

REVIEW ARTICLE

A Study of Cardiovascular Diseases: Types and Risk FactorRavi *¹, Vivek Prakash ², Ram Singh ²

1. College of Pharmacy, Teerthanker Mahaveer University, Moradabad
2. Hygia Institute of Pharmaceutical Education and Research, Lucknow

ABSTRACT

Coronary heart disease (CHD) is the leading cause of death in India and the leading cause of death worldwide. Previously thought to affect primarily high-income countries, CHD now leads to more death and disability in low- and middle-income countries, such as India, with rates that are increasing disproportionately compared to high-income countries. CHD affects people at younger ages in low- and middle-income countries, compared to high-income countries, thereby having a greater economic impact on low- and middle-income countries. Effective screening, evaluation, and management strategies for CHD are well established in high-income countries, but these strategies have not been fully implemented in India.

Keywords: Coronary heart disease (CHD), Atherosclerosis, Heart attack, Cardiomyopathy, Heart failure, Stroke.

INTRODUCTION

Modern medicine, as we know it today grew in prominence through several important landmark events over the last 80 years, such as the discovery of Penicillin in 1941 and Cortisone in 1949. From the demands for the manufacture of these drugs grew the pharmaceutical industry, which over the course of the last century grew to become a large

and powerful industry, focusing much of its work on the development and supply of a constant stream of new drugs for use in modern medicine. By the 1960s over seventy new drugs were being introduced every year but in the 1970s that had reduced to less than 20 a year due to the introduction of legislation requiring much stricter means for testing drugs, due in some part to the serious side-effects of some drugs after being released to the public. In the 1990 drug companies began to focus their attention on techniques for screening millions of chemical compounds for their

Address for correspondence:

Ravi*

College of Pharmacy, Teerthanker Mahaveer University, Moradabad-244001

E-mail: ravi_ra.vns@rediffmail.com

biological activity, hoping to identify the lead components that might have the sort of genuine therapeutic effect that could form the basis of new drugs.^{1,2}

Cardiovascular disease is a complex and multi factorial disease and is characterized by multiple factors. Epidemiologic studies have identified these as elevated serum lipids (cholesterol and triglycerides), increased plasma fibrinogen and coagulation factors, increased platelet activation, alterations in glucose metabolism, and smoking.³ The oxidative modification of LDL by reactive oxygen species (ROS) is also now considered an important mechanism in the development of atherosclerosis, as is the pathogenesis of hypertension.^{4,5} There is also considerable evidence supporting the involvement of platelets in the development of atherosclerosis. Increased platelet

activity has been found in smokers as well as in patients suffering from vascular injury, hyperlipidemia, and hypertension. Increased HDL levels are negatively correlated with cardiovascular disease.

Cardiovascular disease (CVD) includes heart disease (i.e., myocardial infarction and angina), stroke, hypertension, congestive heart failure (CHF), hardening of the arteries, and other circulatory system diseases. CVD is the number one cause of death in America, responsible for more than 40% of annual deaths. An average of 1 death due to CVD occurs every 33 seconds in the United States. CVDs are responsible for over 17.3 million deaths per year and are the leading causes of death in the world.

Distribution of major causes of death including CVDs is shown in Figure 1 below.

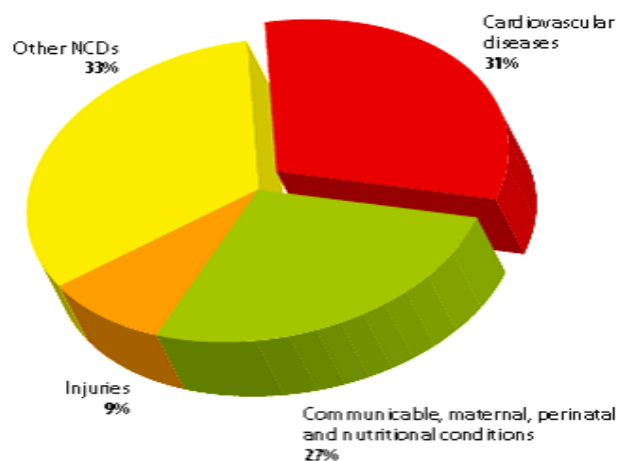


Figure 1: Distribution of major causes of death including CVDs

The different types of CVDs are listed below.

1. CVDs due to atherosclerosis:

- ❖ Ischaemic heart disease or coronary artery disease (e.g. heart attack)
- ❖ Cerebrovascular disease (e.g. stroke)
- ❖ Diseases of the aorta and arteries, including hypertension and
- ❖ Peripheral vascular disease.

2. Other CVDs:

- ❖ Congenital heart disease
- ❖ Rheumatic heart disease
- ❖ Cardiomyopathies
- ❖ Cardiac arrhythmias.

A major contributor to CHD is atherosclerosis, a degenerative and inflammatory syndrome promoting the vascular accumulation of cholesterol and cellular waste products that remodel peripheral blood vessels and impair coronary vessel functioning and blood flow dynamics.⁶⁻⁸

Psychological stress also plays an important role in the development of coronary heart disease (CHD). As a slowly developing chronic illness, CHD results chiefly from a progressive narrowing of the blood vessels that supply oxygenated blood to the heart.⁹⁻¹²

Cardiovascular Disease statistics:

Cardiovascular disease refers to all diseases of the heart and blood vessels. It includes coronary heart disease, stroke, other vascular disease and heart failure. The two major clinical forms of coronary heart disease are heart attack and angina.¹³ More than half of the deaths in the USA and New Zealand are due to coronary Cardiovascular Disease or “hardening of the arteries” resulting in heart attacks and what is called SCD, or sudden cardiac death (discussed in more detail below). Another almost 20% is from the stroke.¹⁴ The third most common cause of death, congestive heart failure (pump failure) has a number of causes, but in 75% of the cases it is a consequence of extensive heart damage following a heart attack. Thus nearly 80% of deaths from cardiovascular disease come from diseases that take years to become clinically apparent and are caused by the development of atherosclerotic plaque in the heart and its arteries (coronary arteries) or in the arteries that supply blood to the brain.¹⁵

Cardiovascular disease is a complex and multifactorial disease and is characterized by multiple factors. Epidemiologic studies have identified these as elevated serum lipids (cholesterol and triglycerides), increased

The **prevalence** (frequency) of cardiovascular (CV) plasma fibrinogen and coagulation factors,

increased platelet activation, alterations in glucose metabolism, and smoking.¹⁶

Frequency of cardiovascular diseases related to age and gender:

The **prevalence** (frequency) of cardiovascular (CV) diseases in the US is shown in the figure 2 below.

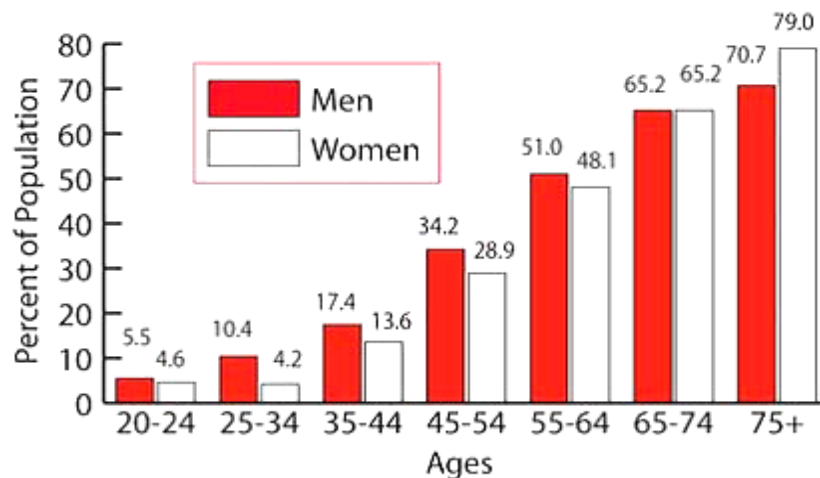


Figure 2: Prevalence of CV Diseases in Men and Women 20 years of age and older—source AHA

In women the prevalence is about 5% at age 20 and increases also to 80% by age 75. However, there is a distinct prevalence of CV Diseases in Men and differences between men and women in the years. The frequency in women remains generally lower than in men until around age 50 (the timing of the onset of menopause) and then accelerate to equal that of men. Between the ages of 25 and 55, the prevalence of CV disease in women, which is mainly Cardiovascular Disease related, is roughly ten years behind that of men of the same age. Coronary Cardiovascular Disease rates in women

As you can see, CV diseases generally uncommon in the very young and the statistics begin around age 20. The frequency increases dramatically with age. In men, the prevalence is only about 6% at age 20 and increases to 80% by age 75.

after menopause are 2-3 times those of women the same age before menopause. Cardiovascular Disease and general atherosclerotic disease (the most common causes of CV disease deaths) increases in frequency as we get older. Symptomatic atherosclerotic disease takes years to develop, although data in heart transplant donors (who died mostly from accidents or suicide) have shown that 25% of teenagers already have developed some atherosclerotic plaque in their coronary arteries. About 600,000 people die of heart disease in the United States every year—that's

1 in every 4 deaths.^{17, 18} The lifetime risk of developing coronary Cardiovascular Disease after age 40 is 49% for men and 32% for women. Coronary Cardiovascular Disease caused more than 1 of every 5 deaths in the United States in 2001. Coronary Cardiovascular Disease is the leading cause of premature, permanent disability in the US labour force, accounting for 19% of disability allowances by the Social Security Administration.¹⁹

Depending on their age and clinical outcome, people who survive a first heart attack have a risk of hospitalization or death that is 1.5 to 15 times higher than that of the general population.

