A RETROSPECTIVE STUDY ON THE EFFECT OF RACE AND GENDER ON THE DEVELOPMENT, PRESENTATION AND MANAGEMENT OF ISCHAEMIC HEART DISEASE

by

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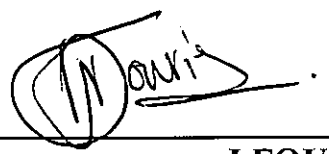
DECLARATION

I declare that the dissertation hereby submitted by me for the Master in Medical Science degree at the University of the Free State is my own independent work and has not previously been submitted by me at another university/faculty. I further more cede copyright of the dissertation in favour of the University of the Free State



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ABSTRACT

Cardiovascular Disease (CVD) is the leading cause of death of both men and women in the United States, with ischaemic heart disease (IHD) being the leading cause of death in women aged 60 and older, outnumbering the next 16 causes combined (Welty, 2001).

Age adjusted Coronary Heart Disease (CHD) death rates per 100 000 of the population for African American men and women were 262.0 and 176.7, 15% and 28% higher than those observed for Whites (Watkins, 2004). Due to the large number of deaths caused by CVD in different race and gender groups, the effect of race and gender on the development and progression of ischaemic heart disease (IHD) is the factor investigated in this study.

Currently, many factors have been associated with increased risk to development and rapid progression of IHD, and many more are being reported. Identification of high-risk populations or individuals, and then controlling these predisposing factors (eg diabetes mellitus, blood pressure, cholesterol and smoking) can delay and reduce progression of IHD and its complications.

The risk factors that are still in controversy are 'race' and 'gender'.

This research revealed that CHD presents among females (especially in the white population), approximately 10 years later than for males. These findings are similar to those already reported in the literature.

In terms of race, this study showed that black males had a higher insidence of myocardial infarctions than the white males. This is also probably responsible for the lower left ventricular function in the black males. The left ventricular dysfunction being more prominent in the black males is surely also responsible for the higher use of Angiotensin Converting Enzyme (ACE)-inhibitors, diuretics and nitrates (drugs commonly used in heart failure) in the black males when compared to the white males.

Unfortunately, race and gender has not been recognized as a major factor in the assessment and management of patients with cardiovascular disease. This is partly due to the conflicting reports on some of these observations, as well as the difficulty in conducting studies on race and gender differences, owing to many factors that often affect and, perhaps, obliterate the race and gender factor.

The purpose of this research is to investigate the role of race and gender on the development (risk factor profiles), presentation and management (treatment and response to treatment) in patients with ischaemic heart disease.

Race and gender in IHD were assessed according to the following objectives:

- A literature evaluation of data on 'race and gender and ischaemic heart disease risk' over the past 10 years (1994 – 2004) to explore the significance of this information to health care. Risk factors for IHD, presentation and treatment of IHD were evaluated.

- A review of the patients undergoing heart catheterization for clinically significant myocardial ischaemia during 2001 and 2002 in the department of Cardiology, Universitas Hospital, Bloemfontein was performed to investigate the effect of race and gender on the following:

  - risk factor profile
  - clinical presentation (including angiographic data)
  - treatment
  - response to treatment.

The information was analysed and compared to the results of the literature evaluation.

Insight into the race and gender differences in CHD in terms of risk factors, presentation and management is sought, to determine the differences in the disease manifestations or in the use of medical resources among various groups, by reviewing the available information in order to illustrate the need for earlier diagnosis and more timely, aggressive and appropriate treatment for specific race and gender groups. This will also be helpful in guiding management and allocation of medical resources.

This research clearly indicates that there is indeed reason for race and gender to be recognized as major factors in the assessment and management of patients with ischaemic heart disease in order to ensure the rapid development of a strategy to optimally manage the growing number of patients with ischaemic heart disease.

cardiovascular disease · coronary heart disease · ischaemic heart disease · race · gender · risk factors for ischaemic heart disease · presentation of ischemic heart disease · treatment of ischemic heart disease · angina · myocardial infarction

ABSTRAK

Kardiovaskulêre siekte is die hoof oorsaak van sterftes by mans en dames in die Verenigde State van Amerika, met isgemiese hartsiekte wat die hoof oorsaak is van sterftes by dames ouer as 60 jaar – dit oortref die volgende 16 oorsake gekombineerd (Welty, 2001).

Ouderdom aangepaste koronêre hartsiekte sterfte syfers per 100 000 vir Afrika-Amerikaanse mans en dames was 262.0 en 176.7, 15% en 28% hoër as dié waargeneem vir blankes (Watkins, 2004). As gevolg van die groot aantal sterftes veroorsaak deur kardiovaskulêre siekte in verskillende ras- en geslagsgroepe, is die effek van ras en geslag op die ontwikkeling en progressie van isgemiese hartsiekte die faktor wat in hierdie studie ondersoek is.

Tans, word verskeie faktore geassosieer met toenemende risiko vir die ontwikkeling en spoedige progressie van isgemiese hartsiekte, en verskeie meer word gerapporteer. Identifisering van hoë risiko populasies of individue, en die kontrolering van die vatbare faktore (bv. diabetes mellitus, bloeddruk, cholesterol en rook), kan die progressie van isgemiese hartsiekte en die komplikasies daarvan verminder. Die risiko faktore waaroor daar steeds polemiek bestaan is 'ras' en 'geslag'.

Hierdie navorsing het getoon dat koronêre hartsiekte presenteer ongeveer 10 jaar later in dames (veral in die wit bevolkingsgroep) as by mans. Hierdie bevindinge stem ooreen met dié wat in die literatuuroorsig gerapporteer is.

Ten opsigte van ras, het hierdie studie getoon dat daar 'n hoër voorkoms van miokardiale infarksies by swart mans is as by blanke mans Hierdie voorkoms is waarskynlik verantwoordelik vir die laer linker ventrikulêre funksie in swart mans. Die linker ventrikulêre disfunksie wat meer prominent is by swart mans is sekerlik ook verantwoordelik vir die hoë gebruik van Angiotensien Omskakelings Ensiem (ACE)-inhibitore, diuretika en nitrate (middles wat algemeen gebruik word vir die behandeling van hartversaking) by swart mans teenoor die van blanke mans.

Ongelukkig is ras en geslag nie erken as belangrike faktore in die skatting en hantering van pasiënte met kardiovaskulêre siekte nie.
Dit is deels as gevolg van die teenstrydige verslae van sommige van die waarnemings, sowel as die probleem om studies op ras en geslagsverskille uit te voer. Dit is te wyte aan verskeie faktore wat dikwels die ras en geslagsfaktore affekteer en kan vernietig.

Die doel van die navorsing is om die rol van ras en geslag op die ontwikkeling, (risiko-faktor profiel), voorstelling en hantering (behandeling en respons op behandeling) in pasiënte met isgemiese hartsiekte te ondersoek.

Ras en geslag in isgemiese hartsiekte is in ooreenstemming met die volgende doelwitte nagevors:

- 'n Literatuur evaluasie van data oor 'ras en geslag en isgemiese hartsiekte risiko' oor die afgelope 10 jaar (1994 – 2004) om die betekenis van die inligting vir gesondheidsorg te bepaal. Risiko faktore vir isgemiese hartsiekte, voorstelling en behandeling van isgemiese hartsiekte is ge-evalueer.
- 'n Oorsig van pasiënte wat hartkateterisasie ondergaan het vir klinies betekenisvolle miokardiale isgemie gedurende 2001 en 2002 in die departement van Kardiologie, Universitas Hospitaal, Bloemfontein is uitgevoer om die effek van ras en geslag op die volgende te bepaal:
  - risiko-faktor profiel
  - kliniese voorkoms (insluitend angiografiese data)
  - behandeling
  - respons op behandeling.

Die inligting is geanaliseer en vergelyk met die inligting wat deur die literatuur evaluasie verkry is.

Insig in die ras- en geslagsverskille in koronêre hartsiekte in terme van risiko faktore, voorstelling en hantering word nagestreef, ten einde die verskille in die siekteverskynsels of in die gebruik van mediese hulpbronne in verskillende groepe te bepaal. Deur die beskikbare inligting na te gaan is die behoefte vir vroeë diagnoses en meer tydige, aggressiewe en geskikte behandeling vir bepaalde rasse en geslagsgroepe vasgestel. Hierdie inligting kan van groot waarde wees om die hantering van pasiënte en allokering van mediese hulpbronne te rig.

Hierdie navorsing het duidelik aangetoon dat ras en geslag belangrike faktore is in die assessering en hantering van pasiënte met isgemiese hartsiekte. Die inligting kan die spoedige ontwikkeling van 'n strategie vir optimale hantering van die toenemende aantal pasiënte met isgemiese hartsiekte verseker.

Kardiovaskulêre siekte · koronêre arterie siekte · isgemiese hartsiekte · ras · geslag · risiko faktore vir isgemiese hartsiekte · kliniese voorkoms van isgemiese hartsiekte · behandeling van isgemiese hartsiekte · angina · miokardiale infarksie

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LIST OF ABBREVIATIONS

ARIC	Atherosclerosis Risk in Communities Study
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
ECG	Electrocardiogram
HDL	High Density Lipoprotein
IDDM	Insulin Dependent Diabetes Mellitus
IHD	Ischaemic Heart Disease
LAD	Left anterior Descending Artery
LDL	Low Density Lipoprotein
LIMA	Left internal Mammary Artery
NHANES I	National Health and Nutritional Examination Survey I
NHANES II	National Health and Nutritional Examination Survey II
NIDDM	Non Insulin Dependent Diabetes Mellitus
PCI	Percutaneous Coronary Intervention
PTCA	Percutaneous Transluminal Coronary Angioplasty
TIA	Transient Ischaemic Attacks
VLDL	Very low Density Lipoprotein

Aristotle (384 – 322 BC) saw the heart as the source of all movement, since the heart links the soul with the organs of life

1 ORIENTATION

1.1 INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death of both men and women, with IHD being the leading cause of death in women aged 60 and older, outnumbering the next 16 causes combined (Welty, 2001). For example, women's age adjusted mortality rates from heart disease are four to six times higher

than their mortality rates from breast cancer (Bedinghaus *et al.*, 2001). In the United States mortality records of 1999, coronary heart disease (CHD) caused 529,659 deaths, amounting to 1 out of every 5 deaths (http://www.in.gov/isdh/publications/minority2001/heart_disease.htm 2002).

Age adjusted CHD death rates per 100 000 of the population for African American men and women were 262.0 and 176.7, 15% and 28% higher than those observed for Whites (Watkins 2004). This research done in the United States of America led the researcher to undertake a similar study in South Africa. Due to the large number of deaths caused by CVD in different race and gender groups, the effect of race and gender on the development and progression of ischaemic heart disease (IHD) form the focal point of this research.

Clarifications of abbreviations used in the literature are given below:
CVD can be described as any disease pertaining to the heart and/or blood vessels (Beers and Berkow, 1999). CHD is due to subintimal deposition of atheromas (see section 3.4.1 for the description of atherosclerosis) in the large and medium sized arteries serving the heart (Beers and Berkow, 1999). IHD is a condition caused by a reduction of coronary flow sufficiently severe that the supply of oxygen to the myocardium (the heart muscle) is inadequate for the oxygen demands of the tissue, leading to an imbalance between the demand of

the myocardium for oxygen and the amount of oxygen being supplied by the coronary arteries (see Section 3.2 for the description of the coronary arteries). These conditions are prominent in the patients investigated in this study.
The white population will be classified as persons of Caucasian descent.
The black population will be classified as persons of African descent.

Several studies have pointed out that gender may be a major risk factor for the development and progression of IHD and its complications. It was, for example, observed that progression of atherosclerosis in men was almost twice that found in women up to the age of 60 years (Davis *et al.*, 1996). According to Milner *et al.*, 2002, presentation of acute coronary syndromes may differ in men and women. This study performed in 2002 found that typical symptoms, such as diaphoresis and chest pain or discomfort, carried significant or borderline-significant positive relative risk for acute coronary syndromes (ACS) in women. In contrast, typical symptoms were not significantly associated with ACS in men. In men, only dizziness/faintness was an independent negative symptom predictor of ACS. Typical symptoms are the strongest symptom predictors of ACS in women. These findings indicate that attention to typical symptoms of acute ischaemia or acute myocardial infarction can provide the most important symptom-based clues on the pretest probability of ACS in women (Milner *et al.*, 2002). Also, one of the most significant differences between men and women is the age of onset of CHD (La Rosa *et al.*, 2002).

In general, CHD has been widely considered a "man's disease" and not a major concern for women, but according to Welty, 2001, CVD was the leading cause of death in adult women in the United States of America (USA).

On the other hand, studies have also shown that race may be another important risk factor in the light of the strikingly lower CHD mortality rates among blacks compared to whites. This is most probably partly due to low dietary fat intake rather than genetic differences between blacks and whites, because earlier data from the USA shows that the prevalence of CHD among whites compared to blacks then was greater in the early to mid 20th century than today. The difference has narrowed markedly and the decline in rates has been notable among both groups, probably because of differences in dietary habits of blacks (Opie & Yellon, 1997).

During the past 50 years there has been a shift in approach to the management of IHD due to the realization that delaying or preventing the onset of IHD is much better than treating the disease itself. It is now known that identification of high-risk populations or individuals, and then controlling the predisposing factors (eg diabetes mellitus, blood pressure, obesity and smoking) can delay the onset and reduce progression of IHD and its complications. Currently, many factors have been associated with an increased risk for the development and rapid progression of IHD, and many more are being reported. However, controversy still exists regarding the effect of 'race' and 'gender' on the presentation and management of ischaemic heart disease. This controversy is the driving force behind this research.

1.2 STATEMENT OF PROBLEM

Literature revealed the perception that CHD has a more benign prognosis in women than in men and this has contributed to less aggressive efforts in prevention, diagnosis and management patterns in women with this disease. As such, since 1980 in the USA, death from CVD has declined dramatically in men, whereas it has increased in women. In the period following 1984, in the USA, annual CVD mortality in women has exceeded that of men by approximately 50 000 a year (Welty, 2001).

The misconception that CVD mainly affects men arose in part from gender differences in the age of onset. CHD begins about a decade later in women than in men, probably due to a protection factor attributed to estrogen before menopause (Redberg, 1998; Welty, 2001).

In terms of race, Watkins 2004 reported that CHD is the leading cause of death among African Americans, but despite the dynamic CHD mortality among African Americans the management of this disease in this population are still being neglected. Literature related to South Africa to contradict or support this information could not be located. For this reason it necessitated this research especially in the Free State, where the researcher conducted the research.

Unfortunately, despite the overwhelming evidence supporting race and gender differences regarding the risk of ischaemic heart disease and its complications,

race and gender have not been recognized as major factors in the assessment and management of patients with IHD. Specifically, there has been no modification in the approach to risk assessment and advice to patients about IHD in relation to race and gender. It is envisaged that this is partly due to the conflicting reports on some of these observations, as well as inconclusive or trivial differences, which, according to some professionals, may appear to be not clinically important. This is compounded by the difficulty in conducting studies on race and gender differences, owing to many factors that often affect and, perhaps, obliterate the race and gender factor. As such, the only option is to review the available information on race and gender as a risk factor to the presentation and progression of IHD.

Insight into the race and gender differences in CHD in terms of risk factors, presentation and management is sought, to determine the differences in the disease manifestations or in the use of medical resources among various groups, by reviewing the available information in order to so illustrate the need for earlier diagnosis and more timely, aggressive and appropriate treatment for specific race and gender groups. This will also be helpful in guiding management and allocation of medical resources.

1.3 AIM OF THIS RESEARCH

The purpose of this research is to investigate the role of race and gender on the development (risk factor profiles), presentation and management (treatment and response to treatment) in patients with ischaemic heart disease. This will be achieved by the evaluation and description of the clinical characteristics, management and in-hospital outcome of black and white patients of both gender groups undergoing heart catheterization for clinically significant myocardial ischaemia during 2001 and 2002.

1.4 SPECIFIC OBJECTIVES

The risk factors in IHD and their relationship to race and gender were assessed according to the following objectives:

a) A literature evaluation of data on 'race and gender and ischaemic heart disease risk' over the past 10 years (1994 – 2004) to explore the significance of this information to health care.

a) A review of the patients undergoing heart catheterization for clinically significant myocardial ischaemia during 2001 and 2002 in the department of Cardiology, Universitas Hospital, Bloemfontein was performed to investigate the effect of race and gender on the following:

- risk factor profile
- clinical presentation (including angiographic data)
- treatment
- response to treatment

The information was analysed and compared to the results of the literature evaluation. The Department of Biostatistics, University of the Free State, Bloemfontein was consulted in this regard.

The following specific objectives were followed:

1.4.1 Objective 1: Risk factor profiles

1.4.1.1 Objective 1a (gender):

To compare the risk factors present in IHD for men with IHD with those of women of the same race with IHD.

The age groups were 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

Outcome: The respective risk factor profiles for caucasian males with IHD vs caucasian females with IHD and black males with IHD vs black females with IHD.

1.4.1.2 Objective 1b (race):

To compare the risk factors present in IHD for blacks with IHD with those of caucasians of the same gender with IHD.

The age groups were 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

Outcome: The respective risk factor profiles for caucasian males with IHD vs black males with IHD compared to caucasian females with IHD vs black females with IHD.

1.4.2 Objective 2: Presentation

1.4.2.1 Objective 2a (gender):

To compare the trend in the presentation of existing IHD for men with IHD with that of women of the same race with IHD.

The age groups were 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

Outcome: The presentation prevalence for caucasian males with IHD vs caucasian females with IHD and black males with IHD vs black females with IHD.

1.4.2.2 Objective 2b (race):

To compare the trend in the presentation of existing IHD for blacks with IHD with that of caucasians of the same gender with IHD.

The age groups were 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

Outcome: The presentation prevalence for caucasian males with IHD vs black males with IHD compared to caucasian females with IHD vs black females with IHD.

1.4.3 Objective 3: Treatment and response

1.4.3.1 Objective 3a (gender):

To compare the trend in the treatment and response to treatment for men with IHD with that of women of the same race with IHD.

The age groups were 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

Outcome: The treatment prevalence and response to this treatment for caucasian males with IHD vs caucasian females with IHD and black males with IHD vs black females with IHD.

1.4.3.2 Objective 3b (race):

To compare the trend in the treatment and response to treatment for blacks with IHD with that of caucasians of the same gender with IHD.

The age groups were 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

Outcome: The treatment prevalence and response to this treatment for caucasian males with IHD vs blacks males with IHD compared to caucasian females with IHD vs black females with IHD.

The gained information was summarized and evaluated in a self-compiled database using Access software.

