



Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Coronary Artery Disease - Angina, Unstable Angina, Myocardial Dystrophy

Discussion paper prepared for

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WSIAT literature search reviewed by Dr. A. Weinberg in 2013, who is of the opinion that this paper still provides a balanced overview of the medical knowledge in this area.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Coronary Artery Disease - Angina, Unstable Angina, Myocardial Infarction

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence. See *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

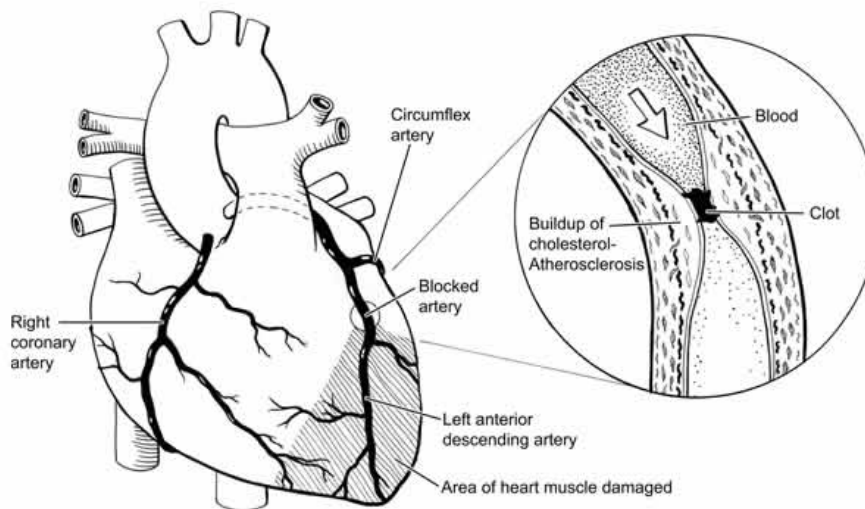
Introduction

Coronary artery disease (CAD) is a major cause of death and disability in Canada. CAD or atherosclerosis is a chronic disorder involving the blood vessels (coronary arteries) that feed blood to the heart muscle. With this disease there are stable and unstable periods.

Anatomy & Physiology

The heart (figure 1) is a pump composed of four chambers - 2 of which receive blood (atria) & 2 which pump blood (ventricles). Blood returns from the body to the right atrium & is pumped into the lungs by the right ventricle. In the lungs, oxygen is added to the blood & carbon dioxide removed. Blood then flows into the left atrium & is then pumped by the left ventricle into the aorta & delivered to the entire body. The coronary arteries (right and left) are the first branches of the aorta and bring the oxygenated blood to the heart muscle itself. To pump blood to the body, the left ventricle has the most muscle in the heart and is the chamber most affected by a heart attack.

Figure 1 - The heart



Angina, Unstable Angina, Myocardial Infarction

1. Causation/Evolution:

Over a long time, the vessel wall becomes thickened with buildup of cholesterol and narrows the lumen. These buildups in the wall or plaques are called atherosclerosis. Plaque in the arteries can become so thick that it severely restricts the flow of blood to the heart. This can result in recurrent chest pain (angina) that's triggered by exertion and relieved by rest. No heart muscle death occurs.

Occasionally a plaque will rupture, triggering the formation of a blood clot. This clot can block blood flow to the heart. This sudden interruption in blood flow leads to inadequate oxygen delivery to the heart muscle and if persistent, myocardial necrosis (heart muscle death) or myocardial infarction ensues. A heart attack can occur anytime — at work or play, while one is resting, or

while one is active. Some heart attacks strike suddenly, but many people who experience a heart attack have warning signs and symptoms (unstable angina) hours, days or weeks in advance.

2. Clinical Presentations:

There are two types of angina-stable and unstable.

Stable angina is the result of a temporary, insufficient blood flow to the heart muscle due to a narrowed vessel impairing flow. Chest pain occurs in a predictable fashion such as during physical activity or emotional stress when the heart is working harder and requiring additional blood flow. Cessation of the precipitating factor results in restoration of adequate blood flow and the symptoms quickly subside without any damage to the heart.