Within 6 years of a first heart attack:

- 18% of men and 35% of women will have a second heart attack
- 7% of men and 6% of women will experience sudden cardiac death
- 22% of men and 46% of women will be disabled with heart failure
- 8% of men and 11% of women will have a stroke²⁰

Cardiovascular Disease from a Medical Perspective:

Cardiovascular disease refers to many different types of heart or blood vessel problems. It is used

most often to describe damage caused to the heart or blood vessels by atherosclerosis. This is a disease that affects the arteries. Arteries are blood vessels that carry oxygen and nutrients from your heart to the rest of the body. Healthy arteries are flexible, strong and elastic.

Over time, however, too much pressure in the arteries can make the walls thick and stiff sometimes restricting blood flow to the organs and tissues. This process is called atherosclerosis, or hardening of the arteries, and atherosclerosis is the most common form of this disorder.

The ultimate causes of atherosclerosis are an unhealthy diet (lots of saturated fats), lack of exercise, being overweight and smoking. All of these are major risk factors for developing atherosclerosis and, in turn, cardiovascular disease. Some forms of cardiovascular disease are not caused by atherosclerosis. Those forms include diseases such as a congenital Cardiovascular Disease, heart valve diseases, heart infections or disease of the heart muscle called cardiomyopathy.

In terms of attributable deaths, the leading cardiovascular risk factor globally is raised blood pressure (to which 13% of global deaths is attributed), followed by tobacco use (9%), raised blood glucose (6%), physical inactivity (6%) and overweight and obesity (5%).²¹

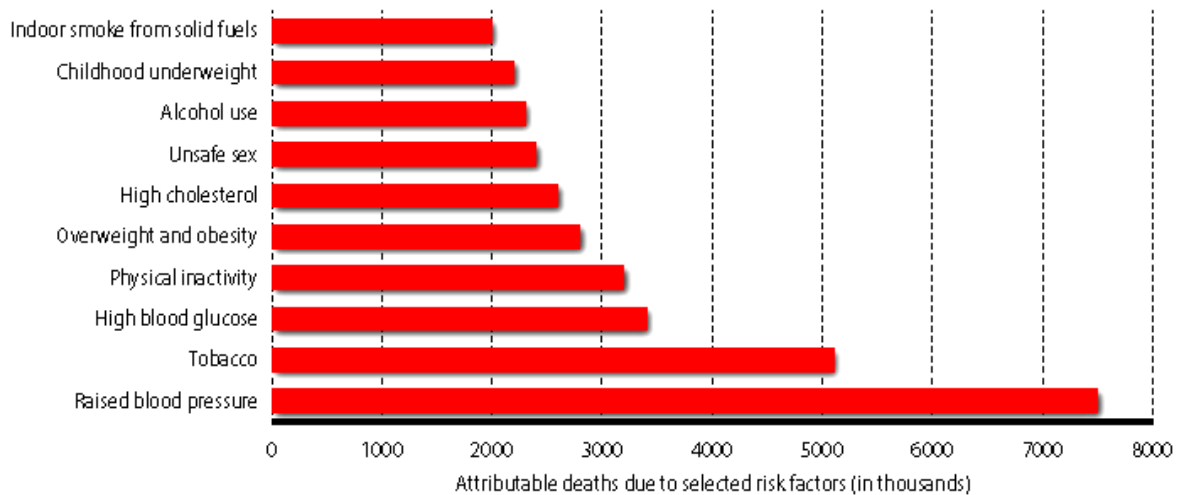


Figure 3: Ranking of 10 selected risk factors of cause of death

Types of cardiovascular disease:

The cardiovascular system consists of the heart and all blood vessels throughout the body. Diseases ranging from aneurysms to valve disease are types of cardiovascular disease. A person may be born with some types of cardiovascular disease (congenital) or acquire others later on, usually from a lifetime of unhealthy habits, such as smoking, which can damage the arteries and cause atherosclerosis.

Some specific terms used to describe the various forms of cardiovascular disease are:

Coronary artery disease:

This is a common form of cardiovascular disease. Coronary artery diseases are diseases of the arteries that supply the heart muscle with blood. Sometimes

known as CAD, coronary artery disease is the leading cause of heart attacks. It generally means that blood flow through the coronary arteries has become obstructed, reducing blood flow to the heart muscle. The most common cause of such obstructions is a condition called atherosclerosis, an largely preventable type of vascular disease. Coronary artery disease and the resulting reduced blood flow to the heart muscle can lead to other heart problems, such as chest pain (angina) and heart attack (myocardial infarction).^{22, 23}

Heart attack:

A heart attack is an injury to the heart muscle caused by a loss of blood supply. The medical term for heart attack is "myocardial infarction," often abbreviated MI. A heart attack usually occurs when a blood clot blocks the flow of blood through a

coronary artery — a blood vessel that feeds blood to a part of the heart muscle. Interrupted blood flow to your heart can damage or destroy a part of the heart muscle.^{24, 25}

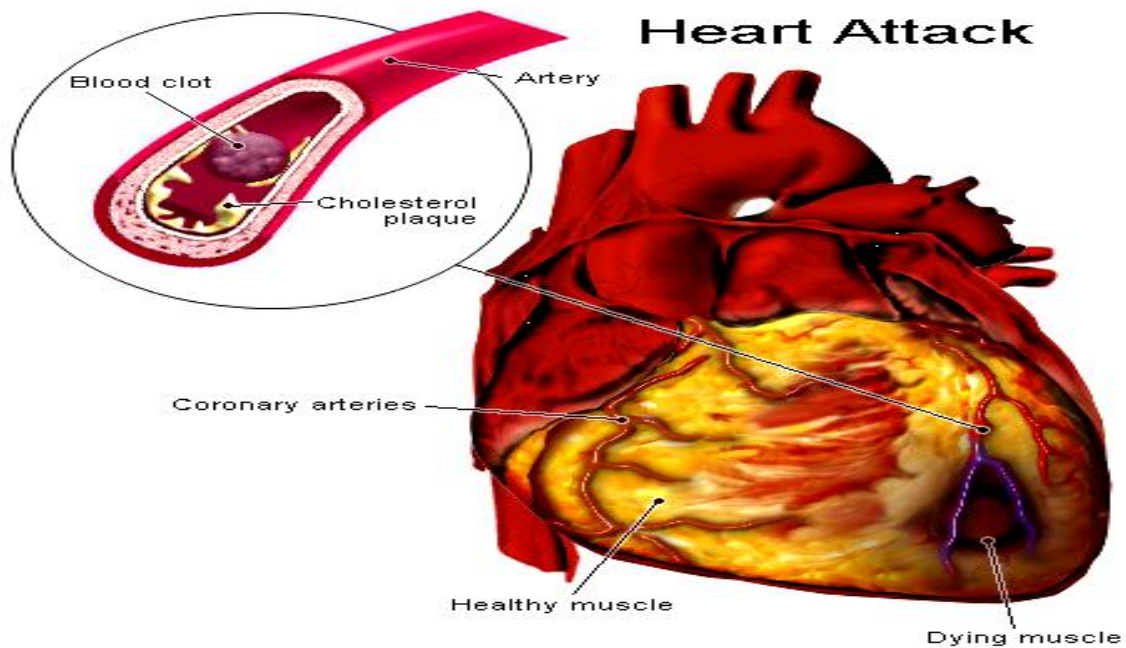


Figure 4: Coronary Artery Disease causing heart attack

Cardiomyopathy:

