similar for males and females of this age group.

With each CPK-MB test, from the first to sixth tests, the distribution of the mean CPK-MB level in males followed an inverted U shape within each age group. For all but the 80-89 year age group, the mean CPK-MB level in males was greatest in the second test and declined steadily with each test thereafter.

Within each CPK-MB test, the distribution of the mean CPK-MB level in males also followed an inverted U shape with

respect to age with the greatest mean CPK-MB values occurring in the 50-59 and 60-69 year age groups in most tests. In males, the Pearson product-moment correlation coefficients between each CPK-MB test and age indicated very weak correlations between these two variables. Explained variance in CPK-MB level due to age in males was very small and failed to reach the .05 level of significance in all CPK-MB tests. These calculations indicate that a linear relationship between CPK-MB and age did not exist in males for all tests.

With each CPK-MB test, from the first to sixth tests, the distribution of the mean CPK-MB level in females followed an inverted U shape within each age group. For all but the 40-49 year age group, the mean CPK-MB level was greatest in the third test and declined steadily with each test thereafter.

Within each CPK-MB test, the mean CPK-MB level in females was lowest in the 50-59 and 60-69 year age groups which created an U shaped distribution of mean CPK-MB level with respect to age. In females, the Pearson product-moment correlation coefficients between each CPK-MB test and age indicated very weak correlations between these two variables. Explained variance in CPK-MB level due to age was small and failed to reach the .05 level of significance in all tests. These calculations indicate that a linear relationship did not exist between CPK-MB and age in females

for all tests.

In all but the 80-89 year age group, the mean CPK-MB level in males was consistently greater than that of females for all tests. The reverse was true in the 80-89 year age group for all tests.

Discussion

No studies were located in the literature in which males and females were compared with respect to CPK-MB following MI. CPK-MB is found primarily in myocardial muscle and, to a lesser extent, in the gastrointestinal tract, skeletal muscle, uterus, diaphragm, thyroid, prostate and urethra (Lee et al., 1986). As such, CPK-MB may be reliable in arriving at qualitative estimates of infarct size but may also be influenced by many factors other than myocardial necrosis (Lee et al., 1986). If the CPK-MB levels observed in this study do correlate with infarct size in both males and females, as has been reported elsewhere (Bleifield et al., 1977; Grande et al., 1982; Shell et al., 1976; Smith et al., 1983; Shibata et al., 1985; Sobel et al., 1972), the findings of this study are consistent with the hypothesis that infarct size may differ both in and between males and females with respect to age. Because infarct size has been observed to be correlated with both infarct location and mortality following MI (Bleifield et al., 1976; Geltman et al., 1979; Kannel et al., 1979; Lee et al., 1986; Pitt, 1981; Shell et al., 1976; Sobel et al.,

1972), testing this hypothesis would provide further insight into differences between males and females with respect to infarct location and mortality observed in this study and in other studies (Kannel et al., 1979; Madsen et al., 1987; Puletti et al., 1984; Roig et al., 1987; Tofler et al., 1987).

In summary, both statistically significant and nonsignificant differences were found both in and between males and females with respect to CPK-MB following MI. These differences are consistent with the hypothesis that infarct size differences may exist both within and between the genders with respect to age. The testing of this hypothesis may provide further insight into differences between males and females with MI.

Summary

In nonstratified analysis, males and females were found to be significantly different with respect to several factors: age, marital status, employment status, history of cardiovascular disease, cigarette smoking, diabetes, hypertension, complication following MI, mortality, and certain LD1 and CPK-MB blood tests. In age stratified analysis, males and females were not found to be statistically different in terms of these factors, except for certain age strata, with regard to the following factors: marital status, employment status, cigarette smoking, uric acid level, and type of MI.

With respect to those factors associated with changes in the probability of occurrence of MI, a greater percentage of females than males were widowed and not employed. A greater percentage of females than males had a positive family history of MI and history of cardiovascular disease. The percentages of those who were being treated for diabetes and hypertension were greater in females than in males. The percentages of females who had quit smoking or had never smoked were greater than those of males while, in the youngest age group, the percentages of males and females who were current smokers were almost identical. Uric acid level more strongly correlated with age in females than in males and was greater in females than males in the older age groups.

With respect to those events which followed the occurrence of MI, males and females differed in terms of type of MI. In most age groups, the percentage of females diagnosed with an anterior MI was greater than that of males while the reverse was observed with respect to an inferior MI. This finding may have implications with respect to the differences between males and females observed in this study in terms of complications following MI and mortality following MI. In females, the most prevalent complications following MI were cardiac related. In males, the most prevalent complications following MI were respiratory related. In almost all age groups, the percentage of

females who died following their MI was greater than that of males. Finally, males and females were similar with respect to almost all LDH, LD1, and CPK-MB serum levels drawn following the occurrence of MI. For many of these blood tests, the trends of the serum levels of these biochemical parameters differed both in and between males and females with respect to age.

CHAPTER V

Summary, Conclusions and Recommendations

Summary

The purpose of this study was to gain knowledge of some of the possible differences between males and females with MI with respect to certain factors which are associated with changes in the probability of occurrence of this disease. An answer to the following question was sought: Do males and females differ in terms of certain factors which have been found to be associated with changes in the probability of occurrence of MI? If so, how?

Using a cross sectional design with a prospective component, a sample of 153 male and 147 female MI patients was identified through the hospital records. Using data from the hospital records of this sample, males and females were compared in terms of certain risk factors identified within the literature as being associated with changes in the probability of occurrence of MI. For the purpose of enhancing the comparison between males and females, males and females were compared in terms of mortality and of certain events which followed the occurrence of MI and, although do not influence the probability of occurrence of MI, may be significant in influencing prognosis. Nonparametric and parametric statistics were used to determine if significant differences existed between the genders in terms of those factors and events. Both data

which were nonstratified and stratified by age were analyzed.

For several factors, statistically significant differences between males and females were observed. These factors included age, marital staus, employment status, history of cardiovascular disease, cigarette smoking, diabetes, hypertension, complications following MI, mortality, and certain serum enzyme levels. When data were stratified into age groups and analyzed, these statistically significant differences were, for the most part, no longer evident.

Conclusions

There was sufficient evidence in this study to indicate that males and females may, indeed, differ with respect to certain factors associated with changes in the probability of occurrence of MI. There was also sufficient evidence to indicate that males and females may differ with respect to certain aspects of prognosis.

Generalizations of the findings of this study must be kept within the limitations of the study design. Such limitations included the highly unstandardized nature of the data collected, the incompleteness of the data recorded within the medical records of some cases, the crude categories of measurement used for many factors, prevalence bias, and the possibility that the samples were of insufficient size when the data were stratified by age.

When taking these limitations into consideration, the findings of this study may be generalizable to males and females who are discharged, with the diagnosis of MI, from the CCU of a large urban hospital in which diagnostic and coding procedures are similar to those which are employed by the hospital in which this study was conducted.

Recommendations

If this study were to be replicated, certain changes, with respect to sample identification, should be made. Male and female subjects considered suitable for inclusion in the study should be recruited into the study as they are admitted to hospital. This would allow the collection of data to occur during the time period in which the subjects are hospitalized.

The advantage of choosing subjects suitable for inclusion into the study as they are admitted to hospital is several fold. First, such a strategy would allow the researcher to design a data collection instrument which could be tested, to assess its validity and reliability. Second, this strategy would eliminate dependence on highly unstandardized data contained within an institution's medical records thereby enhancing the validity and reliability of the data to be collected. Third, this strategy would allow the researcher more flexibility in terms of what and how data are to be collected as he or she would not be limited to the data contained within the

medical records of subjects. Fourth, recruitment of subjects as they are admitted to hospital may increase the number of incident cases included in the study thereby reducing prevalence bias. Finally, such a study design would be applicable to more than one hospital. This would serve to increase the number of subjects suitable for inclusion into the study and aid in analysis and interpretation when data are stratified by a particular variable such as age.

Further research should be directed at investigating those aspects of risk of MI in females for which this study did not provide insight. This study did provide insight into how males and females with MI may differ with respect to several factors. It did not provide insight into how these differences contribute to the risk of MI in each gender since the study sample was restricted to male and female cases of MI only.

Once knowledge of how the differences, between males and females, observed in this study (and other possible differences) contribute to the risk of MI in each gender is attained, effective preventative health care strategies, aimed at reducing or eliminating this risk, may be devised. Case control and prospective study designs are two approaches by which males and females may be compared with respect to how these differences contribute to the risk of MI in each gender.

Because the factors measured in this study were common to both genders, it did not provide insight into what factors, unique to the female gender, may contribute to the risk of MI in females. Such factors include oral contraceptive use, noncontraceptive estrogen use, surgical and natural menopause, and pregnancy. As the incidence of MI in females is very low, case control study designs would be most useful in investigating how these factors contribute to the risk of MI in females.

This study did provide insight into how males and females may differ in terms of various aspects of prognosis. Males and females were observed to differ with respect to type of MI, complications following MI, mortality, and certain serum enzyme levels. These aspects of prognosis may be interrelated. Because no attempt was made during analysis to assess how these aspects of prognosis may be related, the findings of this study did not provide insight into the nature and magnitude of these possible relationships. Prospective study designs would be most useful in this regard.

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APPENDIX A. DATA PRESENTATION AND STATISTICS

Description of Data Presentation and Analysis

minal (Categorical) Variables

Descriptive statistics for all nominal level variables are arranged in rows for each gender and age group. The first contains frequency counts, the second row contains expected frequencies, and the third row contains row percent

Chi uare analysis results for all nominal level variables are arranged under the following headings: chi square value, degrees of freedom, minimum expected frequency, and the number of cells within the table with an expected frequency of less than five.

Fisher's exact test was used when the sample size of a chi square table was less than twenty. Yates' correction for continuity was calculated for all tables in which the degrees of freedom was equal to one.

Ratio (Continuous) Variables

Descriptive statistics for all ratio level variables for each gender and age group are arranged under the following headings: number of cases, mean, median, mode, minimum value, maximum value, range, standard deviation, variance, and standard error.

