Module 4: Development of Coronary Artery Disease and Atherosclerosis

Cardiology

Self Learning Package

Module 4: Development of Coronary Artery Disease and Atherosclerosis
Acknowledgement

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for

Compiling the originally Cardiac Orientation Education Manual: Development of Coronary Artery Disease & Care of the Patient with Angina, it has been a valuable resource.
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INTRODUCTION

Welcome to Module 4: Development of Coronary Artery Disease and Atherosclerosis. This self learning package is designed to as tool to provide and insight for nurse of the evidence of risk factors for the development of Coronary Artery Disease and to provide insight into current understanding of atherosclerosis or plaque formation.

The goal of this module is to review:

- Coronary Artery Disease
- Atherosclerosis
- Coronary Artery Disease Risk Factors
- How risk Coronary Artery Disease Risk Factors affect the Heart

Learning outcomes form this module are:

- Describe atherosclerosis and coronary artery disease.
- List non-modifiable and modifiable risk factors that slowly lead to the development of coronary artery disease.
- Define biochemical risk factors for cardiac disease.
- Identify a treatment plan to manage the risk factors.
- Discuss the usefulness of early detection of coronary artery disease at the presymtomatic phase.
- What is atherosclerosis?
- Describe modifiable and non-modifiable cardiac risk factors.
- Identify biochemical cardiac risk factors.

This module will contribute to the foundation of your cardiac knowledge and enable you to understand Cardiovascular Disease and risk factors which will assist you in completing other modules.
HOW TO USE THIS SELF LEARNING PACKAGE

Follow the step outline to complete the Self Learning Package:

1) Read the journal articles provided.

2) Read the information provided in the Self Learning Package.

3) Complete Questions at the end of the package.

4) Once the above are completed return to your Clinical Nurse Educator or Clinical Nurse Specialist for marking.

Following the completion of this module, you will receive 6 hours professional Development hours, which will be credited to you education data base (CDHB).
INTRODUCTION

More than 68 million Americans currently have one or more forms of cardiovascular disease, according to the latest estimates from the federal government’s National Center for Health Statistics. Many more are said to be at risk for developing one of these serious diseases. The concept of risk factors has evolved only over the past 45 years or so, and new factors are periodically added to the list as our comprehension of the disease process grows. To understand who is at risk and what risk actually means to an individual, one first needs to understand how diseases of the heart and circulatory system—particularly heart attacks—develop.

All heart attacks, with rare exceptions, are caused by atherosclerosis, or a narrowing and “hardening” of the coronary arteries resulting from fatty deposits called plaque. This process, by which the wall of the artery is infiltrated by deposits of cholesterol and calcium, narrows the lumen (the internal orifice) of the artery. When the degree of narrowing reaches a critical level, blood flow to the portion of the heart supplied by that artery is stopped and injury to the heart muscle—a heart attack—occurs. If the reduction in blood flow is not total and is only temporary, the heart muscle recovers. If the blood flow is obstructed completely, the muscle becomes necrotic (dead). Chest pain, usually located in the center of the chest, can be a result of too little blood and oxygen to a portion of the heart in response to its needs (a process called ischemia). Atherosclerosis also occurs in other blood vessels, such as the carotid artery, which carries blood to the brain, or the arteries that provide blood to the legs, and can lead to similar problems. Significant atherosclerosis in the arteries supplying the brain may cause transient ischemic attacks (TIAs) or strokes, while peripheral arterial blood vessel disease, with intermittent claudication (pain on walking or similar activity), occurs when there is significant atherosclerosis in the arteries in the legs.

The fact that atherosclerotic plaque is largely made up of cholesterol has been known since the middle of the 19th century. Only in the 20th century, however, when general hygienic measures greatly reduced the toll from infectious diseases and allowed people to live considerably longer, did we realize the enormous impact of atherosclerosis on general health. By the 1930s and 1940s, the death rate in the United States from atherosclerotic heart disease was increasing at an alarming rate and it was clear that we were in the grips of a cardiovascular disease epidemic. The reasons for this epidemic were not entirely clear. Some scientists were convinced that there was a single cause for atherosclerosis—dietary fat and cholesterol—while others were more impressed by the association of high blood pressure or cigarette smoking with heart attacks. Most researchers fa-
vored the theory that there had to be multiple causes for atherosclerosis, although precisely what they were was debatable.

After World War II, the first large-scale, comprehensive study to determine the causes of atherosclerotic heart disease, the Framingham Heart Study, was begun. In 1948, researchers in the town of Framingham, Massachusetts, a suburb of Boston, enrolled 5,209 local residents, ranging in age from 30 to 62, in the study. They began examining the participants every two years, and they continue to do so. In the early 1970s, 5,135 adult offspring of the original participants joined the study.

Within a short time, the Framingham investigators established that there are, indeed, many factors that predispose an individual to the development of atherosclerosis. The list of these factors, now called cardiovascular risk factors (a term coined by Dr. William Kannel, the first director of the Framingham study), continues to grow as the information from Framingham and numerous other studies becomes available and we learn more about the possible causes of atherosclerotic disease.

This chapter defines cardiovascular risk factors, classifies them, briefly describes how they interact, and discusses what individuals and their physicians can do about them.

## HOW RISK FACTORS ARE IDENTIFIED

A cardiovascular risk factor is a condition that is associated with an increased risk of developing cardiovascular disease. The association is almost always a statistical one, and so the fact that a particular person has a particular factor merely increases the probability of developing a certain type of cardiovascular disease; it does not mean that he or she is certain to develop heart or blood vessel disease. Conversely, the fact that an individual does not have a particular cardiovascular risk factor (or for that matter, any of the known cardiovascular risk factors) does not guarantee protection against heart disease. Even today, a number of individuals who have heart attacks or strokes have none of the identified risk factors.

The box “Cardiovascular Risk Factors” lists the currently accepted cardiovascular risk factors. To understand how this list was compiled, one must know a little about epidemiology and how its techniques have been applied to identify risk factors.

<table>
<thead>
<tr>
<th>Cardiovascular Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Risk Factors That Cannot Be Changed</strong></td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Heredity</td>
</tr>
<tr>
<td><strong>Risk Factors That Can Be Changed</strong></td>
</tr>
<tr>
<td>High blood pressure</td>
</tr>
<tr>
<td>Elevated serum cholesterol</td>
</tr>
<tr>
<td>Lipoprotein (a)</td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Glucose intolerance</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Fibrinogen</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td>Cocaine</td>
</tr>
<tr>
<td>Behavioral factors (stress, Type A)</td>
</tr>
<tr>
<td><strong>Protective Factors</strong></td>
</tr>
<tr>
<td>HDL cholesterol</td>
</tr>
<tr>
<td>Exercise</td>
</tr>
<tr>
<td>Estrogen</td>
</tr>
<tr>
<td>Moderate alcohol intake</td>
</tr>
</tbody>
</table>

The epidemiologist studies populations. He or she begins by selecting a group that is representative of the population to which the information will later be applied. To examine the cause of atherosclerosis, for example, the study group selected should be largely composed of young and middle-aged adults who have no evidence of cardiovascular disease when the study begins. Because the differences between individuals will be small, the group must be large enough to allow the relationships between the factors being studied and the disease to become evident and to enable researchers to draw conclusions about these relationships. While earlier studies were limited to much smaller groups, the advent of computers has enabled epidemiologists to collect and analyze enormous amounts of data and to study very large groups or populations, sometimes numbering hundreds of thousands.

The study group must be followed for a considerable length of time. A chronic disease such as atherosclerosis, which has many causes and usually requires years for signs or symptoms of heart disease to develop, requires multiple observations over many years to determine how each potential risk factor is changing and interacting with the others.

For any epidemiological survey to be helpful, the appropriate factors must be studied. None of the risk factors on the currently accepted list got there by chance; each resulted from careful observations and
Cardiovascular Risk Factors

A statistical technique called multivariate analysis allows researchers to tease out true associations from those that appear to contribute but do not do so independently. A good example is coffee drinking, which seemed at first to be associated with an increased risk of heart disease. Multivariate analysis showed that the association was not independent, but rather due to the fact that many people smoke cigarettes when they drink coffee. When this fact was taken into account, it became clear that the real villain is the cigarette, not the caffeine.

Some cardiovascular risk factors are dichotomous; that is, they are either present or absent. Male gender and family history are two examples. Most risk factors, however, are continuous; that is, above a certain threshold level, risk rises as the strength or severity of the risk factor rises. For example, the more cigarettes smoked a day, the greater the risk of heart disease. This is also called a "dose-response."