Unstable angina results from the sudden rupture of a plaque, which precipitates a rapid accumulation of platelets at the rupture site with abrupt restriction of blood flow in the coronary artery. Consequently, symptoms occur suddenly, in an unexpected or unpredictable fashion. The symptoms may be new, prolonged, more severe, or occur with little or no exertion. Unstable angina is less responsive to nitroglycerin medication than stable angina. The accumulation of platelets and obstruction to blood flow can result in a myocardial infarction.

Myocardial infarction (MI) or a heart attack implies myocardial cell death; this occurs as result of prolonged ischemia (impairment of blood flow with resultant inadequate oxygen delivery to the heart muscle). With the onset of myocardial ischemia, cell death is not immediate, but takes a finite period to develop : as little as 20 minutes. Complete death of all myocardial cells at risk requires at least 2-4 hours or longer.

Finally, CAD and its sequelae account for 80% of sudden cardiac deaths. While this may occur in individuals with unrecognized CAD, a history of prior infarction is present in about 50%. The most common mechanism is ventricular tachyarrhythmia (a catastrophic failure of coordinated electrical activity with prompt loss of cardiac pumping function and death).

3. Risk factors:

The presence of certain factors, called coronary risk factors, increase an individual's risk of a heart attack. These factors contribute to the unwanted buildup of the deposits that narrow the arteries throughout the body, including the arteries to the heart.

Coronary risk factors include:

Tobacco smoke. Smoking and long-term exposure to second hand smoke damage the interior walls of arteries and increases the risk of blood clots forming.

High blood pressure. Over time, high blood pressure can damage the arteries and accelerate atherosclerosis. The risk of high blood pressure increases with age and obesity.

High blood cholesterol or triglyceride levels. Cholesterol is a major part of the deposits that can narrow arteries. A high level of the wrong kind of cholesterol increases the risk of a heart attack. Low-density lipoprotein (LDL) cholesterol (the "bad" cholesterol) is most likely to narrow arteries. A high LDL level is undesirable and is often a byproduct of a diet high in saturated fats and cholesterol. A high level of triglycerides, a type of blood fat related to diet, also is undesirable. However, a high level of high-density lipoprotein (HDL) cholesterol (the "good" cholesterol), which helps the body clean up excess cholesterol, is desirable and lowers the risk of heart attack.

Lack of physical activity. An inactive lifestyle contributes to high blood cholesterol levels and obesity. Conversely, people who get regular aerobic exercise have better cardiovascular fitness,

which decreases their overall risk of heart attack. Exercise is also beneficial in lowering high blood pressure.

Obesity. Obese people have an excess of body fat (a body mass index -BMI of 30 or higher). BMI is a measure of body fat based on height and weight that applies to adult men and women. Abdominal obesity as measured by waist circumference - men >102 cm and women >88 cm - is associated with an elevated risk of CAD. Obesity raises the risk of heart disease because it's associated with high blood cholesterol levels, high blood pressure and diabetes.

Diabetes. Diabetes is the inability of the body to adequately produce or respond to insulin properly. While diabetes can occur in childhood, it appears more often in middle age and in overweight individuals.

Stress. An individual's response to stress may increase the risk of a heart attack. Under stress, an individual may overeat or smoke from nervous tension. Too much stress, as well as anger, can also raise blood pressure.

Alcohol. Alcohol in moderation helps to raise HDL levels and can have a protective effect against heart attack. Men should have no more than two drinks a day, and women should have no more than one. Excessive drinking can raise blood pressure and triglyceride levels, increasing the risk of a heart attack.

Family history of heart attack. If family members have had heart attacks at an early age (<55 years), an individual's risk is greater. This may be related to genetic conditions that raise blood cholesterol levels or blood pressure.

Increasing age

Male sex. While men are generally at greater risk than women of heart attacks, women are not immune and their risk increases after menopause and in the presence of the above risk factors.

4. Symptoms:

Common signs and symptoms of angina, unstable angina and heart attack include:

- pressure, fullness or a squeezing pain in the center of the chest
- pain or discomfort that extends beyond the chest to the shoulders, arms, back, or jaw
- discomfort in the upper abdomen
- shortness of breath

Typically, the symptoms of stable angina will subside promptly with stopping physical activity and resting or taking nitroglycerin. If the symptoms persist for more than 15 minutes, are more intense, rapidly accelerating and associated with other symptoms (sweating, impending sense of doom, dizziness or fainting, nausea and vomiting) or not relieved with nitroglycerin, medical attention should be sought as the symptoms may be those of unstable angina or a heart attack rather than stable angina.