2 RESEARCH METHODOLOGY

2.1 STUDY DESIGN

2.1.1 A Literature Evaluation

The major risk factors for ischaemic heart disease were investigated by means of a literature evaluation. Data were obtained from different published studies, using MEDLINE and PubMed (1966-2004).

The criteria for inclusion of the articles in the literature evaluation were as follows:

a) Studies with the aim or results indicating a comparison of gender and/or race in the development, presentation and management of ischaemic heart disease

b) Study design – prospective studies

c) Studies in which selection of subjects was based on a representative (random) sample of the study population and controls.

2.1.2 Patient Study (Retrospective)

Here, a retrospective study was preferred because it is the quickest and least expensive study to undertake. It involved reviewing the files of patients with existing ischaemic heart disease undergoing heart catheterization at the department of Cardiology, Universitas Hospital, Bloemfontein, from January 2001 until December 2002. The information was used to determine and compare the risk factor profile, presentation, treatment and response to treatment for IHD. For this project, the relevant information was extracted from the files (Hearts database) and documented on a self-compiled template (Appendix A, page 78). This information was typed into a self compiled database using Access software.

2.1.2.1 Selection criteria for patient reports:

- Patients with proven coronary artery disease/history of IHD/at least one occluded vessel.
- Patients that underwent catheterization from January 2001 until December 2002.

Patients' drug treatment, surgery status and 1-, 2- or 3 vessel disease status were documented in the self-compiled template and database.

The data collected about each patient will fall under the following headings:

a) Demographic data

b) Risk factor profile

c) Clinical presentation (angiographic data included)

d) Treatment and response

The data recorded in the Access-database will be analysed using analytical statistics in order to indicate a trend (if present) regarding the effect of race and gender on the risk factors, presentation (common symptoms) and management (treatment and response) of ischaemic heart disease.

2.2 AREA OF STUDY (SAMPLE SIZE)

Catheterization files (2001-2002) from the catheterization room of the Universitas Hospital, Bloemfontein, were accessed. Data of all the patients that underwent catheterization in 2001 and 2002 were retrieved. It included names (initials and surname), birthdate, height, weight and catheterization date. The age of each patient was calculated from the birthdate up to the first catheterization date. Only heights and weights recorded on the first catheterization date were used.

Each file was scrutinized for the clinical status of the patient in terms of ischaemic heart disease. Patients with a history of ischaemic heart disease, coronary heart disease, single, double or triple vessel disease or at least one occluded vessel, were selected. However, whenever it was mentioned in the report that the patient had normal coronary arteries with no evidence of ischaemic heart disease, the patient was immediately deleted from the list. In

this way, only patients that underwent catheterization in 2001 and 2002 with evidence of ischaemic heart disease were studied.

Reports evaluated included catheterization, specialist consultation, operation, ultrasound or stress ECG reports or any combination of the above-mentioned.

The list of names compiled from the catheterization files from the catheterization room amounted to 1820 files (a total of 922 files during 2001 and 898 files during 2002).

According to the selection criteria (see Section 2.1.2.1), 1102 patient files were selected for review. The following two tables indicate the number of patients in a specific race and gender group after statistical evaluation of the 1102 patient files. This data were used to eliminate and to have a final sample size. The criteria used in the elimination process are described below.

Table 2.1 Race frequencies

The following table summarizes the frequency of a specific race group generated from this research

Race	*Frequency*	*Percent*	*Cumulative frequency*	*Cumulative percent*
Black	99	8.98	99	8.98
Coloured	30	2.72	129	11.71
Indian	10	0.91	139	12.61
Other	11	1.00	150	13.61
White	952	86.39	1102	100.00

Table 2.2 Gender frequencies

The following table summarizes the frequency of a specific race group generated from this research

Gender	*Frequency*	*Percent*	*Cumulative frequency*	*Cumulative percent*
Female	324	29.40	324	29.40
Male	778	70.60	1102	100.00

The age groups selected were: 35 to 44 years, 45 to 54 years, 55-64 years, 65 to 74 years and 75-84 years. The ages of a few subjects were outside these ranges and they had to be eliminated.

Due to the small number of patients in the "coloured", "Indian" and "other" racial groups, these patients were excluded form the statistical analysis.

The final sample size used for the statistical analysis was 1039 patients.

Table 2.3 Race/Gender breakdown of final sample size

Race/Gender group	*Frequency*	*Percentage (%)*
Black Females	37	3.6
Black Males	61	5.9
White Females	265	25.5
White Males	676	65

2.3 METHODS OF DATA COLLECTION

2.3.1 Data from most recent report

The following data were collected from the most recent report and documented on the self-compiled format (see attached Appendix A, page 78):

- Common symptoms
- Ventricular function
- ECG
- Heart Sonar
- Final medication

2.3.2 Data from oldest report

The following information was collected from the oldest available report:

- Risk factors
- Cardiovascular status
- Respiratory status
- Gastrointestinal status
- Central Nervous System status
- Affected vessels
- Treatment

2.3.3 Normal/abnormal electrocardiogram and heart sonar

The ECG and Heart Sonar were classified as either normal or abnormal according to the following criteria:

2.3.3.1 Normal ECG

- Sinus rhythm
- No significant abnormalities

2.3.3.2 Abnormal ECG

- Left bundle branch block (LBBB)
- ST segment changes
- Q-waves present

2.3.3.3 Normal Heart Sonar

- Normal left ventricular function
- Normal size
- Normal Doppler examination

2.3.3.4 Abnormal Heart Sonar

- Hypertrophy
- Ventricle or atrium enlarged
- Left ventricular function below 50%

2.3.4 Myocardial Infarction

Myocardial infarction was classified as Q-wave when Q-waves were present on the 12 lead electrocardiogram, if described as anterior/posterior, anteroseptal or lateral infarction or if ST-segment elevation was present.

2.3.5 Effort test

The effort test was classified as normal or abnormal according to the most recent report. The result of treatment was also taken from the most recent report available.

2.3.6 Treatment

Any treatment after the initial bypass or PCI treatment was documented as follow-up and repeat surgery procedures.

2.3.7 Response to treatment

A patient's response to treatment was documented as being symptomatic if angina or any cardiovascular symptoms were present (from latest available report) following medical, angioplasty or bypass treatment.

2.4 MEASUREMENTS

The data from the patient files were documented on the self-compiled template and recorded in the self-compiled database for statistical analysis under the following sections:

2.4.1 Demographic data

The demographic data that were collected for each patient from the patient reports included name and surname, birth date, computer number, age, race, gender, height, weight and place of origin (city or rural). See self-compiled template (Appendix A) and details in Section 4.2.

2.4.2 Risk factor profile

The risk factors that were investigated in the patient reports for this study included a family history of ischaemic heart disease (if mentioned), hypertension (if mentioned), smoking (if mentioned), cholesterol (if mentioned) and diabetes mellitus (if mentioned). See self-compiled template (Appendix A) and details in Section 4.2.

2.4.3 Angiographic presentation

The information collected from the patient reports regarding the angiographic presentation include the following:

Ventricular function, stable or unstable angina, Q-wave or non Q-wave myocardial infarction, normal or abnormal effort test and affected vessels. See self-compiled template (Appendix A) and details in Section 4.2.

2.4.4 Treatment and response

The treatment of the patient included drug treatment, angioplasty (PTCA with or without stent) or bypass surgery.

The response was documented as being either symptomatic or asymptomatic. See self-compiled template (Appendix A, page 78) and details in Section 4.2.

2.5 ANALYSIS

Results were summarized by frequencies and percentages per age/sex and race category. The following age groups were used: 35 to 44 years, 45 to 54 years, 55 to 64 years, 65 to 74 years and 75 to 84 years.

The Cochran-Mantel-Haenszel test of association was performed to test the association between gender and outcomes (in white patients) and race and outcomes (in males), adjusted for age groups. Adjusted odds ratios (males versus females in the white patients, blacks versus whites in the male patients) were calculated with 95% confidence intervals (CI).

2.6 ETHICS

2.6.1 Ethics Committee Approval

This research study was submitted to the Ethics Committee of the University of the Free State for approval and was approved in writing by the Ethics Committee. After the protocol for this study was finalised the biostatistitian and researcher proposed some changes and Amendment no 1 was approved in writing by the Ethics Committee.

2.6.2 Senior Executive Officer, Universitas Tertiary Hospital Approval

This research study was submitted to the Senior Executive Officer, Universitas Tertiary Hospital, for approval, and is was approved. Amendment no 1 was submitted to the Senior Executive Officer, Universitas Tertiary Hospital, for his notification.

3 LITERATURE EVALUATION

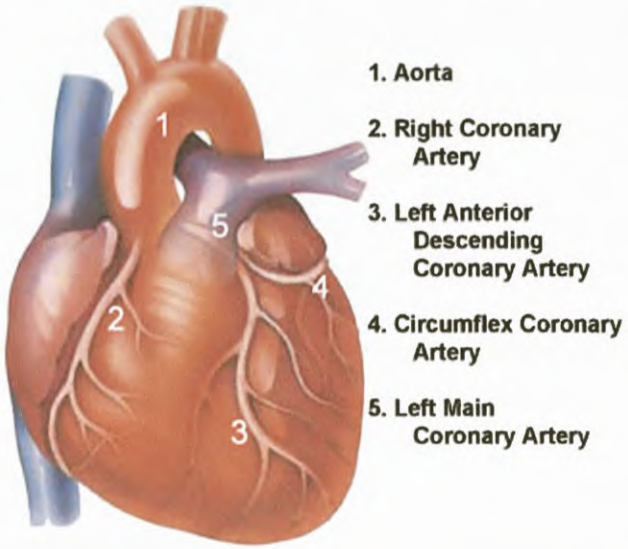
3.1 INTRODUCTION

A literature evaluation was conducted to investigate ischaemic heart disease, the risk factors in the development of IHD, the presentation of IHD and the treatment of this disease. This information was used to compile a template for the patient study to be performed.

3.2 ISCHAEMIC HEART DISEASE

"Ischaemia" means too little blood. The heart muscle itself requires oxygen to function. The heart muscle, like all other muscles, receives its oxygen from arteries. The blood vessels which supply the oxygen-rich blood are called the coronary arteries (see Figure 3.1). They are located on the surface of the heart. The right coronary artery curves around the right side of the heart to supply blood to the front and back of that side. The right coronary artery, which divides into the right posterior descending artery and a large marginal branch, supplies blood to the lower third of the interventricular septum. The left coronary artery has two distinct branches, namely the left anterior descending artery (LAD) (supplies blood to the front of the heart on the left side) and the circumflex branch (supplies blood to the back of the heart on the left side) *(http://www.stanfordhospital.com/healthLib/atoz/cardiac/arteries.html)*.

Figure 3.1 *The Coronary Arteries of the Heart*
(http://www.stanfordhospital.com/healthLib/atoz/cardiac/arteries.html)



IHD is a condition caused by a reduction of coronary flow sufficiently severe that the supply of oxygen to the myocardium is inadequate for the oxygen demands of the tissue, leading to an imbalance between the demand of the myocardium for oxygen and the amount of oxygen being supplied by the coronary arteries. This disease is very common in western societies and is often asymptomatic (Ritter *et al.*, 1995).

To ensure the heart's survival and to maintain optimal function, the blood supplied through the coronary artery system must be adequate to meet the demands of the heart for oxygen. More oxygen is needed when the heart is beating faster or contracting more vigorously. To meet the increased demand, the coronary arteries dilate to ensure a greater area for cross-sectional blood flow.

Diseases such as IHD can limit this dilatation by three processes, namely atherosclerosis, thrombosis and spasm. As ischaemic heart disease is nearly always caused by atheroma in one or more of the coronary arteries, it is important to understand the process of atherosclerosis. This process is described in Section 3.4.1.

In general, patients with ischaemic heart disease commonly die of complications such as myocardial infarction, cardiac failure and stroke. As such, the management of ischaemic heart disease aims at delaying progression of the disease and prevention of complications. For instance, drugs such as anti-hypertensive agents are used to reduce disease progression, while aspirin is used

to prevent thrombotic complications such as stroke or transient ischaemic attacks (TIA) and the acute ischaemic syndromes.

3.3 THE RISK FACTORS THAT INFLUENCE THE DEVELOPMENT AND PROGRESSION OF ISCHAEMIC HEART DISEASE – AN OVERVIEW

3.3.1 The non-modifiable risk factors

Among the most important risk factors for CVD are the irreversible or non-modifiable risks, which include age and inherited characteristics such as race, gender and others implicated by a family history of ischaemic heart disease.

3.3.1.1 Age

Cholesterol and triglyceride levels rise with age and coronary mortality rates are uniformly higher among men than among women at all ages (Lindsay & Gaw, 1997). As already mentioned coronary artery disease increases among women after menopause, suggesting that women are more protected against this disease before menopause, which occurs at the age of approximately 55 years. At 55 years almost all women have experienced the menopausal symptoms and this age is also the age at which sex hormones reach post-menopausal levels in most women (Sunayama *et al.*, 1996).

CHD incidence and mortality among African Americans increase with age. The age adjusted risk is greater among African American women 25-54 years of age than among white women, but lower among African American men than among white men of similar age. The higher risk at younger ages among African American women than among African American men can be explained by the higher prevalence of CHD risk factors in younger women (Watkins, 2004).

In general age is a well-known risk factor for CVD among both men and women.

3.3.1.2 Race

"African" American and "Black" will be regarded as synonyms in this research project.

Racial factors appear to be important in ischaemic heart disease, with one recent American study showing the male-to-female mortality ratio from coronary heart disease to be 3.3 to 1 among white patients and 1.7

to 1 among African-American patients (Jones *et al.*, 2001). The National Health and Nutritional Survey (NHANES) showed that urban black men have a higher prevalence of hypertension when compared to other groups (the urban black females, white males and white females), and even in the USA black people have a far higher prevalence of hypertension compared to the Caucasian population (Opie & Yellon, 1999).

According to Mathew *et al.*, 1997, there are racial differences in the prevalence of risk factors for CAD. Hypertension, obesity and diabetes mellitus are more common among blacks than in whites. A study showed that the total serum cholesterol levels are not significantly different in blacks and whites, but the serum high density lipoprotein cholesterol may be higher in blacks. Published data also show that smoking is more prevalent among blacks, particularly among black men.

Hsia, 1998 reported that the relative risk of coronary artery death associated with risk factors is similar among African-American and white women, although the prevalence of risk factors, such as hypertension, is quite different which confirms the above findings.

It is recognized that mortality rates from IHD among blacks may exceed those among whites for the age group 25 to 64 years, but scarcity of data and controversy over the pathogenesis of IHD among blacks, still exist. Clinical manifestations of the disease are influenced by cultural and socio-economic factors, as well as by other diseases in the community and selective survival (Mathew *et al.*, 1997). CHD has reached epidemic proportions among Indians living in South Africa. By contrast, it is rare among rural blacks, although the prevalence may recently have increased among urban blacks (Ranjith *et al.*, 2002). It has also been noted that peak mortality from CHD occurs a decade earlier among Indian descendants than among Whites (Ranjith *et al.*, 2002).

According to the California Cardiovascular Disease Prevention Coalition (http://www.dhs.cahwnet.gov/ps/cdic/cdcb/chronic/CHDSP/documents/CVDinAFAM.pdf 2003) heart disease occurs at an earlier age among African Americans. Below the age of 50, death rates are 50% higher among African American men and 100% higher among African American women than among their white counterparts.

Budoff *et al.*, 2002 recorded that in a symptomatic population, Whites and Asian-Americans have a higher burden of atherosclerosis, both angiographically and by electron beam tomography (EBT), when compared with Blacks and Hispanics.

The findings in the morbidity and mortality weekly report of 13 November 1998 indicate that IHD death rates declined for all age groups during 1981-1995; however, these decreases were greater for whites than for blacks, causing an increase in the black:white IHD mortality ratios. Black:white mortality ratios were particularly high for young women; black women in the 35-44 and 45-54 year age groups experienced IHD death rates of more than twice those of white women in the same age groups (Ward *et al.*, 1998).

According to Oka RK *et al.*, 1996 recent studies have found that young white males are more likely to receive diagnostic and revascularization procedures than are women or white males who are older or are of Mexican or African descent.

Historically, IHD was considered to be a disease of the white population, and an uncommon cause of death in blacks. In 1997 it was recognized that mortality rates from IHD are similar for blacks and whites; that the rates may be excessive for blacks in the age group 25 to 64 years and that the proportion of sudden cardiac death is greater for blacks than for whites (Mathew *et al.*, 1997). In 2001, disease of the heart and stroke accounted for 30.8% of deaths among African American men and 36.5% of deaths among African American women in the United States of America (Watkins, 2004). The racial factor is very evident in this disease according to the above-mentioned information.

3.3.1.3 Gender

According to Chrysohoou *et al.*, 2003, CHD is more common in men than in women. By the age of 60 years, only 1 in 17 women in the United States has had a coronary event, as compared with 1 in 5 men. After the age of 60, however, coronary heart disease is the primary cause of death among women. In this age group, one in four women, as well as one in four men, die of coronary heart disease (Rich-Edwards *et al.*, 1995). Welty, 2001 reports that CVD mortality rates have decreased in

men during the past 20 years, but have steadily increased in women. In the United States more than 500 000 women die of CAD every year.

Goldberg *et al.*, 2000 confirmed that there are differences between symptoms at presentation of men and women and little data on possible age and sex differences in presentation of symptoms for patients with acute coronary disease. Clinicians should be aware of these differences when managing patients suspected of having CHD. For this reason, further investigation in this field is essential.

CHD mortality has declined since 1968, but the rate of decline has slowed in respect of African Americans, especially African American women, in recent years (Watkins, 2004).

It was observed by Davis *et al.*, 1996, that progression of atherosclerosis in men was almost twice that found in women up to the age of 60 years. In another report, the incidence of coronary heart disease in women was found to be only a third of that in men of similar age. Furthermore, women have been found to have a higher HDL:LDL ratio than men (see Section 3.2.2.2). More than 55% of myocardial infarctions observed in women occur at the age of 70 years and older. Although chest pain is a common complaint, Jones *et al.*, 2001 reported that patients presenting with acute myocardial infarction indicate that a higher percentage of women experience pain radiating to the neck, back and jaw. A higher percentage of women were also found to present with nausea, vomiting and shortness of breath (Jones *et al.*, 2001).

The protection that women have in terms of risk for IHD can be explained by the fact that a larger proportion of cholesterol in their plasma is present as high density lipoprotein (HDL) giving women a higher HDL:LDL ratio than men. Postmenopausal rises in LDL cholesterol tend to eliminate this advantage of women over men (Thompson & Wilson, 1992).

