UNIVERSITY OF CALGARY
FACULTY OF NURSING

UNIT 9

Alterations in Cardiovascular Function

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Unit 9 Table of Contents

UNIT 9
Alterations in Cardiovascular Function

Although there are numerous cardiovascular conditions which could be addressed in this unit it is important for you to gather a basic understanding of how the cardiac system functions. Many cardiovascular conditions are so closely interrelated it is very difficult to clearly separate them. At times, therefore, throughout this unit you may find some areas choppy. Try to keep in mind how conditions may be linked together in the overall functioning of the cardiovascular system.

For the purposes of this unit, four components will be addressed. These will include:

- atherosclerosis
- hypertension
- angina and myocardial infarction
- heart failure

Overview

Aim

Upon completing this unit you will be familiar with the terminology specific to this area and be able to describe the development and implications of the specified cardiac disorders.

Objectives

Objectives will be presented individually for each section of this unit.

Resources

Resources will be presented individually for each section of this unit.

Web Links


All web links in this unit can be accessed through the Web CT system.

Section 1: Atherosclerosis

Objectives

Upon completing this section you should be able to:

1. Describe the etiology, risk factors and pathophysiology of atherosclerosis.

2. Identify the clinical pathological conditions resulting from atherosclerosis.

3. Describe the management of atherosclerosis.

Introduction

Atherosclerosis has its beginnings at an early age. Autopsies on infants have revealed the presence of fatty streaks on the intimal lining of blood vessels which is the earliest sign of atherosclerosis. Although atherosclerosis has its beginnings in the first decade of life, its process is insidious with clinical manifestations not being evident for 20-40 years or longer.

Definition

Atherosclerosis, from the Greek word ***athere*** refers to gruel or porridge while ***sclerosis*** means hardening. Atherosclerosis is a common arterial disorder characterized by thickening and hardening of the inner walls and the tunica media layers of large and medium sized arteries. The thickening of the inner wall is caused by deposits of yellowish plaques. The plaque is composed of cholesterol, lipids, fibrin and cellular debris that will harden over time.

Prevalence

Atherosclerosis is not a single disease entity but can take several forms. Atheromatous lesions are major causes of hypertension, cerebral ischemia, coronary heart disease, angina pectoris, myocardial infarction, and other cardiac disorders. In 1995, cardiovascular disease (CVD) was responsible for 79,117 (37%) deaths in Canada (21% was from ischemic heart disease, 7% from stroke, 9% other CVD, and 63% all other diseases). (Heart and Stroke Foundation of Canada, 1997).

Pathophysiology

Populations at Risk

Non-modifiable Risk Factors

Gender: Males are affected more frequently than women. Women are protected due to higher estrogen levels up until menopause. Following menopause the gender distribution of atherosclerosis becomes equal.

Age: Atherosclerosis is an insidious progressive pathological process which begins at birth and most often becomes evident only in the third to the seventh decades of life.

Familial: A number of genetically determined conditions such as hypertension, hyperlipidemia, hypercholesterolemia, and diabetes appear to increase the risk for the predisposition to atherosclerosis. It has also been suggested that there might be an inherent genetic weakness of the intimal arterial wall lining.

Race: Race which is also an inherited risk factor has been implicated in the development of atherosclerosis. It is thought that the predisposition of some races, e.g. Black Americans to hypertension, increases their risk for atherosclerosis and coronary heart disease.

Modifiable Risk Factors

Cigarette Smoking
Hyperlipidemia
Hypertension
Diabetes Mellitus
Obesity
Sedentary life style
Hormone therapy
Alcohol
Type A Personality

Refer to McCance and Huether (2001) pp. 1000-1005 for a detailed discussion of these risk factors

Etiology

The etiology of atherosclerosis is not clear. Although risk factors have been identified by epidemiological studies the exact mechanisms by which they contribute to the development of atherosclerosis remains controversial. Two major hypotheses regarding the pathogenesis of atherosclerosis are:

- injury to arterial endothelium
- accumulation and infiltration of cholesterol and lipids

The fatty streak (the earliest atherosclerotic lesion) is often referred to as an atheroma or an atheromatous plaque. Refer to McCance and Huether (2001), pp. 980-984 for further discussion.

Breaking News: More recently research is being done looking at the possibility of bacterial and infectious processes and their role in the development of altherosclerosis.

Pathogenesis

A diagrammatic sketch of the pathogenesis of atherosclerosis is clearly outlined in McCance and Huether (2001), p. 982. The occlusion of the arterial lumen is a gradual and insidious process. The clinical condition resulting from atherosclerosis will depend upon the anatomical location of the atherosclerotic lesion. It most commonly affects the terminal portion of the aorta and iliac arteries but can also significantly affect other arteries such as: the coronary, femoral, popliteal, internal carotid, vertebral, basilar, thoracic aorta, and the circle of Willis.

Clinical Manifestations

Clinical manifestations of atherosclerosis will depend on the vessel involved, the site of the lesion, and the risk factors involved (as previously discussed). It has been suggested that 60% of the arterial lumen is already occluded when symptoms are manifested (McCance & Huether, 2001). Clinical manifestations will be subdivided into two sections: those affecting the coronary arteries and those affecting peripheral arteries. The primary focus of this module is the effects of atherosclerosis on the coronary arteries, although the effects on the peripheral arteries are mentioned in less detail.

Common Manifestations of Coronary Atherosclerosis

Chest pain or angina is the most common symptom that usually occurs with coronary atherosclerosis. It has been described in a variety of ways and affects each person individually. It can occur at rest but initially usually begins or occurs with exercise. As atherosclerosis progresses

and gradually further occludes the coronary arteries the likelihood of the person suffering a myocardial infarction increases.

Although angina is the most common symptom of atherosclerosis it usually occurs when 60% or more of the coronary artery affected is occluded. Other symptoms may include shortness of breath, cyanosis, pallor, exercise intolerance, diaphoresis and nausea. These symptoms and others are further described under the clinical manifestation section of myocardial infarction.

Common Manifestations of Atherosclerosis in Peripheral Arteries

Pain
An aching, burning, persistent, cramp-like pain may occur, in the lower extremities with exercise, which is caused by intermittent claudication. Severe ischemia of tissues, and ischemic neuropathy, may cause shooting or severe ache/pain or a gnawing pain during rest.

Cold Extremities or Cold Sensitivity
Patients often complain of coldness in their fingers and toes. Exposure to a cold environment may precipitate blanching or cyanosis of the digits.

Impaired Arterial Pulsations
Significant alterations or more specifically diminished pulsations may be evident in the posterior tibial and/or the dorsalis pedis arteries.

Color Changes
In advanced disease, cyanosis or an abnormally red color (rubor) may be seen, particularly in the skin of the lower extremities. The degree of color change is related to the extent and severity of vascular occlusion.

Ulceration and Gangrene
These lesions may occur either spontaneously or as a result of trauma to the affected ischemic extremity.

Edema
Edema of the feet and legs may occur in severe cases. It primarily occurs, though, when the legs are in a dependent position.

Other Trophic Changes
Moderate to severe chronic ischemia may result in finger/toe nail thickening and deformity; or conversely, the nails may become paper-thin.

