Chapter 2

Literature review

2.1 INTRODUCTION

The researcher conducted a literature review on the pathophysiology and prevalence of risk factors of CHD. Epidemiological studies on CHD and the physical, biomedical and environmental characteristics of the population or individuals defined predictive variables, termed risk factors, shown to be associated with the development of CHD. The risk factors include age, gender, positive family history and lifestyle. In reviewing the literature the researcher intended to highlight the

- pathology of the risk factors
- prevalence of CHD among the Indian community.

2.2 PATHOPHYSIOLOGY OF CHD

Coronary heart disease (CHD) “remains a persistent public health burden in the United States of America and is the cause of one out of every five deaths each year. The link between lipids and CHD has been firmly established, first by epidemiological studies and more recently, by long-term outcomes trials that demonstrated that lowering low-density lipoprotein cholesterol levels significantly reduced the risk of major coronary events. Based on this evidence, the National Cholesterol Education Program recommends lowering the low-density cholesterol (LDL-C) level to reduce CHD risk, particularly for patients at highest risk. Recently, evidence has emerged that suggests that C-reactive protein may be a mediator of arteriosclerosis and its presence may be indicative of increased risk of CHD. Although these data are intriguing, their relevance has yet to be established in prospective outcome trials. Until then, lipid lowering through lifestyle modification and the use of safe and effective modes of therapy should be the emphasis of CHD risk reduction strategies” (Chilton 2004:8).
CHD, once considered the result of vessel-occluding deposition of lipids, appears to be “a manifestation of a chronic inflammatory response to injury or infection. Elevated plasma cholesterol levels have long been established as risk factors for CHD, and lowering cholesterol levels, particularly LDL-C, has been the focus of the prevention of CHD and its sequel for almost 25 years. However, the complex mechanisms by which these molecules act are only beginning to be appreciated. Evidence suggests that lipid-lowering modes of therapy also reduce inflammation, which may reduce the risk of cardiovascular events, even for individuals with LDL-C levels in the normal range (<130mg/dl) based on the National Cholesterol Education Programme Audit Treatment Panel III guidelines” (Chilton 2004:5)

2.2.1 Role of lipids in plaque development

Injury or infection “can disrupt normal endothelial function and initiate the formation of atherosclerotic lesions known as fatty streaks. Fatty streaks typically consist of macrophages and T cells embedded in a thin layer of lipids on the arterial wall. Macrophages engulf lipids, becoming activated foam cells that release an array of chemo attractant molecules, cytokines and growth factors. More lymphocytes are attracted to the lesion and, in turn, add to the pool of effector molecules that expand and perpetuate the inflammatory response. As this cycle is repeated, the plaque develops a fatty core covered by a fibrous matrix that stabilizes the structure ”(Chilton 2004:5).

The frequent presence of “fatty streaks in young children are consistent with the chronic nature of atherosclerotic progression. Although the possible events that can initiate fatty streak formation remain controversial, LDL-C, modified by oxidation, glycation and association with proteoglycans and immune complexes, can become trapped in the arterial wall, injuring the endothelium and vascular smooth muscle. Once trapped, LDL-C particles become progressively more oxidized, form lipid peroxides and facilitate accumulation of cholesterol esters. Also, modified LDL-C is chemotactic for circulating monocytes and stimulates the proliferation of macrophages already in the lesion. Inflammatory mediators increase the binding of LDL-C to endothelial cells and vascular smooth muscle cells that have migrated into the lesion. As the plaque becomes thicker, the arterial wall responds by “remodeling” that is, generally dilating to maintain the diameter of the vessel lumen. Eventually, macrophages may be stimulated to release
metalloproteinases that degrade the fibrous cap and render the plaque vulnerable to rupture”.

Naghavi, Libby & Falk (2003: 108 stated that” although several types of plaque can result in serious coronary events, retrospective analyses have demonstrated that 70% of all fatal acute myocardial infarctions and sudden coronary deaths are attributable to plaque rupture or plaque erosion. Plaque destabilization is often accompanied by the release of prothrombotic factors. However, all types of atherosclerotic plaques can result in coronary events and sudden death. Vulnerable plaques are defined as thrombosis-prone or at risk of rapid progression and exhibit some combination of the following: active inflammation, thinning cap with a large lipid core, endothelial denudation with superficial platelet aggregation, fissures, or greater than 90% stenosis”. The authors further conclude “that the thrombotic status of the blood and the electrical instability of the myocardium are important” (Naghavi 2003:108).