The female hormone estrogen tends to lower LDL levels and increases HDL cholesterol levels and, by maintaining this lower LDL level, reduces the risk for CVD. This situation is not common in men. The protective effect of estrogen was shown in studies when results confirmed a 50% reduction in coronary artery disease risk in post-

menopausal women using estrogen (Barret-Conner & Bush, 1991; McCrohon *et al.*, 1996; Miller *et al.*, 1995; Sbarouni *et al.*, 1998; Herrington *et al.*, 2000). The Heart and Estrogen/progestin Replacement Study (HERS), a randomized trial involving postmenopausal women with known coronary vascular disease, evaluated the effect of hormones on cardiovascular events. According to HERS, the risk of cardiovascular events increases significantly during the first year of treatment with an oral conjugated estrogen plus progestin preparation. This risk declines as the study progresses, with a decrease in cardiovascular events occurring in the fourth and fifth years of therapy. Whether these results can be applied to all hormone regimens is uncertain (Sadovsky, 2002).

Hormone replacement therapy tends to lower the fibrinogen levels, which is a blood clotting factor associated with heart attacks and strokes. Estrogen helps to relax blood vessels and responds to exercise and stress and increasing blood flow (Eisenhard, 1997). With menopause estrogen is not produced naturally anymore and a rise in LDL cholesterol levels occurs, increasing the risk for women of CVD. The mechanisms of this apparent benefit of hormone therapy most likely include lipoprotein effects: orally administered estrogen raises plasma levels of HDL cholesterol and lowers plasma levels of LDL cholesterol and lipoprotein(a), and protects LDL from oxidation. These lipoprotein effects may account for improvement in coronary and systemic vasomotor responsiveness due to the reduction in inhibitory effects of LDL and lipoprotein(a) in the vessel wall and facilitatory effects of HDL, in addition to enhanced nitric oxide bioactivity (Koh *et al.*, 1999).

Lipid lowering medications and hormone replacement therapy have raised great hopes for primary prevention of coronary artery disease (i.e. prevention of a first myocardial infarction or the onset of symptomatic coronary artery disease). However, in caring for women, physicians await evidence from randomized, controlled trials to support the effectiveness of these measures, while sorting among conflicting sets of recommendations (Bedinghaus et al., 2001).

According to Kitler, 1992, women may be referred for coronary angiography much less often and to coronary artery bypass surgery at a much more advanced age and with greater severity of illness, and when hospitalized for coronary heart disease, they undergo fewer major diagnostic and therapeutic procedures than do men.

The mortality rate after myocardial infarction and complications are more frequent in females than in males when procedures such as angioplasty or bypass surgery are performed (Waters *et al.*, 1995; http://www.in.gov/isdh/publications/minority2001/heart_disease.htm 2002).

The assessment and correction of these risk factors by lifestyle and medical means is an enormous challenge in health care today.

3.3.1.4 A family history of cardiovascular disease

It is assumed that a family history of premature CHD in first degree relatives has a similar impact on increasing risk among African Americans (Watkins, 2004).

According to Lindsay and Gaw, 1997, the prevalence of coronary artery disease is much greater among men than among women up to the age of 65 years and this is particularly the case with those patients that have a family history.

According to Robinson *et al.*, 2004, family history of myocardial infarction is a known risk factor for CAD. A random sample was drawn from the population aged 20-79 years. From 3793 subjects with siblings, 34 (0.9%) reported a history of myocardial infarction in one parent and one sibling. Multivariable analysis revealed an independent association between dual parental and sibling history of myocardial infarction and plasma fibrinogen levels. This study concluded that plasma fibrinogen levels may indicate inheritable risk for CAD in subjects with a strong family history of myocardial infarction.

A family history of premature CHD is common among individuals with that disorder and among the possible explanations is the occurrence of familial similarities in serum lipids (Thompson & Wilson, 1992).

3.3.2 The modifiable risk factors

There are four main coronary heart disease risk factors over which we, as individuals have control. These include hypertension, abnormal plasma lipid concentrations, smoking and diabetes:

3.3.2.1 Hypertension

Hypertension affects approximately 50 million individuals in the United States and approximately 1 billion individuals worldwide. As the population ages, the prevalence of hypertension will increase even further unless broad and effective preventative measures are implemented. Recent data from the Framingham Heart Study suggest that individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension (Chobanian *et al.*, 2003). The relationship between blood pressure and risk for CVD events is continuous, consistent and independent of other risk factors. The higher the blood pressure the higher the risk of myocardial infarction, heart failure and stroke (Chobanian *et al.*, 2003).

According to Marques-Vidal & Tuomilento, 1997, reduction of highly or moderately elevated blood pressure levels has been shown to result in a decrease in stroke and myocardial infarction rates.

Research suggests that different aspects of blood pressure may be significant as risk predictors in men and women. A specific study indicated that mortality in men was positively correlated with systolic blood pressure, while among females diastolic blood pressure was a determinant of cardiovascular mortality (cited in http://www.mmhc. com/jgsm/articles/JGSM0008/news.htm, 2000).

In 1992 Thompson and Wilson reported that the prevalence of hypertension was highest in blacks, intermediate in whites and lowest in Hispanics.

According to Mathew *et al.*, 1997, hypertension is more common among blacks than among whites. Research by Watkins, 2004 revealed that hypertension has an age-adjusted prevalence of 36.7% among African American men 20-74 years of age and 36.6% among African American women in NHANES 1999-2000. Furthermore, African Americans develop hypertension earlier in life and have a higher prevalence of more severe hypertension. In the Atherosclerosis Risk in Communities (ARIC) study, hypertension was a strong predictor of CHD incidence, more so among African Americans than among white subjects and especially among women (Watkins, 2004), which is in accordance with previous research results in 1997.

The following table gives an indication of the classification that can be made at different blood pressure levels according to the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure:

***Table 3.1 Blood Pressure Classification for Adults 18 years or older* (Chobanian *et al.*, 2003)**

	Systolic blood pressure	*Diastolic blood pressure*
Normal blood pressure	<120 mmHg	<80 mmHg
Prehypertension	120-139 mmHg	80-89 mmHg
Stage 1 Hypertension	140-159 mmHg	90-99 mmHg
Stage 2 Hypertension	≥160 mmHg	≥100 mmHg

According to the literature there are racial differences in the occurrence of hypertension. Black Africans who live in westernized societies have higher blood pressure and more strokes than their Caucasian counterparts have.

In California high blood pressure is more prevalent among African American men (41.5%) compared to White (22.8%), Hispanic (22.1%) and other (15.6%) men (http://www.dhs.cahwnet.gov/ps/cdic/cdcb/chronic/CHDSP/documents/CVDinAFAM.pdf 2003).

This finding can partly be explained by means of the following tables:

Table 3.2: Hypertension among blacks and whites; biochemical differences (Opie & Yellon, 1997)

Feature	In Blacks
Total cholesterol	Lower
Triglycerides	Lower
High-density lipoproteins	Higher
Low-density lipoproteins	Lower
Very low density lipoproteins	Lower
Response to Na^+ load	Delayed
Urine Na^+/K^+ ratio	Higher
Plasma Na^+/K^+ ratio	Higher
Transport Na^+/K^+	High intracellular Na^+

Table 3.3: Hypertension among blacks and whites; hormonal differences (Opie & Yellon, 1997)

Feature	In Blacks
Plasma rennin activity	Lower
Plasma noradrenaline	Equal
Dopamine β-hydroxylase	Lower
Aldosterone	Higher
Kallikrein	Lower
Circulating inhibition of Na/K ATPase	Higher

3.2.2.2 Plasma Lipid Concentrations

Cholesterol and triglycerides are common types of fats (lipids) that are essential for good health when present in normal amounts. Cholesterol is a waxy fat carried through the bloodstream by lipoproteins. Both cholesterol and triglycerides are either consumed through food or manufactured in the liver and then transported through the bloodstream by the proteins (apolipoproteins) present in various types of lipoproteins. Cholesterol is mostly transported by high-density lipoproteins (HDLs), low-density lipoproteins (LDLs) or very low-density lipoproteins (VLDLs).

A key element of atherosclerosis is the build-up of lipids which come from lipoproteins such as LDL. The lipids invade an artery wall and are retained in the matrix surrounding the cells. Their presence and modification by oxidizing enzymes causes local inflammation. Eventually, the artery wall becomes loaded with lipids and the site develops a cholesterol-rich lesion, called an atheroma, which later develops into a plaque. Sooner or later this cholesterol-rich plaque may rupture, causing a blood clot to develop. This may block an artery completely, which can result in a heart attack or stroke. Normally this whole process is slowed down by another lipoprotein called HDL. HDL helps to get rid of the accumulated lipids through a complex process referred to as the reverse transport of cholesterol to the liver.

Higher levels of HDL are associated with reduced risk for coronary heart disease, which is the case in women because of the fact that they have

higher HDL levels than men (Haffner *et al.*, 1995, Davis *et al.*, 1996, Byrne *et al.*, 1995). Low HDL cholesterol levels and elevated triglyceride levels have been reported to be stronger predictors of coronary artery disease in women than in men (Bass *et al.*, 1993).

On average, higher levels of HDL cholesterol are observed among African American adults compared to white adults, explaining the differences between the races. National data and data from the Multiple Risk factor intervention Trial (MRFIT) participants demonstrate and inverse relationship between HDL levels and education, income and measures of socio-economic status that are common to both African Americans and Whites. In ARIC, there is a similar, though slightly less protective effect of HDL among African Americans than among White persons (Watkins, 2004).

Hormonal factors account for the differences between the sexes in lipoprotein levels; estrogen lowers LDL levels and rises HDL levels whilst androgen lowers HDL levels and rises LDL levels. Environmental influences other than diet include cigarette smoking (HDL cholesterol decreases) and alcohol consumption (HDL cholesterol rises). An increased level of HDL cholesterol (>2mmol/litre; >77mg/dL) appears to be harmless. At any given level of serum cholesterol the risk of CHD in men is roughly three times that of women of comparable age. The greater protection that females enjoy is due to their lower triglyceride and higher HDL cholesterol levels when compared to males. This immunity decreases markedly after menopause.

Plasma or serum cholesterol concentration has been shown to correlate with CHD. The risk of CHD rises appreciably when serum cholesterol exceeds 6.5 mmol/litre and even more when it gets above 7.8 mmol/litre. Serum cholesterol of below 5.2 mmol/litre seems to be safe (Thompson & Wilson, 1992).

LDL is not homogenous and comprises a spectrum of particles which vary in size, density and lipid content. As LDL density increases, particle size, the relative content of total and polar lipids and flotation rate decrease (Opie & Yellon, 1999).

The relative amounts of LDL cholesterol and HDL cholesterol in our bloodstream are therefore very important as they determine our ability to resist atherosclerosis.

3.2.2.3 Smoking

In 2001, self-reported data from the National Health Interview Survey indicated that approximately 22.8% of American adults were current smokers. The results are similar to those in the 1999-2001 National Survey on Drug Use and Health, wherein 25.7% of African American adults, aged 18 and older reported current cigarette use. Cigarette smoking was a significant predictor of CHD mortality in the combined NHANES I/NHANES II follow-up amongst men, in the Charleston Heart Study for men, and in a New York hypertension work site cohort. In ARIC, the adjusted hazard rate ratios for CHD incidence were 1.9 and 2.6 when African American men and women smokers were compared with those who had never smoked (Watkins, 2004).

According to Mathew *et al.*, 1997, smoking is more prevalent among blacks and particularly among black men.

In California smoking is more prevalent among African American women than among any other race-gender group. 26.8% of African American women in California smoke, as opposed to 19.2% of white women (http://www.dhs.cahwnet.gov/ps/cdic/cdcb/chronic/CHDSP/documents/CVDinAFAM.pdf 2003).

When focusing on the gender factor, smoking is a major risk factor for the development of cardiovascular disease in women. More than 60 percent of myocardial infarctions in women younger than 50 years are attributable to smoking, as are 21 percent of all deaths from coronary artery disease. Investigators in the Nurses'Health Study found that the risk of coronary artery disease decreases by one third, two years after smoking cessation. For women seeking to quit smoking, combination therapy with bupropion (Zyban) and the nicotine patch appears to produce the best long-term results (Bedinghaus *et al.*, 2001). According to Serrano *et al.*, 2003, the risk of acute myocardial infarctions is three times higher in patients who continue to smoke after an acute coronary event compared with patients who quit.

Other forms of smoking, like pipes or cigars, increase the incidence rate of oral and bronchial diseases and cancer. Although the ingredients in cigarettes that induce CHD are not identified, it seems that the tar and

nicotine content are secondary in importance to inhaled carbon monoxide.

The death rate as a result of CHD for smokers is two to three times that of non-smokers with between 35% and 40% of the deaths occurring before retirement age.

Smoking is associated with both aspects of atherosclerosis: it promotes the development of lesions, thus creating sites susceptible to blockage, and promotes the occurrence of triggering events that lead to blockages.

Steyn *et al.*, 2002, reported that in 1998, 24.6% adults (44.2% of males and 11.0% of females) smoked regularly. Coloured women had a higher rate (39%) than African women (5.4%). About 24% of the regular smokers had attempted to quit, with only 9.9% succeeding. African women (13.2%) used smokeless tobacco more frequently than others. Of the nonsmokers 28% and 19% were exposed to environmental tobacco smoke in their homes and workplaces, respectively.

3.2.2.4 Diabetes Mellitus

The two major forms of diabetes are type 1 [insulin-dependent diabetes mellitus (IDDM)] and type 2 [non-insulin dependent diabetes mellitus (NIDDM)].

Patients with diabetes mellitus are at a particularly high risk of contracing ischaemic heart disease (Will & Casper, 1996).

In terms of the racial factor, diabetes is more prevalent among African Americans (14.5%) than any other ethnic group (Hispanics-12.9%; Whites - 4.3%; Other - 7.6%) (http://www.dhs.cahwnet.gov/ps/cdic/cdcb/chronic/CHDSP/documents/CVDinAFAM.pdf 2003).

The gender factor is also evident according to Kaseta *et al.*, 1999 who reported for individuals 50 to 59 years old, diabetes is a greater CHD risk factor among women than among men. The risk of death from CHD among women with diabetes is more than 3 times that of nondiabetic women.

Diabetes causes glucose intolerance leading to hyperglycaemia. Diabetes is a predisposing factor for atherosclerotic disease, particularly

CHD. Cardiovascular risk varies widely among diabetics depending on the presence of other risk factors. Though many factors (including endothelial dysfunction, increased vascular oxidative stress and abnormalities of platelet function, coagulation, fibrinolysis and lipoproteins) contribute to the increased risk of CVD in men and women, diabetes is a greater coronary heart disease mortality (coronary heart disease mortality) risk factor among women than among men aged 50-59 years. The impact of diabetes is substantially greater for women as, when present, it eliminates their advantage over men.

People with diabetes have higher levels of other atherogenic risk factors including hypertension, ratio of total: HDL-cholesterol, triglyceride, VLDL cholesterol, uric acid, left ventricular hypertrophy (LVH), high haematocrit and blood fibrinogen.

CHD incidence is increased two- to threefold among diabetic populations and the increased risk is evident at diagnosis. The immediate prognosis of myocardial infarction in diabetic patients is considerably worse than in non-diabetics. CVD is by far the largest cause of morbidity and mortality among the diabetic population. The concurrence of hypertension and proteinuria in diabetes is particularly important in predicting risk with a five-fold excess among men and an eight-fold excess risk among women when compared to those without proteinuria and hypertension (Opie & Yellon, 1999).

In ARIC, wherein diabetes was more than twice as common in African Americans as in Whites, the relative risk of CHD incidence over 4 to 7 years of follow-up conferred by diabetes was 2.52 for men and 3.45 for women, lower for African Americans than for Whites (Watkins, 2004). However, because of the higher prevalence of diabetes, 27.8% of African American female cases were attributable to diabetes, as opposed to 8% of African male cases. Over 22 years of follow-up of the NHANES I cohort, heart disease was listed as the cause of death of 69.5% of diabetic subjects and mortality rates were 27% higher for diabetic African Americans than for diabetic Whites (Watkins, 2004).

A study performed by Duarte *et al.,* in 2003 on 521 patients suffering from acute coronary syndrome (including 159 diabetic patients) indicated that diabetes constitutes a powerful risk factor (as evident from the literature reviewed) for CHD and its complications and should

therefore be taken into consideration in clinical approaches to this pathology.

3.4 PRESENTATION

3.4.1 Atherosclerosis

Atherosclerosis, a slow degenerative process affecting the aorta, epicardial coronary arteries, carotids and other large arteries with high blood flow, starts early in life and generally progresses slowly unless accelerated by cigarette smoking, or disease such as diabetes mellitus, hypercholesterolemia or hypertension. Atherosclerosis is characterized by 3 major abnormalities: firstly, abnormal vasomotion with predominant vasoconstriction; secondly, structural alterations of the vascular wall (with outward and later inward remodeling), and thirdly, thrombus formation (Opie & Yellon, 1999). With increasing age atheromatous plaques progressively narrow one or more of the coronary arteries and the obstruction to blood flow may eventually become so severe that when exercise increases the oxygen consumption of the heart, not enough blood can pass through the arteries to supply it. According to Watkins, 2004, the distribution of 1-, 2- and 3 vessel disease among African Americans was similar to that observed among White patients from reports on series of patients undergoing cardiac catheterization at a variety of institutions, including urban public hospitals and veterans Administration hospitals in the 1980s and 1990s. The ischaemic muscle then produces the characteristic symptoms of angina pectoris ("pain of the chest", Latin) (Neal, 1997) and occlusion of a coronary artery may lead to irreversible acute myocardial damage (myocardial infarction).

Angina pectoris is therefore caused whenever the oxygen demand exceeds the supply and factors that are likely to cause angina are all those that increase oxygen demand.

3.4.2 Angina Pectoris

When obstruction caused by an uncomplicated atheromatous plaque exceeds a critical value, myocardial oxygen demand during exercise exceeds the ability of the stenosed vessel to supply oxygenated blood. Such patients complain of intermittent chest pain, brought on by exertion and relieved within a few minutes on resting. Such pain is probably caused by products of anaerobic metabolism in the working myocardium, formed as a result of the temporary imbalance between oxygen supply and demand (Ritter *et al.*, 1995). According

to the results of a study performed by Milner *et al.*, 1999, chest pain was the most frequently reported symptom in women.

3.4.2.1 Stable angina

Chest pain, brought on by exertion and relieved within a few minutes of resting.

3.4.2.2 Unstable angina

Unstable angina is defined as the presence of one or more of the following: a) crescendo angina, which is represented by more frequent, severe, or prolonged episodes of chest pain (ie an alteration in a past, stable pattern and when attacks occur with increased frequency and severity on less and less exertion); b) angina pectoris of new onset (usually within the last month); c) episodes of angina pectoris at rest (Jones *et al.*, 2001). Unstable angina may lead to myocardial infarction.