Table 9.1 Clinical and Pathologic Effects of Atherosclerosis in Different Anatomic Sites

Site	Clinical and Pathologic Effects
Abdominal/terminal aorta	Ischemic effects in lower extremities; gangrene of toes, feet; effects of fusiform abdominal aneurysm; embolism of atherosclerotic debris to smaller arteries
Aortoiliac and femoral arteries	Intermittent claudication; gangreme of toes, feet; aneurysm formation in iliac arteries
Coronary arteries	Angina pectoris; conduction disturbances; myocardial infarction
Carotid and vertebral arteries	Transient ischemic attacks; cerebrovascular accident (CVA) or stroke
Renal artery	Hypertension; renal ischemia (hematuria, proteinuria)
Mesenteric arteries	Intestinal ishcemia (ileus, bowel perfromation with peritonitis)

Adapted from Pathophysiology adaptations and alterations in function (2nd ed.). (p. 358) by B. L. Bulloch and P. P. Rosendahl, 1988, Glenview, Illinois: Scott Foresman Company. Copyright 1988 by B. L. Bulloch. Reprinted by permission.

Evaluation and Treatment

Essential components of the treatment and surveillance regimen include a complete health history and physical exam. Affected vessels may be identified through the use of X-ray films, electrocardiography, ultrasonography, nuclear scanning, and angiography. Optimal daily cholesterol intake should not exceed 250-300 mg. It is important to monitor the risk factor modification strategies and revise as necessary.

There is no cure for atherosclerosis. Progression of the disorder may continue despite all interventions. Strategies targeted at risk factor modification may prove beneficial in alleviating symptoms associated with coronary heart disease and peripheral atherosclerosis. These strategies may improve coronary and peripheral arterial blood flow in addition to improving the client's general health status.

Segments of severely damaged or obstructed coronary arteries may be reopened by performing a percutaneous transluminal angioplasty or replaced by patch grafts or bypassed, as in coronary artery bypass surgery. An endarterectomy may be performed to clear a major peripheral artery (not done with coronary arteries) that is blocked by a clot or accumulation of plaque by surgically removing the intimal lining of the artery. A more detailed description of interventions aimed at treating patients with coronary occlusion will be discussed in the component on myocardial infarction that follows later in this unit.

Risk factor modification may play a key role in the management of coronary atherosclerosis. A diet low in cholesterol, calories, and saturated fats, adequate exercise, and the avoidance of smoking and stress may help prevent further development of atherosclerosis. Antilipidemic agents may also be used to decrease the lipid levels but will not reverse the atherosclerotic process.

Prognosis/Trajectory

The atherosclerotic process may progress despite all interventions. Atherosclerosis in the vessels of the brain or the heart, leading to cerebral or myocardial ischemia may present as a life-threatening event. Atherosclerosis resulting in cardiovascular disorders is the leading cause of death in North America.

Section 2: Hypertension

Introduction

Hypertension has for years been recognized as a major risk factor in the development of kidney failure as well as cardiovascular disorders (particularly stroke, heart attack and heart failure). Individuals with hypertension may often be symptom free, therefore they may fail to seek medical attention prior to the development of complications. The widespread nature of the condition demands that nurses as health care professionals be cognizant of the pathological process and systemic ramifications of hypertension.

Requirements

A basic understanding of the normal anatomy and physiology of the arterial system is presumed. Learners are encouraged to review an anatomy and physiology text if necessary prior to proceeding. The term hypertension will be used synonymously with high blood pressure throughout the component.

- McCance and Huether (2001), pp. 958-966, 984-994

Objectives

Upon completing this section you should be able to:

1. Define hypertension and its classifications
2. Describe the etiology, pathophysiology, and clinical manifestations of hypertension
3. Describe the management of hypertension

Definition

Hypertension, a common, often asymptomatic disorder, is characterized by a persistently elevated systemic blood pressure greater than 140/90..

Categorization Scheme

A variety of classifications for hypertension exist. High blood pressure can be classified according to the level of severity, the cause, or the type of hypertension. A hypertension classification system which depicts systolic and diastolic pressure readings is illustrated in Table 9.2.

Table 9.2 Recommended scheme for categorizing arterial pressure in individuals aged 18 years and older.

Range mm Hg	**Category***
Diastolic	
< 85	Normal blood pressure
85 - 89	High normal blood pressure
90 -104	Mild hypertension
105 –114	Moderate hypertension
≥115	Severe hypertension
Systolic, when diastolic blood pressure is <90	
<140	Normal blood pressure
140- 159	Borderline isolated systolic hypertension
≥160	Isolated systolic hypertension

*Classification based on the average of two or more readings on two or more occasions.

*A classification of borderline isolated systolic hypertension (SBP 140 TO 159 mm Hg) or isolated systolic hypertension (SBP ≥ 160 mm Hg) takes precedence over high normal blood pressure (diastolic blood pressure, 85 to 89 mm Hg) when both occur in the same person. High normal blood pressure (DBP 85 to 89 mm Hg) takes precedence over a classification of normal blood pressure (SBP < 140 mm Hg) when both occur in the same person

Note: This is based on the WHO and the Canadian Coalition of High Blood Pressure definitions that elevated blood pressure is one that is greater than 140/90 mmHg. Blood pressure consistently >130/85 on home readings is also a significant finding which needs follow up.

Hypertension is often described as being either primary or secondary depending on whether or not there is a known cause or underlying disease/condition.

1. ***Primary Hypertension*** — Often called essential or idiopathic hypertension is manifested by a chronic elevation of blood pressure without evidence of other disease. It effects 90-95% of all hypertensive patients and has no known cause.

4. ***Secondary Hypertension*** — Develops from a specific underlying disorder (refer to McCance & Huether, 2001, pp. 989-991, Table 29-2, p. 990).

Prevalence

Hypertension is a significant public health problem in Canada. Approximately one in five Canadians are at increased risk of developing cardiovascular disease due to high blood pressure, with about one in three being unaware of their condition, (National Institutes of Health, 1988).

Risk Factors

The prevalence of high blood pressure is linked to a number of risk factors which include:

- cigarette smoking
- family history of hypertension
- advancing age — hypertension has been found to rise with age, except for men where it seems to plateau beyond age 45
- male gender
- black race
- obesity
- high sodium intake
- glucose intolerance (diabetes mellitus)
- changeable (labile) blood pressure or borderline hypertension
- heavy alcohol consumption

These risk factors are discussed in detail in McCance and Huether (2001), p. 1000-1004. It is important to remember that these risk factors are not unique to hypertension but are associated with a variety of cardiovascular conditions.

Etiology

The cause of primary hypertension remains in question. It is believed that numerous complex mechanisms interact in the body producing long term elevations of high blood pressure. These factors include hemodynamic, neural, humoral, and renal mechanisms. It is improbable that there is a single factor responsible for the development of primary hypertension.

Several hypotheses for the development of primary hypertension have been proposed and are discussed in McCance and Huether (2001), p. 987.

Secondary hypertension is caused by a systemic disease process. This process raises peripheral vascular resistance or cardiac output. Although secondary hypertension affects less than 10% of all hypertensive adults, more than 80% of the hypertension that occurs during childhood is secondary to a specific physiological condition For more details on specific underlying diseases and/or disorders which may cause secondary hypertension refer to McCance and Huether (2001), pp. 989-991, p. 990.

Pathophysiology

Pathogenesis

You must have a good understanding of the normal mechanisms controlling blood pressure in order to understand the pathophysiology of hypertension. Blood pressure is a product of cardiac output and total systemic vascular resistance. Numerous factors play a part in the regulation of these two major variables to balance blood pressure.

Cardiac output is affected by changes in heart rate and contractility, as well as changes in extracellular fluid volume (affected by alterations in  um intake, renal function, and mineralocorticoid activity).