The risk may rise dramatically when the strength of the risk factor exceeds a certain level. Blood pressure and blood cholesterol levels are typical of such risk factors. For both of these, there is a very small increase in risk as the level rises within the range considered "normal." This increased risk is so small that any attempt to lower it would not improve overall outlook. At the other end of the scale, there is a point (90 mm Hg for diastolic blood pressure and 240 mg/dl for serum cholesterol) above which risk increases substantially.

It is now possible to estimate quantitatively an individual’s cardiovascular risk. This technique employs data gathered from epidemiologic surveys attributing varying levels of risk to such factors as blood pressure, serum cholesterol, age, and number of cigarettes smoked per day. (See Table 3.1.) Within seconds, an individual’s probability of having a heart attack in a defined period of time can be calculated. This approach also shows that the impact of risk factors is at least additive and possibly multiplicative. What this means is that an individual’s risk is determined in part by the number of risk factors present, as well as the level of each individual factor. (See Figure 3.1.) For example, someone who has mildly elevated blood pressure and serum cholesterol may be at greater risk of sustaining a heart attack or stroke than would an individual with even higher blood pressure whose serum cholesterol is normal.

This compounding effect has a number of important implications for individuals. First, it is not sensible to view the risk of having heart disease as great or small on the basis of a single risk factor. Second, a treatment program for risk factor reduction must...
### Table 3.1
Coronary Heart Disease Risk Factor Prediction Chart-Framingham Heart Study

#### 1. Find Points for Each Risk Factor

<table>
<thead>
<tr>
<th>Age (if female)</th>
<th>HDL cholesterol</th>
<th>Total cholesterol</th>
<th>systolic blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Pts.</td>
<td>HDL C Pts.</td>
<td>Total C Pts.</td>
<td>SBP Pts.</td>
</tr>
<tr>
<td>30 -12</td>
<td>47 - 48</td>
<td>5</td>
<td>30 - 2</td>
</tr>
<tr>
<td>31 -11</td>
<td>49 - 50</td>
<td>6</td>
<td>31 - 1</td>
</tr>
<tr>
<td>32 -9</td>
<td>51 - 52</td>
<td>7</td>
<td>32 - 3 - 0</td>
</tr>
<tr>
<td>33 -8</td>
<td>53 - 55</td>
<td>8</td>
<td>34 - 1</td>
</tr>
<tr>
<td>34 -6</td>
<td>56 - 60</td>
<td>9</td>
<td>35 - 36 - 2</td>
</tr>
<tr>
<td>36 -4</td>
<td>68 - 74</td>
<td>11</td>
<td>39 - 4</td>
</tr>
<tr>
<td>37 -3</td>
<td>40 - 41</td>
<td>5</td>
<td>40 - 41 - 5</td>
</tr>
<tr>
<td>39 -1</td>
<td>44 - 45</td>
<td>7</td>
<td>44 - 45 - 7</td>
</tr>
<tr>
<td>40 - 0</td>
<td>46 - 47</td>
<td>8</td>
<td>46 - 47 - 8</td>
</tr>
<tr>
<td>42 -33</td>
<td>50 - 51</td>
<td>10</td>
<td>50 - 51 - 10</td>
</tr>
<tr>
<td>44 - 3</td>
<td>52 - 54</td>
<td>11</td>
<td>52 - 54 - 11</td>
</tr>
<tr>
<td>45 - 46</td>
<td>55 - 56</td>
<td>12</td>
<td>55 - 56 - 12</td>
</tr>
</tbody>
</table>

#### 2. Sum Points For All Risk Factors-Framingham Heart Study

<table>
<thead>
<tr>
<th>Age</th>
<th>HDL C</th>
<th>Total C</th>
<th>SBP</th>
<th>Smoker</th>
<th>Diabetes</th>
<th>ECG-LVH</th>
<th>Point total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Minus points subtract from total.

#### 3. Look Up Risk Corresponding to Point Total

<table>
<thead>
<tr>
<th>Probability of CHD</th>
<th>Probability of CHD</th>
<th>Probability of CHD</th>
<th>Probability of CHD</th>
<th>Probability of CHD</th>
<th>Probability of CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pts. 5 Yr. 10 Yr.</td>
<td>Pts. 5 Yr. 10 Yr.</td>
<td>Pts. 5 Yr. 10 Yr.</td>
<td>Pts. 5 Yr. 10 Yr.</td>
<td>Pts. 5 Yr. 10 Yr.</td>
<td>Pts. 5 Yr. 10 Yr.</td>
</tr>
</tbody>
</table>

#### 4. Compare to Average 10-Year Risk

<table>
<thead>
<tr>
<th>Probability</th>
<th>Age</th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1%</td>
<td>30 - 34</td>
<td>&lt;1%</td>
<td>3%</td>
</tr>
<tr>
<td>1% - 2%</td>
<td>35 - 39</td>
<td>&lt;1%</td>
<td>5%</td>
</tr>
<tr>
<td>2% - 3%</td>
<td>40 - 44</td>
<td>2%</td>
<td>6%</td>
</tr>
<tr>
<td>3% - 4%</td>
<td>45 - 49</td>
<td>5%</td>
<td>10%</td>
</tr>
<tr>
<td>4% - 5%</td>
<td>50 - 54</td>
<td>8%</td>
<td>14%</td>
</tr>
<tr>
<td>5% - 6%</td>
<td>55 - 59</td>
<td>12%</td>
<td>16%</td>
</tr>
<tr>
<td>6% - 7%</td>
<td>60 - 64</td>
<td>13%</td>
<td>21%</td>
</tr>
<tr>
<td>7% - 8%</td>
<td>65 - 69</td>
<td>9%</td>
<td>30%</td>
</tr>
<tr>
<td>8% - 9%</td>
<td>70 - 74</td>
<td>12%</td>
<td>24%</td>
</tr>
</tbody>
</table>
Using Table 3.1

Table 3.1 was created using data from the Framingham Heart Study to help individuals determine their risk of developing coronary heart disease over five or ten years. It represents a first attempt at developing a data-based tool that patients and their physicians can use as a starting point for a discussion of modifying behavior.

Although the Framingham database is one of the most comprehensive available, it has some limitations. For example, it may be less accurate for African-Americans than for whites. The table has been criticized by some for its inclusion of both total cholesterol and HDL cholesterol, thereby perhaps giving extra weight to cholesterol as a risk factor. The table also indicates that an electrocardiogram is necessary to determine if left ventricular hypertrophy is present.

Nevertheless, the table is useful as a general tool for individuals to use in estimating their risk of developing coronary heart disease and comparing their risk to the average. They can also use it to see how changing a modifiable risk factor may affect their total risk. For example, a person who is a smoker can look at the difference in risk if smoking is stopped. Likewise, someone with elevated cholesterol can look at the effect of lowering it. Modifying a single risk factor may affect life expectancy by as much as eight years; when there are strong and multiple risk factors the effect can be substantial. Life expectancy is not the only reason to consider changing risk-prone behavior. Behavioral changes can also have a very positive effect on the quality of life.

be comprehensive. Third, it is likely that measures to prevent atherosclerotic heart disease and stroke will be most beneficial in those with the highest risk, and difficult to prove in those with only a minimally increased chance of developing these diseases.

THE EFFECT OF MODIFYING RISK FACTORS

Taking action that modifies a risk factor does not necessarily imply that the probability of a heart disease or stroke will be eliminated. Furthermore, when a strong risk factor is present, treating it—even if the treatment is very effective—does not necessarily mean that the risk is reduced. Fortunately, treatment of the major risk factors—smoking, high blood pressure, and elevated cholesterol levels—has been shown to reduce the possibility of a heart attack.

In general, it is a monumental scientific undertaking to demonstrate that treatment or modification of a risk factor reduces the number of heart attacks, strokes, or other cardiovascular diseases. Because atherosclerosis has many causes and is almost always present in some degree in all of us, studies to show that a specific treatment works are difficult to design. Furthermore, the results may be hard to interpret and apply to the general population.

For a study of a proposed treatment (usually called a clinical trial) to be valid, it must have a control: The treatment must be tested against another treatment or against no treatment at all. ("Treatment" in a clinical trial might mean a drug or a modification in behavior such as exercising more or eating less saturated fat.) Volunteers enrolled in such a study must be representative of the patients in whom the treatment will be used. For example, if the subjects already have advanced atherosclerosis, the treatment used may appear ineffective, when in fact it might have been successful if started earlier in the course of the disease. If the subjects are at very low risk, the treatment may not appear to work because the like-
lihood that disease would develop is so small. It would be hard in this case to show a difference between the treatment and the control groups.

Investigators who conduct clinical trials must carefully define the population to be studied and the particular cardiovascular benefit they hope to achieve. Some treatments studied have mistakenly been judged ineffective when, in fact, the trial was simply too small or did not last long enough to show the benefit expected.