3.4.3 Myocardial infarction

Myocardial infarction is ischaemia that has reached the point of irreversibility and is the consequence of an imbalance between myocardial oxygen supply and demand. Sustained ischaemia will result in myocardial cell death or necrosis. Unstable angina and myocardial infarction occur as a result of fissuring of an atheromatous plaque in a coronary artery. Initially platelets adhere to the underlying subendothelium and a white thrombus, consisting mainly of platelet/fibrinogen aggregates, develops and extends into the lumen of the artery. Total occlusion of the artery is usually caused by a fibrin or red thrombus. In addition to mechanical obstruction caused by atheroma, with or without adherent thrombus, spasm of smooth muscle in the vascular media can contribute to ischaemia (Ritter *et al.*, 1995).

3.4.3.1 Q-wave myocardial infarction

This type of myocardial infarction is characterized by the development of Q-waves on the 12-lead electrocardiogram.

3.4.3.2 Non-Q-wave myocardial infarction

Non-Q-wave myocardial infarction was defined as an elevation of serum creatine-kinase (CK) values to a level greater than or equal to twice the upper limit of normal and CK-MB value greater than normal according

to the laboratory in the hospital, without the development of Q-waves on the 12-lead electrocardiogram (Stone *et al.*, 1996).

Table 3.4 Comparison of Q-wave and non-Q wave myocardial infarction (Vacek, 2002)

Q-wave	*Non-Q wave*
Presents with ST-segment elevation	Presents with ST-segment depression
High in-hospital mortality	Lower in-hospital mortality
Lower reinfarction rate after hospital discharge	High reinfarction rate after hospital discharge
Thrombolysis beneficial	Thrombolysis not recommended
Sustained coronary artery occlusion	Early spontaneous reperfusion common
Large infarct size	Small infarct size
Acute complications common	Acute complications uncommon
1-mo mortality rate, 10%-15%	1-mo mortality rate, 3%-5%
2-yr mortality rate, 30%	2-yr mortality rate, 30%

Only recently the pessimistic view that atherosclerotic disease is an inevitable result of the aging process, has been replaced by a far more optimistic concept that atherosclerosis can be prevented – or its onset and progression significantly delayed.

Because coronary artery disease is so common and because its main clinical manifestation, myocardial infarction, has such a high mortality rate, it is essential to detect those at high risk before the event occurs.

3.5 TREATMENT

The treatment of IHD includes the following:

3.5.1 Drug treatment

- Organic nitrates
- β-adrenergic antagonists
- Calcium channel blockers
- Aspirin

- Fibrinolytic drugs

Catheterization can identify patients who would benefit from surgery or PCI. Patients with significant disease in the left main coronary artery and patients with triple vessel disease usually survive longer when operated on (Ritter *et al.*, 1995).

The principles of the pharmacological treatment of angina pectoris include the following:

1) Reduction of oxygen demand
   - reduce heart rate by beta blockade
   - reduce afterload by beta blockade, calcium antagonists and control of hypertension
   - reduce metabolic demand by calcium antagonists, beta blockade
   - reduce preload by nitrates
2) Increase oxygen supply
   - coronary vasodilators (calcium antagonists, nitrates)
   - promote growth of collaterals (exercise)
   - change anatomy of coronary disease [coronary bypass grafting, percutaneous coronary intervention (PCI)]

The non-pharmacological treatment of angina pectoris includes the following:

- Exercise with controlling of resting and recording of blood pressure during exercise
- Weight reduction and avoidance of excess sodium intake [and reduction in serum cholesterol levels (dietary measures)]

Drugs to reduce ischaemia:

- Increase blood supply
- Reduce demand

To discontinue smoking (smoking increases the heart rate, which increases oxygen demand and components of tobacco act as vasoconstrictors, causing blood pressure to rise).

3.5.2 Percutaneous Transluminal Coronary Angioplasty (PTCA)

Percutaneous means putting a soft plastic tube (catheter) through the skin and into a blood vessel

Transluminal means the procedure is done inside the blood vessel

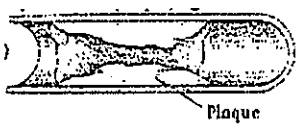
Coronary is the artery of the heart to be treated

Angioplasty is the procedure used to enlarge the opening of the artery

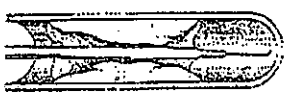
3.5.2.1 Angioplasty without stent

During PTCA, a catheter with a very small balloon at the tip passes through a main catheter in the aorta into the narrowed coronary artery (see Figure 3.2) *(http://www.viahealth.org/rgh/departments/cardiac/cardiacptca.htm).*

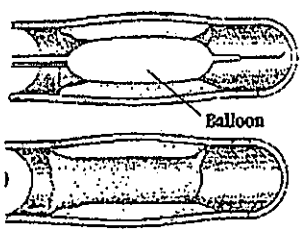
Figure 3.2 *Percutaneous Transluminal Coronary Angioplasty (PTCA) procedure*
(http://www.viahealth.org/rgh/departments/cardiac/cardiacptca.htm)



Coronary artery where fatty material (plaque) has deposited against the wall of the artery



A catheter with a small balloon at the tip passes through a main catheter in the aorta into the narrowed coronary artery



The balloon is inflated and deflated to flatten the fatty material. The opening of the artery is enlarged and more blood flow is allowed

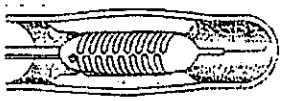
3.5.2.2 Angioplasty with a stent

A stent is a surgical stainless steel coil. The coil is wrapped tightly around a balloon-tipped catheter. The catheter is then inserted into the artery. Once in place, the balloon is inflated which then expands the coil. Once expanded, the permanent coil acts like a scaffold. It supports the coronary artery walls and keeps the artery open. This allows blood flow to the heart muscle and relieves symptoms *(http://www.viahealth.org/rgh/departments/cardiac/coronarystents.htm)*.

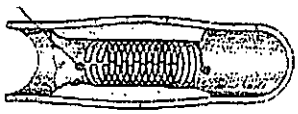
A stent may be used instead of, or along with, angioplasty. Stents are used depending on certain features of the artery blockage. This includes the size of the artery and where the blockage is.

Figure 3.3 *Coronary Stent Procedure*
(http://www.viahealth.org/rgh/departments/cardiac/coronarystents.htm)

A catheter with preloaded stent and angioplasty balloon is inserted into the narrowed artery



The balloon is inflated which expands the coil



The permanent coil supports the coronary artery walls and keeps the artery open

Angioplasty is a less demanding alternative to surgery and involves the dilatation of arterial segments with accessible lesions.

3.5.3 Surgery

Bypass surgery – bypass grafting using a patient's own saphenous vein or preferably mammary artery which is used to circumvent the diseased segment(s) of the coronary artery (Ritter *et al.*, 1995).

Coronary artery bypass graft (CABG) or heart bypass is a surgical procedure performed in patients with coronary artery disease, for the relief of angina and reduction in the risk of heart attack. Veins or arteries from elsewhere in the patient's body are grafted from the aorta to the coronary arteries, bypassing blockages caused by atherosclerosis and improving the blood supply to the myocardium (heart muscle).

First, the sternum is cut and the chest opened (a procedure known as "cracking the chest." The heart is usually stopped and the patient is connected to a bypass machine, which takes over the heart's functions for the duration of the operation. Blood vessels are harvested from elsewhere in the body for grafting.

Typically, the saphenous vein from the leg and the left internal mammary artery (LIMA) are used for the bypass. Veins used either have their valves removed or are turned around so that the valves in them do not occlude blood flow in the graft. LIMA grafts are longer-lasting than vein grafts, both because the artery is more robust than a vein and because, being already connected to the aorta, the LIMA need only be grafted at one end. For this reason, the LIMA is usually grafted to the left anterior descending artery (LAD), which supplies the left ventricle, the part of the heart that pumps oxygenated blood around the body.

Prognosis following CABG depends on a variety of factors, but successful grafts typically last around 10-15 years (http://www.fact-index.com/c/co/coronary_artery_bypass_surgery.html).

3.6 SUMMARY

From the reviewed literature it is clear that one of the most significant differences between men and women is the age of onset of CHD, which has caused a misconception that CVD mainly affects the male population. It is noted in the literature that the annual CVD mortality rate of women is in fact rising and exceeding that of men in the United States, which is a statistical issue that needs further investigation.

Risk factors

From the literature it can be seen that hypertension is more common among blacks than among their white counterparts and is a strong predictor of CHD especially in women.

Smoking is more prevalent among blacks, particularly black males, and is a major risk factor to the female population.

According to the literature, the death rate from CHD for smokers is two to three times that of non-smokers with 35% and 40% of deaths occurring before retirement age.

Diabetes in women is a risk factor that has a negative influence on the presentation and development of coronary artery disease in this population.

Diabetes is a greater coronary heart disease mortality (coronary heart disease mortality) risk factor among women than among men aged 50-59 years. The impact of diabetes is substantially greater for women as, when present, it eliminates their advantage over men.

Literature reported that diabetes is more prevalent among African Americans than any other ethnic group.

The findings in the literature review regarding risk factors could unfortunately not be compared to the retrospective study results due to the high percentage of unknown information in this regard in the patient files in the retrospective study.

Presentation

The literature reports that progression of atherosclerosis among men was almost twice that found among women up to the age of 60 years. The findings in the retrospective study will be compared with this statement from the literature.

From the literature study it is evident that coronary heart disease develop approximately 10 years later among women than it does among men. Women generally have higher HDL cholesterol levels and lower trigliseride levels than men, which is a protective factor for women.

A larger proportion of cholesterol in the plasma of women is high density lipoprotein (HDL), which protects women against IHD. After menopause LDL cholesterol rises and the advantage that women have is eliminated.

Estrogen lowers LDL levels and increases HDL levels, reducing the risk of cardiovascular disease, a situation that is not common for men.

The literature also revealed that African Americans have higher levels of HDL levels compared to their White counterparts.

Literature reports that the White population have higher burden of atherosclerosis and that angina is the most common presenting symptom in the female population.

Treatment

According to literature, young white males are more likely, to receive diagnostic and revascularization procedures than women, and males who are older or of African descent or women.

Complications are reported to be more prevalent in the female population following treatment.

4 *PATIENT REPORTS (RETROSPECTIVE)*

4.1 INTRODUCTION

In the retrospective study the files of patients with existing ischaemic heart disease undergoing heart catheterization at the department Cardiology, Universitas Hospital, Bloemfontein, from January 2001 until December 2002 were examined. The information was used to determine and compare the risk factor profile, presentation, treatment and response to treatment for IHD. For this project, the relevant information was extracted from the files (Hearts database) and documented on a self-compiled template (Appendix A). This information was typed into a self compiled database using Access software.

4.2 CLASSIFICATION OF PATIENT DATA

4.2.1 Demographic data

The demographic data that were collected for each patient from the patient reports included name and surname, birth date, computer number, age, race, gender, height, weight and place of origin (city or rural). The age of each patient was calculated as being from the date of birth up to the catheterization date (the catheterization date indicated on the name lists). The height and weight were documented as indicated in the Universitas Hospital catheterization room files.

4.2.2 Risk factors

The risk factors that were investigated in the patient reports for this study included a family history of ischaemic heart disease (if mentioned), hypertension (if mentioned), smoking (if mentioned), cholesterol (if mentioned) and diabetes mellitus (if mentioned). The above-mentioned risk factors were documented as being present (yes) or not present (no), or if nothing regarding a specific risk factor was mentioned in the patient reports, the risk factor was recorded as having an unknown status.

4.2.3 Aniographic presentation

The information collected from the patient reports regarding the angiographic presentation included the following:

It was documented whether the patient presented with stable or unstable angina (see definition used in Sections 3.4.2.1 and 3.4.2.2, respectively), Q-wave or non-Q-wave myocardial infarction (see definition used in Section 3.4.3.1 and 3.4.3.2, respectively) and the status of the effort test was also classified as either normal or abnormal. Further, the ventricular function was documented as either good, normal or impaired as indicated in the patient report. The amount of vessels affected was obtained from the first available catheterization report.

4.2.4 Treatment and response to treatment

The treatment of the patient could be either medical treatment for which the most recent regimen of medication was documented, angioplasty (PTCA or stent) or bypass surgery. Should bypass surgery have been performed on a patient, the number of vessels that the bypass was performed on were documented and also the number that were patent following the operation. If this information was not mentioned in the report it was documented as being unknown. Should more than one bypass have been performed, the initial bypass operation information was documented.

The response was documented as being either symptomatic (angina pectoris, PND, ortopnee, shortness of breath) or asymptomatic (no symptoms related to ischaemia). Should PTCA or bypass have been repeated, this was documented as repeat surgery and follow-up procedure.

4.3 RESULTS

A p-value below 0.05 indicates a significant difference. In the male patient group, an odd ratio below 1 implies that white males are more likely to undergo the specific procedure, have the condition or be on the specific drugs than black males. An odd ratio above 1 implies that the black males are more likely to undergo the specific procedure, have the condition or be on the specific drugs than white males.

In the white patient group, an odd ratio below 1 implies that white females are more likely to undergo the specific procedure, have the condition or be on the

specific drugs than white males. An odd ratio above 1 implies that the white males are more likely to undergo the specific procedure, have the condition or be on the specific drugs than white females.

4.3.1 Risk factors

Due to the high percentages of unknown values in Section 4.3.1, this data is only reported as a matter of interest and no statistical assessments were performed on this data. This data is reported in Appendix B (page 80).

4.3.2 Presentation

4.3.2.1 Stable angina, unstable angina, Q-wave myocardial infarction and non-Q wave myocardial infarction

Table 4.3.2.1.1 Stable angina, unstable angina, Q-wave myocardial infarction and non-Q wave myocardial infarction among White Males

The following table summarizes stable angina, unstable angina, Q-wave myocardial infarction and non Q-wave myocardial infarction among white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males White (Total 676 subjects)	*Stable angina*			*Unstable angina*			*Q-wave myocardial infarction*			*Non Q-wave myocardial infarction*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	23	42.6	54	12	22.2	54	19	35.2	54	0	0.0
45-54	142	44	31.0	142	47	33.1	142	46	32.4	142	5	3.5
55-64	216	97	44.9	216	57	26.4	216	58	26.9	216	4	1.9
65-74	195	110	56.4	195	42	21.5	195	39	20.0	195	4	2.1
75-84	69	28	40.6	69	17	24.6	69	21	30.4	69	3	4.4

Table 4.3.2.1.2 Stable angina, unstable angina, Q-wave myocardial infarction and non-Q wave myocardial infarction among White Females

The following table summarizes stable angina, unstable angina, Q-wave myocardial infarction and non Q-wave myocardial infarction among white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females White (Total 265 subjects)												
	Stable angina			*Unstable angina*			*Q-wave myocardial infarction*			*Non Q-wave myocardial infarction*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	10	6	60.0	10	3	30.0	10	0	0.0	10	1	10.0
45-54	24	11	45.8	24	5	20.8	24	6	25.0	24	2	8.3
55-64	71	43	60.6	71	16	22.5	71	10	14.1	71	2	2.8
65-74	119	75	63.0	119	31	26.1	119	10	8.40	119	3	2.5
75-84	41	23	56.1	41	13	31.7	41	5	12.2	41	0	0.0

Table 4.3.2.1.3 Stable angina, unstable angina, Q-wave myocardial infarction and non-Q wave myocardial infarction among Black Males

The following table summarizes stable angina, unstable angina, Q-wave myocardial infarction and non Q-wave myocardial infarction among black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males Black (Total 61 subjects)												
	Stable angina			*Unstable angina*			*Q-wave myocardial infarction*			*Non Q-wave myocardial infarction*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	5	0	0.0	5	1	20.0	5	4	80.0	5	0	0.0
45-54	22	10	45.5	22	4	18.2	22	6	27.3	22	2	9.1
55-64	21	8	38.1	21	4	19.1	21	9	42.9	21	0	0.0
65-74	10	5	50.0	10	1	10.0	10	4	40.0	10	0	0.0
75-84	3	1	33.3	3	0	0.0	3	2	66.7	3	0	0.0

WHITE MALES VS WHITE FEMALES

Stable angina

The White population indicated small differences (p-value is 0.0011) between males and females (see Table 4.3.2.1.1 and 4.3.2.1.2). With an odd ratio of 0.80 (*95% CI 0.70; 0.91), the results indicate that white females are more likely to experience stable angina than white males.

*95% CI: 95% Confidence Interval

BLACK MALES VS WHITE MALES

Stable angina

The male population indicated small differences (p-value is 0.7168) between white and black males (see Table 4.3.2.1.1 and 4.3.2.1.3). With an odd ratio of 0.94 (95% CI 0.68; 1.31), the results indicate that white males are more likely to experience stable angina than black males (very similar for both gender groups).

WHITE MALES VS WHITE FEMALES

Unstable angina

The White population indicated small differences (p-value is 0.8340) between males and females (see Table 4.3.2.1.1 and 4.3.2.1.2). With an odd ratio of 1.00 (*95% CI 0.93; 1.10), the results are very similar for both gender groups.

*95% CI: 95% Confidence Interval

BLACK MALES VS WHITE MALES

Unstable angina

The male population indicated small differences (p-value is 0.0598) between white and black males (see Table 4.3.2.1.1 and 4.3.2.1.3). With an odd ratio of 0.59 (95% CI 0.33; 1.06), the results indicate that white males are more likely to experience unstable angina than black males.

WHITE MALES VS WHITE FEMALES

Q-wave myocardial infarction

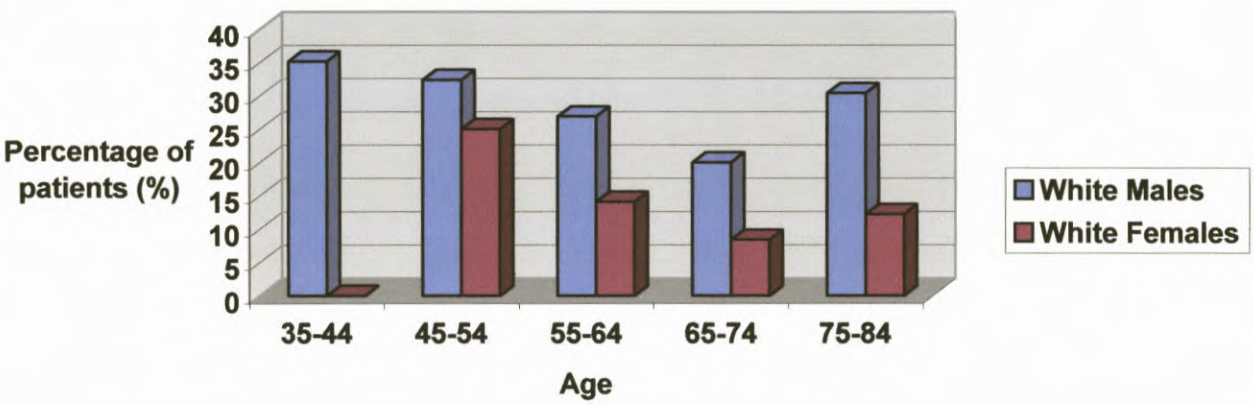
The White population indicated significant differences (p-value is <0.0001) between males and females (see Table 4.3.2.1.1 and 4.3.2.1.2). With an odd ratio of 2.12 (95% CI 1.49; 3.01), the results indicate that white males are more likely to suffer a Q-wave myocardial infarction than white females.