Systemic vascular resistance is influenced by sympathetic nervous system stimulation, humoral controls including the renin-angiotensin-aldosterone system, catecholamines, E_2 prostaglandins, kinins, corticosteroids, and vasopressin (antidiuretic hormone). Autoregulation is an intrinsic property of vascular smooth muscle which regulates blood flow in accordance to tissue metabolic needs. Autoregulation therefore may also serve as a possible link between cardiac output and peripheral resistance. Alterations in any of these factors which lead to elevation in either cardiac output and/or total systemic vascular resistance can cause an increase in blood pressure resulting in sustained hypertension. For further discussion on this area you may want to skim McCance and Huether 2001, pp. 958-966, 984-994.

Clinical Manifestations

Hypertension is often termed the "silent killer" since there are few signs and no symptoms until it becomes severe and target organ damage has occurred. The only sign is the presence of elevated arterial blood pressure readings.

Untreated hypertension may lead to the damage of small arterioles. This in turn may cause target organ dysfunction. Target organs that are subjected to vascular damage may include the heart, eyes, brain and kidneys. See Table 9.3 for the effects of hypertension on target organs.

Table 9.3 Hypertensive Effects on Target Organs

Organ	Effect	Manifested by
Heart	Myocardial infarction	ECG changes; enzyme elevations
	Congestive failure	Decreased cardiac output; S3 or summation gallop auscultated; cardiomegaly on radiograph
	Myocardial hypertrophy	Increased voltage R wave in V_3-V_6; increased frequency of angina; left ventricular strain, manifested by ST and T wave changes
	Dysrhythmias	Usually ventricular dysrhythmias or conduction defects
Eyes	Blurred or impaired vision	Nicking arteries and veins; hemorrhages and exudates on visual examination
		Papilledema
Brain	Encephalopathy	Severe occipital headache, paralysis, speech difficulties, coma
Kidney	Cerebrovascular accident	Rapid development of confusion, agitation, convulsions, death
	Renal insufficiency	Nocturia, proteinuria, elevated BUN, creatinine
	Renal failure	Fluid overload, accumulation of metabolites, metabolic acidosis

Evaluation of high blood pressure should be done on at least two separate readings/occasions. Ongoing home evaluation of blood pressure is often helpful to avoid the "white coat" syndrome (increase of blood pressure when an individual is in the presence of a health care professional). Evaluation of blood pressure using a 24 hour home monitoring system is helpful in detecting daily fluctuations and baseline (BP) readings.

Evaluation and Treatment

Hypertension is a chronic, lifelong disease which requires ongoing blood pressure monitoring to ensure adequate treatment. Medications may be adjusted (added or deleted) as necessary based on the individual's response. Clinical tests such as blood work, X-ray films and other diagnostic tests may be useful in the detection of secondary hypertension or associated complications.

The management of hypertension focuses on the treatment of the underlying cause. An extensive assessment should include a careful history and physical examination to determine target organ involvement, the presence of cardiovascular risk factors other than hypertension, and the type of hypertension (essential or secondary).

The main goal of treatment is to achieve and maintain an arterial blood pressure below 140/90. In primary hypertension this may be accomplished by risk factor modification strategies and/or pharmacological agents. In secondary hypertension the treatment goals are aimed at the control or correction of the underlying disease process.

Classifications of drugs that are available for the treatment of hypertension include:

- diuretics
- angiotensin-converting enzyme (ACE) inhibitors
- adrenergic antagonists which include beta blockers, central acting adrenergic inhibitors, Alpha-1 adrenergic blockers, and combined alpha-beta adrenergic blockers
- vasodilators, which include venodilators, arteriodilators and arterio-venodilators
- calcium channel blockers
- antianxiety agents or benzodiazepines

Each of these drugs has its own mechanisms of action either alone or in combination when individualized to specific patient needs in the treatment of hypertension. Drugs are often added in a step-wise approach until the blood pressure is reduced to the goal level. Following this, maintenance doses of drugs are established.

Other nonpharmacologic interventions can include:

- regular physical exercise
- reduced alcohol intake
- decrease in salt intake (especially in the elderly)
- adequate dietary intake of calcium and potassium
- increase in diet with high fiber, fruit, vegetables and low saturated fat
- stress and coping with stress
- weight reduction (for individuals with BMI >25)

Prognosis

The prognosis of hypertensive individuals worsens with higher blood pressure levels. Arteries and arterioles throughout the body experience the wear and tear effects associated with hypertension. Stroke (cardiovascular accident) remains the leading sequela of hypertension with diastolic pressures above 110 mmHg. Other major morbid sequelae include myocardial infarction, renal failure, and encephalopathy. A downward trend in deaths due to these sequelae has been seen since the introduction of antihypertensive drug therapy and the development of risk factor modification strategies.

Section 3: Myocardial Ischemia (Angina) and Infarction

Introduction

Myocardial ischemia occurs when blood flow through the coronary arteries falls below the level required for normal cellular metabolism. The ischemia that occurs can be reversed if oxygen demands decrease or blood flow is increased. If these factors do not change irreversible cell damage and muscle death occurs, that is, myocardial infarction (MI). MI is the leading cause of death in North America.

Objectives

Upon completing this section you should be able to:

1. Describe the etiology and pathogenesis of myocardial infarction
2. Discuss the clinical manifestations, management and complications of MI
3. Differentiate between right and left ventricular heart failure
4. Discuss the clinical implications of MI in relation to ventricular heart failure, dysrhythmias and cardiogenic shock

Requirements

A requirement of this module is that you complete the readings from McCance and Huether (2001) and relevant articles listed in the references. You may also find it helpful to review the terms outlined in the glossary.

Prior to commencing it is recommended that you complete and review the component on atherosclerosis. You may also find it necessary to review the structure and function of the cardiovascular system as discussed in McCance and Huether (2001), pp. 930-952. For more detailed anatomy and physiology, refer to an anatomy and physiology text.

Read: McCance and Huether (2001), pp. 980-984, 1005-1016, 1029-1035.

Albert, M. (1999). Heart failure: The physiologic basis for current therapeutic concepts. *Critical Care Nurse Supplement,* June, 3-15.

Dracup, K.A. (1999). Combination treatment strategies for management of acute myocardial infarction: New directions with current therapies. *Critical Care Nurse Supplement,* April, 12-13.

Fruth, R.M. (1991). Differential diagnosis of chest pain. *Critical Care Nursing Clinics of North America, 3* (1), 59.67.

Wright, S.M. (1990). Pathophysiology of congestive heart failure. *Journal of Cardiovascular Nursing, 4* (3), 1-18.

Angina

Definition

Angina is the clinical syndrome of transient myocardial ischemia that occurs with an imbalance between oxygen supply and demand.

Types of Angina

1. **Stable Angina Pectoris (exertional angina)**
   Stable angina is often precipitated by physical exertion, cold, or emotional stress. It can last from a few minutes to 15 minutes and is usually relieved by rest, removal of the provoking factors, or administration of nitroglycerine.

5. **Unstable Angina Pectoris**
   Unstable angina may also be referred to as crescendo angina, preinfarction angina, angina decubitus, and nocturnal angina. Unstable angina may occur without obvious provoking factors. It can occur with exertion but often occurs at rest. Each attack of unstable angina is unique in its onset and course of pain. Pain usually occurs more frequently and is of greater intensity and of longer duration with each subsequent attack.