2.2.2 Inflammation and atherosclerosis

Ridker, Rifai & Rose (2002: 1557) state that” an accumulating body of evidence suggests that atherosclerotic progression results from micro inflammation mediated by pro inflammatory cytokines. The observation that monocytes and T lymphocytes are present at all stages of plaque development is consistent with active inflammation. Chronic low-level inflammation increases atherosclerotic plaque deposition in animal models. In addition, heightened levels of the acute-phase reactant C-reactive (CRP) protein is believed to be a marker of the inflammatory process and may also be of value in the prediction of coronary events”. A recently published report by Danesh, & Wheeler suggests, “CRP is more than a marker and may be a mediator of atherosclerosis”.

The association between elevated levels of CRP and cardiovascular risk has been the object of extensive research and is the topic of much current debate. Some evidence suggests that CRP is an independent predictor of the risk of cardiovascular events (Danesh et al: 2004: 1387). In their study, Ridker et al (2004:11)” found that the CRP level was a stronger predictor than the LDL-C level for myocardial infarction, ischaemic stroke, coronary revascularization, or death due to cardiovascular causes. However, because CRP and LDL-C levels appeared to identify somewhat different risk groups, the combined risk assessment was superior to that of either marker alone".
Ridker et al (2004: 11) found “further that CRP levels were associated with increased risk of cardiovascular events at all levels of estimated risk based on the 1984 Framingham risk score. Although the women had high CRP and low LDL-C levels, only the latter group was considered eligible for aggressive therapy”.

Danesh, Wheeler, Hirschfield and Eda (2004:1388) studied circulating inflammatory markers that evaluated the relevance of CRP to the prediction of CHD by observing 18 569 individuals enrolled in the Reyk-Javik Heart Study and measuring inflammatory markers in blood samples obtained at baseline from 2 459 patients who had a non-fatal myocardial infarction or died of CHD during the study and from 3 969 control subjects without CHD. Danesh et al (2004:1397)” found that CRP was not as strong a predictor for CHD as more traditional risk factors, such as total cholesterol level or cigarette smoking, and consequently maintain that recommendations on the use of the CRP level in predicting the likelihood of CHD should be reviewed”.

Chilton (2005:8) found that” CRP is a modulator of inflammation and may have both pro- and anti-inflammatory actions, which may directly contribute to endothelial dysfunction by including cytokine release and surface expression of adhesion molecules. Through a conformational rearrangement in CRP from a penameric to a monomeric structure, the atherogenic effects of CRP were noted on endothelial cells. C-reactive protein thus appears to be more than a marker of cardiovascular events”.

2.3 FRAMINGHAM HEART STUDY, 1984

Disease causation is rarely a single entity factor. Several factors need to be present before the disease process occurs and becomes clinically evident (Katzenellenbogen, Joubert & Abdool 1999:10). The epidemiological Framingham heart study, which began in 1984 as cited in Hudak (1998: 398) and is continuing with second-generation subjects, found an association between specific risk factors and the development of CHD (Thelan et al 2000:482). Participants had their blood cholesterol levels measured, their smoking and activity histories recorded, and blood pressure and electrocardiography results checked on a regular basis. As a result of this and other similar studies, specific lifestyle habits, referred to as coronary risk factors, were
identified as associated with an increased probability of developing CHD. These factors were further delineated into non-modifiable and modifiable risk factors. A theory of cardiac risk factors developed from these studies. The present study was guided by cardiac risk factors identified in the literature. Table 2.1 illustrates the risk factors for the development of CHD.

### Table 2.1 Cardiac risk factors in CHD

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Source: Hudak, Gallo & Morton (1998:398)

### 2.4 CLASSIFICATION OF CARDIAC RISK FACTORS

Cardiac risk factors “are classified as modifiable and non-modifiable. Modifiable risk factors are those risk factors caused by circumstances under an individual’s control. Non-modifiable risk factors are those beyond the individual’s control “(Thelan et al 2000:483).