BLACK MALES VS WHITE MALES

Q-wave myocardial infarction

The male population indicated small differences (p-value is 0.0357) between white and black males (see Table 4.3.2.1.1 and 4.3.2.1.3). With an odd ratio of 1.45 (95% CI 1.05; 2.00), the results indicate that black males are more likely to suffer a Q-wave myocardial infarction than white males.

Figure 4.1 Comparison of the percentage of patients that experienced Q- wave myocardial infarctions in each age group for white males compared to white females



WHITE MALES VS WHITE FEMALES

Non Q-wave myocardial infarction

The White population indicated significant differences (p-value is 0.4507) between males and females (see Table 4.3.2.1.1 and 4.3.2.1.2). With an odd ratio of 0.73 (95% CI 0.33; 1.64), the results indicate that white females are more likely to suffer a non Q-wave myocardial infarction than white males.

BLACK MALES VS WHITE MALES

Non Q-wave myocardial infarction

The male population indicated small differences (p-value is 0.7441) between white and black males (see Table 4.3.2.1.1 and 4.3.2.1.3). With an odd ratio of 1.29 (95% CI 0.28; 5.82), the results indicate that black males are more likely to suffer a non Q-wave myocardial infarction than white males.

4.3.2.2 Normal ventricular function of Black Males

Table 4.3.2.2.1 Normal ventricular function of black males

The following table summarizes normal ventricular function of black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males			
Black			
	Normal ventricular function		
Years	*Total*	*n*	*%*
35-44	4	2	50.0
45-54	21	10	47.6
55-64	20	11	55.0
65-74	9	8	88.9
75-84	1	0	0.0
Unknown (%)	4.6 – 66.7		

4.3.2.2.2 Normal ventricular function of White Males

The following table summarizes normal ventricular function of white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males			
White			
	Normal ventricular function		
Years	*Total*	*n*	*%*
35-44	48	35	72.9
45-54	129	102	79.1
55-64	201	142	70.7
65-74	183	135	73.8
75-84	65	49	75.4
Unknown (%)	6.2 – 11.1		

4.3.2.2.3 Normal ventricular function in White Females

The following table summarizes normal ventricular function in white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females			
White			
	Normal ventricular function		
Years	*Total*	*n*	*%*
35-44	9	6	66.7
45-54	23	20	87.0
55-64	60	49	81.7
65-74	109	95	87.2
75-84	38	35	92.1
Unknown (%)	4.2 – 15.5		

WHITE MALES VS WHITE FEMALES

Normal ventricular function

The White population indicated significant differences (p-value is 0.0002) between males and females (see Table 4.3.2.2.2 and 4.3.2.2.3). With an odd ratio of 0.54 (95% CI 0.39; 0.76), the results indicate that white females are more likely to have normal ventricular function than white males.

BLACK MALES VS WHITE MALES

Normal ventricular function

The Male population indicated significant differences (p-value is 0.0044) between black and white males (see Table 4.3.2.2.1 and 4.3.2.2.2). With an odd ratio of 0.58 (95% CI 0.41; 0.82), the results indicate that white males are more likely to have normal ventricular function than black males.

4.3.2.3 Body Mass Index (BMI)

4.3.2.3.1 Body Mass Index (BMI) for White Males

The following table summarizes the body mass index for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups. Body mass index (BMI) was calculated as follows: $BMI = \frac{\text{weight (in kg)}}{[\text{height (in m)}]^2}$

A BMI value of ≤ 19 = underweight
20 –24 = normal
25-29 = overweight
30+ = obese

Males												
White												
	≤ 19			20 – 24			25 – 29			30+		
Years	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	0	0.0	54.0	12	22.2	54	22	40.7	54	20	37.0
45-54	137	4	2.92	137.0	32	23.4	137	51	37.2	137	50	36.5
55-64	215	7	3.26	215.0	64	29.8	215	88	40.9	215	56	26.1
65-74	192	10	5.21	192.0	46	24.0	192	94	49.0	192	42	21.9
75-84	66	3	4.55	66.0	19	28.8	66	32	48.5	66	12	18.2
Unknown (%)	0.5											

4.3.2.3.2 Body Mass Index for White Females

The following table summarizes the body mass index for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups. Body mass index (BMI) was calculated as follows: $BMI = \frac{\text{weight (in kg)}}{[\text{height (in m)}]^2}$

A BMI value of ≤ 19 = underweight
20 –24 = normal
25-29 = overweight
30+ = obese

Females												
White												
	≤ 19			20 – 24			25 - 29			30+		
Years	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	10	0	0.0	10	3	30.0	10	4	40.0	10	3	30.0
45-54	24	2	8.33	24	7	29.2	24	3	12.5	24	12	50.0
55-64	70	4	5.71	70	22	31.4	70	25	35.7	70	19	27.1
65-74	113	2	1.77	113	26	23.0	113	47	41.6	113	38	33.6
75-84	39	2	5.13	39	19	48.7	39	13	33.3	39	5	12.8
Unknown (%)	0.5											

4.3.2.3.3 Body Mass Index for Black Males

The following table summarizes the body mass index for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups. Body mass index (BMI) was calculated as follows: $BMI = \frac{\text{weight (in kg)}}{[\text{height (in m)}]^2}$

A BMI value of ≤ 19 = underweight
20 –24 = normal
25-29 = overweight
30+ = obese

Males												
Black												
	≤ 19			20 – 24			25 - 29			30+		
Years	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	5	0	0.0	5	2	40.0	5	2	40.0	5	1	20.0
45-54	21	1	4.8	21	6	28.6	21	12	57.1	21	2	9.52
55-64	21	2	9.5	21	8	38.1	21	5	23.8	21	6	28.6
65-74	10	0	0.0	10	4	40.0	10	3	30.0	10	3	30.0
75-84	3	0	0.0	3	2	66.7	3	1	33.3	3	0	0.0
Unknown (%)	0.5											

WHITE MALES VS WHITE FEMALES

Body Mass Index

The White population indicated significant differences (p-value is 0.0776) between males and females (see Table 4.3.2.3.1 and 4.3.2.3.2). With an odd ratio of 0.82 (95% CI 0.65; 1.03♠), the results indicate that white females are more likely to be obese than white males.

♠ BMI obese vs non-obese

BLACK MALES VS WHITE MALES

Body Mass Index

The Male population indicated small differences (p-value is 0.1972) between black and white males (see Table 4.3.2.3.1 and 4.3.2.3.3). With an odd ratio of 0.69 (95% CI 0.41; 1.56♠), the results indicate that white males are more likely to be obese than black males.

♠ BMI obese vs non-obese

4.3.2.4 Affected vessels

4.3.2.4.1 Affected vessels (1-, 2- or 3 vessel disease) of Black Males

The following table summarizes the affected vessel status (1-, 2- or 3 vessel disease) of black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males									
Black									
	1 Affected vessel			*2 Affected vessels*			*3 Affected vessels*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	5	1	20.0	5	4	80.0	5	0	0.0
45-54	21	4	1.1	21	4	19.1	21	13	61.9
55-64	20	2	10.0	20	5	25.0	20	13	65.0
65-74	9	4	44.4	9	2	22.2	9	3	33.3
75-84	3	0	0.0	3	1	33.3	3	2	66.7
Unknown (%)	4.6 – 10.0								

4.3.2.4.2 Affected vessels (1-, 2- or 3 vessel disease) of White Males

The following table summarizes the affected vessel status (1-, 2- or 3 vessel disease) of white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males									
White									
	1 Affected vessel			*2 Affected vessels*			*3 Affected vessels*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	53	12	22.6	53	9	17.0	53	32	60.4
45-54	138	14	10.1	138	34	24.6	138	90	65.2
55-64	203	18	8.9	203	40	19.7	203	145	71.4
65-74	187	13	7.0	187	29	15.5	187	145	77.5
75-84	69	4	5.8	69	17	24.6	69	48	69.6
Unknown (%)	1.85 – 6.02								

4.3.2.4.3 Affected vessels (1-, 2- or 3 vessel disease) of White Females

The following table summarizes the affected vessel status (1-, 2- or 3 vessel disease) of white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

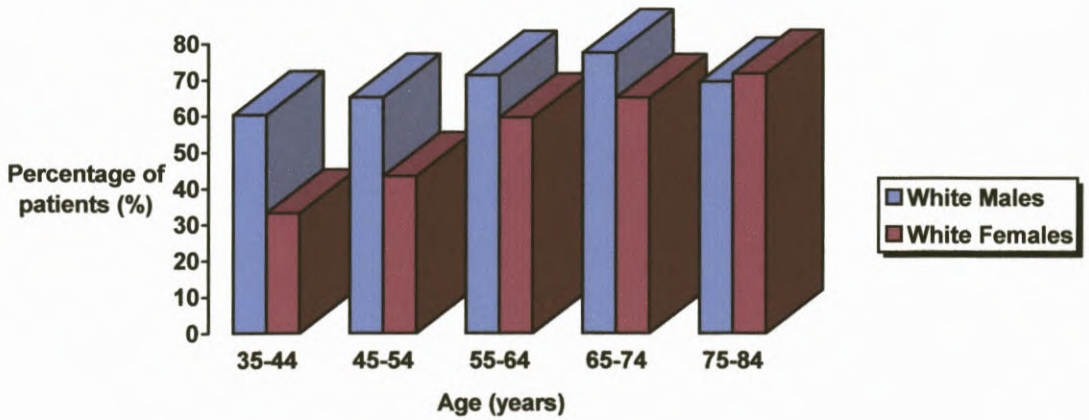
Females									
White									
Years	*1 Affected vessel*			*2 Affected vessels*			*3 Affected vessels*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	9	1	11.1	9	5	55.6	9	3	33.3
45-54	23	9	39.1	23	4	17.4	23	10	43.5
55-64	67	12	17.9	67	15	22.4	67	40	59.7
65-74	109	15	13.8	109	23	21.1	109	71	65.1
75-84	39	5	12.8	39	6	15.4	39	28	71.8
Unknown (%)	4.2 – 10.0								

WHITE MALES VS WHITE FEMALES

3 Affected vessels

The White population indicated significant differences (p-value is <0.0001) between males and females (see Table 4.3.2.4.2 and 4.3.2.4.3). The percentage of white females with 3 affected vessels is higher for the older age groups than for white males.

Figure 4.2 Comparison of percentages of patients with three affected vessels in each age group for white males compared to white females



2 Affected vessels

The percentages of white females with 2 affected vessels is higher than for white males except for the 45-54 and 75-84 years age groups.

1 Affected vessel

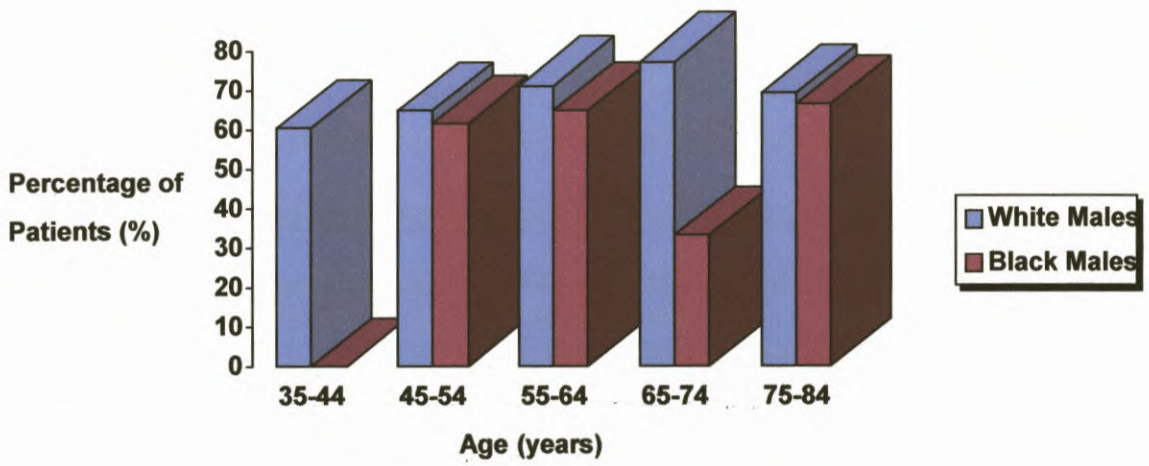
The percentages of white females with 1 affected vessel is higher than for white males except for the 35-44 years age group.

BLACK MALES VS WHITE MALES

3 Affected vessels

The Male population indicated significant differences (p-value is 0.0272) between blacks and whites (see Table 4.3.2.4.1 and 4.3.2.4.2). The percentages of 3 vessel disease are lower among black males than among white males, especially in the 35-44 and 65-74 years age groups.

Figure 4.3 Comparison of percentages of patients with three affected vessels in each age group for white males compared to black males



2 Affected vessels

The percentages of white males and black males with 2 affected vessels is very similar in all the age groups except for the 35-44 years age group where the percentages for black males are much higher than for white males.

1 Affected vessel

The percentages of white males with 1 affected vessel is higher than for black males in the 35-44, 45-54 and 75-84 years age groups.

4.3.3 Treatment

4.3.3.1 Medical, PTCA, Stent or Bypass treatment

4.3.3.1.1 Medical, PTCA, Stent or Bypass treatment for Black Males

The following table summarizes the treatment status [medical, PTCA (with and without stent) or bypass] of black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males												
Black												
Years	*Medical*			*PCI - PTCA*			*PCI – Stent*			*Bypass*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	4	4	100.0	5	0	0.0	5	0	0.0	5	0	0.0
45-54	19	19	100.0	22	5	22.7	22	6	27.3	22	2	9.1
55-64	17	17	100.0	21	6	28.6	21	5	23.8	21	3	14.3
65-74	10	10	100.0	10	5	50.0	10	2	20.0	10	0	0.0
75-84	2	2	100.0	3	2	66.7	3	2	66.7	3	1	33.3
Unknown (%)	13.6 – 33.3			0.00			0.00			0.00		

4.3.3.1.2 Medical, PTCA, Stent or Bypass treatment for White Males

The following table summarizes the treatment status [medical, PTCA (with and without stent) or bypass] of white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males												
White												
Years	*Medical*			*PCI - PTCA*			*PCI – Stent*			*Bypass*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	50	49	98.0	54	18	33.3	54	14	25.9	54	16	29.6
45-54	124	124	100.0	141	43	30.5	141	46	32.6	142	53	37.3
55-64	193	193	100.0	212	52	24.5	212	41	19.3	214	86	40.2
65-74	171	171	100.0	192	43	22.4	193	37	19.2	195	102	52.3
75-84	61	61	100.0	68	23	33.8	69	17	24.6	68	28	41.2
Unknown (%)	7.4 – 12.7			0.7 – 1.9			0.7 – 1.9			0.9 – 1. 5		

4.3.3.1.3 Medical, PTCA, Stent or Bypass treatment for White Females

The following table summarizes the treatment status [medical, PTCA (with and without stent) or bypass] in white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

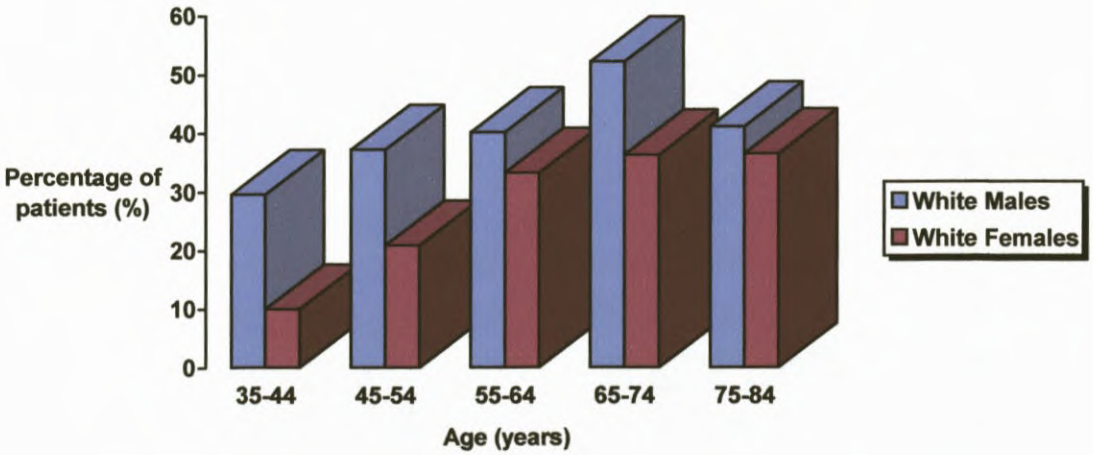
Females												
White												
Years	*Medical*			*PCI - PTCA*			*PCI - Stent*			*Bypass*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	7	7	100.0	10	1	10.0	10	2	20.0	10	1	10.0
45-54	19	19	100.0	23	4	17.4	23	5	21.7	24	5	20.9
55-64	61	61	100.0	68	16	23.5	69	14	20.3	69	23	33.3
65-74	106	105	99.1	117	29	24.8	116	25	21.6	118	43	36.4
75-84	36	36	100.0	41	11	26.8	41	8	19.5	41	15	36.6
Unknown (%)	10.9 – 30.0			1.7 – 4.2			2.5 – 4.2			0.8 – 2.8		

WHITE MALES VS WHITE FEMALES

Bypass surgery

The White population indicated significant differences (p-value is 0.0007) between males and females (see Table 4.3.3.1.2 and 4.3.3.1.3). With an odd ratio of 1.38 (95% CI 1.14; 1.68), the results indicate that white males are more likely to undergo bypass surgery than white females.

Figure 4.4 Comparison of percentages of patients that underwent bypass surgery in each age group for white males compared to white females



PTCA without a stent

The White population indicated small differences (p-value is 0.3789) between males and females (see Table 4.3.3.1.2 and 4.3.3.1.3). With an odd ratio of 1.12 (95% CI 0.86; 1.46), the results indicate that white males are more likely to undergo PTCA without a stent treatment than white females.