T-test analysis results are arranged by age group under the following headings: F value, two tail probability of the F value, pooled variance estimate of the t-test statistics, and separate variance estimate of the t-test

statistics. The F value was used to test the hypothesis that the two sample (i. e., male and female) variances were equal for a given variable. If the probability of the F value is small, the separate variance estimate of the t-test statistic should be used for interpretation of the result. The pooled variance estimate of the t-test statistics should be used in the interpretation of the result if the probability of the F value is large (Norusis, 1983). Both the pooled and separate variance estimate of the t-test statistic are contained within the appendices for the purpose of comparison.

Linear regression analysis results are arranged under the following headings: r, R^2, and significance of R^2. Pearson product-moment correlation coefficient is represented by r, and the proportion of explained variance of the dependent variable due to age is represented by R^2.

APPENDIX B. AGE

Descriptive Statistics For Age - Males

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	153	58.29	59.00	59.00	29.00	92.00	63.00	12.68	160.75	1.03
Stratified										
20-29	1	29.00	29.00	29.00	29.00	29.00	0.00	0.00	0.00	0.00
30-39	11	36.55	37.00	38.00	34.00	39.00	5.00	1.86	3.47	.56
40-49	26	45.15	46.00	47.00	40.00	48.00	8.00	2.53	6.38	.50
50-59	47	54.89	56.00	55.00	50.00	59.00	9.00	2.99	8.93	.67
60-69	40	64.30	64.00	66.00	60.00	69.00	9.00	2.77	7.65	.44
70-79	21	74.24	75.00	75.00	70.00	79.00	9.00	2.55	6.49	.56
80-89	4	82.00	81.50	80.00	80.00	85.00	5.00	2.16	4.67	1.08
90-99	3	91.33	91.00	91.00	91.00	92.00	1.00	.58	.33	.33

Descriptive Statistics For Age - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	147	69.31	71.00	72.00	23.00	92.00	69.00	11.60	134.61	.96
Stratified										
20-29	1	23.00	23.00	23.00	23.00	23.00	0.00	0.00	0.00	.00
30-39	1	35.00	35.00	35.00	35.00	35.00	0.00	0.00	0.00	.00
40-49	5	43.20	44.00	45.00	40.00	45.00	5.00	2.17	4.70	.97
50-59	19	55.47	56.00	55.00	50.00	59.00	9.00	2.99	8.93	.69
60-69	44	64.91	65.00	67.00	60.00	69.00	9.00	2.74	7.48	.41
70-79	47	74.32	74.00	72.00	70.00	79.00	9.00	2.57	6.61	.38
80-89	29	83.45	83.00	82.00	80.00	88.00	8.00	2.21	4.90	.41
90-99	1	92.00	92.00	92.00	92.00	92.00	0.00	0.00	0.00	.00

T-Test Analysis - Age

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.19	.281	-7.85	298	.000	-7.86	297.30	.000
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.79	10	.446	2.75	10.00	.020
40-49	1.36	.849	1.61	29	.117	1.79	6.29	.121
50-59	1.03	.893	-.72	64	.473	-.72	32.91	.478
60-69	1.02	.939	-1.01	82	.314	-1.01	81.06	.314
70-79	1.02	1.000	-.12	66	.905	-.12	38.85	.904
80-89	1.05	1.000	-1.23	31	.228	-1.25	3.92	.280
90-99	0.00	1.000	-1.00	2	.423	-2.00	2.00	.184

APPENDIX C. MARITAL STATUS

Descriptive Statistics - Marital Status

	Single	Commonlaw	Married	Divorced	Widowed
Nonstratifed					
Male	13	1	121	7	11
	9.2	1.0	96.7	6.1	39.9
	8.5	.7	79.1	4.6	7.2
Female	5	1	68	5	67
	8.8	1.0	92.3	5.9	38.1
	3.4	.7	46.6	3.4	45.9
Stratified					
20-29					
Male	0	0	1	0	0
	.5	0	.5	0	0
	0.0	0.0	100.0	0.0	0.0
Female	1	0	0	0	0
	.5	0	.5	0	0
	100.0	0.0	0.0	0.0	0.0
30-39					
Male	3	0	7	1	0
	2.8	0	7.3	.9	0
	27.3	0.0	63.6	9.1	0.0
Female	0	0	1	0	0
	.3	0	.7	.1	0
	0.0	0.0	100.0	.5	0.0
40-49					
Male	4	0	21	1	0
	3.4	0	21.8	.8	0
	15.4	0.0	80.8	3.8	0.0
Female	0	0	5	0	0
	.6	0	4.2	.2	0
	0.0	0.0	100.0	0.0	0.0

50-59

Male	4	0	35	5	3
	3.6	0	36.3	3.6	3.6
	8.5	0.0	74.5	10.6	6.4
Female	1	0	16	0	2
	1.4	0	14.7	1.4	1.4
	5.3	0.0	84.2	0.0	10.5

60-69

Male	1	1	37	0	1
	1.0	1.0	28.4	1.4	8.2
	2.5	2.5	92.5	0.0	2.5
Female	1	1	22	3	16
	1.0	1.0	30.6	1.6	8.8
	2.3	2.3	51.2	7.0	37.2

70-79

Male	1	0	14	0	6
	.6	0	9.0	.6	10.8
	4.8	0.0	66.7	0.0	28.6
Female	1	0	15	2	29
	1.4	0	20.0	1.4	24.2
	2.1	0.0	31.9	4.3	61.7

80-89

Male	0	0	3	0	1
	.1	0	1.5	0	2.4
	0.0	0.0	75.0	0.0	25.0
Female	1	0	9	0	19
	.9	0	10.5	0	17.6
	3.4	0.0	31.0	0.0	65.5

90-99

Male	0	0	3	0	0
	0	0	2.3	0	.8
	0.0	0.0	100.0	0.0	0.0
Female	0	0	0	0	1
	0	0	.8	0	.3
	0.0	0.0	0.0	0.0	100.0

Chi Square Analysis - Marital Status

	Chi Square	Degrees of Freedom	Significance	Min. E. F.	Cells With E.F. LT 5
Nonstratified					
	58.825	4	0.000	.977	2 of 10
Stratified					
20-29	FISHER'S EXACT TEST		one tail (.761)	two tail (1.000)	
30-39	.545	2	.761	.083	5 of 6
40-49	1.146	2	.564	.161	5 of 6
50-59	2.682	3	.443	1.439	6 of 8
60-69	19.967	4	.001	.964	6 of 10
70-79	8.442	3	.038	.618	4 of 8
80-89	2.959	2	.288	.121	4 of 6
90-99	FISHER'S EXACT TEST		one tail (.250)	two tail (.250)	

APPENDIX D. EMPLOYMENT STATUS

Descriptive Statistics - Employment Status

		Not Employed	Employed
Nonstratified			
	Male	65	87
		99.1	52.9
		42.8	57.2
	Female	130	17
		95.9	51.1
		88.4	11.6
Stratified			
20-29			
	Male	0	1
		0.0	1.0
		0.0	100.0
	Female	0	1
		0	1.0
		0.0	100.0
30-39			
	Male	0	11
		.9	10.1
		0.0	100.0
	Female	1	0
		.1	.9
		100.0	0.0
40-49			
	Male	3	23
		3.4	22.6
		11.5	88.5
	Female	1	4
		.6	4.4
		20.0	80.0

50-59			
	Male	8	38
		14.2	31.8
		17.4	82.6
	Female	12	7
		5.8	13.2
		63.2	36.8
60-69			
	Male	26	14
		31.4	8.6
		65.0	35.0
	Female	40	4
		34.6	9.4
		90.9	9.1
70-79			
	Male	21	0
		21.0	0
		100.0	0.0
	Female	47	0
		47.0	0
		100.0	0.0
80-89			
	Male	4	0
		3.9	.1
		100.0	0.0
	Female	28	1
		28.1	.9
		96.6	3.4
90-99			
	Male	3	0
		3.0	0
		100.0	0.0
	Female	1	0
		1.0	0
		100.0	0.0

Chi Square Analysis - Employment Status

	Chi Square	Degrees of Freedom	Significance	Min E.F.	Cells With E.F. LT 5
Nonstratified					
	66.719	1	.000	51.130	none
	68.718	1	.000	(Before Yates Correction)	
Stratified					
20-29	---	---	---	---	----
30-39	FISHER'S EXACT TEST	one tail (.083)	two tail (.083)		
40-49	0.000	1	1.000	.645	3 of 4
	.267	1	.605	(Before Yates Correction)	
50-59	11.160	1	.001	5.846	none
	13.221	1	.000	(Before Yates Correction)	
60-69	6.886	1	.009	8.571	none
	8.354	1	.004	(Before Yates Correction)	
70-79	---	---	---	---	---
80-89	0.000	1	1.000	.121	3 of 4
	.142	1	.706	(Before Yates Correction)	
90-99	---	---	---	---	---

APPENDIX E. FAMILY HISTORY OF MI

Descriptive Statistics - Family History Of MI

		Not Present	Present
Nonstratified			
	Male	84	67
		76.0	75.0
		55.6	44.4
	Female	64	79
		72.0	71.0
		44.8	55.2
Stratified			
20-29			
	Male	0	1
		0	1.0
		0.0	100.0
	Female	0	1
		0	1.0
		0.0	100.0
30-39			
	Male	8	3
		7.3	3.7
		72.7	27.3
	Female	0	1
		.7	.3
		0.0	100.0
40-49			
	Male	14	12
		11.7	14.3
		53.8	46.2
	Female	0	5
		2.3	2.7
		0.0	100.0

50-59			
	Male	22	25
		22.1	24.9
		46.8	53.2
	Female	9	10
		8.9	10.1
		47.4	52.6
60-69			
	Male	18	22
		15.2	24.8
		45.0	55.0
	Female	14	30
		16.8	27.2
		31.8	68.2
70-79			
	Male	16	4
		13.1	[illegible]
		80.0	[illegible]
	Female	26	[illegible]
		28.9	[illegible]
		59.1	[illegible]
80-89			
	Male	4	0
		2.3	1.8
		100.0	0.0
	Female	14	14
		15.8	12.3
		50.0	50.0
90-99			
	Male	2	0
		2.0	0
		100.0	0.0
	Female	1	0
		1.0	0
		100.0	0.0