6. **Variant (Prinzmetal) Angina**
   Variant angina can occur at rest in early hours of the morning and is often associated with ST segment elevations. This type of angina is often referred to as cyclical, occurring at the same time each day. Variant angina is thought to be caused by vasospasm of one or more of the major coronary arteries. It is believed that a decrease in myocardial oxygen consumption during sleep or rest may lead to coronary artery vasoconstriction which is responsible for the spasm.

Angina can often occur off and on over years, then become unstable, and finally result in a MI. Angina, may also occur suddenly and result in myocardial infarction immediately. It is important to remember that there is no set pattern that leads to myocardial infarction and each individual must be treated separately.

Myocardial Infarction (MI)

Definition

Angina may lead to MI. MI refers to the process by which myocardial tissue is destroyed in regions of the heart that are deprived of their blood supply after closure of the coronary artery or one of its branches either by a thrombus or by obstruction of the vessel lumen by atherosclerosis. The key factor contributing to MI is the imbalance between myocardial oxygen supply and demand.

Prevalence

According to statistics Canada, in 1985 MI killed 26,945 people (16,597 males, 10,448 female). In Alberta in 1986 heart disease was responsible for 39% of male deaths and 42% of female deaths.

Populations at Risk

The populations at risk for MI are discussed in detail in McCance and Huether (2001), pp. 974-979. Please review information provided in the module dealing with atherosclerosis and hypertension for further explanations since these areas are closely linked.

Etiology

The etiology of MI may vary with one or more of the following factors being implicated.

Progression of Coronary Artery Atherosclerosis
MI may occur as a natural sequela of advancing atherosclerosis. It has been suggested that at least 60% of the coronary tissue blood supply usually is occluded prior to the development of signs and symptoms of MI. See Figure 29-1, p. 981, Figure 29-21, p. 1011 and Figure 29-22, p. 1012.

Acute Coronary Thrombosis
The rupture of an atheromatous plaque in a coronary artery may lead to secondary thrombosis superimposed on the ulcerated plaque.

Coronary Artery Embolism
Coronary embolism is a rare cause of MI but when it does occur it is believed that the clot often is formed in the left side of the heart. This clot may develop due to mitral valve prolapse, mitral stenosis, or infective endocarditis.

Coronary Artery Spasm
Although implicated as a cause in a limited number of MI patients, the genesis of coronary artery spasm remains a controversial issue. Factors such as increased sympathetic nervous activity, irritation of the arterial wall from an atheroma, production of histamine and serotonin causing vasoconstriction, increase in calcium flux within arterial smooth muscle, and an imbalance in the manufacture or release of prostaglandins such as thromboxane A_2 (causes vasoconstriction), and prostacyclin (causes vasodilation) have all been implicated in the cause of spasm.

Decrease in Oxygen Supply in the Presence of Coronary Artery Disease (CAD)
Any event that causes a decrease in oxygen supply such as an alteration of pulmonary function or anemia which develops as a result of haemorrhage can precipitate the occurrence of MI. This is especially evident with individuals experiencing some degree of CAD.

Increase in Myocardial Oxygen Demand in the Presence of Significant CAD
Myocardial oxygen demand may be increased by a number of factors/situations. Some of these factors include: physical exertion, pain, and tachyarrhythmias. In general any event which increases heart rate, blood pressure and/or causes an increase in catecholamine release can, in the presence of CAD cause an increase in myocardial oxygen demand and consumption therefore potentially leading to MI.

Pathophysiology

Pathogenesis
The pathophysiology of MI is clearly documented in McCance and Huether (2001) pp. 1011-1013. Key points related to this section are listed under the following headings.

Cellular Injury

- hypoxia can cause electrocardiographic changes within 30-60 seconds but death of cardiac cells occurs in approximately 20 minutes
- myocardial contractility is influenced by oxygen and electrolyte (calcium, potassium, magnesium) balance
- Cellular Death

Zones

- **zone of ischemia** is reversible and therefore leaves no permanent damage;
- **zone of hypoxic injury** may progress to necrosis or return to normal depending on the treatment and its effect;
- **zone of necrosis** occurs within 6 hours. This necrotic tissue cannot be revived.
- the boundaries of these zones may change dependent on:
  - time between onset of symptoms and treatment
  - successful treatment resulting in revascularization of the ischemic and hypoxic areas
  - development of collateral circulation

Structural and Functional Changes

- Refer to Table 29-22, p. 1012, McCance and Huether (2001)
- impairment of myocardial function is dependent on:
  1. size of infarct
  2. location and extent of infarct
  3. collateral blood supply
  4. previous history of MI

Repair

- wound healing and tissue repair begins with inflammation
- refer to McCance and Huether (2001), chapter 7 for review of the wound healing process and the wound healing unit
- fibrous scar tissue forms within 3-6 weeks. This scar is nonfunctioning and permanent
- the myocardium cannot regenerate

Types of Infarctions

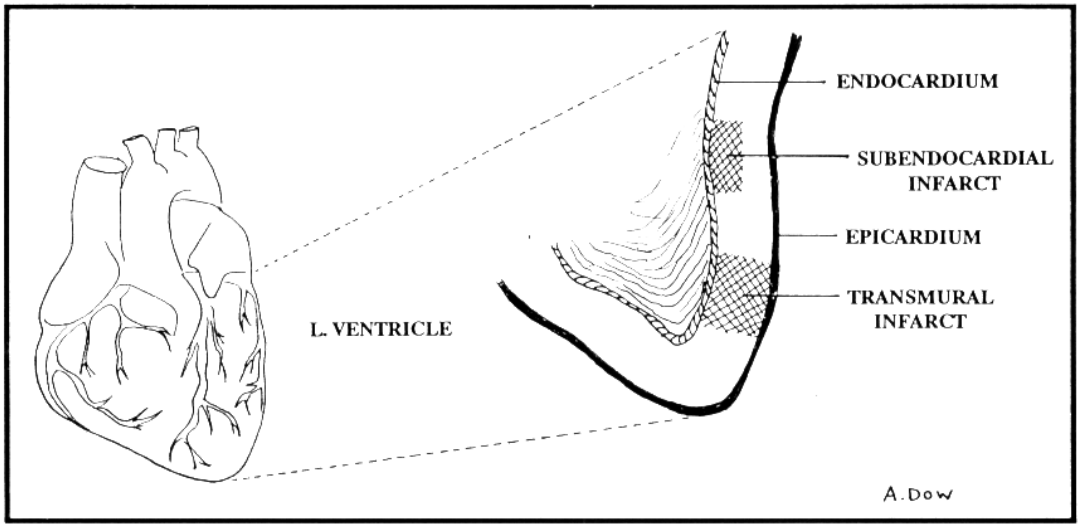
1. Endocardial (endocardium)
   - affects the inner wall of the heart

7. Transmural
   - includes all layers of the myocardium (endocardium, myocardium, epicardium)

8. Subendocardial
   - mmon and involves necrosis of the inner layers of the myocardium
   - often see signs and symptoms of heart failure and mural thrombi
   - myocardium is injured and begins to die in subendocardial layers and then extends outward on a wavefront pattern to the epicardial region

9. Epicardial (epicardium)
   - affects the outside wall of the heart
   - may result in signs and symptoms of pericarditis
   - most common area affected is the left ventricle

Figure 9.1 Types of Infarctions



Location of Infarcts
Anatomical location of MI depends on which coronary artery is affected (see Table 9.4).