5. Diagnostic Tests:

The clinical diagnosis of MI has traditionally required an integrated assessment of the history, with some combination of indirect evidence for heart muscle death using biochemical, electrocardiographic, and imaging modalities. These tests include:

Electrocardiogram (ECG). This is the first test done to diagnose a heart attack. This test records the electrical activity of the heart through electrodes attached to the skin. Injured heart muscle does not conduct electrical impulses normally and the ECG may show that a heart attack has occurred or is in progress.

Blood tests. Certain biomarkers slowly leak into the bloodstream if the heart has been damaged by a heart attack. These biomarker levels can help to determine the size and timing of the heart damage.

Echocardiogram. This test uses sound waves to produce an image of the heart. The sound waves bounce off the heart and are reflected back through the chest wall and help to identify whether an area of the heart has been damaged by a heart attack

Nuclear scan. This test helps to identify blood flow problems to the heart. Small amounts of radioactive material are injected into the bloodstream. Special cameras detect the radioactive material.

Coronary catheterization (angiogram). This test shows whether the coronary arteries are narrowed or blocked. A liquid contrast agent is injected into the coronary arteries through a long, small tube (catheter) that has been advanced to the heart from the leg or arm. The arteries become visible on X-ray, revealing any areas of disease.

Stress test. Subsequent to a heart attack, a stress test may be carried out days or weeks later to assess how the heart responds to physical stress. This would involve walking on a treadmill or pedaling a stationary bike while attached to an ECG machine.

CAD and Stress: When individuals refer to “stress,” they may be referring to either physical stress or emotional stress. Most of the medical literature on stress and heart disease refers to physical stress. Most people, however, are referring to the emotional variety when they talk about stress.

Physical stress (i.e., exertion) is, in general, good and is to be encouraged. The lack of physical stress – a sedentary lifestyle contributes to development of CAD. If an individual already has CAD then excessive physical exertion may precipitate angina or even a heart attack. Numerous studies over several decades have shown the role of heavy exertion — from snow shoveling to recreational exercise — in triggering sudden myocardial events and the protective role of regular exercise in preventing them. Firefighters have episodic exposure to extreme levels of physical exertion, and they face occupational hazards that may add to or amplify their risk of death due to cardiovascular causes. These hazards include chemicals, thermal and emotional stress. Overall, firefighters do not have an excess risk of dying from heart disease. However, emergency firefighting duties have been associated with a risk of death from coronary heart disease that is markedly higher than the risk associated with non-emergency duties. The majority of these individuals had received a diagnosis of vascular disease or had a high prevalence of coronary risk factors.

While individuals who exercise rarely may be more likely to suffer a heart attack after strenuous exertion than those who exercise regularly, the absolute risk of a cardiac event after any single bout of activity remains rare. In summary, physical stress does not cause CAD and while angina is commonly associated with exertion, heart attacks do not usually happen during exercise.

Emotional stress: The evidence for whether emotional stress, either severe or chronic, can cause heart disease has been hard to come by.

Circumstantial evidence suggests that chronic emotional stress may be a risk factor for cardiac disease and early death. The emotional stress caused by a lack of a sense of control over one's life is especially bad, as is an individual's response to such stress. Emotional distress, along with natural disasters, war and sporting events may also trigger heart attacks in vulnerable individuals.

British investigators have shown recently that chronic work stress can produce chronic increases in adrenaline levels, and have related those changes to an increased risk of heart disease. Work stress is associated with poor health behaviors, low physical activity, smoking, poor diet, and the metabolic syndrome that can lead indirectly to CAD.

Emotional or physical stress causes an increase in heart rate, elevation of blood pressure, and release of stress hormones. These hemodynamic, homeostatic, and endothelial abnormalities may culminate in the development of a thrombus (blood clot) and heart attack.

Acute emotional stress such as unexpected death of a loved one can trigger a surge of stress hormones with resultant acute myocardial infarction in the setting of normal coronary arteries.