Chi Square Analysis - Family History of MI

	Chi Square	Degrees of Freedom	Significance	Min E.F.	Cells With E.F. LT 5
Nonstratified					
	3.053	1	.081	71.014	none
	3.474	1	.062	(Before Yates Correction)	
Stratified					
20-29	---	---	---	---	---
30-39	FISHER'S EXACT TEST		one tail (.333)	two tail (.333)	
40-49	2.976	1	.085	2.258	2 of 4
	4.910	1	.027	(Before Yates Correction)	
50-59	0.000	1	1.000	8.924	none
	.002	1	.967	(Before Yates Correction)	
60-69	1.035	1	.309	15.248	none
	1.544	1	.214	(Before Yates Correction)	
70-79	1.819	1	.178	6.875	none
	2.665	1	.103	(Before Yates Correction)	
80-89	1.814	1	.178	1.750	2 of 4
	3.556	1	.059	(Before Yates Correction)	
90-99	---	---	---	---	---

APPENDIX F. HISTORY OF CARDIOVASCULAR DISEASE

Descriptive Statistics -- History of Cardiovascular Disease

	None	Angina	CVA	Previous Cardiac Sx	Previous MI	CHF
Nonstratified						
Male	91	26	2	2	32	0
	76.0	28.0	4.6	3.6	35.2	5.6
	59.5	17.0	1.3	1.3	20.9	0.0
Female	58	29	7	5	37	11
	73.0	26.9	4.4	3.4	33.8	5.4
	39.5	19.7	4.8	3.4	25.2	7.5
Stratified						
20-29						
Male	1	0	0	0	0	0
	1.0	0	0	0	0	0
	100.0	0.0	0.0	0.0	0.0	0.0
Female	1	0	0	0	0	0
	1.0	0	0	0	0	0
	100.0	0.0	0.0	0.0	0.0	0.0
30-39						
Male	8	2	0	0	1	0
	7.3	2.8	0	0	.9	0
	72.7	18.2	0.0	0.0	9.1	0.0
Female	0	1	0	0	0	0
	.7	.3	0	0	.1	0
	0.0	100.0	0.0	0.0	0.0	0.0
40-49						
Male	19	4	0	0	3	0
	17.6	3.4	0	.8	4.2	0
	73.1	15.4	0.0	0.0	11.5	0.0
Female	2	0	0	1	2	0
	3.4	.6	0	.2	.8	0
	40.0	0.0	0.0	20.0	40.0	0.0

50-59

Male	27	7	0	0	13	0
	27.8	6.4	0	.7	12.1	0
	57.4	14.9	0.0	0.0	27.7	0.0
Female	12	2	0	1	4	0
	11.2	2.6	0	.3	4.9	0
	63.2	10.5	0.0	5.3	21.1	0.0

60-69

Male	21	8	1	1	9	0
	18.6	8.1	1.9	1.9	9.0	.5
	52.5	20.0	2.5	2.5	22.5	0.0
Female	18	9	3	3	10	1
	20.4	8.9	2.1	2.1	10.0	.5
	40.9	20.5	6.8	6.8	22.7	2.3

70-79

Male	10	5	0	1	5	0
	7.7	3.4	.9	.3	6.2	2.5
	47.6	23.8	0.0	4.8	23.8	0.0
Female	15	6	[illegible]	0	15	8
	17.3	7.6		.7	13.8	5.5
	31.9	12.8		0.0	31.9	17.0

80-89

Male	3	0	0	0	1	0
	1.6	1.3	.1	0	.8	.1
	75.0	0.0	0.0	0.0	25.0	0.0
Female	10	11	1	0	6	1
	11.4	9.7	.9	0	6.2	.9
	34.5	37.9	3.4	0.0	20.7	3.4

90-99

Male	2	0	1	0	0	0
	1.5	0	.8	0	0	.8
	66.7	0.0	33.3	0.0	0.0	0.0
Female	0	0	0	0	0	1
	.5		.3	0	0	.3
	0.0	0.0	0.0	0.0	0.0	100.0

Chi Square Analysis - History of CV Disease

	Chi Square	Degrees of Freedom	Significance	Min E.F.	Cells With E.F. LT 5
Nonstratified					
	22.787	5	.000	3.430	4 of 12
Stratified					
20-29	-	---	---	---	---
30-39	3.272	2	.195	.083	5 of 6
40-49	8.753	3	.032	.161	7 of 8
50-59	2.967	3	.397	.288	4 of 8
60-69	3.159	5	.676	.476	6 of 12
70-79	9.545	5	.089	.309	6 of 12
80-89	3.289	4	.511	.121	7 of 10
90-99	4.000	2	.135	.250	6 of 6

APPENDIX G. CIGARETTE SMOKING STATUS

Descriptive Statistics - Cigarette Smoking Status

		Never Smoked	Past Smoker	Current Smoker
Nonstratified				
	Male	22	52	71
		53.2	42.1	55.7
		18.5	34.4	47.0
	Female	77	31	39
		51.8	40.9	54.3
		52.4	21.1	26.5
Stratified				
20-29				
	Male	1	0	0
		.5	0	.5
		100.0	0.0	0.0
	Female	0	0	1
		.5	0	.5
		0.0	0.0	100.0
30-39				
	Male	0	1	10
		0	.9	10.1
		0.0	9.1	90.9
	Female	0	0	1
		0	.1	.9
		0.0	0.0	100.0
40-49				
	Male	3	7	16
		3.4	5.9	16.8
		11.5	26.9	61.5
	Female	1	0	4
		.6	1.1	3.2
		20.0	0.0	80.0

50-59				
	Male	8	11	28
		10.7	11.4	24.9
		17.0	23.4	59.6
	Female	7	5	7
		4.3	4.6	10.1
		36.8	26.3	36.8
60-69				
	Male	6	22	10
		11.6	14.4	12.0
		15.8	57.9	26.3
	Female	19	9	16
		13.4	16.6	14.0
		43.2	20.5	36.4
70-79				
	Male	6	9	6
		10.8	6.2	4.0
		28.6	42.9	28.6
	Female	29	11	7
		24.2	13.8	9.0
		61.7	23.4	14.9
80-89				
	Male	2	1	1
		2.7	.8	.5
		50.0	25.0	25.0
	Female	20	6	3
		19.3	6.2	3.6
		69.0	20.7	10.3
90-99				
	Male	2	1	0
		2.3	.8	0
		66.7	33.3	0.0
	Female	1	0	0
		.8	.3	0
		100.0	0.0	0.0

Chi Square Analysis - Cigarette Smoking Status

	Chi Square	Degrees of Freedom	Significance	Min E.F.	Cells With E.F. LT 5
Nonstratified					
	37.442	2	.000	40.943	none
Stratified					
20-29	FISHER'S EXACT TEST	one tail (.500)	two tail (1.000)		
30-39	FISHER'S EXACT TEST	one tail (.917)	two tail (1.000)		
40-49	1.800	2	.407	.645	4 of 6
50-59	3.705	2	.157	4.318	2 of 6
60-69	13.228	2	.001	11.585	none
70-79	6.383	2	.041	4.015	1 of 6
80-89	.843	2	.656	.485	4 of 6
90-99	FISHER'S EXACT TEST	one tail (.750)	two tail (1.000)		

APPENDIX H. OBESITY

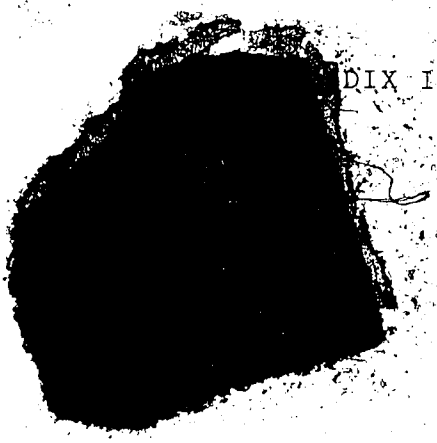
Descriptive Statistics - Obesity Status

		Not Present	Present
Nonstratified			
	Male	90	63
		90.7	62.3
		58.8	41.2
	Female	86	58
		85.3	58.7
		59.7	40.3
Stratified			
20-29			
	Male	1	0
		.5	.5
		100.0	0.0
	Female	0	1
		.5	.5
		0.0	100.0
30-39			
	Male	3	8
		3.7	7.3
		27.3	72.7
	Female	1	0
		.3	.7
		100.0	0.0
40-49			
	Male	13	13
		14.3	11.7
		50.0	50.0
	Female	4	1
		2.7	2.3
		80.0	20.0

50-59			
	Male	22	25
		20.7	26.3
		46.8	53.2
	Female	7	12
		8.3	10.7
		36.8	63.2
60-69			
	Male	27	13
		24.6	15.4
		67.5	32.5
	Female	24	19
		26.4	16.6
		55.8	44.2
70-79			
	Male	18	3
		14.1	6.9
		85.7	14.3
	Female	27	19
		30.0	15.1
		58.7	41.3
80-89			
	Male	3	1
		3.0	1.0
		75.0	25.0
	Female	21	7
		21.0	7.0
		75.0	25.0
90-99			
	Male	3	0
		3.0	0
		100.0	0.0
	Female	1	0
		1.0	0
		100.0	0.0

Chi Square Analysis - Obesity Status

	Chi Square	Degrees of Freedom	Significance	Min E.F	Cells With E.F. LT 5
Nonstratified					
	.002	1	.969	58.667	none
	.025	1	.875	(Before Yates Correction)	
Stratified					
20-29	---	---	---	---	---
30-39	FISHER'S EXACT TEST		one tail (.333)	two tail (.333)	
40-49	.533	1	.457	2.258	2 of 4
	.524	1	.217	(Before Yates Correction)	
50-59	.216	1	.642	8.348	none
	.546	1	.460	(Before Yates Correction)	
60-69	.752	1	.386	15.422	none
	1.195	1	.279	(Before Yates Correction)	
70-79	3.626	1	.057	6.896	none
	7.772	1	.029	(Before Yates Correction)	
80-89	0.000	1	1.000	1.000	2 of 4
	0.000	1	1.000	(Before Yates Correction)	
90-99	---	---	---	---	---