Table 9.4 Primary Location of MI

Artery Affected	MI Type	Key
LAD (distal occlusion)	Anterior MI	LAD Left anterior descending coronary artery
RCA	Inferior Wall MI	RCA Right coronary artery
Circumflex (CX)	Lateral Wall MI	LCA Left circumflex coronary artery
LMS occlusion	Death usually occurs since left main stem supplies 70% of left ventricle	LMS Left main stem
LAD: (more proximal occlusion)	Anterolateral MI	
LCA: (mid and posterior margin branch occlusion)	Posterior MI	
LCA – Left coronary arterial-posterior marginal branches RCA occlusion – right coronary artery (usually proximal occlusion)	Right Ventricular Infarction. (Usually occurs with an inferior infarction of posterior interventricular septum.)	

Clinical Manifestations

Chest pain

- chest pain due to MI generally differs from the pain of angina mainly in intensity and duration
- location of pain—substernal, midsternal or in the shoulder, jaw, epigastric, neck or arms. Pain may occur in any of the above areas in isolation or in combination
- sensation—burning, squeezing, heaviness, smothering, aching, pressure
- duration—constant, 30 minutes or longer, not relieved by rest or nitrates (e.g., nitroglycerine)
- quality—mild to severe
- onset varies, occurring at rest or with exertion

Breaking News: It is important to realize that not every patient who having an acute myocardial infarction will present the typical symptoms of chest pain pallor and diaphoresis. Elderly men (75 yea and older) are at a high risk for presenting *atypically* with vague symptoms of fatigue cough perhaps nausea. [Then, Rankin & Fofon (in press). Atypical presentation of acute myocardial infarction in three age groups. Heart & Lung: The Journal of Critical Care.

Nausea and vomiting

- occurs due to stimulation of vomiting centres (vagal stimulation), pain or decreased cardiac output
- should be avoided if possible as it can cause vasovagal response resulting in a decrease in myocardial oxygen supply as a result of decreased BP and decreased heart rate

Dyspnea and Orthopnea

- shortness of breath is more common in the elderly but can occur at any age
- dependent on the degree of imbalance of oxygen supply/oxygen demand

Anxiety, Severe Apprehension, and Denial

- may be related to fear of the unknown, pain, death and/or stimulation of sympathetic nervous system

Skin

- diaphoresis, cold, clammy, cyanosis, mottling
- skin changes are due to decreased cardiac output and increased peripheral vascular resistance

Vital signs

- blood pressure — hypotension may occur secondary to a decrease in cardiac output or from beta-adrenergic stimulation (causing vasodilation) in response to severe pain
- pulse — depending on the location of the MI and duration of the chest pain the pulse may be tachycardiac, bradycardiac, or irregular and is often weak and thready
- temperature — may rise within 24 hours and last up to 7 days. The temperature correlates with the extent of myocardial damage

Dysrhythmias

- the occurrence of dysrhythmias is high during the first 24 hours after infarct
- the type and severity of the dysrhythmia is dependent upon the location and extent of myocardial damage

Heart sounds

- heart sounds are often muffled
- S_4 gallop is common in MI, indicating possible ventricular dysfunction
- development of murmurs may signify papillary muscle dysfunction, thus valve dysfunction
- pericardial friction rub may indicate pericarditis

Other

Other clinical manifestations may include jugular vein distension, ankle, and sacral edema, and a decrease in urine output. These are all signs of ventricular dysfunction and heart failure.

Electrocardiographic (ECG) Findings

See Figure 9.2 for a diagram depicting ECG changes during MI

Ischemia

- ST segment depression
- T wave inversion

Injury

- stage beyond ischemia, but still reversible
- ST segment elevation — epicardial injury and ischemia
- T wave elevation — epicardial injury and ischemia
- ST segment returns to isoelectric position within a few days

Infarction

- Q waves — changes may indicate pathology
- ST segment changes
  - elevated in lead over or facing infarcted area

- reciprocal changes (ST segment depression) will be found in leads 180 degrees from area of infarction
- may occur hours to weeks after infarct

- T wave changes may last for weeks and return to normal or remain inverted for the patient's life
-

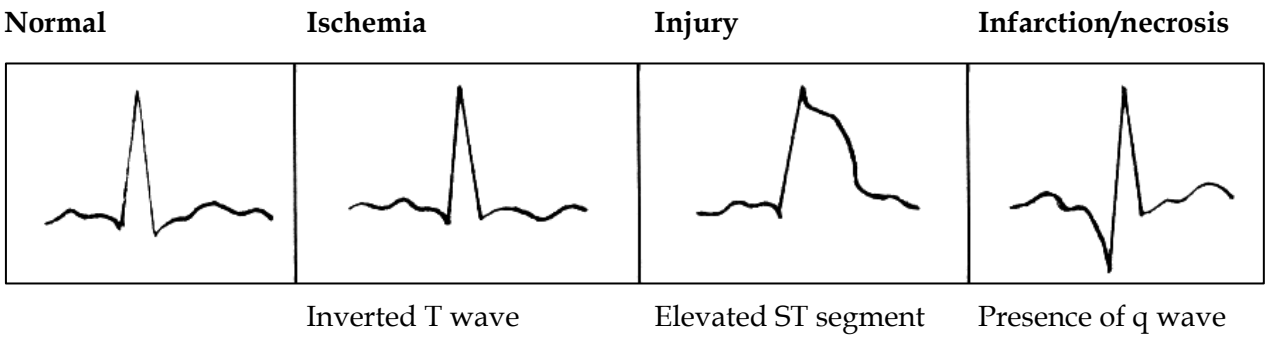


Figure 9.2 Electrocardiographic Changes with Myocardial Infarction

Evaluation and Treatment

Laboratory Tests

- elevations in white blood count may be proportional to myocardial tissue damage
- diagnosis of MI depends on the measurement of cardiac enzymes and isoenzymes

Refer to Table 9.5 and Figure 9.3 to assist in understanding enzymes and their characteristic pattern of fluctuation during the evolution of MI.

Enzyme	Onset	Peak	Return to Normal
CPK (0-225 units/L)	2-5 hours	24 hours	2-3 days
SGOT (0-41 units/L)	6-8 hours	24-48 hours	4-8 days
LDH (60-200 unites/L)	6-12 hours	48-72 hours	7-10 days
CPK-MB (1-10)	4-8 hours	16-24 hours	48-72 hours
LDH1 (17.5-28.3%)	6-24 hours	24-48 hours	72-96 hours

Table 9.5 Evolution of Cardiac Enzymes Following MI

- CPK alone not indicative of MI
- CPK-MB very specific for MI except in marathon athletes

- LDH1 specific for MI — if LDH increased and LDH1 is the predominant isoenzyme then diagnosis of MI can be made. This allows detection of MI several days old