Unfortunately, too, clinical trials designed to evaluate the benefits or risks of therapy with respect to clinical events take a long time to complete. Because of the enormous effort and cost, it is impossible to devise ideal tests for every new and allegedly better approach to therapy. Physicians must analyze the findings from both epidemiologic surveys and clinical trials, synthesize the data, incorporate new information, and then apply it to individual patients. That is a difficult task.

RISK FACTORS THAT CANNOT BE CHANGED

AGE

The risk of cardiovascular events increases as we get older. In many epidemiologic surveys, age remains one of the strongest predictors of disease. More than half of those who have heart attacks are 65 or older, and about four out of five who die of such attacks are over age 65.

Of course, nothing can be done to reduce age. However, careful attention to diet and maintaining fitness may delay the degenerative changes associated with aging.

GENDER

Men are more likely than women to develop coronary heart disease, stroke, and other cardiovascular diseases that are manifestations of atherosclerosis. Whether this is because male hormones—andro gens—increase risk or because female hormones—estrogens—protect against atherosclerosis is not completely understood. It is likely that both play a role, but that the protective role of estrogens is the predominant factor. This seems to be supported by the fact that heart disease risk for women rises dram atically after menopause, when their bodies stop producing estrogen. Nevertheless, coronary heart disease is the number one cause of death among American women.

Women in the United States currently live an average of six years longer than men. Recently, some studies have suggested that much of the difference in life expectancy can be explained by the fact that more men than women smoke cigarettes. As more teenage girls are starting to smoke than are teenage boys, this advantage may disappear. Should this trend go unchecked, women may soon have as much coronary heart disease and other complications of cigarette smoking as do men, or more.

HEREDITY

There is no question that some people have a significantly greater likelihood of having a heart attack or stroke because they have inherited a tendency from their parents. In some instances, such as familial hypercholesterolemia (very high levels of cholesterol in the blood), the pattern of inheritance is well understood and the specific biochemical defects are well characterized. For most cardiovascular risk factors, however, the specific way in which inheritance plays a role is not at all clear. As in almost all situations in medicine, both heredity and environment play a role and it is often difficult to know where one stops and the other begins. Prior generations did not have the level of medical care we now enjoy, nor the general awareness about health; the details of the illness that one’s grandparents or even parents had may not be precise. Prior to the 1960s, many more people smoked and little attention, if any, was paid to diet and fitness. So it is possible that environmental factors, not genes, were responsible for Grandpa’s heart attack or stroke.

In practical terms, anyone who has a family history of heart disease that occurred at an early age (below 55) should be especially careful to reduce the impact of any risk that can be controlled. Even if one can successfully control known risk factors, there are, unfortunately, a number of inherited characteristics that we have not yet identified and so cannot favorably affect. Individuals with a history of atherosclerotic cardiovascular disease in the family simply have to be more vigilant if they wish to avoid heart attacks and strokes. We should remember, however, that almost every family has some member who died of a heart or blood vessel disease, since about half of all deaths are attributable to these diseases. If these ep-
isides occurred in relatives who were 75 or 80, it may not be a major cause for concern.

Hereditity also includes race. For reasons that are not completely understood, African-Americans have considerably higher rates of diabetes and both moderate and severe high blood pressure, adding to their overall risk of heart disease. (For more information, see below and Chapter 22.)

RISK FACTORS THAT CAN BE CHANGED

HIGH BLOOD PRESSURE

High blood pressure, or hypertension, is the risk factor that affects the greatest number of Americans and the one we know the most about. Estimates vary according to the source, but anywhere from 35 million to more than 60 million Americans have elevated blood pressure.

There are several ways to classify hypertension. It is generally agreed that high blood pressure is defined as readings that consistently exceed 140/90 mm Hg, when measured over a period of time with a blood pressure cuff (sphygmomanometer). Experts focused on diastolic blood pressure, the lower of the two numbers, which represents the resting pressure between heartbeats. Anyone with a reading equal to or greater than 90 mm Hg has diastolic hypertension, regardless of the level of the higher number, which represents the systolic, or pumping, pressure.

Some individuals, particularly those over 65 or 70 years of age, have what is called isolated systolic hypertension. The most recent expert committee defines this as a systolic blood pressure of 160 mm Hg or more, when the diastolic blood pressure is less than 90 mm Hg.

Actually, the levels of both systolic and diastolic blood pressures determine an individual’s risk. In fact, of the two readings, the systolic blood pressure may be the superior predictor of all the complications we attribute to hypertension.

The most reliable early information on high blood pressure comes from the Framingham Heart Study, which showed early on that as both the systolic and diastolic blood pressure levels rise, the likelihood that an individual might develop coronary heart disease, stroke, congestive heart failure, peripheral vascular disease, and kidney problems rises as well. The association is strongest for stroke, although it is highly significant for other cardiovascular diseases, too. The Framingham Heart Study also showed that people with hypertension had a higher death rate, when all causes were added together, than did those with normal readings. All of these findings have been amply confirmed by many other studies and apply to both men and women, as well as to people in their 60s and 70s and beyond.

Hypertension is a special problem for African-Americans. Overall, the percentage of blacks in the United States with hypertension is 50 percent greater than that of whites or Asians. Black men under the age of 45 are particularly prone to developing kidney failure from hypertension, eventually requiring dialysis or a kidney transplant. Blacks are also more likely than whites to have heart enlargement as a result of hypertension and ultimately to have congestive heart failure.

Hypertension often occurs together with other cardiovascular risk factors, particularly obesity, elevated levels of cholesterol and triglycerides, and diabetes mellitus. This suggests that there may be a common cause for these conditions, but it may simply be that an environmental factor, such as overeating, may lead to some or all of these problems.

There is a wealth of studies to show that successfully treating hypertension will substantially reduce the increased risk associated with it. Fortunately, too, we now have many well-tolerated antihypertensive medications that lower blood pressure and can be taken indefinitely. Although most of the treatment data are based on drugs, such measures as weight loss, salt restriction, and exercise may also lower blood pressure. As yet, however, no long-term studies have shown convincingly that these life-style changes are as successful as drugs in preventing strokes and other complications of hypertension. (For more information, see Chapter 12.)

HIGH BLOOD CHOLESTEROL AND RELATED LIPID PROBLEMS

Elevated levels of serum lipids (cholesterol and triglycerides) are extremely common and are one of the most important of the heart disease risk factors that can be changed. Yet, there is considerable confusion about the role of cholesterol as a cardiovascular risk factor. (See Chapter 4.)

Epidemiologic studies have shown that the level of total cholesterol in the blood is a strong predictor of the likelihood that an individual will develop coronary heart disease and, to a much lesser degree, a stroke. Most experts consider levels under 200 mg/dl
to be normal and those between 200 and 239 mg/dl to be borderline high. Levels above 240 mg/dl present an increased risk for a heart attack more than double the risk of levels below 200 mg/dl. About one out of four Americans falls into this latter category.

Total cholesterol levels are made up of several fractions. The most important and best studied are high-density lipoproteins (HDL cholesterol, or HDL-C) and low-density lipoproteins (LDL-C). These levels and their relationship to each other maybe more important than total cholesterol levels in predicting heart disease risk. LDL levels over 160 mg/dl are definitely associated with increased risk, while values from 130 to 159 mg/dl are borderline. In contrast, HDL cholesterol is the fraction of cholesterol that appears to protect against coronary heart disease. The higher the level of HDL, the lower the risk. Ideally, it should be at least 35 mg/dl. A ratio of LDL to HDL greater than 3.5 or 4.1 is generally agreed to increase risk.

Many studies have failed to show an independent contribution to coronary heart disease risk from an elevation of triglycerides, another fatty component in the blood. Recent data, however, suggest that triglycerides may be an important predictor of risk, especially in women and those with diabetes mellitus.

While an individual's lipid profile is affected by age (total cholesterol rises with the years), gender (women tend to have higher levels of HDL), and heredity (elevated cholesterol and triglycerides tend to run in families, and certain families have extremely high levels), the picture can be significantly changed by lifestyle modifications. A diet low in saturated fat and cholesterol will lower serum cholesterol an average of 5 percent, but this diet may be more effective in some people. The general rule of thumb is that risk of coronary heart disease decreases by 2 percent for every 1 percent drop in total serum cholesterol.

Reducing alcohol intake in heavy drinkers and (for those who are overweight) body weight can significantly reduce triglyceride levels. Regular exercise will lower triglycerides and increase HDL cholesterol, and stopping smoking will also raise HDL cholesterol. For people with very high total cholesterol and LDL cholesterol levels, diet and exercise alone may not result in a great enough reduction, and these lifestyle measures may need to be combined with cholesterol-lowering drugs. (See Chapter 23.)