Cardiomyopathy means diseases of the heart muscle. Some types of cardiomyopathy are genetic, while others occur for reasons that are less well understood. Types of cardiomyopathy include ischemic, which is caused by loss of heart muscle from the reduced coronary blood flow; dilated, which means the heart chambers are enlarged;

hypertrophic, which means the heart muscle is thickened; and idiopathic, which means the cause is unknown. One of the most common types of cardiomyopathy is idiopathic dilated cardiomyopathy — an enlarged heart without a known cause.^{26, 27}

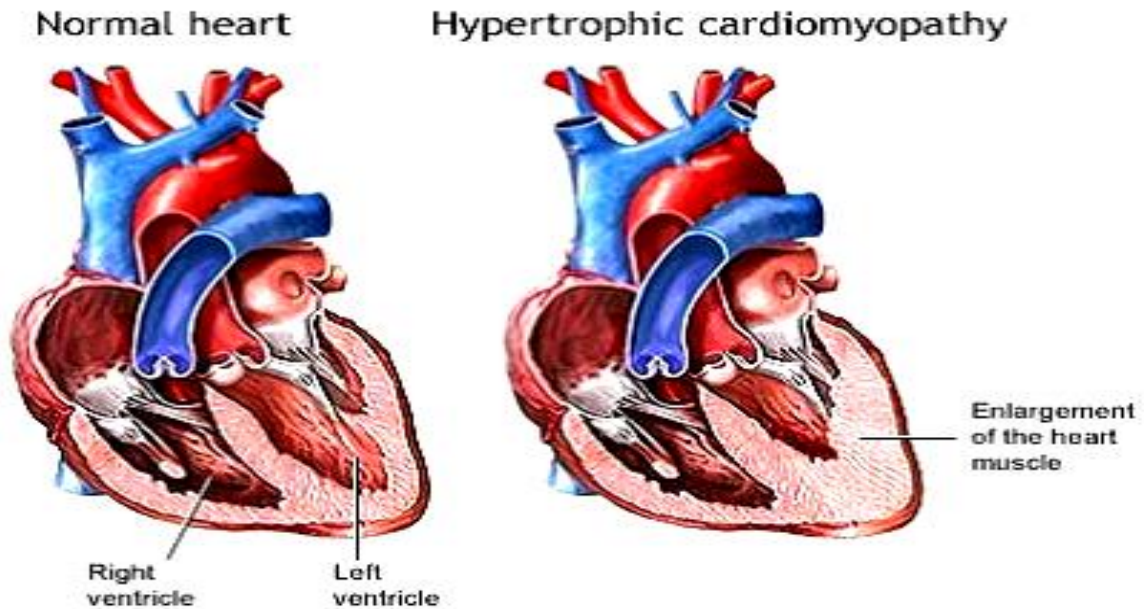


Figure 5: Cardiomyopathy

Congenital heart disease:

Congenital heart disease refers to a form of heart disease that develops before birth (congenital). Congenital heart disease is a broad term and includes a wide range of diseases and conditions. These diseases can affect the formation of the heart muscle or its chambers or valves. They include such conditions as narrowing of a section of the aorta (coarctation) or holes in the heart (atrial or ventricular septal defect). Some congenital heart defects may be apparent at birth, while others may not be detected until later in life.²⁸⁻³⁰

Aneurysm:

An aneurysm is a bulge or weakness in a blood vessel (artery or vein) wall. Aneurysms usually get bigger over time. Because of that, they have the potential to rupture and cause life-threatening bleeding. Aneurysms can occur in arteries in any location in your body. The most common sites include the abdominal aorta and the arteries at the base of the brain.^{31,32}

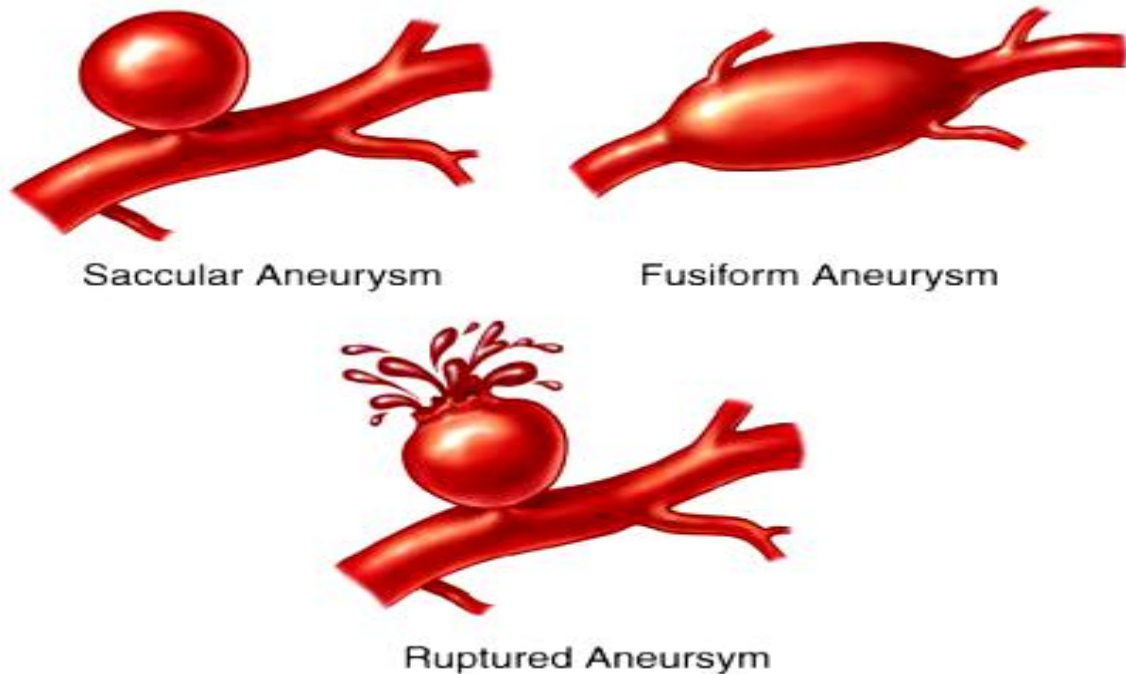


Figure 6: Aneurysms

Valvular heart diseases:

These are diseases of the heart valves. Four valves within your heart keep blood flowing in the right direction. Valves may be damaged by a variety of conditions leading to narrowing (stenosis), leaking (regurgitation or insufficiency) or improper closing (prolapse).

You may be born with valvular disease, or the valves may be damaged by such conditions as rheumatic fever, infections (infectious endocarditis), connective tissue disorders, and

certain medications or radiation treatments for cancer.^{33, 34}

Pericardial diseases:

These are diseases of the sac that encases the heart (pericardium). Pericardial disorders include inflammation (pericarditis), fluid accumulation (pericardial effusion) and stiffness (constrictive pericarditis). These can occur alone or together. The causes of pericardial disease vary, as do the problems they may lead to. For instance, pericarditis can occur after a heart attack and, as a result, lead to pericardial effusion or chest pain.^{35, 36}

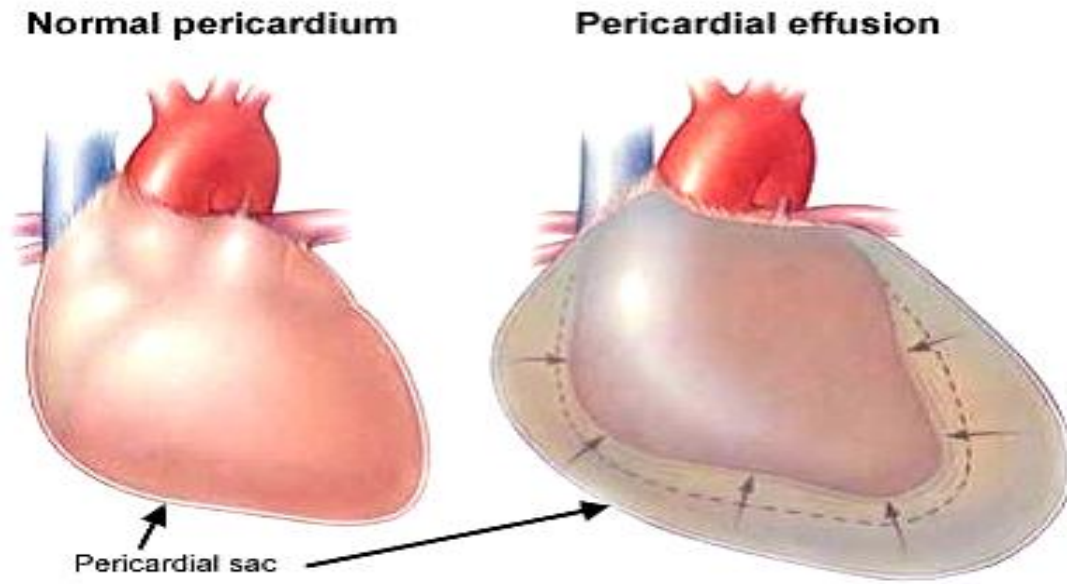


Figure 7: Pericardial effusion

Heart failure:

Heart failure, often called congestive heart failure, is a condition in which the heart can't pump enough blood to meet the needs of your body's organs and tissues. It doesn't mean your heart has failed and can't pump blood at all. With this less effective pumping, vital organs don't get enough blood, causing such signs and symptoms as shortness of breath, fluid retention and fatigue. "Congestive"

heart failure is technically reserved for situations in which heart failure has led to fluid buildup in the body. Not all heart failure is congestive, but the terms are often used interchangeably. Heart failure may develop suddenly or over many years. It may occur as a result of other cardiovascular conditions that have damaged or weakened the heart, such as coronary artery disease or cardiomyopathy.^{37, 38}

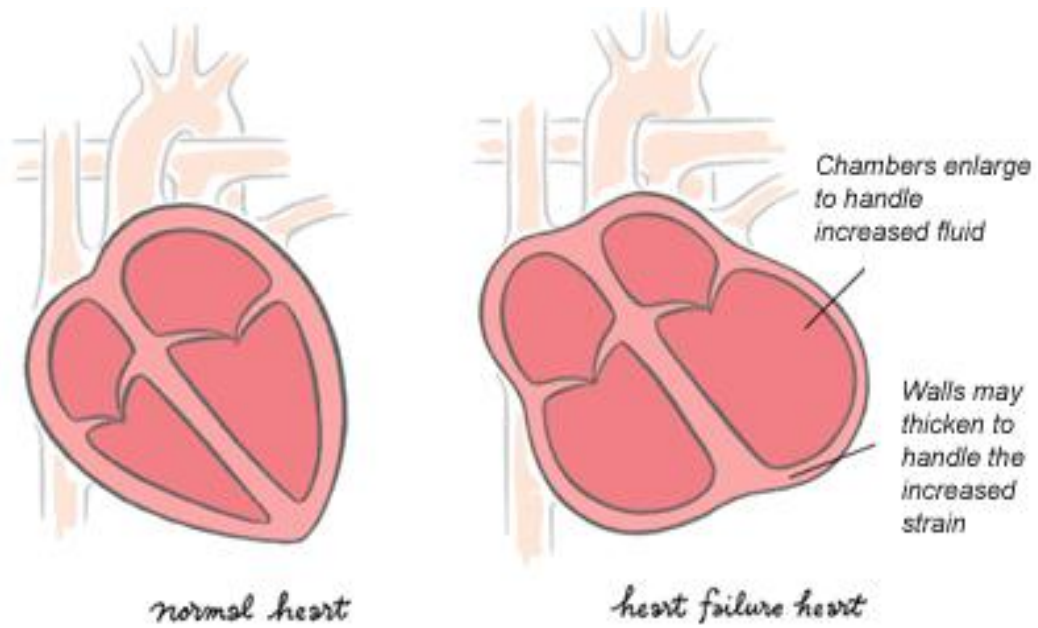


Figure 8: Congestive Heart Failure

High blood pressure:

High blood pressure (hypertension) is the excessive force of blood pumping through your blood vessels. It's perhaps the most common form of cardiovascular disease in the Western world,

affecting about one in four Americans. Although potentially life-threatening, it's one of the most preventable and treatable types of cardiovascular disease. High blood pressure also causes many other types of cardiovascular disease, such as stroke and heart failure.^{39,40}

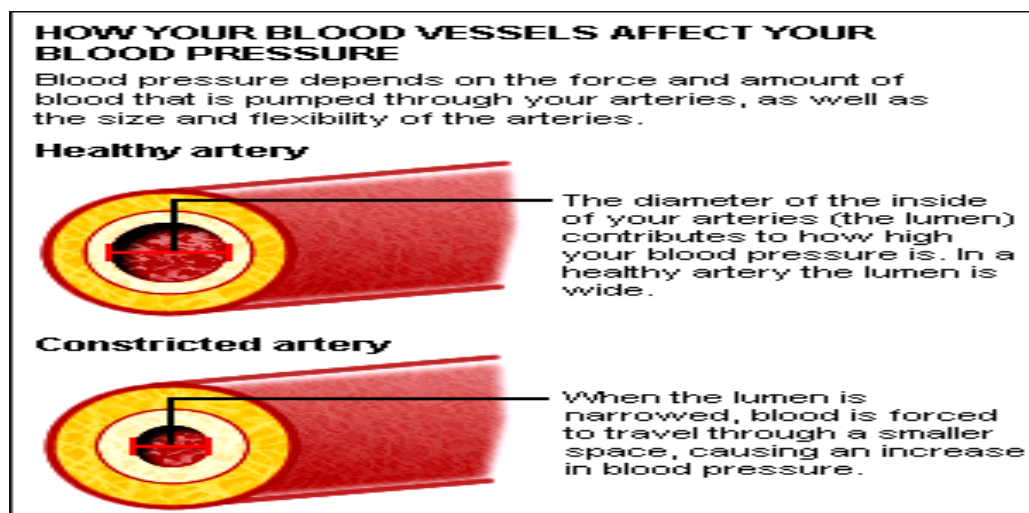


Figure 9: Blood Pressure

Stroke:

A stroke occurs when blood flow to the brain is interrupted (ischemic stroke) or when a blood vessel in the brain ruptures (hemorrhagic stroke). Both can cause the death of brain cells in the affected areas. Stroke is also considered a neurological disorder because of the many complications it causes. Other forms of cardiovascular disease, such as high blood pressure, increase your risk of stroke.^{41,42}

Peripheral arterial disease and claudication:

You may be more familiar with the term "claudication" — which usually refers to pain in your legs during exercise — than you are the term "peripheral arterial disease." Strictly speaking, claudication is a symptom of peripheral arterial disease.

However, claudication is often referred to as a disease itself. Peripheral arterial disease is a disorder in which the arteries supplying blood to your limbs — usually your legs — become narrowed or blocked. When this happens, your legs

Arrhythmias:

Abnormal electrical activity in the heart is known as cardiac arrhythmia; the heartbeat may be too fast or too slow, and may be regular or irregular. It may originate from the region of the atria or ventricles.

receive less blood than they need to keep up with demand. Claudication may then develop. When the obstruction is mild, you may have such symptoms as pain in your legs only during strenuous exercise. As the disease progresses and arteries become more obstructed, you may have pain or cramping in your legs even at rest.^{43,44}

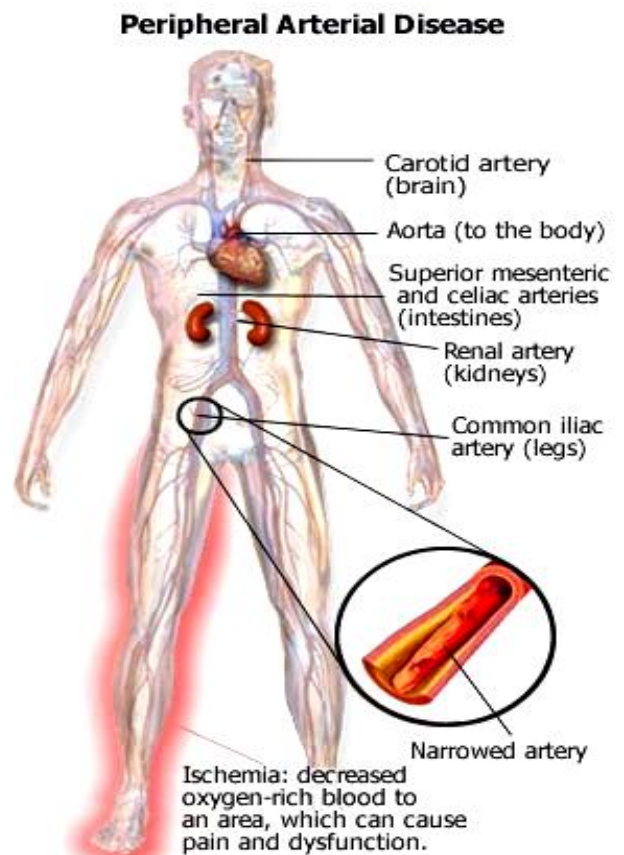


Figure 10: Peripheral arterial disease

Sometimes cardiac arrhythmia is life threatening and causes medical emergencies, sometimes it may not cause symptoms or it may give rise to palpitations. Atrial fibrillation is one of the common tachyarrhythmias arising from the atria. It is characterized by predominantly uncoordinated

atrial activation with consequent deterioration of mechanical function of the heart.⁴⁵

Stable Angina:

Also known as Angina pectoris is chest pain caused by myocardial ischemia. It usually last from 3 to 5 minutes and if the blood flow is restored no permanent change or damage results. It is usually experienced by chest discomfort ranging from a sensation of heaviness or pressure to moderately severe pain. Discomfort may radiate to the neck, lowerjaw, left arm and left shoulder, or occasionally to the back or down the right arm. Discomfort is commonly mistaken for indigestion.