#### 2.4.1 Non-modifiable risk factors

##### 2.4.1.1 Age

Symptomatic CHD “appears in clients over 40 years of age. Although the disease was more prevalent in people over 60 years as a result of degeneration of the intima of the blood vessels, the pattern seems to be changing especially in the Indian population. Referring to CHD as the “silent killer” that “stalks younger men”, Naidoo (2004:11) states that up to 20% of Indian patients in their late 20s and 30s are having heart
attacks and being treated for these condition Ranjith, Verho, Verho and Winkelmann 
“(2000:242) report similar findings at the RK Khan Hospital. According to Naidoo 
(2004:11), “culturally, the Indian population uses a lot of fat in their cooking. Ghee, a 
type of saturated fat, commonly used by Indians for cooking, is said to be unhealthy”.

The risk of coronary atherosclerosis increases with age, but evidence suggests that the 
condition may start to develop very early in life. About four out of five people who die of 
coronary heart disease are 65 or older. At older ages, women who have heart attacks 
are more likely than men are to die from them within a few weeks (Edward & Havranek 

2.4.1.2 Genetic/hereditary factors

• Diabetes

Hammersmith & Earling (1999:76) stated that “diabetes is a disease that can either 
result from a person’s genetic make up or be acquired. There is a higher likelihood of 
developing diabetes if one or both parents have the condition. Genetic studies indicate 
that juvenile diabetes has significantly higher proportions of certain genetically 
controlled antigenic cell marks. According to Armstrong and McKay (2001:461), 
“although genetic factors are important, some environmental factors are critical in 
triggering the expression of the genetic predisposition”. Insulin stimulates fatty acid 
synthesis and triglyceride formation in adipose tissue. One of the long-term 
complications of diabetes is atherosclerosis as a result of progressive thickening of the 
basement membrane of capillaries in the affected area. The formation of these deposits 
is believed to be the result of glucose bonding chemically with amino groups of body 
proteins. Thickening of the capillaries results in diminished perfusion to the areas 
involved. In the coronary arteries this could lead to angina pectoris eventually leading to 
myocardial infarction. This mechanism applies to both juvenile and adult diabetes 

Diabetes was strongly associated with cardiac disease in both men and women, with 
diabetic men having a 2.2-fold increased risk of CHD and diabetic women, a 3.5-fold 
increased rate compared to non-diabetic individuals. Insulin concentration was a 
significant predictor in men but not in women (Lee. 1999:255).
• **Gender**

Smeltzer and Bare (2000:595) stated that women of childbearing age display one-fourth the risk of developing CHD, compared to men of the same age. This difference in susceptibility diminishes after menopause; however, even after 65, women continue to be less likely than men to develop CHD. Women who take oral contraceptives are more likely to develop CHD. This risk is particularly significant in women who smoke. When oral contraceptives are discontinued, the increased risk of CHD does not continue. Women with an early menopause face three times the risk of CHD as women with late menopause. Two lifestyle changes during the past decade may increase the incidence of CHD among women. More women (many with full responsibility of the household and children) have entered the workforce. Also, more women begin smoking at an earlier age.

Women before menopause rarely suffer from heart disease (the hormone oestrogen present in women appears to protect them). Coronary heart disease mainly afflicts adult males and manifests itself in the Middle Ages between 45-55 years (Edward & Havranek 1999:1467).

### 2.4.2 Modifiable risk factors

A modifiable risk factor is one over which individuals may exercise control.

#### 2.4.2.1 Major risk factors

• **Cigarette smoking**

Goodfellow (2002:75) stated “cigarette smoking is a major risk factor in CHD. The nicotine in cigarettes promotes vasoconstriction resulting in intima hypoxia. People who smoke ten (10) cigarettes per day are at almost double the risk as those smoking five (5) per day. Tobacco inhaled during smoking is the major cause of erosion of the inner lining of the coronary arteries. The tar and alkaloids in tobacco-nicotine lead to this damage and make the layer more susceptible to cholesterol and fat deposits. Male adult smokers have a 70% higher mortality rate than male non-smokers, and all smokers
have more than twice the risk of a heart attack than non-smokers. Clients who smoke have two to four times the risk of sudden cardiac death. Clients who quit smoking lose their increased risk within 24 months"(Goodfellow 2002:75).

People who smoke are four times more prone to develop heart disease compared to non-smokers The Framingham heart study, carried out in the small town of Framingham, USA, studied more than 5 000 people and their families for more than three decades and confirmed that smoking substantially increases the risk of heart attacks (Goodfellow 2002:75).