Lp(a)

Lipoprotein (a) or "Lp little a" was discovered in 1963, but its importance was not appreciated until recently.

Lp(a) is a molecule composed of the protein portion of low-density lipoprotein (LDL), which is called apoB, and another protein called apo(a). Apo(a) is very similar chemically to plasminogen, a naturally occurring substance that participates in dissolving clots that form in the bloodstream. Lp(a) has the opposite effect, however. It interferes with the normal process of clot lysis (dissolving) and thus may increase the likelihood that once a clot forms, a heart attack or stroke will occur.

Recent epidemiologic studies have shown that increased Lp(a) levels are associated with a greater frequency of coronary artery disease, increased clogging (stenosis) of coronary artery bypass grafts, and stroke (cerebrovascular disease). The impact of Lp(a) levels on the risk of coronary heart disease is as strong as that seen with total cholesterol levels or reduced high-density lipoprotein (HDL) levels, and the increase in risk attributable to high Lp(a) levels is independent of other risk factors. At this time, of the drugs available, only nicotinic acid seems to lower Lp(a) levels. Whether this reduction decreases the risk of developing disease is still unclear.

CIGARETTE SMOKING

Cigarette smoking is a major contributor to coronary heart disease, stroke, and peripheral vascular disease—even though smokers tend to be thinner and to have lower blood pressure than nonsmokers. Overall, it has been estimated that 30 to 40 percent of the approximately 500,000 deaths from coronary heart disease each year can be attributed to smoking.

Individuals who smoke, regardless of their level of other risk factors or family history, are at significant risk of premature coronary disease and death. Smokers, for example, have less of a chance of surviving a heart attack than nonsmokers. Evidence from the Framingham Heart Study shows that the risk of sudden death increases more than tenfold in men and almost fivefold in women who smoke. Smoking is the number one risk factor for sudden cardiac death and for peripheral vascular disease.

Smoking cigarettes that are low in nicotine and tar does not decrease the risk of heart disease, which is increased by the effect of smoke on blood vessel walls. In fact, some people tend to smoke more and inhale deeply when they switch to this type of cigarette, increasing their exposure to the carbon monoxide in the smoke itself.

Fortunately, the risk of heart disease begins to de-
OBESITY

Any level of overweight appears to increase heart disease risk. Obesity can predispose the development of other risk factors, and the greater the degree of overweight, the greater the likelihood of developing other antecedents of atherosclerosis (such as high blood pressure and diabetes) that will increase the probability that heart disease will develop. Those who are obese (more than 30 percent over their ideal body weight) are the most likely to develop heart disease, even if they have no other risk factors. One recent study that examined more than 100,000 women age 30 to 55 showed that the risk for heart disease was more than three times higher among the most obese group than among the leanest group.

It also appears that how our weight is distributed may be even more important than exactly how much we weigh. There are two basic patterns of obesity—one in which excess fat is found primarily in the abdominal area (the "beer belly" or apple shape) and one in which excess fat deposits form around the hips and buttocks (the pear shape). The former type is called male-pattern obesity or android obesity; the latter, female-pattern or gynecoid obesity. Android obesity, which is also found in some women (especially after menopause), is associated with an increased risk of cardiovascular disease, specifically, coronary heart disease and stroke. A general rule of thumb is that a man's waist measurement should not exceed 90 percent of his hip measurement and that a woman's waist measurement should be no more than 80 percent of her hip measurement.

Android obesity appears to be most closely related not only to risk but also to other cardiovascular risk factors—namely hypertension, elevated triglycerides, low HDL cholesterol, elevated blood sugar levels, and diabetes mellitus. The common feature of all these conditions is an elevation in the level of insulin (the hormone that regulates the metabolism of sugar in the body) in the blood and a condition called insulin resistance, in which body tissues (especially the large muscles) do not respond normally to insulin. The likelihood that fat distribution and insulin resistance are related to genetics again points to the pivotal role of heredity in disease risk.

DIABETES MELLITUS AND INSULIN RESISTANCE

Individuals with diabetes mellitus, especially those whose diabetes occurs in adult life, have an increased incidence of coronary heart disease and stroke. Those who have slightly elevated blood sugar levels but do not have detectable diabetes also have an increased risk of developing these problems. Many individuals whose diabetes begins after age 40 or 50 (so-called adult-onset or Type II diabetes) often have higher than normal levels of circulating insulin. The primary role of insulin, a hormone produced by the pancreas, is to maintain blood sugar at normal levels and to assist this body fuel in entering each of the body's cells. For some reason, some individuals do not respond as readily to insulin, and more is required to do the job; they have insulin resistance. Elevated levels of insulin can raise blood pressure and assist in the deposition of and reduce the removal of cholesterol from plaques in the arteries. Both these actions increase the likelihood that atherosclerosis and its complications will develop.

Fortunately, weight reduction and exercise can improve the burning up of blood sugar (glucose) and prevent or slow down the onset of diabetes.

Individuals who develop diabetes in childhood (so-called juvenile-onset or Type I diabetes) are more likely to develop kidney and eye problems than coronary heart disease or strokes. In this type of diabetes, insulin is absent due to disease in the pancreas.

FIBRINOGEN

Serum fibrinogen is a component of the blood that plays a central role in the clotting process. Recent results from the Framingham Heart Study and elsewhere have shown that the level of fibrinogen is an independent cardiovascular factor. Why higher levels of this clotting factor increase risk is not yet known, but it is likely that individuals with higher levels may be more prone to develop clots in their arteries, thereby increasing the risk of a heart attack or stroke. Fibrinogen levels rise with age, and in that sense are not a risk factor that can be modified. However, fibrinogen levels are also adversely affected by cigarette smoking, which can be controlled.

BEHAVIORAL FACTORS

Coronary-prone behavior, sometimes referred to as "Type A behavior, is felt by some, but not all, experts to be an important risk factor for coronary heart dis-
PROTECTIVE FACTORS

EXERCISE
While it is not clear that a sedentary life-style is a cardiovascular risk factor, the evidence is convincing that regular exercise will reduce the likelihood of a heart attack and may improve the chances of survival if one does occur. Exercise also seems to have a positive effect on a number of other risk factors. Whether its benefit lies in the fact that it helps control weight, improves the body's ability to use insulin, conditions the heart muscle, increases levels of protective HDL cholesterol, moderates stress, or lowers blood pressure—or a combination of these effects—is not clear. Whatever the reason, regular exercise can lower cardiovascular risk and should be encouraged for everyone within the limits of each individual. (See Chapter 7.)

ESTROGEN
Estrogen (the major female sex hormone) protects against heart attacks and other forms of cardiovascular disease. Estrogen increases HDL cholesterol, which may explain how the hormone reduces the incidence of heart attacks in premenopausal women. It is now clear that once menopause occurs, women are at the same risk for heart attacks as are men. Thus, it is reasonable to advise that postmenopausal women receive estrogen replacement therapy unless it is medically contraindicated. Although it is likely that estrogen replacement therapy reduces the frequency of heart attacks, such therapy may increase the risk of cancer of the uterus. This risk can be reduced or eliminated by combining estrogen with progesterone, another female sex hormone. In fact, recent studies indicate that combined hormone therapy may actually reduce the possible risk of breast or uterine cancer. As an added advantage, postmenopausal estrogen replacement reduces the severity of osteoporosis—the bone thinning that is a leading cause of death and disability in older women. (See Chapter 19.)

ALCOHOL
In moderation—that is, no more than one or two drinks a day—alcohol may protect against coronary heart disease and atherosclerosis. Although the exact
mechanism is not understood, it appears that alcohol raises HDL cholesterol. The association is certainly not strong enough to recommend that nondrinkers take up alcohol consumption. Furthermore, drinking four or more drinks per day can have deleterious effects. It raises blood pressure and puts the individual at significant risk of liver damage, central nervous system complications, and a number of other serious problems, some of which are cardiovascular. (See Chapter 6.)

A PROGRAM FOR CARDIOVASCULAR RISK FACTOR MODIFICATION

How should you use the information presented in this chapter to make certain that you are doing everything possible to avoid a heart attack, stroke, or other complication of atherosclerosis? The first step is to assess, with the help of a physician, whether or not you are a high- or low-risk individual.

For some answers, you do not need a doctor. Do you smoke cigarettes? Are you overweight? Do you drink too much? Is there heart disease or high blood pressure in the family? To fully assess risk, however, a physician is needed. He or she will measure blood pressure, send blood for serum cholesterol, triglyceride, and glucose measurements, and perform a history and physical examination. An electrocardiogram or more specialized procedures can be done to determine if the heart is enlarged. With this information, a table such as Table 3.1 may be helpful in assessing the interaction of various factors to determine total risk.