Summary

Stable angina, unstable angina and heart attacks are all caused by CAD, however they are not the same.

Angina is a symptom that occurs as the result of the gradual accumulation of plaque in a coronary artery with resultant restriction of sufficient blood flow to the heart. Angina is classified into two types- stable and unstable. Stable angina is usually predictable, associated with physical and emotional stress, and relieved with rest and/or nitroglycerin pills. Stable anginal symptoms do not cause permanent damage to heart muscle.

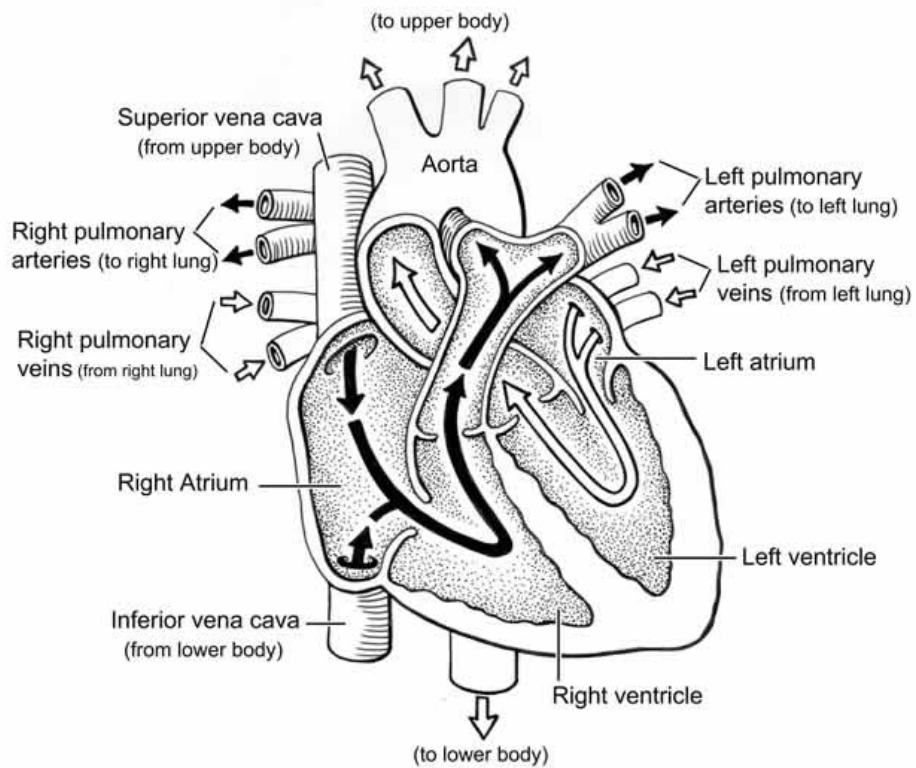
Unstable angina is precipitated by the sudden rupture of a plaque, which results in a rapid accumulation of platelets at the plaque with increasing obstruction of blood flow in the coronary artery. Unstable angina symptoms occur in an unexpected and unpredictable manner. The symptoms are more severe and less responsive to nitroglycerin medication. This is a medical emergency as it may culminate in a heart attack.

A myocardial infarction or heart attack occurs when heart muscle dies. This is the result of a CAD plaque rupturing. Any CAD plaque can rupture, even one that is not severe enough to cause angina. When this occurs, a blood clot develops resulting in complete blockage of the artery, and heart muscle death. The symptoms of a MI are similar to unstable angina, but are usually more severe and prolonged. Symptoms alone may not distinguish between unstable angina and MI, thus both are considered medical emergencies, and immediate medical attention is important.

Minimizing the overall risk involves primary and secondary prevention measures. These measures include promoting healthy behaviors (such as a heart-healthy diet, no tobacco or excessive alcohol, and regular exercise) and modifying conditions (such as hypertension (high blood pressure), hyperlipidemia (elevated blood cholesterol), diabetes, and obesity) that pose additional risks.

The illustration (Figure 2) shows a cross-section of a healthy heart and its inside structures. The black arrow shows the direction in which low-oxygen blood flows from the body to the lungs. The white arrow shows the direction in which oxygen-rich blood flows from the lungs to the rest of the body.

Figure 2 - Cross-section of a healthy heart and its inside structures



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