Edward&Havranek (1999:1466) in their study observed that “smoking tends to increase blood cholesterol levels. Cigarette smokers also have raised fibrinogen levels and platelet counts, which makes the blood stickier. Carbon monoxide attaches itself to haemoglobin much more easily than oxygen does. This reduces the amount of oxygen available to the tissues. All these factors make smokers more at risk of developing various forms of atherosclerotic disease. As the atherosclerotic process progresses, blood flows less easily through rigid and narrowed arteries and the blood is more likely to form a thrombosis. This sudden blockage of an artery may lead to a fatal heart attack”.

Doll & Pete (1994:901) in their study stated that “inhaling tobacco smoke causes several immediate responses within the heart and its blood vessels within one minute of starting to smoke, the heart rate begins to rise, it may increase by as much as 30 percent during the first 10 minutes of smoking. Carbon monoxide in tobacco exerts a negative effect on the heart by reducing the blood’s ability to carry oxygen”.

Habitual cigarette smoking is well recognized as a major factor in CHD. Paradoxically, smokers have been noted to improve procedural and long-term outcomes following direct angioplasty in acute myocardial infarction. Barker and Siegal (2003:643) studied the test “smoker’s paradox”, using baseline clinical risk profiles. A twelve-month clinical trial using 1 085 consecutive patients (476 and 608 non-smokers) within twelve hours of onset of acute myocardial infarction, found the existence of “smoker’s paradox” in patients undergoing direct infarct intervention. Despite identical procedural outcomes, habitual smokers paradoxically demonstrated superior in-hospital and twelve-month clinical outcomes compared to non-smokers. The significantly younger age of smokers
at presentation and the more favourable clinical and angiographic profiles may account for the potential “confounding effect” of smoking (Barker & Siegal 2003:724).

- **Hypertension**

High blood pressure affects nearly 60 million adults and children worldwide. Men over 45 with blood pressures exceeding 140/90 and all adult women with pressures above 160/95 have a 50% higher chance of mortality. As blood pressure increases, the risk for cardiovascular events also escalates. When hypertension is controlled, the risk of CHD decreases. Therefore, hypertension should be treated to lower the risk of CHD and premature death (WHO 2002: 355).

The normal blood pressure in adults is between 100/60 and 140/90 mmHg. If the blood pressure is consistently more than 140/90 on two or more separate occasions, it is called high blood pressure or hypertension. Hypertension is a very common disease, and 20% to 30% of adults suffer from hypertension. However, most people are not even aware that they have this disease because many of them do not have any symptoms. This is why this disease is called the “silent killer”. High blood pressure puts an extra strain on the heart and the arteries supplying blood to the other organs of the body. Many diseases, such as stroke (damage to the brain), heart attacks and damage to the eyes, are caused by high blood pressure. High blood pressure can be classified as mild, moderate and severe. It is also one of the major causes of deposition of cholesterol and fat in the coronary arteries. It damages the endothelial lining of arteries, making them more prone to fat deposition. Besides a high intake of salt, psychological stresses are an important cause of high blood pressure (WHO 2002:3).

2.4.3 **Diet high in saturated fats and calories**

- **Elevated serum cholesterol**

An elevated serum cholesterol level increases the risk of developing CHD. A client with a serum cholesterol level greater than 259mg/dl is three times more likely to develop CHD than one with a serum level of 200 mg/dl. The body produces endogenous cholesterol, primarily in the liver. Additional cholesterol is ingested through dietary intake, primarily from dairy products and meats. Cholesterol circulates in the blood in
combination with triglycerides and protein-bound phospholipids. This complex is called a lipoprotein. There are four basic groups of lipoproteins, all produced in the intestinal wall. Elevation of lipoproteins is called hyperlipoproteinemia. Elevation of lipids, a component of lipoproteins, is called hyperlipidemia. Lipoproteins and their functions are as follows Lee, ET (1999:248).

- Chylomicrons primarily transport dietary triglycerides and cholesterol.
- Very low-density lipoprotein mainly transports the triglycerides synthesized by the liver.
- Low-density lipoprotein (LDL) has the highest concentration of cholesterol and transports endogenous cholesterol to body cells.
- High-density lipoprotein (HDL) has the lowest concentration of cholesterol and transports endogenous cholesterol to the body cells.