PTCA with a stent

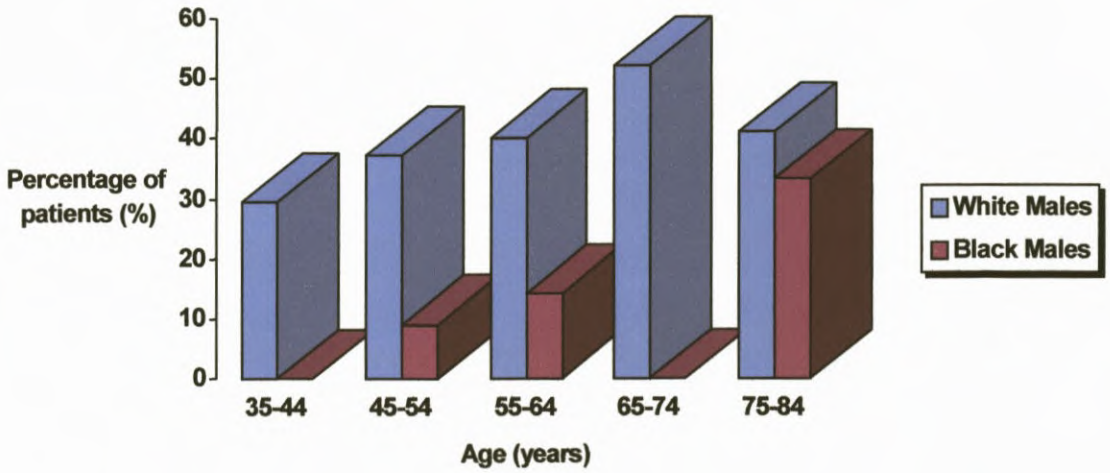
The White population indicated small differences (p-value is 0.7504) between males and females (see Table 4.3.3.1.2 and 4.3.3.1.3). With an odd ratio of 1.05 (95% CI 0.79; 1.39), the results indicate that white males are more likely to undergo PTCA with a stent treatment than white females.

BLACK MALES VS WHITE MALES

Bypass surgery

The Male population indicated significant differences (p-value is <0.0001) between blacks and whites (see Table 4.3.3.1.1 and 4.3.3.1.2). With an odd ratio of 0.24 (95% CI 0.11; 0.52), the results indicate that white males are more likely to undergo bypass surgery than black males.

Figure 4.5 Comparison of percentages of patients that underwent bypass surgery in each age group for white males compared to black males



PTCA without a stent

The Male population indicated small differences (p-value is 0.6864) between blacks and whites (see Table 4.3.3.1.1 and 4.3.3.1.2). With an odd ratio of 1.09 (95% CI 0.73; 1.63), the results indicate that black males are more likely to undergo PTCA without a stent treatment than white males.

PTCA with a stent

The Male population indicated small differences (p-value is 0.9840) between blacks and whites (see Table 4.3.3.1.1 and 4.3.3.1.2). With an odd ratio of 1.00 (95% CI 0.63; 1.57), the results indicate that white males and black males undergo PTCA with a stent treatment at very similar rates.

Due to the small sample sizes in the following section (drug treatment), it was decided to report only the statistically significant results in this section. The remaining tables are listed in Appendix D (page 86).

4.3.3.2 Positive inotropic and anti-arrhythmia drug treatment

4.3.3.2.1 Positive inotropic and anti-arrhythmia drug treatment for White Males

The following table summarizes the drug treatment [positive inotropic and anti-arrhythmia drugs] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
White						
Years	+Inotropic drug			Anti-arrhythmia		
	Total	n	%	Total	n	%
35-44	54	3	5. 6	54	3	5.6
45-54	142	9	6.3	142	6	4.2
55-64	216	25	11.6	216	24	11.1
65-74	195	32	16.4	195	24	12.3
75-84	69	6	8.7	69	7	10.1

4.3.3.2.2 Positive inotropic and anti-arrhythmia drug treatment for White females

The following table summarizes the drug treatment [positive inotropic and anti-arrhythmia drugs] for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females						
White						
Years	+Inotropic drug			Anti-arrhythmia		
	Total	n	%	Total	n	%
35-44	10	0	0.0	10	0	0.0
45-54	24	1	4.2	24	3	12.5
55-64	71	4	5.6	71	1	1.4
65-74	119	7	5.9	119	10	8.4
75-84	41	3	7.3	41	2	4.9

WHITE MALES VS WHITE FEMALES

Positive inotropic drugs

The White population indicated significant changes (p-value is 0.0026) between males and females (see Table 4.3.3.2.1 and 4.3.3.2.2). With an odd ratio of 2.21 (95% CI 1.29; 3.79), the results indicate that white males are more likely to be on positive inotropic drug treatment than white females.

Anti-arrythmic drugs

The White population indicated significant changes (p-value is 0.0333) between males and females (see Table 4.3.3.2.1 and 4.3.3.2.2). The association was different in different age groups and therefore an odd ratio was not calculated.

4.3.3.3 ACE inhibitor and Diuretic drug treatment

4.3.3.3.1 ACE inhibitor and Diuretic drug treatment for White Males

The following table summarizes the drug treatment [ACE inhibitor and diuretic drugs] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
White						
	ACE inhibitor			*Diuretics*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	25	46.3	54	12	22.2
45-54	142	65	45.8	142	29	20.4
55-64	216	110	50.9	216	70	32.4
65-74	195	96	49.2	195	66	33.9
75-84	69	25	36.2	69	21	30.4

4.3.3.3.2 ACE inhibitor and Diuretic drug treatment for Black Males

The following table summarizes the drug treatment [ACE inhibitor and diuretic drugs] for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
Black						
	ACE inhibitor			*Diuretics*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	5	3	60.0	5	1	20.0
45-54	22	17	77.3	22	12	54.6
55-64	21	15	71.4	21	9	42.9
65-74	10	6	60.0	10	4	40.0
75-84	3	3	100.0	3	3	100.0

BLACK MALES VS WHITE MALES

ACE inhibitor drugs

The Male population indicated significant changes (p-value is 0.0003) between blacks and whites (see Table 4.3.3.3.1 and 4.3.3.3.2). With an odd ratio of 1.51

(1.26; 1.81), the results indicate that black males are more likely to be on ACE inhibitor drug treatment than white males.

Diuretics

The Male population indicated significant changes (p-value is 0.0012) between blacks and whites (see Table 4.3.3.3.1 and 4.3.3.3.2). With an odd ratio of 1.72 (1.28; 2.32), the results indicate that black males are more likely to be on diuretic drug treatment than white males.

4.3.3.4 Anti-platelet and Calcium channel blocker drug treatment

4.3.3.4.1 Anti-platelet and Calcium channel blocker drug treatment for White Males

The following table summarizes the drug treatment [anti-platelet and calcium channel blocker drugs] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
White						
	Anti-platelet drugs			*Ca channel blocker*		
Years	*Total*	*N*	*%*	*Total*	*n*	*%*
35-44	54	48	88.9	54	3	5.6
45-54	142	116	81.7	142	10	7.1
55-64	216	171	79.2	216	32	14.8
65-74	195	150	76.9	195	33	16.9
75-84	69	50	72.5	69	12	17.4

4.3.3.4.2 Anti-platelet and Calcium channel blocker drug treatment for White Females

The following table summarizes the drug treatment [anti-platelet and calcium channel blocker drugs] for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females						
White						
	Anti-platelet drugs			*Ca channel blocker*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	10	6	60.0	10	2	20.0
45-54	24	17	70.8	24	2	8.33
55-64	71	48	67.6	71	16	22.5
65-74	119	90	75.6	119	33	27.7
75-84	41	26	63.4	41	9	22.0

WHITE MALES VS WHITE FEMALES

Anti-platelet drugs

The White population indicated significant changes (p-value is 0.0139) between males and females (see Table 4.3.3.4.1 and 4.3.3.4.2). With an odd ratio of 1.11 (95% CI 1.01; 1.22), the results indicate that white males are more likely to be on anti-platelet drug treatment than white females.

Calcium channel blocker drugs

The White population indicated significant changes (p-value is 0.0036) between males and females (see Table 4.3.3.4.1 and 4.3.3.4.2). With an odd ratio of 0.65 (0.48; 0.87), the results indicate that white females are more likely to be on calcium channel blocker drug treatment than white males.

4.3.3.5 Lipid lowering and nitrate drug treatment for Black Males

4.3.3.5.1 Lipid lowering and nitrate drug treatment for Black Males

The following table summarizes the drug treatment [lipid lowering drugs and nitrates] for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
Black						
	Lipid lowering drugs			*Nitrates*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	5	1	20.0	5	4	80.0
45-54	22	5	22.7	22	7	31.8
55-64	21	4	19.1	21	13	61.9
65-74	10	0	0.0	10	4	40.0
75-84	3	0	0.0	3	2	66.7

4.3.3.5.2 Lipid lowering and nitrate drug treatment for White Males

The following table summarizes the drug treatment [lipid lowering drugs and nitrates] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
White						
	Lipid lowering drugs			*Nitrates*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	29	53.7	54	20	37.0
45-54	142	74	52.1	142	38	26.8
55-64	216	71	32.9	216	74	34.3
65-74	195	56	28.7	195	86	44.1
75-84	69	16	23.2	69	28	40.6

4.3.3.5.3 Lipid lowering and nitrate drug treatment for White Females

The following table summarizes the drug treatment [lipid lowering drugs and nitrates] for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females						
White						
	Lipid lowering drugs			*Nitrates*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	10	6	60.0	10	4	40.0
45-54	24	10	41.7	24	8	33.3
55-64	71	28	39.4	71	26	36.6
65-74	119	52	43.7	119	44	37.0
75-84	41	12	29.3	41	18	43.9

WHITE MALES VS WHITE FEMALES

Lipid lowering drugs

The White population indicated significant changes (p-value is 0.0234) between males and females (see Table 4.3.3.5.2 and 4.3.3.5.3). With an odd ratio of 0.81 (95% CI 0.67; 0.97), the results indicate that white females are more likely to be on lipid lowering drug treatment than white males.

BLACK MALES VS WHITE MALES

Lipid lowering drugs

The Male population indicated significant changes (p-value is 0.0002) between blacks and whites (see Table 4.3.3.5.1 and 4.3.3.5.2). With an odd ratio of 0.40 (95% CI 0.23; 0.72), the results indicate that white males are more likely to be on lipid lowering drug treatment than black males.

Nitrates

The Male population indicated significant changes (p-value is 0.0151) between blacks and whites (see Table 4.3.3.5.1 and 4.3.3.5.2). With an odd ratio of 1.46 (95% CI 1.11; 1.92), the results indicate that black males are more likely to be on nitrate drug treatment than white males.

4.3.4 Response to treatment

4.3.4.1 Symptomatic response to treatment

4.3.4.1.1 White Males with symptomatic response to treatment

The following table summarizes the symptomatic response to treatment of white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males			
White			
Years	Symptomatic		
	Total	*n*	*%*
35-44	54	31	57.4
45-54	142	94	66.2
55-64	216	146	67.6
65-74	195	127	65.1
75-84	69	42	60.9

4.3.4.1.2 Black Males with symptomatic response to treatment

The following table summarizes the symptomatic response to treatment of black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males			
Black			
Years	Symptomatic		
	Total	*n*	*%*
35-44	5	4	80.0
45-54	22	15	68.2
55-64	21	16	76.2
65-74	10	8	80.0
75-84	3	2	66.7

4.3.4.1.3 White Females with symptomatic response to treatment

The following table summarizes the symptomatic response to treatment of white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females			
White			
Years	Symptomatic		
	Total	*n*	*%*
35-44	10	9	90.0
45-54	24	13	54.2
55-64	71	45	63.4
65-74	119	81	68.1
75-84	41	27	65.9

WHITE MALES VS WHITE FEMALES

The White population indicated small differences (p-value is 0.8776) between males and females (see Table 4.3.4.1.1 and 4.3.4.1.3). With an odd ratio of 0.99 (95% CI 0.89; 1.10), the results indicate, following treatment, that white females are more likely to be symptomatic than white males.

BLACK MALES VS WHITE MALES

The Male population indicated small differences (p-value is 0.2039) between blacks and whites (see Table 4.3.4.1.1 and 4.3.4.1.2). With an odd ratio of 1.12 (95% CI 0.96; 1.32), the results indicate that, following treatment, black males are more likely to be symptomatic than white males.

4.4 SUMMARY OF ASSOCIATIONS

Cochran-Mantel-Haenszel tests of association were performed to test the association between gender and outcomes (in white patients) and race and outcomes (in males), adjusted for age groups. Odds ratios with 95% confidence intervals were calculated for males versus females (in white patients) and Blacks vs Whites (in male patients) adjusting for age groups.

4.4.1 Summary of associations between gender and outcomes

Table 4.4.1 summarises the associations found between gender and outcomes.

Table 4.4.1 Summary of associations between gender and outcomes

WHITES		
Variable	**p-value**	**Odd ratio M vs F (95% CI)**
BMI grp	***0.0776***	***0.82 (0.65; 1.03)****
Stable angina	0.0011	0.80 (0.70; 0.91)
Unstable angina	0.8340	1.00 (0.93; 1.10)
Q-wave	***<0.0001***	***2.12 (1.49; 3.01)***
Non- Q- wave	0.4507	0.73 (0.33; 1.64)
Normal ventr function	***0.0002***	***0.54 (0.39; 0.76)***
Bypass	***0.0007***	***1.38 (1.14; 1.68)***
PCI-stent	0.7504	1.05 (0.79; 1.39)
PCI-PTCA	0.3789	1.12 (0.86; 1.46)
Symptomatic	0.8776	0.99 (0.89; 1.10)
Affected vessels	***<0.0001***	
+ Inotropic drugs	***0.0026***	***2.21 (1.29; 3.79)***
Anti arrhythmia drugs	***0.0333***	***association different in different***
ACE_inhibitors	0.5779	1.05 (0.89; 1.22)
ACE_inhibitors + calcium channel blockers	data too sparse	
ACE_inhibitors + diuretics	0.7578	0.85 (0.31; 2.37)
Anti hypertensive drugs	data too sparse	
Anti platelet drugs	***0.0139***	***1.11 (1.01; 1.22)***
Angiotensin receptor blockers	0.4486	0.72 (0.31; 1.69)
Beta blockers	0.0927	1.18 (0.97; 1.44)
Calcium channel blockers	***0.0036***	***0.65 (0.48; 0.87)***
Diuretics	0.1285	0.85 (0.70; 1.04)
Lipid lowering drugs	***0.0234***	***0.81 (0.67; 0.97)***
Nitrates	0.8121	1.02 (0.85; 1.23)

*BMI obese vs non-obese

4.4.2 Summary of associations between race and outcomes

Table 4.4.2 summarises the associations found between race and outcomes.

Table 4.4.2 Summary of associations between race and outcomes

MALES		
Variable	**p-value**	**Odd ratio Blacks vs Whites(95% CI)**
BMI grp	0.1972	0.69 (0.41; 1.56)*
Stable angina	0.7168	0.94 (0.68; 1.31)
Unstable angina	0.0598	0.59 (0.33; 1.06)
Q-wave	0.0357	1.45 (1.05; 2.00)
Non- Q- wave	0.7441	1.29 (0.28; 5.82)
Normal ventr function	***0.0044***	***0.58 (0.41; 0.82)***
Bypass	***<0.0001***	***0.24 (0.11; 0.52)***
PCI-stent	0.9840	1.00 (0.63; 1.57)
PCI-PTCA	0.6864	1.09 (0.73; 1.63)
Symptomatic	0.2039	1.12 (0.96; 1.32)
Affected vessels	***0.0272***	
+ Inotropic drugs	***0.7359***	association different in different age groups
Anti arrhythmia drugs	0.1545	0.38 (0.09; 1.58)
ACE_inhibitors	***0.0003***	***1.51 (1.26; 1.81)***
ACE_inhibitors + calcium channel blockers	data too sparse	
ACE_inhibitors + diuretics	data too sparse	
Anti hypertensive drugs	data too sparse	
Anti platelet drugs	0.5737	0.96 (0.83; 1.11)
Angiotensin receptor blockers	Data too sparse	
Beta blockers	0.1993	association different in different age groups
Calcium channel blockers	0.0734	1.67 (0.97; 2.89)
Diuretics	***0.0012***	***1.72 (1.28; 2.32)***
Lipid lowering drugs	***0.0002***	***0.40 (0.23; 0.72)***
Nitrates	***0.0151***	***1.46 (1.11; 1.92)***

*BMI obese vs non-obese

4.5 SUMMARY

In the Black population the sample size of the females was too small for the assessments on this group to be meaningful. Therefore, the statistical evaluation of gender was only performed on the White population, and the evaluation of race was only done with regards to the male population.

Concerning the White population for males compared to females, the BMI group, Q-wave myocardial infarctions, normal ventricular function, bypass surgery, affected vessels, positive inotropic drugs, anti arrythmia drugs, anti-platelet drugs, calcium channel blockers and lipid lowering drugs showed significant differences (indicated in bold and italics in Section 4.4). The results for each of these groups are discussed under the relevant tables in Section 4.3.

Significant differences between blacks and whites among the male population include normal ventricular function, bypass surgery, affected vessels, ACE inhibitor drugs, diuretics, lipid lowering drugs and nitrates (indicated in bold and italics in Section 4.4).

5 *DISCUSSION, RECOMMENDATIONS AND CONCLUSION*

5.1 INTRODUCTION

In this chapter the findings of this study will be discussed and recommendations will be made. Further investigations will be identified and an overall conclusion will be drawn after completion of this research.

5.2 DISCUSSION

- According to the literature 1,-2, and 3 vessel disease distribution is similar for African Americans and Whites. The results from this study showed that the distribution of 1-, 2- and 3 vessel disease differed in white males when compared to black males and the percentage of 3 vessel disease is lower among black males than among white males, especially in the 35-44 and 65-74 year age groups. These findings are in agreement with statements in the literature that whites have a greater burden of atherosclerosis than blacks.

- When the age groups are investigated in terms of 3 vessel disease among males compared to females it is evident that CHD presents among females (especially in the white population), approximately 10 years later than for males. These findings are similar to those already reported in the literature.

- According to the literature, angina is the most common presenting symptom among women and this research indicated that stable angina is more prevalent among white females than white males. White males however had higher prevalence of myocardial infarction which is also reflected in the worse left ventricular function found in the white males compared to white females.

- At the time of presentation of the clinical ischaemic event to hospital, black males presented more often with myocardial infarction than white males. This is also probably responsible for the lower left ventricular function in the black males. The left ventricular dysfunction being more prominent in the black males is surely also responsible for the higher use of ACE-inhibitors, diuretics and nitrates (drugs commonly used in heart failure) in the black males when compared to the white males.