Stable angina is caused by gradual luminal narrowing and hardening of the arterial walls, so that affected vessels cannot dilate in response to increased myocardial demand associated with physical exertion or emotional stress.⁴⁶

Prinzmetal angina:

Prinzmetal angina is chest pain attributed to transient ischemia of the myocardium that occurs unpredictably and almost exclusively at rest. Pain is caused by vasospasm of one or more major coronary arteries with or without associated atherosclerosis. The pain often occurs at night during rapid eye-movement sleep and may have a cyclic pattern of occurrence. The angina may occur from hyperactivity of the sympathetic nervous system, increases calcium flux in arterial smooth

muscle or impaired production or release of prostaglandin or thromboxane.^{47, 48}

Silent Ischemia:

Myocardial ischemia often does not cause detectable symptoms such as angina. Ischemia can be totally asymptomatic which is referred to as silent ischemia. People who do not experience angina often have silent episodes of myocardial ischemia.⁴⁹

Mental Stress induced Ischemia:

Recent study has suggested that an artery is occluded in some individuals duringmental stress. Rozansky documented myocardial ischemia by radionuclide angiography (RNA) during mental stress; the majority of cases (83%) were silent. These observations confirmed in similar studies, suggest that the increases in blood pressure induced by mental stress and the increase in myocardial oxygen demand play a role in the pathophysiology of mental stress-induced myocardial ischemia.

Chronic stress has been linked to a hypercoagulable state that may contribute to acute ischemic events. Stress management has been associated with a significant reduction in CAD events in men.^{50, 51}

Hypertension:

Hypertensive Cardiovascular Disease is generally

1. Left ventricular hypertrophy in the absence of other cardiovascular pathology and
2. A history of hypertension.

Hypertension strongly predisposes to atherosclerosis and so most patients with elevated blood pressure have significant coronary atherosclerosis. The vascular disease increases peripheral resistance and viscosity in the arteries. The heart then must maintain a normal cardiac output against this increased peripheral resistance and can accomplish this only by hypertrophy of myofibres causing cardiac enlargement.

Hypertension is present in over 50% of patients with pheochromocytoma, and may be sustained or paroxysmal. Higher variability of blood pressure has been demonstrated in pheochromocytoma compared to patients with essential hypertension and is associated with a higher incidence of target organ damage. Resolution of hypertension has been reported in about 50% of patients after successful surgical treatment of pheochromocytoma.⁵²

The prevalence of raised blood pressure was highest in the WHO African Region, where it was 46% for males and females combined. The lowest prevalence of raised blood pressure was in the WHO Region of the Americas, with 35% for both males and females.⁵³

Evaluation of Cardiovascular Disease:

This is generally done by taking a complete history, including risk factors, a physical examination, laboratory tests. Judicious use of x-ray, electrocardiography, ultrasonography, nuclear scanning and angiography may be necessary to identify affected vessels, particularly coronary vessels.

Development of Cardiovascular Disease

The medical model:

The factors that cause Cardiovascular Disease are classified into modifiable and nonmodifiable. The nonmodifiable risk factors refer to variables that cannot be altered by persons wishing to decrease their risk of cardiovascular disease. Examples of this are genetic polymorphisms. Numerous types of genetic susceptibilities to CAD have been identified in individuals with a family history of Cardiovascular Disease. Modern medicine looks at several factors which contribute to Cardiovascular Disease.

Atherosclerosis

The word Atherosclerosis is derived from the Greek ather (porridge) and scleros (hardness). It is a form of arteriosclerosis in which soft deposits of intra arterial fat and fibrin on the vessels walls of medium to large arteries harden over time. It is the most common vascular disease, being the main cause of death in approximately 50% to 60% of people in the developed world. It is an

inflammatory process affecting medium and large-sized blood vessels throughout the cardiovascular system.⁵⁴⁻⁵⁶

It is the main cause of ischaemic coronary Cardiovascular Disease, carotid artery stenosis by occlusion or thromboembolism. In turn, this can cause ischaemia in the cerebral arteries, presenting

either as transient ischaemic attack or cerebral infarction. Abdominal aortic atherosclerotic aneurysm, where the main artery in the abdomen becomes abnormally wide with a thin wall, can burst. Peripheral vascular disease (PVD), which causes intermittent claudication or gangrene of the extremities, pain in the legs when walking, can be relieve by rest.

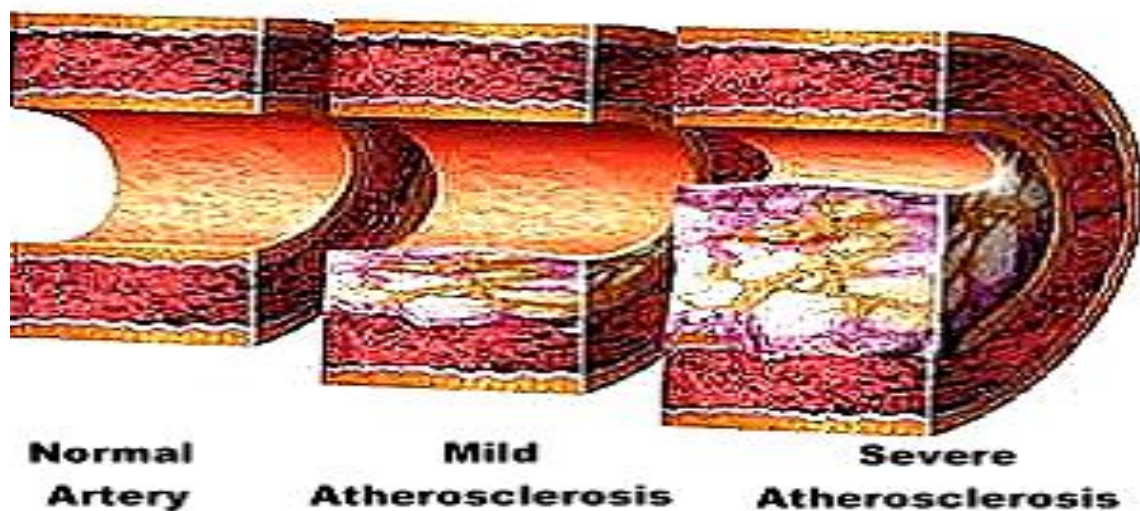


Figure 12: Atherosclerosis

Other Diseases

Heart attack and stroke are very loose terms. "Heart attack" most commonly indicates myocardial infarction. It is sometimes used to denote dysrhythmia.

Coronary thrombosis is typically due to plaque rupture. In this process, the atherosclerotic lesion (abnormal area of arteries) has a thin fibrous lining

that is more mechanically weak, has increased macrophage cells, and has reduced vascular smooth muscle cells (which normally make collagen).

In plaque rupture, the plaque inflammation erodes the fibrous lining (fibrous cap) to the point where it breaks apart (possibly under added hemodynamic stresses), releasing necrotic debris containing tissue factor in the lipid necrotic core into contact with blood. This stimulates clotting that block off the

artery (occlusive thrombus). Unless reopened by drugs or percutaneous coronary intervention the blocked artery causes the heart muscle to die of lack of nutrients and oxygen. This is termed a myocardial infarction.

Risk Factors

Established adult risk factors

Table: Adult Risk Factors

Unmodifiable traits that predict risk		Age	Sex	Race	
Behaviors that affect risk	Smoking	Alcohol consumption	Diet	Psychological Factors	Exercise
Physiological conditions increasing risk		Hypertension	Hypercholesterolemia	Obesity	Diabetes
Cardiovascular endpoints	CHD	MI	Stroke	CHF	Death

Major Risk Factors:

High Blood Pressure (Hypertension). High blood pressure increases the risk of cardiovascular disease, heart attack, and stroke. Though other risk factors can lead to high blood pressure, you can have it without having other risk factors. If obese, smoking, or there is high blood cholesterol levels

Risk factors from a medical perspective:

The major modifiable atherosclerotic risk factors include smoking, hypertension, hyperlipidaemia, diabetes and obesity. Obesity is a mild independent risk factor and very common, and contributes to development of hypertension, hyperlipidaemia and diabetes.

along with high blood pressure, the risk of cardiovascular disease or stroke greatly increases.