The presence of lipoproteins may predispose the body to the development of CHD. The ratio of total cholesterol to HDL or of LDL to HDL is the best test for predicting the risk of CHD. High concentrations of HDL seem to have a protective effect against the development of CHD. Exercise and low-fat, low-cholesterol diets increase the amount of HDL in the blood (Coopoo 1995:43). In a wellness-designed study, Whaley, Kaminsky, Getchell, Treloar & Kelly (1992:42) investigated the changes in total cholesterol concentration following endurance training. The subjects comprised of 215 women and 327 men aged 20 to 75, and the training consisted of individualized aerobic exercise at approximately 75% to 85% of maximal heart rate for 30 to 50 minutes four times per week for sixteen weeks. After adjustments “for pre-training, total cholesterol, age, changes in body weight, and body fat were significant first-order predictors of change in total cholesterol for the total cohort. The level of total cholesterol prior to training therefore appeared to have an effect on the degree to which total cholesterol changed during training. The results confirmed the importance of the pre-training, total cholesterol levels in assessing the potential for a favorable change in total cholesterol subsequent to aerobic training. The results also may explain the equivocal results found in previous studies. This may have been due in part to the use of samples with normal pre-training total cholesterol levels or of samples exhibiting a wide range of pre-training total cholesterol levels. Further, it was reported that lower initial levels of HDL-C resulted in greater increases following exercise. Only LDL-C deviated from this pattern of strong correlations”.
2.5 MINOR RISK FACTORS

A number of risk factors are clinically recognized in the pathogenesis of CHD, and many other factors interact in increasing the possibility of the development of CHD. The risk of CHD may be doubled or tripled when more than one risk factor is present (Jackson 1999:497).

2.5.1 Obesity, lack of exercise and sedentary lifestyle

Obesity and lack of exercise increase the risk of CHD. Obesity places an extra burden on the heart, requiring the muscle to work harder to pump enough blood to support added tissue mass. In addition, obesity is often associated with a sedentary lifestyle, elevated serum cholesterol, and high blood pressure.

Effective, routine aerobic exercise may decrease the likelihood of a coronary event. According to Fuster, Badimon, Badimon and Chesebro (1998:242-250), a “sedentary lifestyle potentiates the lethality of a myocardial infarction, and is considered a significant risk factor in the development of CHD. Exercise may reduce the risk of CHD by decreasing weight, reducing blood pressure, and elevating the protective HDL. The prevailing thought is that exercise, along with general body conditioning, makes the heart use oxygen more efficiently. To be effective, aerobic exercise should raise the heart rate from 50 to 100 % of baseline (depending on age and physical condition) for at least 20-30 minutes. Such exercise must be performed at least three times a week to be beneficial.”

Adams, Lenk, Sandri, Lenz, Tamok, Scheinert, Sculer and Hambrecht (2003:700) found that in patients with coronary heart disease exercise training is regularly associated with a decrease in exercise-induced myocardial ischaemia.

A sedentary lifestyle or lack of exercise is the main cause of heart disease because low physical activity is often associated with a high incidence of CHD. Regular exercise can break down the fat consumed, decrease cholesterol, reduce blood sugar, control blood pressure, reduce weight and make the heart healthy. Without exercise more and more people will have heart disease, diabetes, high blood pressure, obesity and low fitness.
Besides, lack of physical activity will lead to less flexibility, joint diseases, and other ailments (WHO 2003:3)

Byrne (1991:1-36) points out that in many instances, the elimination of these risk factors has not resulted in a retardation or reversal of CHD. Physical activity, on the other hand, improves a number of these risk factors, and its apparent beneficial effect on CHD could reduce or retard the severity of this disease. Chandrashekhar and Anand (1995:1723) emphasise that progressive aerobic activity, engaged in on a regular basis, plays a role in the primary and secondary prevention of cardiovascular disease. According to the AHA (1992:240), inactivity is a recognized risk factor for CHD.

Various organic systems in the body can alter their function as part of an adaptive process in response to physical activity. These alterations may in some way assist in the retardation of CHD or may protect from the effects of the disease (Chandrashektar & Anand 1995:1723).

A number of physiological changes in the cardio respiratory system may occur as a result of aerobic training, which may indirectly or directly reduce the development of CHD. Only exercise-induced cardio-respiratory changes will be discussed.

Mattos and Ribero (2003:629) found that “moderate intensity training resulted in larger improvements in the exercise capacity and perception of quality of life than low intensity training in elderly post-myocardial infarction women”. Exercise-induced ventricular hypertrophy, unlike pathologic hypertrophy, is not associated with reduced myocardial contractility (Chandrashektar & Anand 1991:1723). In healthy persons “ aerobic training increases left ventricular end-diastolic volume and these changes are lost with deconditioning. These changes increase cardiovascular functional capacity and decrease the myocardial oxygen demand for the same level of work performed. This is achieved by a decrease in the product of heart rate and systolic arterial blood pressure (an index of myocardial oxygen consumption). These adaptations also benefit coronary patients, who after exercise may attain a higher level of physical work before reaching the level of myocardial oxygen requirement that results in myocardial ischaemia”.