DIX I. DIABETES STATUS

50-59						
Male	11	8	7	16	3	2
	11.4	7.1	6.4	17.1	2.8	2.1
	23.4	17.0	14.9	34.0	6.4	4.3
Female	5	2	2	8	1	1
	4.6	2.9	2.6	6.9	1.2	.9
	26.3	10.5	10.5	42.1	5.3	5.3
60-69						
Male	5	8	5	18	2	2
	6.7	3.8	6.7	16.7	2.4	3.8
	12.5	20.0	12.5	45.0	5.0	5.0
Female	9	0	9	17	3	6
	7.3	4.2	7.3	18.3	2.6	4.2

	Chi Square
Nonstratified	
	10.514
Stratified	
20-29	---
30-39	.364
40-49	.692
50-59	.913
60-69	13.352
70-79	5.279
80-89	9.039
90-99	4.000

Descriptive Statistics - Complication Following MI

	None	Intubation	Cardiogenic Shock	Cardiac Arrest	Anoxic Brain Insult
Nonstratified					
Male	126	13	8	6	0
	108.6	13.8	14.8	12.2	3.6
	82.4	8.5	5.2	3.9	0.0
Female	87	14	21	18	7
	104.2	13.2	14.2	11.8	3.4
	59.2	9.5	14.3	12.2	4.8

50-59					
Male	39	5	1	2	0
	38.5	4.3	2.8	1.4	0
	83.0	10.6	2.1	4.3	0.0
Female	15	1	3	0	0
	15.5	1.7	1.2	.6	0
	78.9	5.3	15.8	0.0	0.0
60-69					
Male	35	2	1	2	0
	30.5	3.8	1.9		

Nonstrat

Stratifi

20-29

30-39

40-49

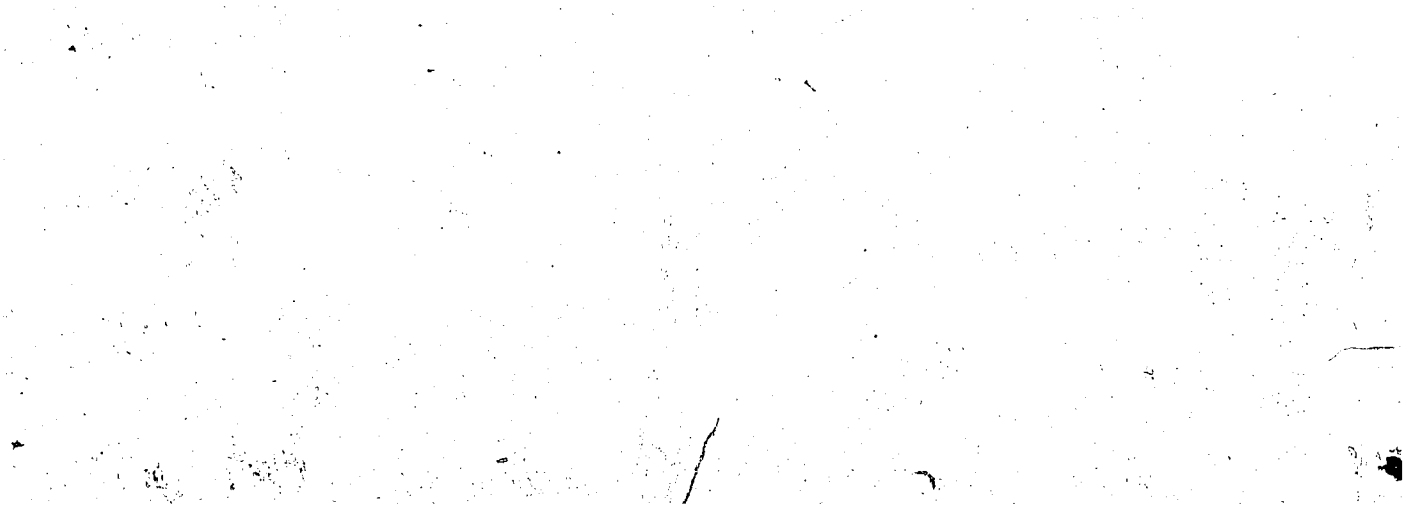
50-59

60-69

70-79

80-89

90-99



Descriptive Statistics - Mortality

Nonstratified

	No	Yes
Male	137	16
	128.0	27.0
	89.5	10.5
Female	110	37

Descriptive Statistics for Uric Acid in Umol/l - Males

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	153	412.49	402.00	376.00	201.00	896.00	695.00	106.00	11236.62	8.68
Stratified										
20-29										
	1	251.00	251.00	251.00	251.00	251.00	0.00	0.00	0.00	.00
30-39										
	11	351.46	368.00	398.00	213.00	420.00	207.00	67.20	4516.07	20.26
40-49										
	26	407.81	389.50	387.00	266.00	565.00	299.00	77.37	5886.64	15.17
50-59										
	47	418.55	411.00	311.00	201.00	869.00	695.00	117.65	13840.77	17.16
60-69										
	40	429.18	416.50	244.00	244.00	748.00	504.00	114.17	13034.64	18.52
70-79										
	21	407.21	388.00	242.00	242.00	592.00	350.00	101.31	10264.62	23.24
80-89										
	4	440.50	404.50	300.00	300.00	653.00	353.00	151.78	23037.67	75.89
90-99										
	3	420.33	410.00	350.00	350.00	501.00	151.00	76.03	5780.33	43.90

Descriptive Statistics for Uric Acid in Umol/l - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	147	421.32	410.00	480.00	155.00	886.00	731.00	153.63	23602.57	12.85
Stratified										
20-29	1	224.00	224.00	224.00	224.00	224.00	224.00	0.00	0.00	0.00
30-39	1	291.00	291.00	291.00	291.00	291.00	291.00	0.00	0.00	0.00
40-49	5	406.00	440.00	284.00	284.00	490.00	206.00	88.09	7760.50	49.40
50-59	19	332.74	306.00	306.00	155.00	567.00	412.00	115.27	13287.54	26.45
60-69	44	366.81	346.00	248.00	200.00	713.00	513.00	115.62	13369.91	18.06
70-79	47	477.11	473.50	497.00	244.00	886.00	642.00	166.77	27812.10	24.59
80-89	29	486.66	483.00	283.00	283.00	865.00	582.00	155.94	24317.16	28.96
90-99	1	283.00	283.00	283.00	283.00	283.00	0.00	0.00	0.00	0.00

T-Test Analysis - Uric Acid Level in Umol/l

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	2.10	.000	-.57	290	.567	-.57	251.11	.570
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.86	10	.409	2.98	10.00	.014
40-49	1.30	.597	.05	29	.963	.04	5.26	.967
50-59	1.04	.964	2.70	64	.827	.25	46.57	.801
60-69	1.03	.941	2.41	77	.018	2.41	76.68	.018
70-79	2.71	.024	-1.70	63	.095	-2.07	53.85	.044
80-89	1.06	1.000	-.56	2	.582	-.57	3.93	.601
90-99	0.00	1.000	1.56	2	.258	3.13	2.00	.089

Correlation Coefficient, R^2, Significance - Uric Acid with Age

	r	R^2	Significance
Male	.149	.022	.035
Female	.330	.109	.000

APPENDIX L. TYPE OF MI

	Anterior	Anterior Lateral	Anterior Septal	Inferior	Posterior	Sub-Endo
Nonstratified						
Male	30	21	19	68	6	9
	27.6	16.9	21.5	64.0	6.7	16.4
	19.6	13.7	12.4	44.4	3.9	5.9
Female	24	12	23	57	7	23
	26.4	16.1	20.5	61.0	6.3	15.6
	16.4	8.2	15.8	39.0	4.8	15.8
Stratified						
20-29						
Male	0	0	0	1	0	0
	0	0	0	1.0	0	0
	0.0	0.0	0.0	100.0	0.0	0.0
Female	0	0	0	1	0	0
	0	0	0	1.0	0	0
	0.0	0.0	0.0	100.0	0.0	0.0
30-39						
Male	1	0	1	8	0	1
	.9	0	.9	8.3	0	.9
	9.1	0.0	9.1	72.7	0.0	9.1
Female	0	0	0	1	0	0
	.1	0	.1	.8	0	.1
	0.0	0.0	0.0	100.0	0.0	0.0
40-49						
Male	8	1	2	15	0	0
	8.4	.8	1.7	15.1	0	0
	30.8	3.8	7.7	57.7	0.0	0.0
Female	2	0	0	3	0	0
	1.6	.2	.3	2.9	0	0
	40.0	0.0	0.0	60.0	0.0	0.0

50-59						
Male	11	8	7	16	3	2
	11.4	7.1	6.4	17.1	2.8	2.1
	23.4	17.0	14.9	34.0	6.4	4.3
Female	5	2	2	8	1	1
	4.6	2.9	2.6	6.9	1.2	.9
	26.3	10.5	10.5	42.1	5.3	5.3
60-69						
Male	5	8	5	18	2	2
	6.7	3.8	6.7	16.7	2.4	3.8
	12.5	20.0	12.5	45.0	5.0	5.0
Female	9	0	9	17	3	6
	7.3	4.2	7.3	18.3	2.6	4.2
	20.5	0.0	20.5	38.6	6.8	13.6
70-79						
Male	5	3	4	8	0	1
	3.4	2.8	2.8	8.0	.9	3.1
	23.8	14.3	19.0	38.1	0.0	4.8
Female	6	6	5	18	3	9
	7.6	6.2	6.2	18.0	2.1	6.9
	12.8	1.8	10.6	38.3	6.4	19.1
80-89						
Male	0	0	0	2	1	1
	.3	.5	.8	1.4	.1	1.0
	0.0	0.0	0.0	50.0	25.0	25.0
Female	2	4	6	9	0	7
	1.8	3.5	5.3	9.6	.9	7.0
	7.1	14.3	21.4	32.1	0.0	25.0
90-99						
Male	0	1	0	2	0	0
	0	.8	.8	1.5	0	0
	0.0	33.3	0.0	66.7	0.0	0.0
Female	0	0	1	0	0	0
	0	.3	.3	.5	0	0
	0.0	0.0	100.0	0.0	0.0	0.0