- It is reported in various articles that fewer African Americans are on lipid lowering drug treatment than white patients are. This study confirmed this fact when the results showed that white male patients are more likely to be on lipid lowering drug treatment compared to black male patients. This can probably be ascribed to the fact that lipid values are generally lower in black patients. Due to the high percentages of unknown values for risk factors investigated in this study (family history, smoking, hypertension, hypercholesterolemia and diabetes mellitus), this data is only reported for interest sake [see Appendix B (page 80) Tables 1.1.1 to 1.1.3 and Appendix E (page 98) Table 1.1.1] and no statistical assessments were performed on this data. However, if these tables are reviewed, it is evident that the hypercholesterolemia values are lower in the black patients. This research also showed that more white females are on lipid lowering treatment and calcium channel blockers compared to white males. No obvious reason is evident for this as also for the higher use of anti-platelet drugs in white males.

- It is very often stated in the literature that African Americans are less likely to receive diagnostic or revascularization procedures than Whites are. This research clearly indicated that white males are more likely to undergo bypass surgery than white females and black males are. This can partly be explained by the fact that 3 vessel disease was more prevalent in white males when compared to black males and younger white females. Only in older white females was there a higher incidence of triple vessel disease.

- The white females and black males remained more symptomatic following treatment when compared to the white males. This may be due

to the greater use of coronary artery bypass graft surgery in white males. More complete revascularization is generally achieved by bypass surgery.

5.3 RECOMMENDATIONS

For further studies it will contribute to the success of the research if the data captured in the patient reports on the database are transferred from the catheterization files without writing errors. Checking this data could avoid these errors.

The medical history (risk factors) of the patients, including smoking, cholesterol status, diabetes mellitus, hypertension, family history of IHD should be transferred to recent patient reports on the database.

5.4 LIMITATIONS OF STUDY

Risk factor information was lacking in the patient reports. This was because the database was only created 10 years ago and the medical history of the patient is usually recorded with the first visit and this was a long time ago for the majority of the patients. For many of the patients this information was documented in more recent reports on the database, but it was lacking in a lot of cases. For this reason, the risk factor data could not be assessed statistically.

A significant limitation of this study was the small number of black and female patients in the Universitas Hospital, Bloemfontein patient files. The numbers in these specific groups were not adequate to perform meaningful statistical evaluations regarding these groups. In the foreseeable future these numbers might increase and an investigational study in this regard may be meaningful.

5.5 FIELDS FOR FURTHER INVESTIGATION

In both the statistical assessments performed (comparison between white males and females and comparison between black and white males) there were significant changes in the bypass surgery. A possible future study could investigate the amount of bypass surgery performed during 2005 and 2006 in terms of gender and race and compare this to the statistics collected in this study.

5.6 CONCLUSION

This research clearly indicates that there is indeed reason for race and gender to be recognized as major factors in the assessment and management of patients with ischaemic heart disease.

In this smaller study there does not appear to be major differences in medical care between the different groups. This is evident for example in the similar incidence of stent placements in black and white males. White males, however, were more likely to undergo bypass surgery in this study. Due to the new dispensation in South Africa, this situation might change in future because of the fact that the black population currently have more access to medical schemes and hospitalization.

More research is needed particularly in the female and black populations to ensure the rapid development of a strategy to optimally manage the growing number of patients with ischaemic heart disease. The differences in medical care received by African Americans as opposed to Whites and also a lower rate of awareness of risk factors by African Americans might explain the slower decline in CHD among African Americans than among Whites over the past few years.

This study shows that approaches to targeting preventative efforts to women's special needs as well as the black population are areas that needs more investigation. Further investigation into the gender and ethnic differences in symptom patterns and recognition will improve screening and earlier identification of cardiac problems and by focusing on the specific problem area for each race and gender group should lead to a new decline in CHD mortality.

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APPENDICES

Appendix A: Patient reports – self compiled template

Demographic data

Initials and Surname:
Birth date:
Age:
Computer number:
Gender:
Race:
Weight:
Length:
City/Rural:

Risk factors:

Family History:
Smoking:
Hypertension:
Hypercholesterolemia:
Total Cholesterol:
Triglycerides:
HDL:
LDL:
Diabetes Mellitus:
Obesity:
CVS: Pulse
Blood pressure:

	Normal	Abnormal
Respiratory tract:	Normal	Abnormal
Gastrointestinal:	Normal	Abnormal
SSS:	Normal	Abnormal

Clinical image:

Stable angina:
Unstable angina:
ST elevation MI (Q wave infarction):
Non ST-segment myocardial infarction (Not Q wave):
Abnormal effort test:
Normal effort test:
Other:

Angiographic:

Ventricular function:
Affected vessels:
Internal mammary arteries Patent
Blocked

Treatment:

Medical
PCI Stent
PTCA
Bypass Yes No
Venous arteries Amount done
Amount patent
Surgery Arterial bypass
Venous bypass
Other

Response to treatment:

Asymptomatic
Symptomatic
Repeat surgery
Follow-up procedure
PCI

ECG:

Heartsonar:

Final medication:

Appendix B: Results not statistically evaluated – Risk factors

1.1 Family History, Smoking, Hypertension, Hypercholesterolemia and Diabetes Mellitus

1.1.1 Family History, Smoking, Hypertension, Hypercholesterolemia and Diabetes Mellitus among White Males

The following table summarizes family history, smoking, hypertension, hypercholesterolemia and diabetes mellitus among white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males

White

Years	*Family History*			*Smoking*			*Hypertension*			*Hypercholesterolemia*			*Diabetes Mellitus*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	29	12	41.4	35	20	57.1	31	14	45.2	33	24	72.7	29	5	17.2
45-54	105	30	28.6	112	60	53.6	108	71	65.7	111	70	63.1	104	24	23.1
55-64	149	43	28.9	162	81	50.0	162	94	58.0	161	81	50.3	150	37	24.7
65-74	138	21	15.2	153	44	28.8	149	99	66.4	155	71	45.8	143	39	27.3
75-84	43	8	18.6	47	11	23.4	47	33	70.2	47	25	53.2	47	8	17.0
Unknown (%)	26.1 – 46.3			21.1 – 35.2			23.6 – 42.6			20.5 – 38.9			26.7 – 46.3		

1.1.2 Family History, Smoking, Hypertension, Hypercholesterolemia and Diabetes Mellitus among Black Males

The following table summarizes family history, smoking, hypertension, hypercholesterolemia and diabetes mellitus among black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males

Black

Years	*Family History*			*Smoking*			*Hypertension*			*Hypercholesterolemia*			*Diabetes Mellitus*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	2	0	0.0	2	2	100.0	2	1	50.0	2	0	0.0	1	0	0.0
45-54	14	1	7.1	14	7	50.0	14	8	57.1	14	4	28.6	14	3	21.4
55-64	14	1	7.1	14	4	28.6	14	9	64.3	14	3	21.4	14	8	57.1
65-74	5	0	0.0	5	2	40.0	7	6	85.7	5	1	20.0	6	3	50.0
75-84	2	0	0.0	2	0	0.0	2	2	100.0	2	1	50.0	2	0	0.0
Unknown (%)	33.3 – 60.0%			33.3 – 60.0%			30.0 – 60.0%			33.3 – 60.0%			33.3 – 80.0%		

1.1.3 Family History, Smoking, Hypertension, Hypercholesterolemia and Diabetes Mellitus among White Females

The following table summarizes family history, smoking, hypertension, hypercholesterolemia and diabetes mellitus among white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

White

Years	*Family History*			*Smoking*			*Hypertension*			*Hypercholesterolemia*			*Diabetes Mellitus*		
	Total	*n*	*%*	*Total*	*N*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	10	3	30.0	10	5	50.0	10	5	50.0	10	6	60.0	9	2	22.2
45-54	17	10	58.8	21	12	57.1	19	12	63.1	20	15	75.0	17	3	17.7
55-64	56	22	39.3	58	24	41.4	59	44	74.6	55	36	65.5	54	12	22.2
65-74	83	26	31.3	86	14	16.3	97	78	80.4	92	61	66.3	87	24	27.6
75-84	31	8	25.8	29	4	13.8	32	24	75.0	30	13	43.3	28	5	17.9
Unknown (%)	21.1 – 30.3%			12.5 – 29.3%			16.9 – 22.0%			16.7 – 26.8%			10.0 – 31.7%		

Appendix C: Results not statistically evaluated – Presentation

1.1 Normal ECG and Normal Heart Sonar

1.1.1 Normal ECG and Normal Heart Sonar in Black Males

Due to the high percentages of unknown values for normal ECG and normal heart sonar, this data is only reported for interest sake and no statistical assessments were performed on this data.

The following table summarizes normal ECG and normal heart sonar in black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
Black						
Years	*Normal ECG*			*Normal Heart Sonar*		
	Total	*n*	*%*	*Total*	*n*	*%*
35-44	2	0	0.0	0	0	0.0
45-54	17	2	11.8	8	2	25.0
55-64	10	3	30.0	3	1	33.3
65-74	6	1	16.7	2	1	50.0
75-84	2	0	0.0	1	0	0.0
Unknown (%)	22.7 – 60.0			63.6 – 100		

1.1.2 Normal ECG and Normal Heart Sonar in White Males

The following table summarizes normal ECG and normal heart sonar in white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
White						
Years	*Normal ECG*			*Normal Heart Sonar*		
	Total	*n*	*%*	*Total*	*n*	*%*
35-44	36	11	30.6	14	6	42.9
45-54	109	33	30.3	51	22	43.1
55-64	168	61	36.3	78	29	37.2
65-74	149	47	31.5	88	35	39.8
75-84	51	16	31.4	29	11	37.9
Unknown (%)	22.1 – 33.3			54.9 – 74.1		

1.1.3 Normal ECG and Normal Heart Sonar in White Females

The following table summarizes normal ECG and normal heart sonar in white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females						
White						
Years	*Normal ECG*			*Normal Heart Sonar*		
	Total	*n*	*%*	*Total*	*n*	*%*
35-44	7	2	28.6	4	4	100.0
45-54	14	7	50.0	9	3	33.3
55-64	54	24	44.4	33	19	57.6
65-74	90	34	37.8	56	26	46.4
75-84	30	18	60.0	16	8	50.0
Unknown (%)	23.9 – 41.7			52.9 – 62.5		

1.2 Normal Cardiovascular System, Normal Respiratory System, Normal Gastrointestinal System and Normal Central Nervous System

1.2.1 Normal Cardiovascular System, Normal Respiratory System, Normal Gastrointestinal System and Normal Central Nervous System in White Males

Due to the high percentages of unknown values for normal cardiovascular system, normal respiratory system, normal gastrointestinal system and normal central nervous system, this data is only reported for interest sake and no statistical assessments were performed on this data.

The following table summarizes normal cardiovascular system, normal respiratory system, normal gastrointestinal system and normal nervous system in white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males

White

Years	*Normal Cardiovascular System*			*Normal Respiratory System*			*Normal Gastrointestinal System*			*Normal Central Nervous System*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*	*Total*	*n*	*%*
35-44	50	49	98.0	36	35	97.2	36	36	100.0	25	25	100.0
45-54	128	123	96.1	101	91	90.1	97	95	97.9	60	60	100.0
55-64	191	175	91.6	153	131	85.6	146	140	95.9	107	104	97.2
65-74	175	153	87.4	147	117	79.6	143	130	90.9	93	89	95.7
75-84	59	56	94.9	48	38	79.2	46	43	93.5	36	34	94.4
Unknown (%)	7.4 – 14.5			24.6 – 33.3			26.7 – 33.3			47.8 – 57.8		

1.2.2 Normal Cardiovascular System, Normal Respiratory System, Normal Gastrointestinal System and Normal Central Nervous System in Black Males

The following table summarizes normal cardiovascular system, normal respiratory system, normal gastrointestinal system and normal nervous system in white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males

Black

Years	*Normal Cardiovascular System*			*Normal Respiratory System*			*Normal Gastrointestinal System*			*Normal Central Nervous System*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	3	2	66.7	1	0	0.0	1	0	0.0	0	0	0.0
45-54	19	17	89.5	12	10	83.3	12	11	91.7	9	9	100.0
55-64	17	16	94.1	8	7	87.5	7	7	100.0	5	5	100.0
65-74	9	6	66.7	4	3	75.0	4	4	100.0	2	2	100.0
75-84	3	2	66.7	2	0	0.0	2	0	0.0	0	0	0.0
Unknown (%)	10.0 – 40.0			33.3 – 80.0			33.3 – 80.0			59.1 – 100		

1.2.3 Normal Cardiovascular System, Normal Respiratory System, Normal Gastrointestinal System and Normal Central Nervous System in White Females

The following table summarizes normal cardiovascular system, normal respiratory system, normal gastrointestinal system and normal nervous system in white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

White

	Normal Cardiovascular System			*Normal Respiratory System*			*Normal Gastrointestinal System*			*Normal Central Nervous System*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	8	8	100.0	7	7	100.0	7	7	100.0	5	5	100.0
45-54	22	20	90.9	15	14	93.3	15	15	100.0	12	12	100.0
55-64	60	45	75.0	48	43	89.6	47	43	91.5	25	25	100.0
65-74	110	95	86.4	88	77	67.5	87	79	90.8	56	53	94.6
75-84	40	34	85.0	33	30	90.9	32	30	93.8	24	22	91.7
Unknown (%)	2.44 – 20.0			19.5 – 37.5			22.0 – 37.5			41.5 – 64.8		

Appendix D: Results not statistically evaluated – Treatment

1.1 Positive inotropic and anti-arrhythmia drug treatment

1.1.1 Positive inotropic and anti-arrhythmia drug treatment for Black Males

The following table summarizes the drug treatment [positive inotropic and anti-arrhythmia drugs] for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males

Black

Years	+Inotropic drug			Anti-arrhythmia		
	Total	n	%	Total	n	%
35-44	5	1	20.0	5	0	0.0
45-54	22	4	18.2	22	1	4.6
55-64	21	1	4.8	21	1	4.8
65-74	10	0	0.0	10	0	0.0
75-84	3	1	33.3	3	0	0.0

1.2 ACE inhibitor + Calcium channel blocker and ACE inhibitor + Diuretic drug treatment

1.2.1 ACE inhibitor + Calcium channel blocker and ACE inhibitor + Diuretic drug treatment for White Males

The following table summarizes the drug treatment [ACE inhibitor + calcium channel blocker and ACE inhibitor + diuretic drugs] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
White						
	ACE inhib + Ca channel blocker			*ACE inhib + Diuretic*		
Years	*Total*	*N*	*%*	*Total*	*n*	*%*
35-44	54	0	0.0	54	0	0.0
45-54	142	0	0.0	142	2	1.4
55-64	216	0	0.0	216	3	1.4
65-74	195	0	0.0	195	4	2.1
75-84	69	0	0.0	69	2	2.9

1.2.2 ACE inhibitor + Calcium channel blocker and ACE inhibitor + Diuretic drug treatment for Black Males

The following table summarizes the drug treatment [ACE inhibitor + calcium channel blocker and ACE inhibitor + diuretic drugs] for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
Black						
	ACE inhib + Ca channel blocker			*ACE inhib + Diuretic*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	5	0	0.0	5	0	0.0
45-54	22	0	0.0	22	0	0.0
55-64	21	0	0.0	21	0	0.0
65-74	10	0	0.0	10	0	0.0
75-84	3	0	0.0	3	0	0.0

1.2.3 ACE inhibitor + Calcium channel blocker and ACE inhibitor + Diuretic drug treatment for White Females

The following table summarizes the drug treatment [ACE inhibitor + calcium channel blocker and ACE inhibitor + diuretic drugs] for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

White

	ACE inhib + Ca channel blocker			*ACE inhib + Diuretic*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	10	0	0.0	10	0	0.0
45-54	24	0	0.0	24	0	0.0
55-64	71	0	0.0	71	1	1.4
65-74	119	0	0.0	119	3	2.5
75-84	41	1	2.4	41	2	4.9

1.3 Anti-hypertensive and Beta blocker drug treatment

1.3.1 Anti-hypertensive and Beta blocker drug treatment for White Males

The following table summarizes the drug treatment [anti-hypertensive and beta blocker drugs] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males

White

	Anti- hypertensives			*Beta blockers*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	0	0.0	54	28	51.9
45-54	142	0	0.0	142	60	42.3
55-64	216	1	0.5	216	90	41.7
65-74	195	0	0.0	195	74	38.0
75-84	69	0	0.0	69	24	34.8

1.3.2 Anti-hypertensive and Beta blocker drug treatment for White Females

The following table summarizes the drug treatment [anti-hypertensive and beta blocker drugs] for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females						
White						
Years	*Anti- hypertensives*			*Beta blockers*		
	Total	*n*	*%*	*Total*	*n*	*%*
35-44	10	0	0.0	10	4	40.0
45-54	24	0	0.0	24	11	45.8
55-64	71	0	0.0	71	19	26.8
65-74	119	0	0.0	119	45	37.8
75-84	41	0	0.0	41	10	24.4

1.3.3 Anti-hypertensive and Beta blocker drug treatment for Black Males

The following table summarizes the drug treatment [anti-hypertensive and beta blocker drugs] for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males						
Black						
Years	*Anti- hypertensives*			*Beta blockers*		
	Total	*n*	*%*	*Total*	*n*	*%*
35-44	5	0	0.0	5	3	60.0
45-54	22	0	0.0	22	14	63.6
55-64	21	0	0.0	21	12	57.1
65-74	10	0	0.0	10	0	0.0
75-84	3	0	0.0	3	2	66.7

1.4 Angiotensin receptor blocker drug treatment for Black Males

1.4.1 Angiotensin receptor blocker drug treatment for Black Males

The following table summarizes the drug treatment [angiotensin receptor blocker drugs] for black males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males			
Black			
	Angiotensin receptor blocker		
Years	*Total*	*n*	*%*
35-44	5	0	0.0
45-54	22	0	0.0
55-64	21	0	0.0
65-74	10	0	0.0
75-84	3	0	0.0

1.4.2 Angiotensin receptor blocker drug treatment for White Males

The following table summarizes the drug treatment [angiotensin receptor blocker drugs] for white males in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Males			
White			
	Angiotensin receptor blocker		
Years	*Total*	*n*	*%*
35-44	54	0	0.0
45-54	142	2	1.4
55-64	216	1	0.5
65-74	195	8	4.1
75-84	69	1	1.5

1.4.3 Angiotensin receptor blocker drug treatment for White Females

The following table summarizes the drug treatment [angiotensin receptor blocker drugs] for white females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females			
White			
	Angiotensin receptor blocker		
Years	*Total*	*n*	*%*
35-44	10	0	0.0
45-54	24	1	4.2
55-64	71	2	2.8
65-74	119	3	2.5
75-84	41	2	4.9