Blood pressure can vary with activity and with age, but a healthy adult who is resting generally has a systolic pressure reading between 120 and 130 and a diastolic pressure reading between 80 and 90 (or below).

High Blood Cholesterol:

One of the major risk factors for cardiovascular disease is high blood cholesterol. Cholesterol, a fat-like substance carried in your blood, is found in all of the body's cells. The liver produces all of the cholesterol which the body needs to form cell membranes and to make certain hormones. Extra cholesterol enters the body when food is eaten, that

comes from animals (meats, eggs, and dairy products).

Although the cholesterol found in foods is blamed for raising blood cholesterol, the main culprit is the saturated fat in food. Foods rich in saturated fat include butter fat in milk products, fat from red meat, and tropical oils such as coconut oil.^{57, 58}

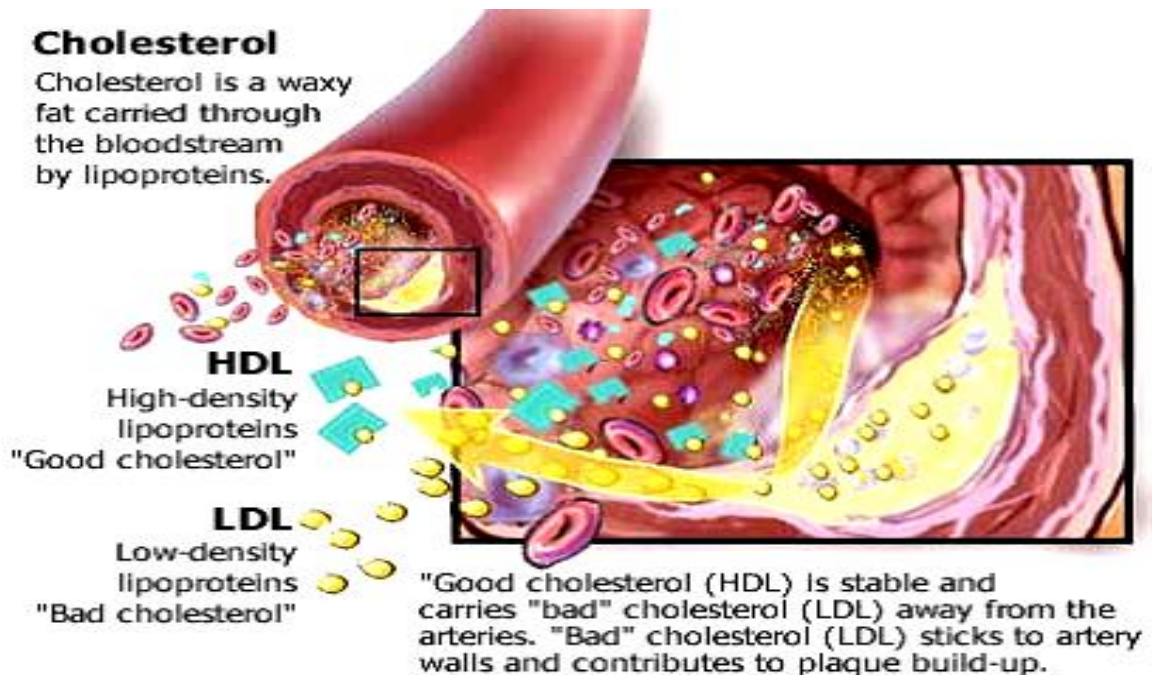


Figure 13: Cholesterol

There are two major types of cholesterol found in the blood: low-density lipoprotein (LDL) cholesterol sometimes referred to as 'bad' cholesterol, and high-density lipoprotein (HDL) cholesterol, or 'good' cholesterol.

- ❖ Low density lipoprotein (LDL) cholesterol is called 'bad' because it is a major contributor to the development of

atherosclerosis - the sticky plaques that can form inside blood vessels and contribute to problems like stroke.

- ❖ High density lipoprotein (HDL) is 'good' because it helps remove cholesterol from these developing plaques, taking it back to the liver to be excreted from the body in bile. Levels of HDL in the body can be

raised for example by things like exercise, and lowered by smoking.

Diabetes:

Heart problems are the leading cause of death among people with diabetes, especially in the case of adult-onset or Type II diabetes (also known as

noninsulin- dependent diabetes). Certain racial and ethnic groups (African Americans, Hispanics, Asian and Pacific Islanders, and Native Americans) have a greater risk of developing diabetes. The American Heart Association estimates that 65% of patients with diabetes die of some form of cardiovascular disease.

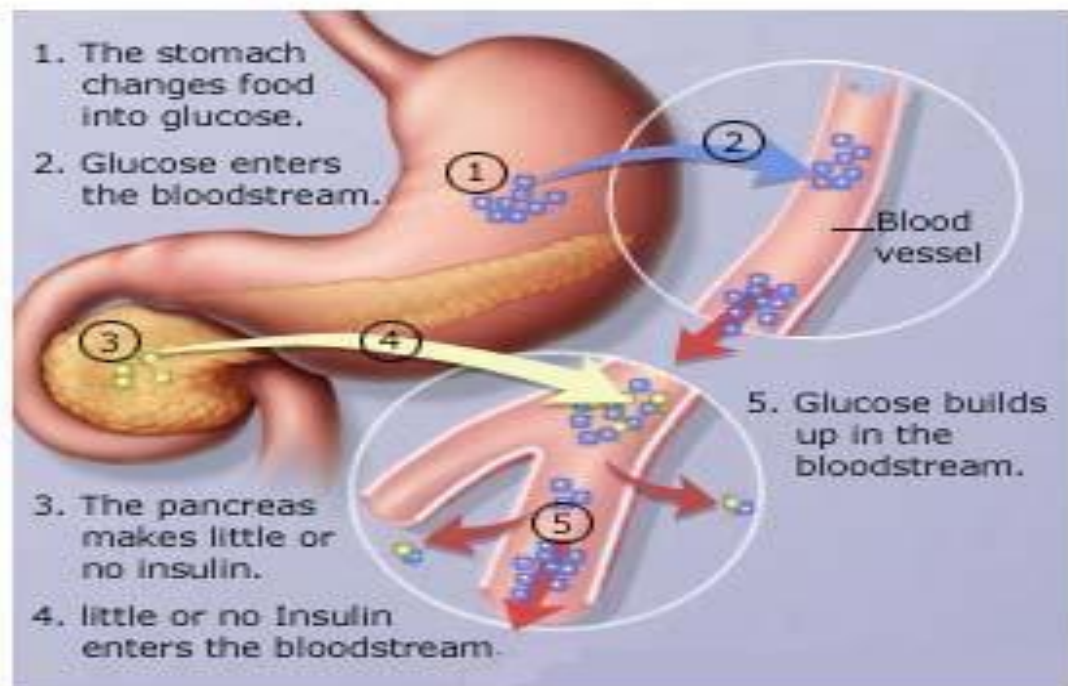


Figure 15: Diabetes – glucose build-up

Obesity and Overweight:

Obesity is a growing health problem in both developed and developing countries. Extra weight is thought to lead to increased total cholesterol levels, high blood pressure, and an increased risk of coronary artery disease. Obesity increases the chances of developing other risk factors for

cardiovascular disease, especially high blood pressure, high blood cholesterol, and diabetes. Many doctors now measure obesity in terms of body mass index (BMI), which is a formula of kilograms divided by height in metres squared ($BMI = W [kg]/H [m^2]$). According to the National Heart, Lung, and Blood Institute (NHLBI), being

overweight is defined as having a BMI over 25. Those with a number over 30 are considered obese.

Obesity is strongly related to major cardiovascular risk factors such as raised blood pressure, glucose intolerance, type 2 diabetes and dyslipidaemia.^{59, 60}

Smoking:

Most people know that cigarette and tobacco smoking increases the risk of lung cancer, but fewer realize that it also greatly increases the risk of cardiovascular disease and peripheral vascular disease (disease in the vessels that supply blood to the arms and legs). According to the American Heart Association, more than 400,000 Americans die each year of smoking-related illnesses. Many of these deaths are because of the effects of smoking on the heart and blood vessels.