Astrand and Rodahl (1990:21), Fox (1993:41) and Wilmore (1997:17) found “an increase in total workload during exercise stress test. This is measured by assessing
oxygen uptake (Vo₂ max) and increases between 15% and 20% have been recorded after aerobic training programmes”.

According to Fox (1993:51) and Wilmore (1997:17), the increase in Vo₂ max is brought about by two main physiological changes, namely

- an increased oxygen delivery to the working muscles through increased cardiac output
- an increased oxygen extraction from the blood by the skeletal muscle.

With the increase in Vo₂ max, a decrease in resting, exercise an intrinsic heart rate occurs as well as an increase in stroke volume, which would improve the functional capacity of the heart (Chandashekar & Anand 1996:1723). It is postulated that exercise triggers the formation of collateral coronary circulation.

Haskell (1993:1076) examined “the response of nitroglycerine administration on the dilating capacity and coronary artery size in ultra-distance runners compared to sedentary subjects, using computer-assisted arteriographical measurements. The results demonstrated for the first time that the coronary arteries of highly trained middle-aged men have a greater dilating capacity in response to nitroglycerine than those of men who are generally sedentary. Given the cross-sectional nature of the study, it is not possible to know whether this difference was caused by running or was due to some other acquired trait or genetic selection. Further research is needed in order to determine the causes of the greater dilating capacity of epicardial coronary arteries in highly trained runners and if any clinical benefits is provided by this increased capacity”.

According to Gordon and Gibbons (1991:707), exercise can have the following positive impact on CHD risk factors:

- increased level of HDL-C
- reduced ratio of total cholesterol to HDL-C
- lowered triglycerides
- reduced risk of developing hypertension
- lowered systolic and diastolic pressure in hypertensive persons
- prevention of obesity by reducing the amount of excess fat
• relief of stress and a modifying influence on type-A behaviour
• prevention or control of diabetes mellitus by aiding carbohydrate metabolism.

2.5.2 Exercise, lipids and lipoproteins

Physical activity plays an independent role in the primary prevention of CHD, and physically active people are at lower risk of CHD than their inactive counterparts (Oberman 1983:37).

Exercise favourably alters lipoprotein profiles thereby reducing CHD. Reported research in this area exhibits many features, which make uniform conclusions difficult. For example, most of the studies used only males as subjects. Many trials were longitudinal, lasting for decades. This may have altered the natural history of the disease, through a better understanding of coronary risk factors, increased population awareness and higher standards of medical care. Several studies examined occupational activity while others assessed leisure activity. Finally, unknown variables could have influenced the outcome in non-randomized trials (e.g., changes in smoking patterns, exercise behaviour, or calorie restriction). Notwithstanding these deficiencies, Berlin and Colditz (2000:612) took the study designs of research into account and concluded that the relative risk of death from CHD was 1.9 times greater among sedentary subjects than ones with active occupations.

Morris, Clayton, Evritte, Semmerce and Burgess (2001:325) reported that “exercise must be vigorous and ongoing to protect against heart disease”. Persons participating in “non-vigorous” activity, such as golf and dancing, did not enjoy a protective effect. The number of hours spent walking on a weekly basis was not compared with CHD rates, but the walking speed tended to show an inverse relationship to increased CHD rates. The CHD rates were not associated with performing callisthenics or with the number of steps climbed daily. Recreational work such as gardening had no effect on CHD rates, regardless of its quantity or intensity. A multivariate analysis adjusting for risk factors, such as family and smoking, did not change the overall finding that vigorous, aerobic exercise protected all men while lesser degrees of aerobic exercise protected older men.
2.5.3 Aerobics

Numerous studies have been published, recording the effects of exercise programmes on serum lipids and lipoproteins. Many of these studies used small samples while others did not use control groups. Further compounding factors included seasonal variations, relative body weight and dietary changes; independent of exercise affects (Mattos & Ribero 2003:629). Mattos, ABM & Ribero, JP. 2003. Effects of two aerobic exercise intensities on exercise capacity and quality of life of elderly post-myocardial infarction women. *Journal of European Cardiology* 29(3): 629.