Once all of this information is collected and evaluated, a treatment program, directed at modifying risk factors, can be started. For those who are free of cardiovascular risk factors or clinical vascular disease, certain simple steps can always help, and will do little if any harm:

- *Eat a heart-healthy diet*—one low in saturated fats and cholesterol. Use monosaturated or polyunsaturated fat.
- *Reduce weight if it is elevated.* Even a small amount of weight loss can be helpful if you are overweight.
- *Moderate your salt intake.* Many people are not sensitive to salt and their blood pressure will not rise even if their intake of table salt and other forms of sodium is high. The problem is, we cannot distinguish who is and is not salt-sensitive without complex testing. Most of us eat more salt than we need. Many foods are naturally high in sodium and others have salt added in processing. Simple measures such as not adding salt to the food as it is cooked or at the table will reduce sodium intake to a reasonable amount. This degree of salt restriction

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<table>
<thead>
<tr>
<th>Table 3.2</th>
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<td><strong>The American Heart Association’s Recommendations for Periodic Health Examinations</strong></td>
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<th>Physical exam</th>
<th>Blood pressure</th>
<th>Plasma lipids</th>
<th>Body weight</th>
<th>Fasting glucose</th>
<th>ECG chest X-ray</th>
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<td>75 and over (every year)</td>
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Blood pressure should be taken every 2½ years in normal patients. Plasma lipids include fasting cholesterol and triglycerides. Optional if baseline levels are well documented.

Note: These recommendations are reviewed periodically and are subject to change. They can, however, be used as a general guideline.
is absolutely safe and does not rob food of its taste, especially if herbs and spices are used as alternative flavorings.

- **Start a regular exercise program.** Virtually everyone can benefit from regular exercise. To be helpful, the program need not be too strenuous and can be tailored to an individual's preferences, schedule, and physical capabilities. Regular walking may be all that is necessary.
- **If you smoke, stop.** Nothing will be more beneficial!
- **If you drink alcohol, do so in moderation.**
- **Learn stress-reduction techniques** and avoid reacting to stressful situations in ways that will only serve to aggravate the problem.
- **Have your risk factor status assessed on a regular basis.** A clean bill of health on one occasion does not guarantee a lifetime of protection. Blood pressure, if normal, should be checked every two years or so, and cholesterol, if normal, should be checked every five years. (These recommendations are reviewed periodically as more is learned about risk. See Table 3.2 for current recommendations from the American Heart Association.)

What about individuals with definite hypertension or elevated cholesterol levels? The time to initiate therapy and the choice of therapy should be left to the physician, but always in consultation with the patient. In general, those who are at high risk because of very high blood pressure or cholesterol level or who have multiple risk factors require drug treatment, although a brief trial of diet, exercise, or other lifestyle changes may be appropriate first.

It is crucial to understand that treatment of cardiovascular risk factors is preventive medicine at its most challenging. After all, the physician is asked to select an effective and affordable regimen that does not make the patient sick and that can be useful for life. The irony is that in their early stages, neither hypertension nor high blood cholesterol produces symptoms, yet therapy for these conditions may interfere with enjoyment of life or, in some cases, actually cause symptoms.

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**Figure 3.2**

*Age-Adjusted Death Rates for Major Cardiovascular Diseases*

![Chart showing age-adjusted death rates for major cardiovascular diseases](chart.png)

*Source: National Center for Health Statistics, U.S. Public Health Service, DHHS and the American Heart Association.*
Nevertheless, dietary or behavioral changes and drug therapy have proved worthwhile. It is clear that modifying cardiovascular risk factors is remarkably successful preventive medicine. In the United States, we have made considerable inroads against the epidemic of cardiovascular disease. Since 1972, we have reduced the death rate from strokes by more than 50 percent and deaths from coronary heart disease by more than 40 percent. (See Figure 3.2.) Other countries that have followed our lead are beginning to do as well. It is likely that with increased understanding and application of the principles discussed here, we can do even better.
Progress in Prevention

Should We Screen for Risk Factors or Disease?

A Current Debate in the Prevention of Cardiovascular Events

Cheryl R. Dennison, PhD, RN, ANP, FAHA; Suzanne Hughes, MSN, RN, FAHA

Cardiovascular disease (CVD) is highly prevalent, and cardiovascular (CV) events often are fatal or disabling and expensive. A debate is underway regarding the most rational approach to preventing CV events among individuals who may be at risk but who have not yet manifested CVD. One view in this debate reflects the current guideline-driven approach and recommends prevention, routine screening, and treatment of traditional CV risk factors to prevent CV events.1-3 Traditional modifiable CV risk factors include elevated blood pressure and cholesterol level, diabetes, tobacco smoking, physical inactivity, and obesity. The opposing view suggests that a more effective approach to preventing CV events would involve identification of asymptomatic individuals with early CVD to initiate treatment. Cardiovascular abnormalities that ultimately result in morbid events are detectable in the arteries and heart through noninvasive testing before the development of symptomatic disease. Therefore, recent efforts have focused on identifying the presence of these abnormalities as a more sensitive and specific guide to the need for therapy.4

Screening for CVD Risk Factors

The evidence that most CVD is preventable continues to grow.1 Results of long-term prospective studies consistently identify persons with low levels of risk factors as having lifelong low levels of CVD.1 Management of CV risk factors remains the cornerstone of primary prevention in CVD. Assessment for the presence of CV risk factors provides an opportunity to identify asymptomatic individuals who are at risk for developing clinical CVD and initiate treatment to reduce risk of CV events. A strong evidence base provides support for guidelines recommending smoking cessation, physical activity, and management of blood pressure, lipids, diabetes, and weight to prevent CVD and CV events.1

The quantitative relationship between these risk factors and CVD risk has been elucidated by the Framingham Heart Study, showing that the major risk factors are additive in predictive power.5 Accordingly, an individual’s total risk can be estimated by summing the risk imparted by each of the major risk factors. The Framingham Risk Score (FRS) is the most commonly used multiple risk factor score. The FRS, based on a number of traditional risk factors including age, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, and cigarette smoking, is expressed as a number reflecting the likelihood of fatal or nonfatal CV event over a 10-year period. Patients who have less than a 10% risk of events are considered low risk, those who have a 10% to 20% likelihood of events are at intermediate risk, and those with more than 20% likelihood are at high risk.5 The FRS serves as the basis for identifying asymptomatic adults who should be managed aggressively for primary prevention.

Approximately one-third of Americans aged 40 to 70 years are at intermediate risk for CVD, as defined by the FRS.5 Although consideration of major risk factors such as serum cholesterol...
levels, blood pressure, and smoking status plays a role in the overall evaluation, this approach may not always provide an accurate risk assessment. The exclusion of significant risk factors such as family history and obesity limits the FRS, particularly among women. In several studies, the FRS has been found to underestimate risk in women by its failure to include family history in the scoring criteria. Moreover, the FRS frequently classifies women as being low risk, even in the presence of significant coronary artery calcification (CAC). Although the assessment of absolute CV risk is advocated in individual risk factor guidelines, the FRS has limitations and may not apply equally to all sex, race, and ethnic groups.

**Screening for Asymptomatic CVD**

Cohn and Duprez argue that traditional CV risk factors are not disease markers, have low sensitivity and specificity for predicting CV events, and therefore provide a crude and imprecise approach to decision making. Because atherosclerotic disease precedes morbidity events by years and can now be recognized by easily performed noninvasive testing, they recommend the Rasmussen Disease Score as a global model for assessing vascular and cardiac health and identifying early CVD. The Rasmussen score consists of 7 vascular and 3 cardiac functional and structural tests, selected because they are predictive of CV morbidity and mortality. The tests comprising the Rasmussen score conceivably can be performed in 1 hour by 1 technician in a single room. The 7 vascular tests include large and small artery elasticity (compliance), sitting blood pressure, blood pressure response during a moderate treadmill exercise test, optic fundus photography, measurement of carotid intimal-media thickness, and microalbuminuria. The 3 cardiac tests include an electrocardiogram, left ventricular ultrasonography for left ventricular volume and mass, and determination of blood N-terminal pro-B-type natriuretic peptide level. Each of the 10 tests is scored as follows: 0 for normal, 1 for borderline abnormal, and 2 for abnormal. The Rasmussen score, therefore, ranges from 0 to 20. Screening of 1,500 individuals has demonstrated that one-third of this population had a Rasmussen score between 0 and 2 (low disease risk), one-third had between 3 and 5, and one-third had 6 and above, which is considered high risk. This screening approach may identify vascular and cardiac abnormalities in asymptomatic individuals who have low FRS scores or even no traditional CV risk factors.