Chi Square Analysis - Type of MI

	Chi Square	Degrees of Freedom	Significance	Min E.F	Cells With E.F. LT 5
Nonstratified					
	10.514	5	.062	6.348	none
Stratified					
20-29	---	---	---	---	---
30-39	.364	3	.948	.083	7 of 8
40-49	.692	3	.875	.161	6 of 8
50-59	.913	5	.969	.864	7 of 12
60-69	13.352	5	.030	2.381	6 of 12
70-79	5.279	5	.038	.976	6 of 12
80-89	9.039	5	.108	.125	9 of 12
90-99	4.000	2	.135	.250	6 of 6

APPENDIX M. COMPLICATION FOLLOWING MI

Descriptive Statistics - Complication Following MI

	None	Intubation	Cardiogenic Shock	Cardiac Arrest	Anoxic Brain Insult
Nonstratified					
Male	126	13	8	6	0
	108.6	13.8	14.8	12.2	3.6
	82.4	8.5	5.2	3.9	0.0
Female	87	14	21	18	7
	104.2	13.2	14.2	11.8	3.4
	59.2	9.5	14.3	12.2	4.8
Stratified					
20-29					
Male	1	0	0	0	0
	1.0	0	0	0	0
	100.0	0.0	0.0	0.0	0.0
Female	1	0	0	0	0
	1.0	0	0	0	0
	100.0	0.0	0.0	0.0	0.0
30-39					
Male	11	0	0	0	0
	11.0	0	0	0	0
	100.0	0.0	0.0	0.0	0.0
Female	1	0	0	0	0
	1.0	0	0	0	0
	100.0	0.0	0.0	0.0	0.0
40-49					
Male	20	4	2	0	0
	20.1	4.2	1.7	0	0
	79.9	15.4	7.7	0.0	0.0
Female	4	1	0	0	0
	3.9	.8	.3	0	0
	80.0	20.0	0.0	0.0	0.0

50-59

Male	39	5	1	2	0
	38.5	4.3	2.8	1.4	0
	83.0	10.6	2.1	4.3	0.0
Female	15	1	3	0	0
	15.5	1.7	1.2	.6	0
	78.9	5.3	15.8	0.0	0.0

60-69

Male	35	2	1	2	0
	30.5	3.8	1.9	3.3	.5
	87.5	5.0	2.5	5.0	0.0
Female	29	6	3	5	1
	33.5	4.2	2.1	3.7	.5
	65.9	13.6	6.8	11.4	2.3

70-79

Male	16	1	3	1	0
	11.7	1.2	3.7	3.1	1.2
	76.2	4.8	14.3	4.8	0.0
Female	22	3	9	9	4
	26.3	2.8	8.3	6.9	2.8
	46.8	6.4	19.1	19.1	8.5

80-89

Male	3	0	0	1	0
	2.1	.4	.7	.6	.2
	75.0	0.0	0.0	25.0	0.0
Female	14	3	6	4	2
	14.9	2.6	5.3	4.4	1.8
	48.3	10.3	20.7	13.8	6.9

90-99

Male	1	1	1	0	0
	1.5	.8	.8	0	0
	33.3	33.3	33.3	0.0	0.0
Female	1	0	0	0	0
	.5	.3	.3	0	0
	100.0	0.0	0.0	0.0	0.0

Chi Square Analysis - Complication Following MI

	Chi Square	Degrees of Freedom	Significance	Min E.F.	Cells With E.F. LT 5
Nonstratified					
	25.896	4	.000	3.430	2 of 10
Stratified					
20-29	---	---	---	---	---
30-39	---	---	---	---	---
40-49	.445	2	.801	.323	5 of 6
50-59	5.432	3	.143	.576	6 of 8
60-69	5.671	4	.225	.476	8 of 10
70-79	6.332	4	.176	1.236	6 of 10
80-89	2.296	4	.682	.242	8 of 10
90-99	1.333	2	.513	.250	6 of 6

APPENDIX N. MORTALITY

Descriptive Statistics - Mortality

Nonstratified

		No	Yes
	Male	137	16
		128.0	27.0
		89.5	10.5
	Female	110	37
		121.0	26.0
		74.8	25.2

Stratified

20-29			
	Male	1	0
		1.0	0
		100.0	0.0
	Female	1	0
		1.0	0
		100.0	0.0
30-39			
	Male	11	0
		11.0	0
		100.0	0.0
	Female	1	0
		1.0	0
		100.0	0.0
40-49			
	Male	23	3
		23.5	2.5
		88.5	11.5
	Female	5	0
		4.5	.5
		100.0	0.0

50-59			
	Male	44	3
		43.4	3.6
		93.6	6.4
	Female	17	2
		17.6	1.4
		89.5	10.5
60-69			
	Male	36	4
		34.8	4.2
		90.0	10.0
	Female	37	7
		38.2	5.8
		84.1	15.9
70-79			
	Male	17	4
		14.2	6.8
		81.0	19.0
	Female	29	18
		31.8	15.2
		61.7	38.3
80-89			
	Male	3	1
		2.7	1.3
		75.0	25.0
	Female	18	10
		19.3	9.7
		65.5	34.5
90-99			
	Male	2	1
		2.3	.8
		66.7	33.3
	Female	1	0
		.8	.3
		100.0	0.0

Chi Square Analysis - Mortality

	Chi Square	Degrees of Freedom	Significance	Min E.F.	Cells With E.F. LT 5
Nonstratifed					
	10.168	1	.001	25.970	none
	11.157	1	.001	(Before Yates Correction)	
Stratified					
20-29	---	---	---	---	---
30-39	---	---	---	---	---
40-49	0.000	1	1.000	.484	3 of 4
	.639	1	.424	(Before Yates Correction)	
50-59	.004	1	.950	1.439	3 of 4
	.332	1	.565	(Before Yates Correction)	
60-69	.228	1	.633	5.238	none
	.643	1	.422	(Before Yates Correction)	
70-79	1.657	1	.198	7.794	none
	2.458	1	.117	(Before Yates Correction)	
80-89	0.000	1	1.000	1.330	2 of 4
	.142	1	.706	(Before Yates Correction)	
90-99	FISHER'S EXACT TEST	one tail (.750)	two tail (1.000)		

APPENDIX O. LDH LEVEL

Descriptive Statistics for First Lactic Dehydrogenase in IU/1 - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	153	316.55	216.00	163.00	90.00	1545.00	1455.00	256.57	65826.82	20.81
Stratified										
20-29	1	294.00	294.00	294.00	294.00	294.00	294.00	0.00	0.00	0.00
30-39	11	434.73	310.00	90.00	90.00	1202.00	1112.00	363.45	132093.00	109.58
40-49	26	294.46	199.50	152.00	126.00	889.00	763.00	225.62	50904.00	44.25
50-59	47	418.55	411.00	311.00	201.00	896.00	695.00	117.65	13840.77	17.16
60-69	44	259.02	213.00	716.00	97.00	716.00	619.00	111.67	12471.09	16.84
70-79	21	308.29	233.00	130.00	130.00	953.00	823.00	214.18	45871.51	46.74
80-89	4	231.33	224.00	196.00	196.00	274.00	78.00	39.51	1561.33	22.81
90-99	3	222.00	205.00	175.00	175.00	286.00	111.00	57.42	3297.00	33.15

Descriptive Statistics for First Lactic Dehydrogenase in IU/1 - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	147	326.75	247.50	175.00	97.00	1655.00	1558.00	247.65	61324.71	20.50
Stratified										
20-29	1	246.00	246.00	246.00	246.00	246.00	0.00	0.00	0.00	0.00
30-39	1	161.00	161.00	161.00	161.00	161.00	0.00	0.00	0.00	0.00
40-49	5	651.20	420.00	175.00	175.00	1655.00	1480.00	617.75	381612.00	276.27
50-59	19	332.74	306.00	306.00	155.00	567.00	412.00	115.27	13787.54	26.45
60-69	40	353.38	212.50	128.00	128.00	1545.00	1417.00	304.54	92747.02	48.15
70-79	47	380.87	277.00	196.00	114.00	1482.00	1368.00	313.89	98528.69	46.28
80-89	29	333.17	291.00	123.00	123.00	875.00	752.00	183.81	33783.86	34.13
90-99	1	283.00	283.00	283.00	283.00	283.00	0.00	0.00	0.00	0.00

Descriptive Statistics for Second Lactic Dehydrogenase in IU/l - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	153	461.54	386.00	227.00	108.00	1805.00	1697.00	288.61	83296.07	28.49
Stratified										
20-29	1	356.00	356.00	356.00	356.00	356.00	0.00	0.00	0.00	0.00
30-39	11	447.91	325.00	114.00	114.00	789.00	675.00	260.34	67778.69	78.50
40-49	26	478.68	364.00	175.00	141.00	1010.00	869.00	299.76	89857.23	59.95
50-59	47	492.02	443.00	167.00	165.00	1251.00	1086.00	263.45	69351.98	38.41
60-69	40	460.95	369.00	108.00	108.00	1805.00	1697.00	367.06	134729.64	58.04
70-79	21	440.76	386.00	386.00	169.00	1074.00	905.00	211.63	44785.39	46.18
80-89	4	251.00	237.00	149.00	149.00	367.00	218.00	109.67	12028.00	63.32
90-99	3	290.00	289.00	267.00	267.00	314.00	47.00	23.52	553.00	13.58