CPK = creatine phosphokinase

LDH = lactate dehydrogenase

SGOT = serum glutamic oxaloacetic transaminase

CPK-MB = creatine phosphokinase myocardial band

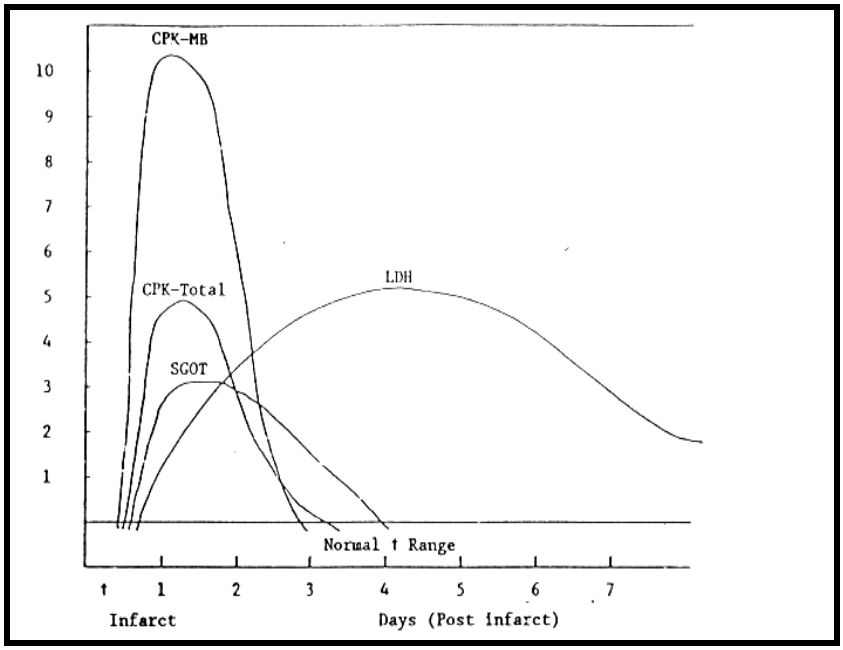


Figure 9.3 Chronological serum enzyme activities following MI

Treatment

The danger of death from MI is greatest during the first two weeks postinfarction but is particularly severe during the first 24 to 48 hours (with most deaths occurring within the first two hours of onset of pain).

Goals

The major goals of care for patients with acute MI include:

- successful treatment of the acute attack and prompt alleviation of symptoms
- prevention of complication and further attacks
- rehabilitation and education of the patient and significant others

Treatment

Treatment involves four main areas:

1. Decrease oxygen demand of the heart
2. Continued relief/prevention of pain
3. Monitoring to prevent electrical problems
4. Treatment of pump failure

1. Decreasing Oxygen Demands

- bedrest is recommended for 2-3 days then progressive gradual ambulation
- a 20% increase in oxygen demand can occur during bathing in a stable ICU patient, therefore supplemental oxygen should be given especially during activity, (e.g., eating, bathing)

2. Pain Relief and Prevention

- Pain increases oxygen consumption by causing tachycardia and increased contractility
- Continued chest pain may signal ongoing myocardial damage

a. Thrombolytic Therapy

- types of thrombolytic therapy include streptokinase, tissue type plasminogen activator (TPA) and urokinase
- the goal of this therapy is to reduce or dissolve thrombus formation(s) occluding the coronary arteries
- the goal is obtained through the lysis of existing thrombi and the reperfusion of myocardial tissue
- it is essential that thrombolytic therapy is commenced within the first few hours following the presentation of chest pain in order to limit infarct size, thus preserving myocardial function and life

b. Nitrates: Nitroglycerine and Isordil

- reduces oxygen demands and improves coronary blood flow
- potent vasodilators
- improves myocardial oxygen supply while decreasing oxygen demand
- this decreases preload more than afterload
- decreases end diastolic volume (EDV) and ventricular wall tension therefore decreases myocardial workload and oxygen consumption
- reduces infarct size especially if given IV within first 4 hours after onset of signs and symptoms, also decreases likelihood of heart failure, myocardial infarction, extensions, or cardiac death

c. Beta-adrenergic agents: Propranolol, Atenolol, Metoprolol, Timolol

- inhibits effects of catecholamines at beta-adrenergic receptor sites, therefore decreases sympathetic tone
- primary therapeutic effect—decreases heart rate and response to exercise
- may reduce mortality rate by 20% and reduce infarct size
- thought to decrease acute myocardial ischemic injury by improving balance between oxygen supply and demand
- blocks sympathetic stimulation of myocardium (B1 receptors)
- results in decreased HR, myocardial contractility, cardiac output, B.P., which results in decreased myocardial oxygen consumption
- enhances myocardial blood supply by redistributing blood flow
- also affects the electrical system of the heart: decreased automaticity in SA, delay through AV node, decreased excitability of atria and ventricles

d. Calcium Channel Blockers: Verapamil, Diltiazem, Nifedipine, Sinoatrial (SA), Atrioventricular (AV)

- relaxes smooth muscle, decreasing resistance and increasing blood flow in coronary arteries and the peripheral circulation
- decreases calcium influx into cells and produces smooth muscle relaxation (decreases contractility)
- results in vasodilation of coronary and peripheral blood vessels, decrease in cardiac contractility, cardiac output, automaticity, atrioventricular conduction, platelet aggregation
- reverses coronary spasm in some patients
- improves blood flow in ischemic areas by vasodilation of coronary arteries
- decreases myocardial oxygen demands by decreasing afterload, heart rate and contractility

e. Analgesics: Morphine

- decreases venous return, therefore decreases congestion
- decreases oxygen demand

f. Oxygen

g. Antiarrhythmic agents: Lidocaine, Procainamide

- a variety of antiarrhythmic drugs are used to treat dysrhythmias occurring following MI

h. Anticoagulation

- Heparin and ASA agents are often used to decrease platelet aggregation and thrombus formation

3. Mechanical Interventions
PTCA: percutaneous transluminal angioplasty

- coronary angioplasty is a procedure used to open or dilate occluded coronary arteries by pressing lipid deposits back against the intimal lining of the artery

Pacemaker

- a variety of conduction defects may result from damaged or ischemic myocardial tissue
- a pacemaker (temporary or permanent) may be necessary to reestablish effective electrical stimulation of myocardial tissue

CABG: coronary artery bypass graft surgery

- severely damaged or obstructed coronary arteries may be bypassed to facilitate the improvement and/or re-establishment of coronary blood flow

IABP: intra-aortic balloon pumping

- a IABP is a counter pulsation device that is placed in the descending aorta
- is used to support circulation temporarily when the injured myocardium cannot generate adequate cardiac output
- principles of therapy include decreasing left ventricular workload, partially supporting the systemic circulation and enhancing oxygen supply to the injured myocardium

Complications

1. Dysrhythmias and conduction disturbances

- 95% of patients experience some disturbance of cardiac rate or rhythm
  - due to immediate ischemic effects on ventricular muscle, cardiac dysrhythmias slow the initial phase of depolarization in the ischemic myocardium or enhance the automaticity of ventricular cells
- most common are ventricular extrasystoles (80%)
- often occurs within 12-14 hours after infarct
- dangerous because have the potential to trigger ventricular tachycardia or fibrillation which is life threatening
- AV conduction disturbances occur 12-15% of the time; occurs 2-3 times more often in patients with inferior infarction (RCA supplies AV node in 90% of patients)
- bundle branch blocks and complete heart blocks are more likely with anterior infarct (LAD supplies conduction system below the Bundle of His)

2. Cardiogenic shock

- heart fails to pump effectively resulting in a decrease in stroke volume causing tissue ischemia and hypoxia
- syndrome of hypotension and peripheral circulatory insufficiency secondary to primary cardiac dysfunction
- most common cause of death in patients in CCU's post MI
- autopsies show that there is usually 40% acute necrosis of the left ventricle
- Unit 18 on shock deals with this in more depth

3. Extension of Infarction

- implies additional myocardial necrosis from transient nonperfusion of compromised portion of the coronary arterial circulation
- frequency -- 17-23% of patients

4. Pericarditis

- usually due to transmural MI (results when damage to entire wall has occurred)
- this pain differs from MI pain in that pericarditis pain increases on deep inspiration
- the pain may not necessarily be severe but annoying and is relieved by ASA or antiinflammatories
- occurs in 15% of MI patients