In semi-longitudinal and longitudinal studies “concerning serum lipids and exercise, results have been both varied and inconsistent. Factors such as duration, frequency, intensity of exercise and mode of training may interact and influence results. There have been wide variations with respect to the magnitude of lipid changes over various time frames. The majority of studies involving exercise training reported differences in lipoprotein levels after 13 weeks (Murray 1990:119)”.

According to Chandrashektar & Anand aerobic training engaged “ by apparently healthy people induces ventricular hypertrophy, which occurs rapidly and is reversed during detraining. Exercise-induced ventricular hypertrophy, unlike pathologic hypertrophy, is not associated with reduced myocardial contractility. In healthy persons aerobic training increases left ventricular end-diastolic volume and these changes are lost with de-conditioning. These changes increase cardiovascular functional capacity and decrease the myocardial oxygen demand for the same level of work performed. This is achieved by a decrease in the product of heart rate and systolic arterial blood pressure (an index of myocardial oxygen consumption). These adaptations also benefit coronary patients, who, after exercise, may attain a higher level of physical work before reaching the level of myocardial oxygen requirement that results in myocardial ischaemia “.

Whaley et al (1992:42-50) emphasise the importance of the pre-training total cholesterol levels in assessing the potential for a favourable change in total cholesterol subsequent to aerobic training.
Studies done by Golding & Rochelle (1961:499) using training frequencies of 5 days per week, with aerobic activity being emphasised in training. In this study “significant reductions in cholesterol were noted following the exercise period”.

2.5.4 Diet and CHD

Data establishing the link between diet and heart disease emerged in publications which appeared in the early nineteen thirties. Byrne (1996:1-36) reports that Nikoloi Anitsckow (1993) was the first to trace the link between cholesterol and dietary factors, using rabbits. It was only after World War 1, when the industrialized nations became aware of the epidermic increase in CHD, that a surge in research occurred concerning dietary factors and the relationship to CHD (Byrne, 1991:1-36).

Keys (1970:1-121), an epidemiologist at the University of Minnesota, and co-workers in several other countries documented fat intakes, serum cholesterol and CHD statistics of various populations during the early nineteen sixties. This was the first concerted effort to establish a relationship between diet and CHD through population studies. This study was known as the “Seven Country Study” and included Finland. Greece, Japan, Italy, Holland, United States and Yugoslavia. Dietary intakes, blood cholesterol levels and death rates from CHD were recorded in all these populations. This pioneering study showed that in populations where serum cholesterol levels were 6.2 to 7.2 mmol/l the incidence of CHD was 10 times higher than the average of 4.1 mmol/l.

Mensink and Katan (1994:439) found “that the effects of trans-fatty acids on the serum lipoprotein profile are at least as unfavorable as that of saturated fatty acids, as they not only raise LDL cholesterol levels but also lower HDL cholesterol levels”.

McMurray (1991:1704) investigated” the effects of an affluent Western diet on the lipid and lipoprotein levels and body weight of Tarahumara Indians. The risk of coronary heart disease increased dramatically and there were large increases in plasma lipid, lipoprotein levels and body weight mass”.

With regard to lipid levels and an affluent diet, Mann (2000:1992) found that “if weight loss occurred, the serum lipid levels would fall. The critical factor was sufficient activity
to burn off excess calories and to avoid weight gain. Besides the excess calories and weight gain of the experimental population, the most important aspect was the composition of the diet. A diet in saturated fats, with good weight control via exercise, may still not improve the lipid profile because of the increased intake through diet”.

Wood & Stefanick (2000:461) examined the effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise. The results indicated “that in overweight men and women, regular exercise further reduced the plasma lipoprotein levels resulting from the adoption of a low saturated, low cholesterol diet. The study used three groups, namely a control group, a low calorie diet group, and a low calorie and exercise group. Weight loss from the calorie diet alone did not significantly change HDL cholesterol levels in either men or women. Plasma HDL increased significantly in men who exercised and dieted. Men who dieted without exercise and men who acted as controls did not register such increases. HDL cholesterol levels remained similar in the women who exercised and dieted, but were higher than the controls. From this it appeared that moderate exercise coupled with a low caloric diet was most effective in reducing selected risk factors in CHD”.

Saini (2003:14-26) states that although most Indians were life-long vegetarians, wealth and a materialistic way of life have exposed them to unhealthy eating habits, resulting in a universal high incidence of CHD among the Indian population.