Research has shown that smoking increases heart rate, tightens major arteries, and can create irregularities in the timing of heartbeats, all of which make the heart work harder. Smoking also raises blood pressure, which increases the risk of stroke in people who already have high blood pressure. Although nicotine is the main active agent in cigarette smoke, other chemicals and compounds like tar and carbon monoxide are also harmful to the heart in a variety of ways. These chemicals lead to the buildup of fatty plaque in the arteries, possibly by injuring the vessel walls. And they also affect cholesterol and levels of fibrinogen, which is

a blood-clotting material. This increases the risk of a blood clot that can lead to a heart attack.

Physical Inactivity:

People who are not active have a greater risk of heart attack than do people who exercise regularly. Exercise burns calories, helps to control cholesterol levels and diabetes, and may lower blood pressure. Exercise also strengthens the heart muscle and makes the arteries more flexible. Those who actively burn 500 to 3500 calories per week, either at work or through exercise, can expect to live longer than people who do not exercise. Even moderate-intensity exercise is helpful if done regularly.

Gender:

Overall, men have a higher risk of heart attack than women. But the difference narrows after women reach menopause. After the age of 65, the risk of cardiovascular disease is about the same between the sexes when other risk factors are similar.

Heredity:

Cardiovascular disease tends to run in families. For example, if parents or siblings had a heart or circulatory problem before age 55, this then creates a greater risk for cardiovascular disease than someone who does not have that family history. Risk factors (including high blood pressure,

diabetes, and obesity) may also be passed from one generation to another.

Race:

Researchers have found that some forms of cardiovascular disease are more common among certain racial and ethnic groups. For example, studies have shown that African Americans have more severe high blood pressure and a greater risk of cardiovascular disease than whites. The bulk of cardiovascular research for minorities has focused on African Americans and Hispanics, with the white population used as a comparison. Risk factors for cardiovascular disease in other minority groups is still being studied. In New Zealand it has been shown that Maori have a greater risk of cardiovascular disease also.

Age:

Older age is a risk factor for cardiovascular disease. In fact, about 4 of every 5 deaths due to cardiovascular disease occur in people older than 65.

As we age, our hearts tend to not work as well. The heart's walls may thicken, arteries may stiffen and harden, and the heart is less able to pump blood to the muscles of the body. Because of these changes, the risk of developing cardiovascular disease increases with age. Because of their sex hormones, women are usually protected from cardiovascular disease until menopause, and then their risk

increases. Women 65 and older have about the same risk of cardiovascular disease as men of the same age.

Contributing Risk Factors

Stress:

Stress is considered a contributing risk factor for cardiovascular disease because little is known about its effects. The effects of emotional stress, behaviour habits, and socioeconomic status on the risk of cardiovascular disease and heart attack have not been proven. That is because we all deal with stress differently: how much and in what way stress affects us can vary from person to person.

Researchers have identified several reasons why stress may affect the heart. Stressful situations raise the heart rate and blood pressure, increasing the heart's need for oxygen. This need for oxygen can bring on angina pectoris, or chest pain, in people who already have cardiovascular disease.

During times of stress, the nervous system releases extra hormones (usually adrenaline). These hormones raise blood pressure, which can injure the lining of the arteries. When the arteries heal, the walls may harden or thicken, making it easier for plaque to build up. Stress also increases the amount of blood clotting factors that circulate in the blood, and makes it more likely that a clot will form. Clots may then block an artery narrowed by plaque and cause a heart attack. Stress may also contribute to

other risk factors. For example, people who are stressed may overeat for comfort, start smoking, or smoke more than they normally would.

Sex hormones:

Sex hormones appear to play a role in cardiovascular disease. Among women younger than 40, cardiovascular disease is rare. But between the ages 40 and 65, around the time when most women go through menopause, the chances that a woman will have a heart attack greatly increase. From 65 onward, women make up about half of all heart attack victims.

Birth control pills:

Early types of birth control pills contained high levels of estrogen and progestin, and taking these pills increased the chances of cardiovascular disease and stroke, especially in women older than 35 who smoked. However birth control pills today contain much lower doses of hormones. Birth control pills are considered safe for women younger than 35, who do not smoke or have high blood pressure.

If smoking or have other risk factors, birth control pills will increase the risk of cardiovascular disease and blood clots, especially if older than 35. According to the American Heart Association, women who take birth control pills should have yearly checkups to test blood pressure, triglyceride and glucose levels.

Alcohol:

Studies have shown that the risk of cardiovascular disease in people who drink moderate amounts of alcohol is lower than in nondrinkers. Experts say that moderate intake is an average of one to two drinks per day for men and one drink per day for women. One drink is defined as 1 fluid ounce (fl oz) or 30 ml of 80-proof spirits (such as bourbon, Scotch, vodka, gin, etc.), 30 ml of 100-proof spirits, 120 ml of wine, or 360 ml of beer. However drinking more than a moderate amount of alcohol can cause heart-related problems such as high blood pressure, stroke, irregular heartbeats, and cardiomyopathy (disease of the heart muscle). And the average drink has between 100 and 200 calories. Calories from alcohol often add fat to the body, which may increase the risk of cardiovascular disease. It is not recommended that nondrinkers start using alcohol or that drinker's increase the amount that they drink.^{61, 62}

CONCLUSION:

Work-related cardiovascular disease is an important cause of work-related morbidity and mortality. However, the extent of the problem is difficult to identify accurately, and currently can only be studied through the use of population methods. Effective prevention activities exist for some exposures, but for others there is insufficient information on prevention activities and their effectiveness.

REFERENCE:

1. Athavale, Vasanat “Cardiology in Ayurveda (Hrud-Vijnanya)” Kawahar, Nagar Delhi: Chaukhamba Sanskrit Pratishthan, 1999.
2. American Heart Association. 2002 heart and stroke statistical update. Available at: <http://www.americanheart.org>. Accessed January 17, 2013.
3. Wood D. Established and emerging cardiovascular risk factors. *Am Heart J.* 2001;141:S49–57.
4. Keaney JF, Jr. Atherosclerosis: from lesion formation to plaque activation and endothelial dysfunction. *Mol Aspects Med.* 2000;21:99–166.
5. Dhawan V, Jain S. Effect of garlic supplementation on oxidised low density lipoproteins and lipid peroxidation in patients of essential hypertension. *Mol Cell Biochem.* 2004;266:109–15.
6. Libby, P., 2002. Inflammation in atherosclerosis. *Nature* 420, 868–874.
7. Libby, P., Theroux, P., 2005. Pathophysiology of coronary artery disease. *Circulation* 111, 3481–3488.
8. Rosamond, W., Flegal, K., Furie, K., Go, A., Greenlund, K., Haase, N., Hailpern, S.M., Ho, M., Howard, V., Kissela, B., Kittner, S., Lloyd-Jones, D., McDermott, M., Meigs, J., Moy, C., Nichol, G., O'Donnell, C., Roger, V., Sorlie, P., Steinberger, J., Thom, T., Wilson, M., Hong, Y., 2008. Heart disease and stroke statistics-2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation* 117, e25–e146.
9. Holmes, S.D., Krantz, D.S., Rogers, H., Gottdiener, J., Contrada, R.J., 2006. Mental stress and coronary artery disease: a multidisciplinary guide. *Prog. Cardiovasc. Dis.* 49, 106–122.
10. Krantz, D.S., Contrada, R.J., Hill, D.R., Friedler, E., 1988. Environmental stress and biobehavioral antecedents of coronary heart disease. *J. Consult. Clin. Psychol.* 56, 333–341.
11. Manuck, S.B., Kaplan, J.R., Adams, M.R., Clarkson, T.B., 1988. Effects of stress and the sympathetic nervous system on coronary artery atherosclerosis in the cynomolgus macaque. *Am. Heart J.* 116, 328–333.
12. AIHW (Australian Institute of Health and Welfare) 2004. Heart, stroke and vascular diseases—Australian facts 2004. AIHW Cat. No. CVD 27. Canberra: AIHW and National Heart Foundation of Australia (Cardiovascular Disease Series No. 22).
14. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *New Engl J Med* 2005; 352: 1685-95.