1.5 The main drug treatment in each race and gender group

1.5.1 The main positive inotropic drug treatment for each race and gender group

The following table summarizes the main positive inotropic drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Positive Inotropic												
	White Males			*Black Males*			*White Females*			*Black Females*		
	Lanoxin			*Digoxin*			*Digoxin*			*Digoxin*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*	*Total*	*n*	*%*
35-44	54	2	3.7	5	1	20.0	10	0	0.0	4	0	0.0
45-54	142	4	2.8	22	3	13.6	24	1	4.2	6	1	16.7
55-64	216	11	5.1	21	0	0.0	71	1	1.4	14	1	7.1
65-74	195	9	4.6	10	0	0.0	119	2	1.7	11	1	9.1
75-84	69	3	4.4	3	0	0.0	41	1	2.4	2	0	0.0

1.5.2 The main anti-arrhythmia drug treatment for each race and gender group

The following table summarizes the main anti arrhythmia drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Anti arrhythmia drugs

Years	White Males			Black Males			White Females			Black Females		
	Cordarone X			Cordarone X			Cordarone X			Cordarone X		
	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	3	5.6	5	0	0.0	10	0	0.0	4	0	0.0
45-54	142	4	2.8	22	1	4.6	24	1	4.2	6	0	0.0
55-64	216	14	6.5	21	0	0.0	71	1	1.4	14	0	0.0
65-74	195	15	7.7	10	0	0.0	119	4	3.4	11	0	0.0
75-84	69	4	5.8	3	0	0.0	41	2	4.9	2	1	50.0

1.5.3 The main ACE inhibitor drug treatment for each race and gender group

The following table summarizes the main ACE inhibitor drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

ACE inhibitors

Years	White Males			Black Males			White Females			Black Females		
	Coversyl			Coversyl			Coversyl			Coversyl		
	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	20	37	5	3	60	10	1	10	4	3	75
45-54	142	41	28.9	22	12	54.6	24	4	16.7	6	3	50
55-64	216	73	33.8	21	13	61.9	71	23	32.4	14	6	42.9
65-74	195	60	30.8	10	3	30	119	32	26.9	11	6	54.6
75-84	69	17	24.6	3	3	100	41	10	24.4	2	2	100

1.5.4 The main ACE inhibitor and calcium channel blocker combination drug treatment for each race and gender group

The following table summarizes the main ACE inhibitor and calcium channel blocker combination drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

ACE inhibitor & Calsium channel blocker												
Years	White Males			Black Males			White Females			Black Females		
	Lanoxin			Triplent Forte			Triplent Forte			Triplent Forte		
	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	0	0.0	5	0	0.0	10	0	0.0	4	0	0.0
45-54	142	0	0.0	22	0	0.0	24	0	0.0	6	0	0.0
55-64	216	0	0.0	21	0	0.0	71	0	0.0	14	0	0.0
65-74	195	0	0.0	10	0	0.0	119	0	0.0	11	0	0.0
75-84	69	0	0.0	3	0	0.0	41	1	2.4	2	0	0.0

1.5.5 The main ACE inhibitor and diuretic combination drug treatment for each race and gender group

The following table summarizes the main ACE inhibitor and diuretic combination drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

ACE inhibitor & Diuretics												
Years	White Males			Black Males			White Females			Black Females		
	Zestoretic			Zestoretic			Zestoretic			Zestoretic		
	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	0	0.0	5	0	0.0	10	0	0.0	4	0	0.0
45-54	142	2	1.4	22	0	0.0	24	0	0.0	6	0	0.0
55-64	216	3	1.4	21	0	0.0	71	1	1.4	14	0	0.0
65-74	195	4	2.1	10	0	0.0	119	3	2.5	11	0	0.0
75-84	69	2	2.9	3	0	0.0	41	2	4.9	2	0	0.0

1.5.6 The main anti-hypertensive drug treatment for each race and gender group

The following table summarizes the main anti hypertensive drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Anti hypertensive drugs	White Males			Black Males			White Females			Black Females		
	Hypotone			Hypotone			Hypotone			Hypotone		
Years	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	0	0.0	5	0	0.0	10	0	0.0	4	0	0.0
45-54	142	0	0.0	22	0	0.0	24	0	0.0	6	0	0.0
55-64	216	1	0.46	21	0	0.0	71	0	0.0	14	0	0.0
65-74	195	0	0.0	10	0	0.0	119	0	0.0	11	0	0.0
75-84	69	0	0.0	3	0	0.0	41	0	0.0	2	0	0.0

1.5.7 The main anti-platelet drug treatment for each race and gender group

The following table summarizes the main anti platelet drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Anti platelet drugs	White Males			Black Males			White Females			Black Females		
	Disprin			Disprin			Disprin			Disprin		
Years	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	54	46	85.2	5	4	80.0	10	6	60.0	4	3	75.0
45-54	142	107	75.4	22	15	68.2	24	17	70.8	6	2	33.3
55-64	216	157	72.7	21	15	71.4	71	45	63.4	14	7	50.0
65-74	195	135	69.2	10	5	50.0	119	83	69.8	11	9	81.8
75-84	69	46	66.7	3	3	100.0	41	22	53.7	2	1	50.0

1.5.8 The main angiotensin receptor blocker drug treatment for each race and gender group

The following table summarizes the main angiotensin receptor blocker drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Angiotensin receptor blockers												
	White Males			*Black Males*			*White Females*			*Black Females*		
	Atacand			*Atacand*			*Atacand*			*Atacand*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*
35-44	54	0	0.0	5	0	0.0	10	0	0.0	4	0	0.0
45-54	142	1	0.7	22	0	0.0	24	1	4.2	6	0	0.0
55-64	216	1	0.5	21	0	0.0	71	1	1.4	14	0	0.0
65-74	195	5	2.6	10	0	0.0	119	1	0.8	11	0	0.0
75-84	69	0	0.0	3	0	0.0	41	2	4.9	2	0	0.0

1.5.9 The main beta blocker drug treatment for each race and gender group

The following table summarizes the main beta blocker drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Beta blockers												
	White Males			*Black Males*			*White Females*			*Black Females*		
	Atenolol			*Dilatrend*			*Atenolol*			*Atenolol*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	12	22.2	5	2	40.0	10	2	20.0	4	0	0.0
45-54	142	24	16.9	22	4	18.2	24	4	16.7	6	1	16.7
55-64	216	25	11.6	21	4	19.1	71	9	12.7	14	5	35.7
65-74	195	10	5.13	10	0	0.0	119	15	12.6	11	5	45.5
75-84	69	9	13.0	3	2	66.7	41	3	7.32	2	0	0.0

1.5.10 The main calcium channel blocker drug treatment for each race and gender group

The following table summarizes the main calcium channel blocker drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Calcium channel blockers												
	White Males			*Black Males*			*White Females*			*Black Females*		
	Adalat			*Adalat*			*Adalat*			*Adalat*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	1	1.9	5	1	20.0	10	1	10.0	4	0	0.0
45-54	142	6	4.2	22	3	13.6	24	2	8.3	6	1	16.7
55-64	216	8	3.7	21	4	19.1	71	10	14.1	14	1	7.1
65-74	195	13	6.7	10	1	10.0	119	14	11.8	11	1	9.1
75-84	69	4	5.8	3	1	33.3	41	3	7.3	2	0	0.0

1.5.11 The main diuretic drug treatment for each race and gender group

The following table summarizes the main diuretic drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Diuretics												
	White Males			*Black Males*			*White Females*			*Black Females*		
	Lasix			*Ridaq*			*Ridaq*			*Ridaq*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	5	9.26	5	0	0	10	3	30	4	1	25
45-54	142	9	6.34	22	5	22.7	24	2	8.33	6	1	16.7
55-64	216	23	10.7	21	6	28.6	71	11	15.5	14	5	35.7
65-74	195	30	15.4	10	0	0	119	14	11.8	11	3	27.3
75-84	69	6	8.7	3	1	33.3	41	3	7.32	2	0	0

1.5.12 The main lipid lowering drug treatment for each race and gender group

The following table summarizes the main lipid lowering drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Lipid lowering drugs

	White Males			*Black Males*			*White Females*			*Black Females*		
	Lipitor			*Lipitor*			*Lipitor*			*Lipitor*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	54	21	38.9	5	1	20.0	10	5	50.0	4	2	50.0
45-54	142	61	43.0	22	4	18.2	24	7	29.2	6	3	50.0
55-64	216	53	24.5	21	3	14.3	71	24	33.8	14	3	21.4
65-74	195	45	23.1	10	0	0.0	119	37	31.1	11	1	9.1
75-84	69	12	17.4	3	0	0.0	41	11	26.8	2	0	0.0

1.5.13 The main nitrate drug treatment for each race and gender group

The following table summarizes the main nitrate drug treatment for each race and gender group, respectively in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Nitrates

	White Males			*Black Males*			*White Females*			*Black Females*		
	Isordil			*Isordil*			*Isordil*			*Isordil*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*
35-44	54	19	35.2	5	3	60.0	10	2	20	4	1	25.0
45-54	142	33	23.2	22	6	27.3	24	5	20.8	6	1	16.7
55-64	216	61	28.2	21	10	47.6	71	22	31	14	4	28.6
65-74	195	62	31.8	10	3	30.0	119	31	26.1	11	5	45.5
75-84	69	21	30.4	3	2	66.7	41	13	31.7	2	0	0.0

Appendix E: Results not statistically evaluated – Black female patients

1.1 Risk factors

1.1.1 Family History, Smoking, Hypertension, Hypercholesterolemia and Diabetes Mellitus for Black Females

The following table summarizes family history, smoking, hypertension, hypercholesterolemia and diabetes mellitus for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

Black

Years	*Family History*			*Smoking*			*Hypertension*			*Hypercholesterolemia*			*Diabetes Mellitus*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*	*Total*	*n*	*%*
35-44	4	0	0.0	4	0	0.0	4	2	50.0	4	1	25.0	4	1	25.0
45-54	3	0	0.0	3	0	0.0	3	3	100.0	3	3	100.0	3	3	100.0
55-64	5	2	40.0	5	0	0.0	7	6	85.7	6	3	50.0	5	4	80.0
65-74	5	0	0.0	4	0	0.0	6	5	83.3	5	2	40.0	6	2	33.3
75-84	2	0	0.0	2	0	0.0	2	2	100.0	2	1	50.0	2	0	0.0
Unknown (%)	50.0 – 64.3			50.0 – 64.3			45.5 – 50.0			50.0 – 57.1			45.5 – 64.3		

1.2 Presentation

1.2.1 Stable angina, unstable angina, Q-wave myocardial infarction and non-Q wave myocardial infarction for Black Females

The following table summarizes stable angina, unstable angina, Q-wave myocardial infarction and non Q-wave myocardial infarction for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

Black

(Total 37 subjects)

Years	*Stable angina*			*Unstable angina*			*Q-wave myocardial infarction*			*Non Q-wave myocardial infarction*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	4	1	25.0	4	0	0.0	4	3	75.0	4	0	0.0
45-54	6	4	66.7	6	0	0.0	6	2	33.3	6	0	0.0
55-64	14	9	64.3	14	2	14.3	14	2	14.3	14	1	7.1
65-74	11	4	36.36	11	1	9.1	11	6	54.6	11	0	0.0
75-84	2	1	50.0	2	0	0.0	2	1	50.0	2	0	0.0

1.2.2 Normal ventricular function, Normal ECG and Normal Heart Sonar for Black Females

The following table summarizes normal ventricular function, normal ECG and normal heart sonar for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females									
Black									
Years	*Normal ventricular function*			*Normal ECG*			*Normal Heart Sonar*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	4	3	75.0	3	2	66.7	1	1	100.0
45-54	5	4	80.0	3	1	33.3	2	0	0.0
55-64	12	9	75.0	6	4	66.7	4	2	50.0
65-74	11	8	72.7	6	0	0.0	3	1	33.3
75-84	2	0	0.0	2	0	0.0	2	0	0.0
Unknown (%)	14.3 – 16.7			25.0 – 57.1			66.7 – 75.0		

1.2.3 Normal Cardiovascular System, Normal Respiratory System, Normal Gastrointestinal System and Normal Central Nervous System for Black Females

The following table summarizes normal cardiovascular system, normal respiratory system, normal gastrointestinal system and normal nervous system for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females												
Black												
Years	*Normal Cardiovascular System*			*Normal Respiratory System*			*Normal Gastrointestinal System*			*Normal Central Nervous System*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	4	4	100.0	2	2	100.0	2	2	100.0	2	2	100.0
45-54	4	2	50.0	2	2	100.0	2	2	100.0	2	2	100.0
55-64	8	7	87.5	4	4	100.0	4	4	100.0	4	4	100.0
65-74	8	7	87.5	4	4	100.0	4	4	100.0	3	2	66.7
75-84	2	2	100.0	2	2	100.0	1	1	100.0	1	1	100.0
Unknown (%)	27.3 – 42.9			50.0 – 71.4			50.0 – 71.4			50.0 – 72.7		

1.2.4 Body Mass Index for Black Females

The following table summarizes the body mass index for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups. Body mass index (BMI) was calculated as follows: $BMI = \frac{\text{weight (in kg)}}{[\text{height (in m)}]^2}$

A BMI value of ≤ 19 = underweight
20 –24 = normal
25-29 = overweight
30+ = obese

Females												
Black												
Years	≤ 19			20 – 24			25 - 29			30+		
	Total	n	%	Total	n	%	Total	n	%	Total	n	%
35-44	4	0	0.0	4	1	25.0	4	1	25.0	4	2	50.0
45-54	6	0	0.0	6	2	33.3	6	2	33.3	6	2	33.3
55-64	13	0	0.0	13	1	7.7	13	5	38.5	13	7	53.9
65-74	11	0	0.0	11	5	45.5	11	4	36.4	11	2	18.2
75-84	2	0	0.0	2	1	50.0	2	0	0.0	2	1	50.0
Unknown (%)	0.5											

1.2.5 Affected vessels (1-, 2- or 3 vessel disease) for Black Females

The following table summarizes the affected vessel status (1-, 2- or 3 vessel disease) for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females									
Black									
Years	1 Affected vessel			2 Affected vessels			3 Affected vessels		
	Total	N	%	Total	n	%	Total	n	%
35-44	4	3	75.0	4	1	25.0	4	0	0.0
45-54	6	1	16.7	6	0	0.0	6	5	83.3
55-64	13	5	38.5	13	2	15.4	13	6	46.2
65-74	11	2	18.2	11	2	18.2	11	7	63.6
75-84	1	0	0.0	1	0	0.0	1	1	100.0
Unknown (%)	7.1 – 50								

1.3 Treatment

1.3.1 Medical, PTCA, Stent or Bypass treatment for Black Females

The following table summarizes the treatment status [medical, PTCA (with and without stent) or bypass] for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females												
Black												
Years	*Medical*			*PCI - PTCA*			*PCI – Stent*			*Bypass*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	3	3	100.0	4	1	25.0	4	0	0.0	4	0	0.0
45-54	3	3	100.0	6	0	0.0	6	0	0.0	6	1	16.7
55-64	9	9	100.0	14	1	7.1	14	1	7.1	14	1	7.1
65-74	9	9	100.0	11	2	18.2	11	2	18.2	11	0	0.0
75-84	2	2	100.0	2	0	0.0	2	0	0.0	2	0	0.0
Unknown (%)	18.2 – 50.0			0.00			0.00			0.00		

1.3.2 Positive inotropic and anti-arrhythmia drug treatment for Black Females

The following table summarizes the drug treatment [positive inotropic and anti-arrhythmia drugs] for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females						
Black						
Years	*+Inotropic drug*			*Anti-arrhythmia*		
	Total	*n*	*%*	*Total*	*n*	*%*
35-44	4	0	0.0	4	0	0.0
45-54	6	1	16.7	6	0	0.0
55-64	14	1	7.1	14	0	0.0
65-74	11	2	18.2	11	0	0.0
75-84	2	0	0.0	2	1	50.0

1.3.3 ACE inhibitor, ACE inhibitor and Calcium channel blocker, ACE inhibitor and Diuretic and Diuretic drug treatment for Black Females

The following table summarizes the drug treatment [ACE inhibitor, ACE inhibitor + calcium channel blocker, ACE inhibitor + diuretic and diuretic drugs] for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

Black

	ACE inhibitor			*ACE inhib + Ca channel blocker*			*ACE inhib + Diuretic*			*Diuretics*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*
35-44	4	4	100	4	0	0	4	0	0	4	2	50
45-54	6	4	66.7	6	0	0	6	0	0	6	3	50
55-64	14	10	71.4	14	0	0	14	0	0	14	8	57.1
65-74	11	7	63.6	11	0	0	11	0	0	11	6	54.6
75-84	2	2	100	2	0	0	2	0	0	2	1	50

1.3.4 Anti-hypertensive, Anti-platelet, Beta blocker and Calcium channel blocker drug treatment for Black Females

The following table summarizes the drug treatment [anti-hypertensive, anti-platelet, beta blockers and calcium channel blocker drugs] for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females

Black

	Anti-hypertensives			*Anti-platelet drugs*			*Beta blockers*			*Ca channel blocker*		
Years	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*	*Total*	*N*	*%*
35-44	4	0	0	4	3	75	4	3	75	4	0	0
45-54	6	0	0	6	3	50	6	3	50	6	1	16.7
55-64	14	0	0	14	7	50	14	8	57.1	14	1	7.14
65-74	11	0	0	11	10	90.9	11	10	90.9	11	2	18.2
75-84	2	0	0	2	1	50	2	2	100	2	0	0

1.3.5 Angiotensin receptor blocker, Lipid lowering and nitrate drug treatment for Black Females

The following table summarizes the drug treatment [angiotensin receptor blocker, lipid lowering drug and nitrates] for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females									
Black									
Years	*Angiotensin receptor blocker*			*Lipid lowering drugs*			*Nitrates*		
	Total	*n*	*%*	*Total*	*n*	*%*	*Total*	*n*	*%*
35-44	4	0	0.0	4	2	50.0	4	1	25.0
45-54	6	0	0.0	6	3	50.0	6	2	33.3
55-64	14	0	0.0	14	3	21.4	14	4	28.6
65-74	11	0	0.0	11	1	9.1	11	6	54.6
75-84	2	0	0.0	2	0	0.0	2	0	0.0

1.4 Response to treatment

1.4.1 Black Females with symptomatic response to treatment

The following table summarizes the symptomatic response to treatment for black females in the 35-44, 45-54, 55-64, 65-74 and 75-84 years age groups

Females			
Black			
	Symptomatic		
Years	*Total*	*n*	*%*
35-44	4	3	75.0
45-54	6	5	83.3
55-64	14	10	71.4
65-74	11	8	72.7
75-84	2	2	100.0