Given the potential limitations of the FRS and the appeal of CV screening with common tests such as vascular ultrasound for intima-media thickness and CAC testing, why hasn’t routine screening for subclinical CVD not become routine practice? Greenland and Lloyd-Jones argue that screening for subclinical CVD among asymptomatic individuals has not been recommended in clinical guidelines because evidence that these screening tests confer a benefit is lacking. They acknowledged that recent reports from Multi-Ethnic Study of Atherosclerosis demonstrated convincingly that CAC testing was predictive of coronary events across ethnic groups and that increased CAC scores were predictive over and above traditional risk factors in both men and women. Although CAC seems to be a promising method for early detection of CVD, sensitivity and specificity were relatively poor, depending on the chosen cutoff for a positive test. In addition, an ongoing study is comparing the Rasmussen score with the classical FRS estimate in the prediction of long-term CV events. However, at present, there is no evidence that this type of screening will improve outcomes over and above treating known risk factors. Furthermore, there also may be risk of harm related to screening with these tests. The risks may include false reassurance due to a test with low sensitivity or anxiety due to a false positive test. The added burden of follow-up invasive angiography, which might or might not be necessary, observed after coronary calcium scoring offers a lesson that screening for vascular disease is not without obvious potential hazards. Greenland and Lloyd-Jones also identify substantial difficulties associated with broad application of these newer technologies in clinical practice, including technician dependence of scanning, reader error and bias, and lack of agreement on the most appropriate parameters to be measured, best instruments to use, and frequency for testing.

The United States Preventive Services Task Force clinical guidelines strongly recommend the routine screening and treatment of traditional CV risk factors to eligible patients on the basis of solid evidence that these services improve important health outcomes and the benefits of such tests substantially outweigh the harms. However, because of the limited supportive evidence, the United States Preventive Services Task Force clinical guidelines recommend against routine screening with resting electrocardiography, exercise treadmill test, or rapid computed tomography scanning for coronary calcium for the prediction of CV events in adults at low risk for CVD events (ie, screening of the general asymptomatic population). The debate regarding which approach is ultimately the most
effective in preventing CV events is ongoing and unlikely to be resolved in the short term. A growing body of evidence supports CV risk factor assessment and intervention as an effective approach to preventing CV events. However, current methods for multiple risk factor assessment are limited. Preliminary data on screening for subclinical CVD to prevent CV events offer promise. Nevertheless, additional studies of screening strategies including CV and cost outcomes are needed before routine CVD screening for the asymptomatic individual can be recommended and incorporated into clinical guidelines.

REFERENCES


OVERVIEW OF CORONARY ARTERY DISEASE (CAD)

One person around the world will experience a coronary artery disease event every 25 seconds (Overbaugh, 2009). NZ Ministry of Health statistics (2003) indicate similar trends that cardiovascular disease is the leading cause of death in NZ, accounting for 40% of all deaths. They suggest the prevalence of cardiovascular disease is greater in Maori, Pacific Islanders, and those living in lower socio-economic areas. The ‘outcome gap’ between Maori and non-Maori is widening (NZGG, 2003).

Emphasis is being placed on primary prevention and specifically reducing modifiable risk factors such as high blood pressure, smoking, raised cholesterol, diabetes, obesity, and inactivity. Factors that lead to acute coronary syndrome such as cardiovascular disease can be reduced through lifestyle change and appropriate drug therapy. People with known cardiovascular disease and those at high risk because of diabetes with renal disease, or some genetic lipid disorders, are clinically defined at very high risk.

Despite progression within cardiology, to reduce heart disease, lifestyle modification remains the most significant factor in the prevention of the development and progression of cardiac disease and in reducing the likelihood of a further cardiac event. For this reason it is essential that nurses caring for cardiology patients have a thorough understanding of the pathophysiology of the disease and are conversant with the recommendations of evidenced-based guidelines that have been developed by the Cardiac Rehabilitation New Zealand guidelines group, (2002). The recommendations developed by the Cardiac Rehabilitation New Zealand guidelines group have formed the basis of the information provided.
The Cardiology Department at Christchurch Hospital is fortunate to have Cardiac Rehabilitation Nurse Specialists, but with the vast amount of education patients require it is essential that all nurses working within the department play an active role in patient education. A teaching session doesn’t need to be long; in fact it is better to be of a short duration and the cardiac rehabilitation checklist for patient education may be used as a guide for patient teaching. Nurses play a significant role in detecting patients at risk for ACS, facilitating their diagnosis and treatment, and providing education that can promote optimal outcomes.

There are many teachable moments that confront nurses every day and it is important to identify them and make the most of them, e.g. when you are showering a patient or making their bed assess their understanding of the angina action plan and/or discuss the use of GTN spray, when to see their doctor or when to call an ambulance following discharge. These teaching moments not only supports the patient but assist nurses in developing a holistic view of our patients, allowing us to provide appropriate care.

It is important to know if the patient knows their own risk factor(s), knows the benefits of modifying these risk factors and has a plan and the support necessary to make changes. This information along with stressing the importance of not running out of medications or stopping them without advice from their doctor is of prime importance and should be restated prior to discharge. Also, remember to take opportunities to include their family/whanau and/or significant others in education sessions.

**WHAT IS CORONARY ARTERY DISEASE**

The heart is a strong muscular pump that is responsible for moving about 11000 litres of blood through the body every day. Like other muscles, the heart requires a continuous supply of blood to work properly. The heart muscle (myocardium) gets the blood it needs to do its job from the coronary arteries. Coronary artery disease is the narrowing or blockage of the coronary arteries, usually caused by atherosclerosis.

Atherosclerosis (sometimes called “hardening” or “clogging” of the arteries) is the build-up of cholesterol and fatty deposits (called plaques) on the inner walls of the arteries. These
plaques can restrict blood flow to the heart muscle by physically clogging the artery or by causing abnormal artery tone and function.

Without an adequate blood supply, the heart becomes starved of oxygen and the vital nutrients it needs to work properly. This can cause chest pain called angina. If blood supply to a portion of the heart muscle is cut off entirely, or if the energy demands of the heart become much greater than its blood supply, a heart attack (injury to the heart muscle) may occur.

**WHAT CAUSES THE CORONARY ARTERIES TO NARROW**

The coronary arteries are shaped like hollow tubes through which blood can flow freely. The muscular walls of the coronary arteries are normally smooth and elastic and are lined with a layer of cells called the endothelium. The endothelium provides a physical barrier between the blood stream and the coronary artery walls, while regulating the function of the artery by releasing chemical signals in response to various stimuli.

Coronary artery disease starts at a young age. Before the teen years, the blood vessel walls begin to show streaks of fat. As we get older, the fat builds up, causing slight injury to the blood vessel walls. Other substances travelling through the blood stream, such as inflammatory cells, cellular waste products, proteins and calcium begin to stick to the vessel walls. The fat and other substances combine to form a material called plaque.

Over time, the inside of the arteries develop plaques of different sizes. Many of the plaque deposits are soft on the inside with a hard fibrous “cap” covering the outside. If the hard surface cracks or tears, the soft, fatty inside is exposed. Platelets (disc-shaped particles in the blood that aid clotting) come to the area, and blood clots form around the plaque.
The endothelium can also become irritated and fail to function properly, causing the muscular artery to squeeze at inappropriate times. This causes the artery to narrow even more.

**CORONARY ARTERIES**

The heart receives its own supply of blood from the coronary arteries. Two major coronary arteries branch off from the aorta near the point where the aorta and the left ventricle meet. These arteries and their branches supply all parts of the heart muscle with blood. The Right Coronary Artery (RCA) branches into the right marginal artery and the posterior descending artery supplying oxygenated blood to the right atrium, right ventricle and the bottom portion of both ventricles and back of the septum.

Whereas the Left Main Coronary Artery (also called the left main trunk) branches into the circumflex artery and the left anterior descending artery (LAD). The circumflex artery, supplies oxygenated blood to the left atrium, side and back of the left ventricle. The left anterior descending artery (LAD), supplies oxygenated blood to the front and bottom of the left ventricle and the front of the septum.

**Collateral circulation** is a network of tiny blood vessels, and under normal conditions, not open. When the coronary arteries narrow to the point that blood flow to the heart muscle is limited (coronary artery disease), collateral vessels may enlarge and become active. This allows blood to flow around the blocked artery to another artery.
nearby or to the same artery past the blockage, protecting the heart tissue from injury.

NORMAL ARTERIES
To understand the process by which atherosclerosis develops, it is necessary to review the composition of the normal artery. Artery walls are constructed of three layers and a hollow internal core called the lumen.

- The internal layer is the tunica interna (intima), and is composed of a lining of endothelium (simple squamous epithelium) that is in contact with the blood and a layer of elastic tissue beneath.
- The middle layer is the tunica media, consisting of elastic fibres and smooth muscle.
- The outer layer is the tunica externa (adventitia), composed principally of elastic and collagenous fibres. Nerves and blood vessels pass through this layer into the media.