15. http://www.hse.ie/eng/services/Publications/Health_Protection/Public_Health_/Health_Status_Report_section_5.pdf access on the March 2013.
16. Wood D. Established and emerging cardiovascular risk factors. *Am Heart J.* 2001;141:S49–57.
17. Kochanek KD, Xu JQ, Murphy SL, Miniño AM, Kung HC. Deaths: final data for 2009. *National vital statistics reports.* 2011;60(3).
18. Dunstan DW, Zimmet PZ, Welborn TA, de Courten MP et al. 2002. The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care* 25(4):829–34.
19. American Heart Association. Heart Disease and Stroke Statistics—2007 Update. Dallas, TX: American Heart Association, 2007.
20. Hurst, W. *The Heart, Arteries and Veins.* 10th ed. New York, NY: McGraw-Hill; 2002.
21. World Health Organization. Global health risks: Mortality and burden of disease attributable to selected major risks. Geneva, WHO, 2009.
22. Gibbons RJ, Abrams J, Chatterjee K et al. ACC/AHA 2002 Guideline update for the management of patients with chronic stable angina—summary article. A report of the American College of Cardiology and the American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol* 2003; 41: 159.
23. Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation in myocardial infarction. A report from the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients with Unstable Angina). *J Am Coll Cardiol.* 2000; 36(0): 970-1062.
24. World Health Organisation. Prevention of recurrent heart attacks and strokes in low and middle income populations: Evidence-based recommendations for policy makers and health professionals. 2003. Geneva, Switzerland. Mallinson, T (2010). "Myocardial Infarction". *Focus on First Aid* (15): 15. Retrieved 2010-06-08.
25. Kosuge, M; Kimura K, Ishikawa T et al. (March 2006). "Differences between men and women in terms of clinical features of ST-segment elevation acute myocardial infarction". *Circulation Journal* 70 (3): 222–6. doi:10.1253/circj.70.222. PMID 16501283. Retrieved 2008-05-31.
26. Kasper, Denis Lh. et al. (2005). *Harrison's Principles of Internal Medicine*, 16th edn. McGraw-Hill. ISBN 0-07-139140-1.
27. *Cardiopulmonary Pharmacology for Respiratory Care*, Jahangir Moini, Ch.2; page 24

28. Hoffman JI, Kaplan S (June 2002). "The incidence of congenital heart disease". *J. Am. Coll. Cardiol.* 39 (12): 1890–900. doi:10.1016/S0735-1097(02)01886-7. PMID 12084585.
29. "Heart Defects: Birth Defects". Merck. Retrieved 30 July 2010.
30. "Congenital Heart Defects in Children Fact Sheet". American Heart. Retrieved 30 July 2010.
31. Kumar, Vinay. *Robbins Basic Pathology*, 8th Edition. W.B. Saunders Company, 052007. 10.7
32. Kumar, Vinay. *Robbins & Cotran Pathologic Basis of Disease*, 7th Edition. Saunders Book tea 082004. 11.2.6
33. Bonow RO, Carabello BA, Kanu C, et al. (2006). "ACC/AHA 2006 guidelines for the management of patients with valvular heart disease. *Circulation* 114 (5): e84–231. doi:10.1161/CIRCULATIONAHA.106.176857. PMID 16880336
34. agavendra R. Baliga, Kim A. Eagle, William F Armstrong, David S Bach, Eric R Bates, *Practical Cardiology*, Lippincott Williams & Wilkins, 2008, page 452.
35. American College of Physicians (ACP). "Pericardial disease". *Medical Knowledge Self-Assessment Program (MKSAP-15): Cardiovascular Medicine*. p. 64. ISBN 9781934465288.
36. AU Corey GR; Campbell PT; Van Trigt P; Kenney RT; O'Connor CM; Sheikh KH; Kisslo JA; Wall TC (August 1993). "Etiology of large pericardial effusions". *American Journal of Medicine* 95 (2): 209–13. doi:10.1016/0002-9343(93)90262-N
37. "Heart failure". *Health Information*. Mayo Clinic. 23 December 2009. DS00061.
38. He J; Ogden LG; Bazzano LA; Vupputuri S, et al. (2001). "Risk factors for congestive heart failure in US men and women: NHANES I epidemiologic follow-up study". *Arch. Intern. Med.* 161 (7): 996–1002. doi:10.1001/archinte.161.7.996
39. Carretero OA, Oparil S (January 2000). "Essential hypertension. Part I: definition and etiology". *Circulation* 101 (3): 329–35.
40. Fisher ND, Williams GH (2005). "Hypertensive vascular disease". In Kasper DL, Braunwald E, Fauci AS, et al.. *Harrison's Principles of Internal Medicine* (16th ed.). New York, NY: McGraw-Hill. pp. 1463–81. ISBN 0-07-139140-1.
41. *Brain Basics: Preventing Stroke*". National Institute of Neurological Disorders and Stroke. Retrieved 2009-10-24.
42. Guercini F, Acciarresi M, Agnelli G, Paciaroni M (April 2008). "Cryptogenic stroke: time to determine aetiology". *Journal of Thrombosis and*

- Haemostasis 6 (4): 549–54. doi:10.1111/j.1538-7836.2008.02903.
43. Peripheral Arterial Disease at Merck Manual of Diagnosis and Therapy Professional Edition, Retrieved on August 9, 2010
44. Norgren L, Hiatt WR, Dormandy JA (2007). "Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II)". *Eur J Vasc Endovasc Surg.* 33 (Suppl 1): S1–75. doi:10.1016/j.ejvs.2006.09.024
45. Sierra C, Coca A, Schiffrin EL. Vascular mechanisms in the pathogenesis of stroke. *Current Hypertension Reports*, 2011, June, 13(3):200–207.
46. Tobin, Kenneth J. (2010). "Stable Angina Pectoris: What Does the Current Clinical Evidence Tell Us?". *The Journal of the American Osteopathic Association* 110 (7): 364–70. PMID 20693568
47. Hombach, V.; Hoher, M.; Kochs, M.; Eggeling, T.; Schmidt, A.; Hopp, H. W.; Hilger, H. H. (1988). "Pathophysiology of unstable angina pectoris—correlations with coronary angioscopic imaging". *European Heart Journal* 9: 40–5.
48. Prinzmetal, Myron; Kenamer, Rexford; Merliss, Reuben; Wada, Takashi; Bor, Naci (1959). "Angina pectoris I. A variant form of angina pectoris". *The American Journal of Medicine* 27 (3): 375–88. doi:10.1016/0002-9343(59)90003-8
49. Kusumoto, Fred M. "Cardiovascular Disorders: Heart Disease". In McPhee, SJ; Hammer, GD. *Pathophysiology of Disease: An Introduction to Clinical Medicine* (6th ed.). ISBN 978-0-07-162167-0.
50. Merck & Co. Occlusive Peripheral Arterial Disease, The Merck Manual Home Health Handbook website, revised and updated March 2010. Retrieved March 4, 2012.
51. Rozanski A; Mental stress and the induction of silent myocardial ischemia in patients with coronary artery disease, *N Engl J Med* 318(16):1005-1012, 1988.
52. Zelinka T, Strauch B, Petra'k O, Holaj R, Vrankova' A, Weisserova' H, et al. Increased blood pressure variability in pheochromocytoma compared to essential hypertension patients. *J Hypertens.* 2005;23:2033–9.
53. Plouin PF, Chatellier G, Fofol I, Corvol P. Tumor recurrence and hypertension persistence after successful pheochromocytoma operation. *Hypertension.* 1997;29:1133–9.
54. Mendis S et al. Report for the Pathobiological Determinants of Atherosclerosis in Youth (PBDAY) Research Group: Atherosclerosis in children and young adults: An overview of the World Health Organization and International Society and Federation of Cardiology Study on Pathobiological Determinants of Atherosclerosis in

- Youth Study (1985– 1995). Prevention and Control, 2005, 1:3–15.
55. Ross R. Mechanisms of disease: Atherosclerosis – an inflammatory disease. New England Journal of Medicine, 1999, 340:115–126.
56. Davis NE. Atherosclerosis: An inflammatory process. Journal of Insurance Medicine, 2005, 37:72–75.
57. Durrington, P (August 2003). "Dyslipidaemia". The Lancet 362 (9385): 717–31. doi:10.1016/S0140-6736(03)14234-1
58. Biggerstaff KD, Wooten JS (December 2004). "Understanding lipoproteins as transporters of cholesterol and other lipids". Adv Physiol Educ 28 (1-4): 105–6. doi:10.1152/advan.00048.2003
59. World Health Organization. Prevention of cardiovascular disease: Guidelines for assessment and management of cardiovascular risk. Geneva, WHO, 2007.
60. Finucane MM et al. National, regional, and global trends in body-mass index since 1980: Systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet, 2011, 337(9765):557–567.
61. Government of Great Britain. Obesity: Third report of session 2003–2004. Volume 1: Report, together with formal minutes. Document HC 23–1. London, House of Commons, 2004.
62. Ezzati M et al. Selected major risk factors and global and regional burden of disease. Lancet, 2002, 360:1347–1360.