Descriptive Statistics for Second Lactic Dehydrogenase in IU/1 - Females

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	147	434.53	359.00	180.00	158.00	1440.00	1282.00	253.68	64351.86	21.07
Sratified										
20-29	1	407.00	407.00	407.00	407.00	407.00	0.00	0.00	0.00	0.00
30-39	1	159.00	159.00	159.00	159.00	159.00	0.00	0.00	0.00	0.00
40-49	3	667.00	463.00	283.00	283.00	1422.00	1139.00	469.85	220755.50	210.12
50-59	19	368.39	302.50	160.00	160.00	716.00	556.00	183.00	33659.55	43.24
60-69	44	389.25	346.50	190.00	158.00	872.00	714.00	180.13	32444.94	27.16
70-79	47	486.20	364.00	178.00	178.00	1440.00	1262.00	321.31	103239.58	47.37
80-89	29	438.52	407.00	171.00	171.00	977.00	806.00	198.48	39393.76	36.86
90-99	1	283.00	283.00	283.00	283.00	283.00	0.00	0.00	0.00	0.00

Descriptive Statistics for Third Lactic Dehydrogenase in IU/1 - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	153	514.87	410.00	320.00	147.00	2115.00	1968.00	331.29	109752.00	27.51
Stratified										
20-29	1	300.00	300.00	300.00	300.00	300.00	0.00	0.00	0.00	0.00
30-39	11	425.09	374.00	240.00	190.00	796.00	606.00	227.08	51565.09	68.47
40-49	26	636.33	468.50	170.00	170.00	1605.00	1435.00	434.97	89196.93	88.79
50-59	47	541.01	441.00	164.00	164.00	1359.00	1186.00	291.93	85220.84	43.04
60-69	40	486.33	394.50	147.00	147.00	2115.00	1968.00	389.06	151364.00	64.84
70-79	21	491.52	414.00	210.00	210.00	961.00	751.00	210.00	44097.86	45.83
80-89	4	267.33	235.00	157.00	157.00	410.00	253.00	129.56	16786.33	74.80
90-99	2	296.00	295.00	285.00	285.00	308.00	23.00	11.52	133.00	6.67

Descriptive Statistics for Third Lactic Dehydrogenase in IU/1 - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	147	482.98	397.00	193.00	152.00	2455.00	2303.00	347.46	120725.71	29.16
Stratified										
20-29										
	1	358.00	358.00	358.00	358.00	358.00	0.00	0.00	0.00	0.00
30-39										
	1	170.00	170.00	170.00	170.00	170.00	0.00	0.00	0.00	0.00
40-49										
	5	588.40	380.00	336.00	336.00	1395.00	1059.00	455.23	207232.30	203.58
50-59										
	19	339.13	255.00	257.00	166.00	839.00	673.00	190.75	36383.72	47.67
60-69										
	44	488.05	416.50	152.00	152.00	1463.00	1311.00	299.07	89441.30	45.09
70-79										
	47	530.85	416.00	269.00	166.00	2455.00	2289.00	466.00	217153.91	68.71
80-89										
	29	482.93	432.50	193.00	193.00	1010.00	817.00	225.05	51053.48	42.70
90-99										
	1	272.00	272.00	272.00	272.00	272.00	0.00	0.00	0.00	0.00

T-Test Analysis - First Lactic Dehydrogenase in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.07	.668	-.35	296	.727	-.35	295.99	.727
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.72	10	.487	2.50	10.00	.032
40-49	7.50	.001	-2.35	29	.026	-1.28	4.21	.268
50-59	1.97	.118	.22	64	.827	.25	46.57	.801
60-69	7.44	.000	1.90	82	.061	1.83	48.46	.073
70-79	2.15	.066	-.96	65	.340	-1.10	54.96	.275
80-89	21.64	.090	-.94	30	.363	-2.48	15.45	.025
90-99	0.00	1.000	-.71	2	.552	-.142	2.00	.292

T-Test Analysis - Second Lactic Dehydrogenase in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.29	.120	0.00	294	1.000	0.00	291.74	.393
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	1.06	10	.313	3.68	10.00	.004
40-49	2.46	.146	-1.17	28	.253	.86	4.67	.431
50-59	2.06	.106	1.83	63	.073	2.14	44.24	.038
60-69	4.15	.000	1.15	82	.252	1.12	55.53	.268
70-79	2.31	.046	-.59	65	.557	-.16	56.46	.495
80-89	3.28	.521	-1.60	30	.121	-2.56	3.56	.070
90-99	0.00	1.000	.88	2	.470	1.77	2.00	.219

T-Test Analysis - Third Lactic Dehydrogenase in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.10	.570	.80	285	.427	.80	283.67	.427
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	1.08	10	.307	3.73	10.00	.004
40-49	1.10	.765	.22	27	.826	.22	5.63	.837
50-59	2.34	.075	2.57	60	.031	3.14	40.45	.003
60-69	1.69	.101	-.02	78	.982	-.02	64.71	.983
70-79	4.92	0.000	-.37	65	.713	-.48	65.00	.636
80-89	3.04	.555	-1.61	29	.119	-2.50	3.49	.076
90-99	0.00	1.000	1.80	2	.213	3.60	2.00	.069

Correlation Coefficient, R^2 , Significance - LDH with Age

	r	R^2	Significance
First Test			
Male	-.076	.004	.210
Female	.030	.001	.341
Second Test			
Male	-.102	.010	.106
Female	.040	.002	.317
Third Test			
Male	-.121	.015	.074
Female	.085	.007	.155

APPENDIX P. LD1 PROPORTION

Descriptive Statistics for First LD One Fraction (Proportion) - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	153	.27	.23	.21	.13	.53	.40	.11	.01	.02
Stratified										
20-29	1	.28	.28	.28	.28	.28	0.00	0.00	0.00	0.00
30-39	11	.27	.19	.18	.12	.55	.43	.15	.02	.05
40-49	26	.27	.23	.21	.13	.53	.40	.11	.01	.02
50-59	47	.28	.24	.21	.14	.54	.40	.09	.01	.01
60-69	40	.30	.28	.21	.14	.53	.39	.12	.01	.02
70-79	21	.28	.24	.21	.17	.54	.37	.10	.01	.02
80-89	4	.26	.26	.20	.20	.31	.11	.06	.00	.03
90-99	3	.25	.25	.23	.23	.26	.03	.02	.00	.00

Descriptive Statistics for First LD One Fraction (Proportion) - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	146	.26	.25	.25	.05	.50	.45	.08	.01	.04
Stratifed										
20-29	1	.40	.40	.40	.40	.40	0.00	0.00	0.00	0.00
30-39	1	.25	.25	.25	.25	.25	0.00	0.00	0.00	0.00
40-49	5	.27	.25	.13	.13	.47	.34	.13	.02	.06
50-59	19	.25	.24	.20	.13	.39	.26	.07	.01	.02
60-69	44	.27	.26	.21	.14	.50	.36	.09	.01	.01
70-79	47	.26	.25	.22	.05	.46	.41	.10	.01	.01
80-89	29	.27	.26	.22	.17	.49	.32	.07	.00	.01
90-99	1	.35	.35	.35	.35	.35	0.00	0.00	0.00	0.00

Descriptive Statistics for Second LD One Fraction (Proportion) - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratifed										
	151	.36	.36	.36	.14	.55	.41	.10	.01	.01
Stratified										
20-29	1	.41	.41	.41	.41	.41	0.00	0.00	0.00	0.00
30-39	11	.27	.19	.18	.12	.55	.43	.15	.02	.05
40-49	25	.34	.36	.18	.16	.52	.36	.11	.01	.02
50-59	47	.37	.36	.30	.14	.53	.39	.10	.01	.02
60-69	39	.37	.38	.46	.17	.53	.36	.10	.01	.02
70-79	21	.36	.37	.32	.18	.55	.37	.10	.01	.02
80-89	3	.26	.26	.20	.20	.31	.11	.06	.00	.03
90-99	3	.25	.25	.23	.23	.26	.03	.02	.00	.01

Descriptive Statistics for Second LD One Fraction (Proportion) - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratifed										
	145	.37	.34	.34	.10	.53	.43	.32	.10	.04
Stratified										
20-29	1	.50	.50	.50	.50	.50	0.00	0.00	0.00	0.00
30-39	1	.23	.23	.23	.23	.23	0.00	0.00	0.00	0.00
40-49	5	.35	.41	.14	.14	.47	.33	.14	.02	.06
50-59	18	.34	.32	.28	.24	.50	.26	.08	.01	.02
60-69	44	.37	.36	.33	.19	.53	.34	.09	.01	.01
70-79	47	.26	.25	.22	.05	.46	.41	.10	.01	.01
80-89	29	.34	.34	.36	.21	.48	.27	.07	.01	.01
90-99	1	.34	.34	.34	.34	.34	0.00	0.00	0.00	0.00

Descriptive Statistics for Third LD One Fraction (Proportion) - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	144	.38	.38	.42	.17	.58	.41	.09	.01	.01
Stratified										
20-29	1	.38	.38	.38	.38	.38	0.00	0.00	0.00	0.00
30-39	11	.36	.37	.50	.22	.50	.28	.10	.01	.03
40-49	24	.40	.41	.30	.18	.57	.39	.10	.01	.02
50-59	46	.40	.41	.38	.17	.58	.41	.09	.01	.01
60-69	35	.36	.37	.42	.20	.55	.35	.09	.01	.02
70-79	21	.38	.40	.40	.18	.57	.39	.09	.01	.02
80-89	3	.29	.26	.18	.18	.42	.24	.12	.02	.07
90-99	3	.31	.32	.32	.30	.32	.02	.01	.00	.01

Descriptive Statistics for Third LD one Fraction (Proportion) - Female

Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified									
142	.35	.36	.24	.07	.55	.48	.11	.01	.03
Stratified									
20-29									
1	.38	.38	.38	.38	.38	0.00	0.00	0.00	0.00
30-39									
1	.25	.25	.25	.25	.25	0.00	0.00	0.00	0.00
40-49									
5	.42	.42	.21	.21	.55	.34	.13	.02	.06
50-59									
16	.33	.32	.24	.21	.48	.27	.10	.01	.02
60-69									
44	.37	.38	.42	.20	.55	.35	.01	.01	.02
70-79									
46	.33	.33	.28	.07	.52	.45	.11	.01	.02
80-89									
28	.36	.36	.36	.20	.52	.32	.09	.01	.02
90-99									
1	.32	.32	32	.32	.32	0.00	0.00	0.00	0.00