2.5.5 Stress and CHD

Stress is associated with elevated blood pressure. Although moderate stress plays a role in modern life, excessive response to stress can be a health hazard. Significant stressors include major changes in residence, occupation or status (Smeltzer & Bare 2000:674-675).

CHD is seven times more prevalent in North America, Australia, Europe, India, South Africa and New Zealand than in Japan, Switzerland and Italy (Smeltzer & Bare 2000:674-675) In developing countries, CHD is most prevalent among the affluent. Physical activity and the resulting physical fitness have a positive effect on the rate and development of coronary artery disease (Chandrashekhar & Anand 1995:1723-1739)).
Brezinka & Kittel (1996: 1351) stated that “persistent stress can lead to tension headache, migraine, backache, triggering of asthma, eczema, arthritis, palpitation, indigestion, diarrhoea, constipation and insomnia, which are not life-threatening but draining. Major stress disorders, like high blood pressure, heart disease or cancer can be life threatening and that “psychological stress or tension is the primary risk factor of coronary heart disease. Excessive stress can lead to increased blood pressure, heart rate, concentration of fat in the blood, blood sugar, cholesterol in the blood, blood clotting and deposition of fat and cholesterol in the arteries as well as spasm of coronary and other arteries. This means that stress on its own can cause CHD. The problem with stress is that there is no means of measuring it.

Stepanovic, Ostojic, Belesin & Todaro (2003:257) found that myocardial ischaemia was related to mental stress in a significant number of patients with severe CHD. Todaro, Biing-Jiun, Niaura, Spiro and Ward (2003:901) found that” negative emotions, such as depression and anxiety, were associated with an increased risk of developing CHD”.

### 2.6 CHD AMONG THE INDIANS

Morar, Sedat, Naidoo & Desai. (2003:313) In their study compared the blood pressure profile in 154 medical students of which 83 were Indians, 71 were blacks. Electrocardiography was performed in all participants and echocardiography in 90 of the students. The results of this study indicated that black people had higher blood pressure readings than young Indian participants in the absence of metabolic abnormalities and had greater cardiac involvement. Borderline hypertension is not innocuous. Metabolic risk factors for CHD in Indian people are apparent at an early age. This study emphasized the need for prevention of risk factors leading to CHD at an early age.

In their study on CHD risks in Indian and European populations, Bhopal, Urwin, White and Yallop (2000:215) found that Indians have lower high-density lipoprotein cholesterol, more diabetes, and a lack of aerobic exercise and greater central distribution of body fat. As a result they are prone to developing hypertension obesity. Moreover, Indians have a higher prevalence of factors potentially leading to stress, particularly economic deprivation and social upheaval following racial discrimination.
Finally, Indians are susceptible to CHD risk factors for genetic reasons. Sewell, Beth, Molosky and Gedney (2002:1368) found increasing incidences of coronary artery diseases and cardiovascular risk factors among the Southern Native American tribes. Sheth, Naire, Nargundkar, Anand and Yusuf (1999:8) research on the prevalence of CHD among the European, Indian and Chinese Canadians. The results indicated that ischaemic heart conditions were highest among Canadians of Indian origin (men 320.2, women 144.5) and European origin (men 319.6, women 109.90) and were markedly lower among Canadians of Chinese origin (men 107.0, women 40.0).

2.7 CONCLUSION

The literature review indicated that CHD is generally due to a combination of several risk factors. CHD is the major cause of premature death in the Indian population, is a serious source of disability and contributes in large part to the escalating costs of health care. The underlying pathology is usually atherosclerosis, which develops insidiously over many years and is usually advanced by the time symptoms occur.

Death, myocardial infarction and stroke frequently occur suddenly and before medical care is available, and many therapeutic interventions are therefore inapplicable or palliative. The rising incidence of CHD relates strongly to lifestyles and modifiable physiological factors. Risk factor modifications reduce mortality and morbidity.

This study investigated the increasing incidence of CHD among Indians in KwaZulu-Natal. With sufficient evidence to indicate that this community is at risk of CHD, proper patient education from nursing and medical staff on causes of heart diseases, modification of lifestyles, diet and medication compliance, and adequate stress management of those at risk are of particular significance. The findings will contribute to understanding the role of proper patient education from a nursing perspective, especially the prevention of CHD and its complications.

Chapter 3 describes the research design and methodology.