CORONARY ARTERY DISEASE - ATHEROSCLEROSIS
(derived from the Greek word for ‘porridge’ or ‘gruel’ (athera), whilst sclerosis infers ‘scar formation’, hence the term hardening of the arteries).

Coronary atherosclerosis or coronary artery disease forms following macroscopic injury to the endothelium of the coronary arteries. This damage to the artery wall allows cholesterol to be deposited within the intima lining. The developing build up is known as an atherosclerotic plaque. Atherosclerotic plaque can be described as “a lesion within the intima of the arterial wall that consists of a lipid pool (atheroma)
surrounded by a fibrous or sclerotic cap, which forms in response to vascular endothelial injury”.

Rather than uniformly thickening arterial walls, atherosclerosis is patchy and unevenly distributed. The specific coronary arteries affected by atherosclerosis vary from person to person, but there is a common feature: within a coronary artery, plaques are found most often at branch points, places where the blood flow naturally becomes turbulent.

The narrowing of coronary arteries usually occurs slowly and, in response, new small collateral arteries have time to grow into the fields of the atherosclerotic arteries to help bolster the local oxygen supply. These collateral arteries will sometimes provide enough extra blood flow to keep the heart muscle working comfortable at a resting rate. The collateral arteries are small, however, and they do not have the capacity to keep up with the oxygen demands of heart muscle during exercise.

Even with the growth of small collateral arteries, the continual narrowing of the coronary arteries by atherosclerosis can eventually produce ischemia and angina pain. Initially, these symptoms occur only when the patient is exercising; later, the symptoms begin to occur even when the patient is at rest.

Besides slowly narrowing the coronary arteries, atherosclerosis can cause a sudden medical crisis. The degeneration of a plaque can seed clots into the bloodstream and can also trigger local vasospasm. These lead to a marked reduction of blood flow, and the resulting damage can range from temporary to permanent and from mild to fatal.

THE PROCESS OF CORONARY ARTERY DISEASE

1. The coronary arteries are shaped like hollow tubes through which blood can flow freely. The walls of the coronary arteries are normally smooth and elastic.
2. Coronary artery disease starts at a young age. Before the teen years, the blood vessel walls begin to show streaks of fat.

3. As we get older, the fat builds up, causing slight injury to the blood vessel walls. To heal the blood vessel walls, the cells release chemicals that make the blood vessel walls stickier. Other substances travelling through your blood stream, such as inflammatory cells, cellular waste products, proteins and calcium, begin to stick to the vessel walls. The fat and other substances combine to form a material called plaque.

4. Over time, with coronary artery disease, the inside of the arteries develop plaques of different sizes. Many of the plaque deposits are soft on the inside with a hard fibrous “cap” covering the outside. If the hard surface cracks or tears, the soft, fatty inside is exposed. Platelets (disc-shaped particles in the blood that aid clotting) come to the area, and blood clots form around the plaque.

5. This causes the artery to narrow even more. Sometimes, the blood clot breaks apart, and blood supply is restored.

6. In other cases, the blood clot may totally block the blood supply to the heart muscle, called a coronary thrombus or coronary occlusion - causing an acute coronary syndrome.

The cause of atherosclerosis isn't known. However, certain traits, conditions, or habits may raise the risk for the disease. These conditions are known as risk factors. You can control
some risk factors, such as lack of physical activity, smoking, and an unhealthy diet. Others you can't control, such as age and a family history of heart disease. Some people who have atherosclerosis have no signs or symptoms. They may not be diagnosed until after a heart attack or stroke.

The main treatment for atherosclerosis is lifestyle changes. Patients also may need medicines and medical procedures. These treatments, along with ongoing medical care, can help them to live a healthier life. In patients with occlusive coronary artery disease, severe atherosclerosis eventually completely occludes the coronary artery causing the heart muscles supplied by the artery to die.

This clinical event is known as myocardial infarction; more commonly called the heart attack, and is accompanied by prolonged chest pain, nausea, sweating, shortness of breath, and weakness. The arterial occlusion and subsequent tissue death may occur silently and without symptoms. Although atherosclerosis occurs in the general population, some people are at greater risk for developing coronary artery disease. Epidemiological studies have identified several cardiac disease risk factors. These risk factors can be classified as either modifiable or non-modifiable.

**RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF CORONARY ARTERY DISEASE**

**NON-MODIFIABLE RISK FACTORS**

**Age and gender:**

Coronary artery disease is more likely to occur as you get older, especially after Age 65 and men have a greater risk of heart attack than women do. Men have heart attacks earlier in life than women. However, beginning at Age 70, the risk is equal for men and women.

**Genetic or familial predisposition:**

You have an increased risk of developing heart disease if you have a parent with a history of heart disease, especially if they were diagnosed before Age 50. Ask your doctor when it’s
appropriate for you to start screenings for heart disease so it can be detected and treated early.

Race:

Indigenous populations have more severe high blood pressure than Caucasians and, therefore, have a higher risk of heart disease. The risk of heart disease is also higher among. This is partly due to higher rates of obesity and diabetes in these populations.

MODIFIABLE RISK FACTORS

Smoking:

Evidence that smoking is responsible for atheroma formation is overwhelming, McBride (1992). The mechanism by which smoking causes this damage is primarily by endothelial dysfunction. Smoking elevates fibrinogen levels which is implicated in the clotting process, promotes vasoconstriction by increasing vascular tone and reduces the protective effects of HDL.

Positive effects of quitting smoking for those with CAD include: reducing the risk of a recurrent myocardial infarction by 50%. Nicotine replacement therapy increases smoking cessation by 1.5 – 2x compared to placebo, regardless of setting.

Second hand smoke is a significant risk factor for non-smokers and should be avoided. It is beneficial to stop smoking at any age but the earlier it is stopped, the greater the health gain. Benefits of quitting are almost immediate as:

- Within 1 day of quitting, the chances of an MI decreases
- Within 2 days smell and taste are enhanced
- The risk of MI is reduced by 50% after 1 year of quitting
- The risk of a major coronary event reduces to the level of a never smoker within 5 years
- Hypertension
Blood lipids:

This refers to cholesterol and triglycerides found in the plasma. High Density Lipoproteins (HDL) and Low Density Lipoproteins (LDL) differ in molecule size. HDL are the smallest molecules that are able to enter and leave the arterial wall and are thought to remove LDL cholesterol from the artery wall and transport it to the liver. Low HDL levels are associated with increased risk of developing atherosclerosis.

The goals of diet modification and or lipid therapy are:

- Total cholesterol < 4.0 mmol
- LDL cholesterol < 2.5 mmol
- Triglycerides < 1.7 mmol
- HDL cholesterol > 1.0 mmol
- TC:HDL ratio 4.5 or less

HDL is lowered by:

- Smoking
- Obesity
- Physical inactivity

LDLs are able to enter the arterial wall, however if these lipoproteins are oxidised by macrophages they are detained within the wall to cause the lipid pools of atherosclerotic plaques. LDL level is increased by the consumption of saturated fats, (animal fats as in red meat, skin on chicken, full fat dairy products etc).

Patients with cardiovascular disease should be encouraged to adopt a cardioprotective dietary pattern, which reduces cardiovascular and total mortality. Modification of dietary fat should not be considered in isolation from a whole diet approach. This dietary pattern
reduces LDL cholesterol but also improves the lipid profile, lowers blood pressure, improves glycaemic control and reduces thrombotic clotting.

**Diabetes:**

Type I and Type II are associated with a markedly increased risk of atherosclerosis. This is possibly due to hyperglycaemia playing a role in acceleration of atheroma formation. Diabetes appears to increase endothelial permeability to calcium and LDL cholesterol and poor glucose control causes abnormalities of lipoprotein metabolism, resulting in an elevation of plasma triglycerides and an enhanced susceptibility to oxidation of LDL cholesterol, which are associated with an increased risk of thrombus formation.

CV risk increases with increasing levels of glucose (e.g. impaired fasting glucose), but overt diabetes confers the greatest risk. Silent ischaemia and infarction are common in diabetic patients. Patients with type 2 diabetes and no evidence of prior MI, have the same prognosis as non-diabetic patients post AMI. The management goal of patients with type 2 diabetes is aggressive CV risk reduction i.e. smoking cessation, tight blood pressure control (<130/80), and aggressive lipid lowering (LDL < 2.0mmol).