T-Test Analysis - First LD One Fraction (Proportion)

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.54	.009	1.29	295	.198	1.29	285.76	.197
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.11	10	.918	.37	10.00	.722
40-49	1.41	.516	-.04	29	.968	-.04	5.15	.973
50-59	1.88	.146	1.10	64	.274	1.26	45.46	.214
60-69	1.80	.064	1.51	81	.134	1.49	69.75	.142
70-79	1.07	.824	.66	65	.509	.66	37.69	.515
80-89	1.46	.974	-.32	30	.754	-.37	2.65	.739
90-99	0.00	1.000	-5.86	2	.028	-11.72	2.00	.007

T-Test Analysis - Second LD One Fraction (Proportion)

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Sepatate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.23	.212	1.13	293	.261	1.13	291.60	.261
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.74	10	.477	2.56	10.00	.028
40-49	1.62	.403	-.19	28	.850	-.16	5.03	.878
50-59	1.61	.283	1.25	63	.215	1.39	39.00	.172
60-69	1.32	.371	-.14	81	.891	-.14	75.86	.892
70-79	1.16	.730	1.14	65	.260	1.17	41.68	.249
80-89	1.05	.729	-1.57	30	.126	-1.54	2.41	.242
90-99	0.00	1.000	-5.86	2	.028	11.72	2.00	.007

T-Test Analysis - Third LD One Fraction (Proportion)

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.18	.314	2.41	284	.016	2.41	281.28	.016
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	1.10	10	.297	3.81	10.00	.003
40-49	.82	.407	-.37	27	.715	-.31	5.08	.766
50-59	1.17	.565	2.42	60	.019	2.33	24.52	.028
60-69	1.11	.761	-.57	77	.570	-.57	74.57	.568
70-79	1.61	.251	1.86	65	.068	2.03	48.50	.048
80-89	2.08	.290	-1.30	29	.204	-.96	2.21	.431
90-99	0.00	1.000	-.50	2	.667	-1.00	2.00	.423

Correlation Coefficients, R2 , Significance - LD1 with Age

	r	R2	Significance
First Test			
Male	-.009	.000	.456
Female	.013	.000	.436
Second Test			
Male	-.032	.011	.349
Female	-.098	.010	.122
Third Test			
Male	-.140	.020	.047
Female	-.051	.002	.273

APPENDIX Q. CPK-MB LEVEL

Descriptive Statistics For First Creatine Phosphokinase Multiband in IU/L - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	152	76.11	39.00	8.00	8.00	612.00	604.00	108.80	11837.54	8.85
Stratified										
20-29										
	1	68.00	68.00	68.00	68.00	68.00	0.00	0.00	0.00	0.00
30-39										
	11	128.45	62.00	8.00	8.00	549.00	541.00	163.17	26624.07	49.20
40-49										
	25	68.12	30.00	8.00	8.00	612.00	604.00	120.33	14479.28	24.07
50-59										
	47	58.72	40.00	8.00	8.00	320.00	312.00	63.44	4024.34	9.25
60-69										
	40	95.15	39.50	8.00	8.00	605.00	597.00	138.28	19121.62	21.86
70-79										
	21	72.19	28.00	8.00	8.00	280.00	272.00	86.80	7534.26	18.94
80-89										
	3	8.33	8.00	8.00	8.00	9.00	1.00	.58	.33	.33
90-99										
	3	51.67	27.00	16.00	16.00	112.00	96.00	52.54	2760.33	30.33

Descritpive Statistics for First Creatine Phosphokinase Multiband in IU/1 - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified.										
	146	64.31	39.50	8.00	8.00	420.00	412.00	75.61	5716.20	6.26
Stratified										
20-29	1	72.00	72.00	72.00	72.00	72.00	0.00	0.00	0.00	0.00
30-39	1	57.00	57.00	57.00	57.00	57.00	0.00	0.00	0.00	0.00
40-49	5	125.40	103.00	33.00	33.00	310.00	277.00	111.26	12378.30	49.76
50-59	19	47.53	24.00	11.00	8.00	220.00	212.00	53.11	2820.15	12.18
60-69	44	55.27	28.00	8.00	8.00	350.00	342.00	73.52	5405.41	11.08
70-79	46	60.17	36.00	8.00	8.00	280.00	272.00	63.28	4004.15	9.33
80-89	29	86.24	58.00	8.00	8.00	420.00	412.00	98.55	9712.69	18.30
90-99	1	26.00	26.00	26.00	26.00	26.00	0.00	0.00	0.00	0.00

Descriptive Statistics for Second Creatine Phosphokinase Multiband in IU/l - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	151	113.29	75.00	8.00	8.00	630.00	622.00	114.55	13120.80	9.32
Stratified										
20-29	1	72.00	72.00	72.00	72.00	72.00	0.00	0.00	0.00	0.00
30-39	11	87.64	74.00	8.00	8.00	298.00	290.00	85.36	7287.06	25.74
40-49	25	127.00	70.00	27.00	15.00	589.00	574.00	139.20	19376.58	27.84
50-59	47	120.32	95.00	8.00	8.00	555.00	547.00	116.00	13456.01	16.92
60-69	40	110.28	76.50	8.00	8.00	630.00	622.00	122.75	15067.23	19.40
70-79	21	124.38	100.00	74.00	20.00	346.00	326.00	89.54	8017.15	19.54
80-89	3	17.67	11.00	8.00	8.00	34.00	26.00	14.22	202.33	8.21
90-99	3	51.67	27.00	16.00	16.00	112.00	96.00	52.54	2760.33	30.33

Descriptive Statistics for Second Creatine Phosphokinase Multiband in IU/1 - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	146	92.53	66.50	24.00	8.00	450.00	442.00	89.18	7953.26	7.38
Stratified										
20-29	1	76.00	76.00	76.00	76.00	76.00	0.00	0.00	0.00	0.00
30-39	1	48.00	48.00	48.00	48.00	48.00	0.00	0.00	0.00	0.00
40-49	5	123.20	97.00	31.00	31.00	321.00	290.00	113.19	12811.20	50.62
50-59	19	69.26	50.00	10.00	8.00	261.00	253.00	62.10	3856.54	14.25
60-69	44	92.91	62.50	8.00	8.00	405.00	397.00	93.71	8781.43	14.13
70-79	46	82.50	55.50	24.00	9.00	360.00	351.00	75.43	5689.28	11.12
80-89	29	119.38	77.00	49.00	13.00	450.00	437.00	111.98	12540.24	20.80
90-99	1	39.00	39.00	39.00	39.00	39.00	0.00	0.00	0.00	0.00

Descriptive Statistics Third Creatine Phosphokinase Multiband in IU/1 - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	151	108.21	70.50	30.00	8.00	468.00	460.00	95.82	9180.65	7.82
Stratified										
20-29	1	78.00	78.00	78.00	78.00	78.00	0.00	0.00	0.00	0.00
30-39	11	85.64	56.00	8.00	8.00	310.00	302.00	93.15	8676.06	28.08
40-49	24	121.13	70.50	150.00	8.00	468.00	460.00	122.04	14894.46	24.91
50-59	47	116.38	72.00	32.00	10.00	405.00	395.00	97.99	9601.42	14.29
60-69	40	102.90	66.00	30.00	8.00	291.00	283.00	83.13	6911.12	13.15
70-79	21	97.00	91.00	70.00	16.00	231.00	215.00	59.72	3566.60	13.03
80-89	3	77.67	15.00	8.00	8.00	210.00	202.00	114.66	13146.33	66.20
90-99	3	149.33	27.00	8.00	8.00	413.00	405.00	228.54	52230.33	131.95

Descriptive Statistics for Third Creatine Phosphokinase Multiband in IU/l - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	145	97.52	57.00	8.00	8.00	680.00	672.00	106.87	11420.67	8.875
Stratified										
20-29										
	1	132.00	132.00	132.00	132.00	132.00	0.00	0.00	0.00	0.00
30-39										
	1	36.00	36.00	36.00	36.00	36.00	0.00	0.00	0.00	0.00
40-49										
	5	104.20	100.00	14.00	14.00	235.00	221.00	81.51	6643.20	46.45
50-59										
	19	93.63	54.00	8.00	8.00	510.00	502.00	120.59	14542.58	27.67
60-69										
	43	106.91	56.00	8.00	8.00	570.00	562.00	111.44	12418.75	16.99
70-79										
	46	85.83	55.00	12.00	9.00	680.00	671.00	107.05	11460.64	15.78
80-89										
	29	106.76	71.00	10.00	8.00	400.00	392.00	102.90	10587.76	19.11
90-99										
	1	31.00	31.00	31.00	31.00	31.00	0.00	0.00	0.00	0.00

Descriptive Statistics for Fourth Creatine Phosphokinase Multiband in IU/1 - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	148	94.28	58.50	30.00	8.00	508.00	500.00	92.65	8583.78	7.62
Stratified										
20-29										
	1	35.00	35.00	35.00	35.00	35.00	0.00	0.00	0.00	0.00
30-39										
	11	64.55	42.00	51.00	8.00	220.00	212.00	72.67	5280.87	21.91
40-49										
	24	86.13	52.50	220.00	8.00	325.00	317.00	85.36	7286.38	17.43
50-59										
	46	114.63	72.50	9.00	9.00	415.00	406.00	109.57	12006.15	16.16
60-69										
	40	89.35	60.50	18.00	8.00	240.00	232.00	72.08	5195.11	11.40
70-79										
	21	74.05	63.00	76.00	12.00	260.00	248.00	54.16	2933.75	11.82
80-89										
	3	70.00	22.00	8.00	8.00	180.00	172.00	95.52	9124.00	55.15
90-99										
	2	265.00	265.00	22.00	22.00	508.00	486.00	343.65	118098.00	243.00