5. Pulmonary Embolism

- the blockage of a pulmonary artery by foreign matter such as thrombus
- can occur with atrial fibrillation, transmural MI or subendocardial MI involving the right ventricle
- treated prophylactically with ASA, heparin, and/or oral anticoagulants

6. Systemic Embolism

- originates in deep veins of lower extremities

7. Myocardial Rupture (Cardiorrhexis)

- 85% of ruptures occur in left ventricle wall (few patients survive)
- a slit-like tear can result in bleeding into pericardial sac (cardiac tamponade) or rupture of the wall
- classic clinical clue indicating free wall rupture is electromechanical dissociation (when electrical heart activity persists without detectable pulse or blood pressure)
- leads to congestive heart failure, cardiogenic shock and death

8. Papillary Muscle Dysfunction

- with decreased coronary perfusion the papillary muscles are susceptible to injury
- dysfunction occurs because of rupture of papillary muscles
- if the entire papillary muscle structure ruptures, it usually is fatal within minutes to hours because of severe mitral regurgitation

9. Rupture of Interventricular Septum

- causes left to right shunting leading to decreased oxygenation of systemic blood, CHF, and shock

10. Ventricular Aneurysm

- a localized dilation or protrusion in the wall of the left ventricle
- complications of ventricular aneurysms are ventricular tachycardia, impaired ventricular function, arterial embolism, and ventricular rupture

11. Musculoskeletal

- localized chest tenderness and pain is often referred to as chest wall pain
- chest wall pain is often difficult to distinguish due to high levels of anxiety
- it can be caused by fractured ribs from CPR, bruising, and anxiety

12. Genitourinary

- a decrease in cardiac blood flow and cardiac output leads to a decrease in blood flow to the kidneys which may result in acute renal failure

13. Gastrointestinal

- hiccoughing (often seen with inferior MI) can interrupt sleep and increase anxiety
- nausea and vomiting often occurs from MI but can be caused by use of opiates and other drugs
- retching may cause a vagal stimulation, therefore increasing the incidence of serious dysrhythmias
- nausea and vomiting are often treated with antiemetics to decrease the incidence of complications

14. Dressler's Syndrome

- an autoimmune disorder that may occur several days after MI or cardiac surgery
- results from body's immunologic response to damaged myocardium and pericardium
- is often treated with ASA and corticosteroids

15. Psychological

- patients may experience and exhibit feelings of denial, anxiety, anger and depression
- though these are often considered normal reactions they must not be overlooked since they can significantly affect the physical well-being of the patient

16. Cardiac Arrest

- the sudden cessation of cardiac output and effective circulation
- when it occurs the delivery of oxygen and removal of waste products stops
- factors affecting the incidence of cardiac arrest include the length of time between the onset of pain and the initiation of treatment, the extent and location of the MI, and past medical history

17. Congestive Heart Failure

- this complication is addressed in more depth in the following section because it is not only a complication of MI but it may also occur in a variety of other conditions

Section 4: Congestive Heart Failure (CHF)

Definition

A state in which the heart is unable to maintain a cardiac output sufficient to meet the needs of the body. Acute pulmonary edema or the presence of excess fluid in the lungs is the direct result of ventricular failure.

Etiology

CHF is often referred to as heart failure and ventricular heart failure. CHF is often further defined as left and right failure. There are many conditions which may contribute to the development of CHF, some of which are outlined in Table 9.6.

Table 9.6 Conditions which may contribute to heart failure

Left Heart Failure	**Right Heart Failure**
atherosclerotic heart disease	left heart failure
acute myocardial infarction	atherosclerotic heart disease
tachycardia/bradycardia	acute myocardial infarction
cardiomyopathy	tachycardia/bradycardia
increased circulating volume	pulmonary embolism
aortic stenosis or insufficiency, mitral insufficiency	fluid overload/excess sodium intake
coarctation of aorta	COPD—pulmonary hypertension
ASD or VSD	mitral stenosis
cardiac tamponade, constrictive pericarditis	ASD or VSD
	pulmonary outflow stenosis

Pathophysiology

Although either right or left heart failure can be precipitated by a variety of conditions the progression of events is similar.

Left heart failure to right heart failure:

- a diseased left ventricular myocardium cannot pump blood returning from the lungs into systemic circulation, therefore causing a decrease in cardiac output
- pressure increases in the lungs owing to the accumulation of blood in the lungs. Fluid therefore leaks into the pulmonary interstitial space, resulting in pulmonary edema
- as pressure increases in the lungs, pressure in the right heart increases owing to the backflow of pressure in the pulmonary vasculature
- the right side of the heart cannot pump its blood into the pulmonary system due to this backflow of pressure, therefore venous return is impeded
- pressure continues to back up, and eventually body organs become congested with venous blood

For further explanations or review on the pathophysiology in CHF please refer to McCance and Huether (2001), pp. 1029-1035. Please pay special attention to the figures depicting the sequence of events of heart failure throughout these pages.

Table 9.7 Clinical Manifestations of Heart Failure

Left Heart Failure	**Right Heart Failure**
anxiety, restlessness air hunger nocturnal dyspnea, dyspnea on exertion, orthopnea cough with frothy sputum tachycardia, diaphoresis basilar crackles, bronchial wheezing (respiratory system) cyanosis or pallor, hypoxia, respiratory acidosis gallop rhythm (S3) insomnia and palpitations hyperventilation pulsus alternans left ventricular heave cheyne-Stokes respirations fatigue weakness	dependent pitting edema, peripheral edemea venous ditention, jugular distention bounding pulses, disorientation, cerebral edema (CNS) oliguria (kidneys) dysrhythmias fatigue split S2 right ventricular gallop tricuspid insufficiency murmur right ventricular heave liver engorgement (GI) nausea anorexia S3 pleural effusion

Clinical Manifestations and Diagnostic Findings

- Weight gain
- Upper abdominal pain (Hepatosplenomegaly)
- Nocturia
- Anorexia and nausea (caused by edema of the bowel)
- Chest x-ray shows pulmonary clouding (interstitial density) and cardiac hypertrophy

Evaluation and Treatment

Key elements of medical management include:

1. Removing or controlling the underlying or precipitating cause of the failure

2. Improving myocardial contractility
   a. cardiac glycosides—Digoxin
      - results in augmenting contractility, increasing stroke volume and cardiac output
   b. sympathomimetic amines—Dopamine, Dobutamine
      - are used in acute situations at specific dosages to increase myocardiac contraction, cardiac output and stroke volume
   c. coxygen therapy

3. Reduction in myocardial workload
   a. reduce preload through the use of diuretics, fluid and sodium restriction, and venous vasodilators
   b. reduce afterload through the use of arterial vasodilators and stress reduction
   c. rest

Nursing Management

1. Place patient in upright position, head and shoulders up, feet and legs hanging down to favour pooling of blood in dependent portions of body by gravitational forces to decrease venous return

2. Ongoing physical assessment

3. Administer oxygen therapy

4. Monitor intake and output closely

5. Weigh patient daily

6. Maintain a relaxed, quiet environment to provide rest

7. Organize nursing care to allow for rest periods

8. Administer drugs as ordered

   For example:

   ***Morphine*—**decreases anxiety, reduces pain, induces vasodilation, decreases venous return to the heart, decreases myocardial oxygen consumption

   ***Diuretics*—**to promote excretion of water from the body

   ***Aminophylline*—**relaxes bronchospasm, increases renal blood flow and enhances diuresis, decreases pulmonary arterial pressure, decreases peripheral venous pressure, and peripheral resistance

10. If patient is on bed rest for prolonged period, institute measures to prevent hazards of immobility

11. Monitor lab data

12. Allow patient to verbalize feelings

13. Give explanations to the patients of treatments being implemented

14. Involve patient in the plan of care

*Although these nursing management strategies are listed under management of the patient with congestive heart failure they are also applicable to the nursing management of the MI patient.