**Hypertension:**

Causes damage to the vascular endothelium, allowing the influx of proteins, lipoproteins and cells into the arterial intima and thereby promoting formation of lipid pools and development of atherosclerosis. A raised blood pressure alters shear stress on the endothelium, leading to damage and subsequent platelet deposition. This then reduces vessel lumen size by altering its morphology or shape. Blood pressure control goal for hypertensive patients is <120-130 / 80-90, or lower if the patient is diabetic.
Lifestyle modification interventions should be promoted such as exercise, weight reduction if overweight, no added salt diet, and compliance with antihypertensive medication. Patients need to be reminded that anti-hypertensive medication is not a cure and that medications should only be stopped or withheld on medical advice.

**Physical Activity:**

The goal is to achieve at least 30 minutes exercise on most days of the week. A gradual increase to this level or beyond is recommended. For those with known coronary disease vigorous exercise is not recommended but the benefits of regular moderate physical activity overall considerably outweighs any risk of sudden death. As many patients are taking beta-blockers, the standard guide to exercise intensity of correlation between age and heart rate is not applicable.

Patients are instead encouraged to exercise at a perceived level of exertion, i.e. initially exercise at a level that they perceive as gentle to moderate, gradually building over days to weeks to a level the patient perceives as moderate to hard. Not vigorous! All other recommendations associated with exercise apply, including warming up and cooling down.

**Obesity:**

Defined as a body mass index (BMI) of >30. About one in six New Zealanders are obese. Obesity is associated with a two-three fold increase in the risk of coronary heart disease, and it also increases risk of hypertension, raised LDL, and causes glucose intolerance, particularly in the presence of central obesity (waist to hip circumference ratio; apple rather than pear shaped).

Conversely a moderate weight reduction will improve blood lipids, blood pressure and blood glucose control and the initial goal of weight loss should be to reduce the patient’s weight by 10% by encouraging exercise and nutrition goals.
Popular high protein weight loss diets are not recommended for long term weight loss because they restrict consumption of healthy foods and do not provide the variety of foods needed to meet nutritional needs. For overweight or obese patients with coronary heart disease, the combination of a reduced-energy diet and increased physical activity is recommended.

**Stress and Psychosocial Issues:**

Depression is associated with a five-fold increase in mortality at six months and a three-fold increase in one year mortality. Major depression following a cardiac event runs a long-term course with the majority of those affected remaining depressed at one year. This situation is vastly different from a few “down days”, and should be treated as a serious illness requiring prompt medical attention.

Marital status, emotional and social support, and social networks are likely to have a protective effect and reduce risk of future fatal and non-fatal coronary heart disease and total mortality. Up to one in four patients will experience a disabling level of anxiety or depression following a myocardial infarction.

**Alcohol:**

A small amount of alcohol may provide health benefits. The protective effect of alcohol is seen at doses as low as one standard drink every second day. Generally patients are advised not to begin drinking alcohol if they don’t normally drink.

**Raised Homocysteine level:**

Homocysteine is an amino acid produced within the body and is a by-product of the metabolism of the dietary protein methionine contained in red meat. Levels are determined by a
blood test and the normal level is considered to be <10 mmols/L. Elevated homocysteine levels are commonly present in men and post-menopausal women, particularly those with coronary artery disease, and are more common with advancing age and in the presence of lower blood folate levels and vitamin B6 & B12 deficiency. A cardio-protective diet is thought to provide adequate folate and B vitamins to control homocysteine levels.

**Infection & Inflammation:**

The inflammatory response is of such significance in the formation of plaque and its vulnerability to rupture that this has led to the consideration of chronic bacterial and viral infections as potential initiating factors. Inflammation and infection as potential causal factors may help explain why some populations without genetic predisposition or other traditional risk factors such as hypercholesterolaemia develop atherosclerosis and its manifestations.

Potential infectious causes are: Helicobacter pylori, Chlamydia pneumonia, Cytomegalovirus Herpes viruses, and Enterovirus.

**Cumulative Risk:**

This refers to the fact that the presence of more than one risk factor, more than doubles the risk of coronary artery disease. With three or more risk factors the chances of developing coronary artery disease are tremendously increased but importantly effects of changing one risk factor can have the following consequences:

- Quitting smoking reduces the risk of a serious cardiac event (including death), by ½ to 2/3
- Hypertension: if systolic pressure is reduced by 20mmHg, risk is reduced by ½
- Hypercholesterolaemia: LDL reduced by 1mmol/L results in a 1/3 risk reduction

Cumulative risk (if all the above risk factors apply to the patient) and if all are modified, the risk is reduced by 5/6ths.

**Dysfunctional Endothelium**
Once factors such as hypertension or smoking have contributed to a dysfunctional endothelium, this damaged area undergoes changes that activate the inflammatory response. Damaged endothelial cells produce surface adhesion molecules and release chemicals that attract leukocytes. These leukocytes then adhere to the adhesion molecules, while the damaged endothelial cells go on to produce growth factors that enhance the proliferation and differentiation of smooth muscle cells at the site of injury.

Monocytes differentiate into macrophages, which accumulate lipids from the blood into the collagen fibres of connective tissue within the evolving plaque. This process tends to be cyclic and results in the development of a large lipid core.
REFERENCES


Module 4: Development of Coronary Artery Disease and Atherosclerosis

Questions

1. Define Coronary artery disease: 
   
   
   
   
   
   

2. What causes Coronary artery disease?
   
   
   
   
   
   
   

3. What are 2 non modifiable risk factors?
   
   
   
   

4. What are 4 modifiable risk factors?
5. How does Hypertension cause damage to the vascular endothelium?

6. Case Scenario:

A fifty-year-old man was admitted into a cardiac ward for investigation of ongoing chest pain. On admission he appeared to be healthy, but complains of experiencing occasional chest pain. He said the chest discomfort was accompanied with feeling of dizziness, shortness of breath and tingling in fingers. It usually occurred during an evening walk, which he took after dinner. The chest ache lasted for a very short time and disappeared if he stopped walking for a short while. He also mentioned that he had started taking the short evening walks recently on the advice of a friend.

Family History:

The patient has a family history of coronary artery disease. His father was diagnosed with coronary artery disease at 60 years of age. He subsequently underwent triple bypass surgery. The patient’s paternal grandfather died of a heart attack at the age of sixty.

Social History:

The patient is married. He is employed as a laboratory manager in a leading clinical diagnostic laboratory. Although he enjoys his work, he works long hours and often takes work home with him. He takes few vacations and even on vacation he maintains contact
with his laboratory. His wife accuses him of being a chronic workaholic who is always under considerable stress.

**Physical Exam:**

The patient was of moderate build and appeared healthy and well hydrated. His height was 5' 5" and his weight was 86kg/190lb. His BMI was 31.

**Vital signs:**
Temperature: 98.7°F
BP: 140/80mm Hg
Pulse: 80 beats/min

Q. From the information provided, what is your assessment of the patient so far?

Q. As this patient’s nurse what would you consider a priority to manage first?

Q. What key points should identify from the information provided and discussed with the patient?

Q. From the information provided by the patient, what would you consider to be significant that could place the patient at high risk of coronary artery disease?
Q. It hasn’t been determined that the cause of the patient’s pain is cardiac at this time, so therefore what would you consider as other cause. What would your differential diagnoses be?

Q. What tests would you expect to be ordered to determine if it is cardiac related?

Diagnosis

The patient underwent an electrocardiogram (ECG) and an exercise treadmill test (ETT). Both tests were within normal limits. He had non-significant S-T depression and no chest pain. The lab test are all normal except for lipids.

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>3.5mmol/L</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.7mmol/L</td>
</tr>
<tr>
<td>Cholesterol (HDL)</td>
<td>0.98mmol/L</td>
</tr>
<tr>
<td>Cholesterol (Total/HDL)</td>
<td>6.1mmol/L</td>
</tr>
<tr>
<td>Cholesterol (LDL)</td>
<td>3.3</td>
</tr>
</tbody>
</table>
Q. What are the optimal level of lipid results and would you consider there is any concern with the results that would need to be discussed with the patient?

Q. Would the above results and the patient’s history suggest he is at a high risk of atherosclerosis?

Management Plan

Q. What suggestion would you make to the patient's to manage his condition?
EVALUATION FORM

Topic: Module 4: Development of Coronary Artery Disease and Atherosclerosis

We want to ensure that the training/education you have received is effective and relevant. We would be grateful if you would complete this evaluation. Please circle the most appropriate rating.

(The response range from 1 for limited use, to 5 for very useful)

Circle your choice

1. Please rate the overall value of the Self Learning Package

   1 2 3 4 5

   Comments:__________________________________________________________
   ___________________________________________________________________

2. Please rate how relevant the information was to your position

   1 2 3 4 5

   Comments:__________________________________________________________
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3. Please rate the presentation of the Self Learning Package

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Please add any further comments you consider would improve the programme.

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