Descriptive Statistics for Fourth Creatine Phosphokinase Multiband in IU/l - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	145	72.60	48.00	8.00	8.00	370.00	362.00	72.85	5307.69	6.05
Stratified										
20-29	1	67.00	67.00	67.00	67.00	67.00	0.00	0.00	0.00	0.00
30-39	1	20.00	20.00	20.00	20.00	20.00	0.00	0.00	0.00	0.00
40-49	5	80.80	60.00	10.00	10.00	165.00	155.00	58.87	3465.70	26.33
50-59	19	64.00	42.00	8.00	8.00	210.00	202.00	64.13	4112.44	14.71
60-69	43	80.77	46.00	8.00	8.00	343.00	335.00	77.60	6022.23	11.83
70-79	46	61.96	42.00	18.00	8.00	370.00	362.00	65.62	4305.87	9.68
80-89	29	85.24	48.00	12.00	10.00	300.00	290.00	86.59	7496.76	16.08
90-99	1	22.00	22.00	22.00	22.00	22.00	0.00	0.00	0.00	0.00

Descriptive Statistics for Fifth Creatine Phosphokinase Multiband in IU/1 - Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	143	65.23	41.00	8.00	8.00	400.00	392.00	68.45	4685:60	5.72
Startified										
20-29	1	34.00	34.00	34.00	34.00	34.00	0.00	0.00	0.00	0.00
30-39	10	43.50	31.00	8.00	8.00	134.00	126.00	43.74	1912.72	13.83
40-49	23	63.70	34.00	8.00	8.00	207.00	289.00	72.38	5239.04	15.09
50-59	44	67.02	37.00	8.00	8.00	400.00	392.00	73.15	3550.95	11.03
60-69	39	73.36	45.00	12.00	8.00	360.00	352.00	71.75	5148.03	11.49
70-79	21	52.57	45.00	45.00	11.00	205.00	194.00	39.10	1528.66	8.53
80-89	3	57.33	14.00	8.00	8.00	150.00	142.00	80.31	6449.33	46.37
90-99	2	265.00	265.00	22.00	22.00	508.00	486.00	343.65	118098.00	243.00

Descriptive Statistics for Fifth Creatine Phosphokinase Multiband Level in IU/l - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	141	52.21	36.00	8.00	8.00	284.00	276.00	52.45	2750.45	4.42
Stratified										
20-29	1	42.00	42.00	42.00	42.00	42.00	0.00	0.00	0.00	0.00
30-39	1	14.00	14.00	14.00	14.00	14.00	0.00	0.00	0.00	0.00
40-49	4	48.00	42.00	8.00	8.00	100.00	92.00	38.23	1461.33	19.11
50-59	18	37.06	18.50	8.00	8.00	136.00	128.00	38.79	1504.29	9.14
60-69	42	65.64	36.00	8.00	8.00	216.00	208.00	59.44	3533.31	9.17
70-79	45	43.78	30.00	8.00	8.00	195.00	187.00	43.52	1893.40	6.49
80-89	29	58.76	40.00	9.00	8.00	284.00	276.00	62.04	3848.40	11.52
90-99	1	15.00	15.00	15.00	15.00	15.00	0.00	0.00	0.00	0.00

Descriptive Statistics for Sixth Creatine Phosphokinase Multiband in IU/1 -Male

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	141	43.65	28.00	8.00	8.00	250.00	242.00	41.76	1743.50	3.52
Stratified										
20-29										
	1	19.00	19.00	19.00	19.00	19.00	0.00	0.00	0.00	0.00
30-39										
	10	30.10	16.50	8.00	8.00	84.00	76.00	27.77	770.99	8.78
40-49										
	21	44.38	26.00	8.00	8.00	170.00	162.00	43.64	1904.65	9.52
50-59										
	44	42.66	27.00	8.00	8.00	250.00	242.00	46.57	2169.02	7.02
60-69										
	39	49.77	31.00	8.00	8.00	210.00	202.00	43.75	1913.66	7.01
70-79										
	21	40.91	41.00	43.00	8.00	162.00	154.00	31.97	1021.89	6.98
80-89										
	3	32.00	8.00	8.00	8.00	80.00	72.00	41.57	1728.00	24.00
90-99										
	2	64.50	64.50	21.00	21.00	108.00	87.00	61.52	3784.50	43.50

Descriptive Statistics for Sixth Creatine Phosphokinase Multiband in IU/l - Female

	Number of Cases	Mean	Median	Mode	Minimum	Maximum	Range	Standard Deviation	Variance	Standard Error
Nonstratified										
	141	34.75	23.00	8.00	8.00	170.00	162.00	32.20	1037.09	2.71
Stratified										
20-29	1	28.00	28.00	28.00	28.00	28.00	0.00	0.00	0.00	0.00
30-39	1	8.00	8.00	8.00	8.00	8.00	0.00	0.00	0.00	0.00
40-49	4	40.25	37.50	8.00	8.00	78.00	70.00	28.90	835.92	14.45
50-59	18	21.89	14.00	8.00	8.00	58.00	50.00	16.65	277.05	3.92
60-69	42	41.60	26.00	8.00	8.00	155.00	147.00	37.34	1393.95	5.76
70-79	45	33.20	20.00	8.00	8.00	170.00	162.00	4.07	1160.71	5.08
80-89	29	36.45	31.00	8.00	8.00	120.00	112.00	28.74	825.83	5.34
90-99	1	10.00	10.00	10.00	10.00	10.00	0.00	0.00	0.00	0.00

T-Test Analysis - First Creatine Phosphokinase Multiband in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	2.07	.000	1.08	295	.280	1.09	268.07	.278
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.42	10	.684	1.46	10.00	.177
40-49	1.17	.990	-.98	28	.336	-1.04	6.04	.340
50-59	1.43	.416	.74	64	.463	.80	39.50	.430
60-69	3.54	.000	1.67	82	.098	1.63	58.14	.109
70-79	1.88	.079	.64	65	.525	.57	30.09	.574
80-89	----	.000	1.35	30	.187	-4.26	28.02	.000
90-99	0.00	1.000	.42	2	.713	.85	2.00	.487

T-Test Analysis - Second Creatine Phosphokinase Multiband in IU/l

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.65	.003	1.74	295	.083	1.75	282.27	.082
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.44	10	.666	1.54	10.00	.155
40-49	1.51	.750	.03	28	.979	.03	6.68	.976
50-59	3.49	.005	1.81	64	.075	2.31	58.81	.025
60-69	1.72	.086	.73	82	.466	.72	72.75	.472
70-79	1.41	.336	1.99	65	.051	1.86	33.50	.071
80-89	61.98	.032	-1.55	30	.132	-4.55	27.91	.000
90-99	0.00	1.000	.25	2	.826	.50	2.00	.668

T-Test Analysis - Third Creatine Phosphokinase Multiband in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.24	.187	.91	293	.366	.90	287.17	.367
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.51	10	.621	1.77	10.00	.108
40-49	2.24	.452	.29	27	.771	.38	8.29	.711
50-59	1.51	.257	.80	64	.428	.73	28.11	.471
60-69	1.80	.067	-.18	81	.854	-.19	77.44	.853
70-79	3.21	.006	.45	65	.657	.55	62.21	.587
80-89	1.24	.609	-.46	30	.647	-.42	2.35	.708
90-99	0.00	1.000	.45	2	.698	.90	2.00	.465

T-Test Analysis - Fourth Creatine Phosphokinase Multiband in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.62	.004	2.23	291	.027	2.23	278.05	.026
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.54	10	.570	2.03	10.00	.069
40-49	2.10	.494	.13	27	.896	.17	8.00	.870
50-59	2.92	.016	1.88	63	.065	2.32	55.38	.024
60-69	1.16	.644	.52	81	.604	.52	81.00	.603
70-79	1.47	.354	.74	65	.464	.79	46.59	.433
80-89	1.22	.623	-.29	30	.775	-.27	2.36	.812
90-99	0.00	1.000	.58	1	.667	1.00	1.00	.500

T-Test Analysis - Fifth Creatine Phosphokinase Multiband in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.70	.002	1.80	282	.073	1.80	265.84	.073
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.64	9	.536	2.13	9.00	.062
40-49	3.59	.320	.42	25	.679	.64	7.51	.538
50-59	3.56	.006	1.64	60	.108	2.09	55.78	.041
60-69	1.46	.239	.53	79	.599	.52	74.01	.601
70-79	1.24	.617	.79	64	.433	.82	43.24	.416
80-89	1.68	.411	-.04	30	.971	-.03	2.25	.976
90-99	0.00	1.000	.60	1	.656	1.04	1.00	.486

T-Test Analysis - Sixth Creatine Phosphokinase Multiband in IU/1

	F Value	2 Tail Prob	Pooled Variance Estimate T Value	Degrees of Freedom	2 Tail Prob	Separate Variance Estimate T Value	Degrees of Freedom	2 Tail Prob
Nonstratified								
	1.68	.002	2.00	280	.046	2.00	263.02	.046
Stratified								
20-29	0.00	1.000	0.00	0	1.000	0.00	0.00	1.000
30-39	0.00	1.000	.76	9	.467	2.52	9.00	.330
40-49	2.78	.544	.18	23	.859	.24	6.00	.819
50-59	7.83	.000	1.84	60	.071	2.58	59.40	.012
60-69	1.37	.321	.91	79	.367	.90	74.99	.370
70-79	1.14	.779	.87	64	.368	.84	41.52	.377
80-89	2.09	.285	-.25	30	.807	-.18	2.20	.872
90-99	0.00	1.000	.72	1	.601	1.25	1.00	.429

Correlation Coefficient, R^2, Significance - CPK-MB with Age

	r	R^2	Significance
First Test			
Male	-.064	.004	.218
Female	.064	.004	.221
Second Test			
Male	-.048	.002	.281
Female	.064	.004	.220
Third Test			
Male	.003	.000	.483
Female	-.033	.001	.344
Fourth Test			
Male	.071	.005	.196
Female	.004	.000	.454
Fifth Test			
Male	.091	.008	.140
Female	.034	.001	.394
Sixth Test			
Male	.084	.007	.161
Female	.061	.003	.236