Ongoing Evaluation and Surveillance

The ongoing assessment of a patient experiencing MI and/or CHF may include the following parameters:

- cardiac rhythm monitoring
- serial enzyme collection
- ongoing physical examination
- radionucleotide imaging
- electrocardiograms

Prognosis/Trajectory

- 25% die before they reach the hospital
- mortality rate in hospitalized patient range from 3-30% depending on the infarct size
- the first coronary event is more often fatal in women than men (39% vs. 31%)
- patients who develop heart failure have higher mortality rates
- prognosis after discharge is determined by:
  1. the degree of left ventricular dysfunction
  2. the extent of residual ischemic myocardium
  3. the presence of ventricular dysrhythmias

- mortality rate after 1 year is 8%, with half of the deaths occurring in first three months
- after that the mortality rate averages 4% per year
- ***Long-term impairment*** depends upon:
  1. Size of infarct
  15. Location and extent of infarct
  16. Coronary collateral blood supply
  17. Previous medical history

Learning Activity #1—Case Study: A Person Suffering from a Myocardial Infarction

Personal/Social History

Name: Kevin Zabou **Age:** 44

Sex: Male **Race/Culture:** Canadian

Occupation: Oil Executive **Marital Status:** Married

Religion: Catholic

Mr. Zabou lives with his wife and two teenagers (one daughter, Kathy, age 15; and one son, Mark, age 13) in a five-bedroom, 3000-square-foot home. He works approximately 10 hours a day, 5 days a week, and often works at home or at the office during the weekend. Mr. Zabou is a top executive at a large oil company and is responsible for supervision of 10 coordinators and 1500 employees. He finds his work challenging but at times "hard on the nerves." Mr. Zabou eats well and states that over the last year he has put on 20 lbs. due to the numerous business lunch and supper meetings. He has tried to compensate for this by not eating breakfast but instead will just have a cup of coffee with cream. His exercise regime includes walking the family dog every evening for about 15 minutes. He is an ex-smoker for the past five years but does state that many of his co-workers do smoke. He also states that he has "no time for hobbies."

Past Health History

Usual childhood diseases; no hospitalizations or surgeries.

- complaints of indigestion for last three months off and on but went away on its own
- takes no medications except Tylenol for headaches
- has not had a physical examination for the last five years

Family History

- Father died at age 74 from an MI
- Mother had breast Ca but underwent surgery with no further problems
- Has two brothers, one who recently underwent PTCA for an 95% blockage for his RCA and Circumflex arteries, and another who is presently undergoing a cardiovascular follow-up due to chest pain
- No family history of diabetes, epilepsy, hypertension, psychiatric problems, muscular problems, or neurological problems

History of Present Illness

While working in his office following lunch Mr. Zabou developed mild midsternal burning pain and pain in his jaw. He phoned his dentist to set up an appointment thinking something was wrong with one of his teeth. He then continued to work and gradually noticed the pain went away. Later on in the day he drove out to one of the oil field sites to consult with his co-workers. On the way back he again developed this pain when sitting in the middle of a traffic jam. He continued on back to work but was convinced by his secretary to go to the hospital and get checked out.

After a short period of time in the Emergency Department he was admitted to a cardiology floor with the diagnosis of possible angina.

Physical Assessment

Height: 6 feet, 1 inch

Weight: 102 kilograms

Vital signs: T-36.9ºC HR: 80 BP: 148/75 R: 16

CVS: S1, S2 audible, no S3, S4 or murmurs, no apical impulse, no jugular venous distention with the head of the bed elevated 30 degrees bilaterally, no ankle edema

Resp: color pink, no cyanosis, breath sounds audible to all lungs fields, no adventitious sounds

GI: abdomen soft, round, no tenderness on palpation, bowel sounds audible X 4 quadrants, no nausea

GU: voiding qs light amber colored urine, specimen sent

CNS: alert, but restless, states he does not feel he needs to be in here and is quite upset

Psychological: angry that he came to hospital for an upset stomach and then to be admitted. States he has to get back to work, people are relying on him and he has five major jobs that he must finish today.

Treatment Ordered

- Admit to cardiac unit
- Cardizem 30 mg QID
- Nitroglycerine .3 sl prn X 3
- Stat ECG with chest pain
- Cardiac profile X 3
- SMA12
- Cholesterol levels
- Activity as tolerated
- Low Na diet
- Captopril 12.5 mg TID

Prior to the nurse starting any of the above orders Mr. Zabou developed the following:

- midsternal burning/pressure radiating to jaw and left arm (7/10)
- diaphoretic
- nausea and vomited 150 cc of brown fluid
- SOB
- Color—pale grey

Mr. Zabou was given 3 Nitroglycerine without effect, a stat ECG was done showing increased ST segments in leads II III and AVF. An intravenous line of D5W was started in his left arm and infusing TKVO. His vital signs were: BP: 95/50; HR: 94; R: 20. He was then given 2.5 mgs IV morphine push by the physician and was given Thrombolytic therapy. He was then transferred to the ICU.

ICU orders:

- Start nitroglycerine gtt at 20 mcg/min and titrate for pain. Maintain BP greater 90 syst.
- Morphine 2.5 mgs IV prn
- O2 at 3L/NP
- Bedrest
- Heparin drip 1100 u/hr to maintain PTT 60-100
- Continue all previous orders

Six hours later blood work showed the following:

CK - 500	Na - 135
LD - 190	K - 4.1
AST - 12	Cholesterol - 7.90
CKMB - 7%	PTT - 92

Questions to Case Study

1. Identify risk factors in Mr. Zabou's life which may have contributed to his myocardial infarction and complete the following table.

Risk Factor	Rationale and Associated Pathophysiology
Nonmodifiable	
Modifiable	

2. Compare Mr. Zabou's experience of "typical" anginal pain to the pain he experienced when he suffered a myocardial infarction.

	Angina	MI
Location		
Factors giving relief		

3. Discuss the rationale and effects for the following medications ordered. (You may need to use a pharmacology book to help you with this.)

   Thrombolytics:

   Cardizem:

   Captopril:

4. After 24 hours in CCU Mr. Zabou again developed midsternal chest pain (6/10). He had no radiation to other areas but stated it increased on deep inspiration and was very sharp. What might Mr. Zabou's chest pain be caused by?

5. What assessment data might help identify the reason for Mr. Zabou's pain.

6. Mr. Zabou's ECG showed an increase in ST segments.
   a. What does an elevation of ST segments indicate?

   b. Mr. Zabou is diagnosed as having an inferior wall MI. Which coronary artery is affected?

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Glossary

preload: determined by myocardial fibre length at end of diastole and before the beginning of a contraction

afterload: resistance to left ventricular ejection; amount of pressure that must be built up to overcome the forces that oppose ejection from the ventricle

akinesis: area of ventricle involved does not move at all

dyskinesis: area of ventricle involved expands and bulges during systole, paradoxical motion of ventricular wall

cardiac index: the cardiac output per square meter of the body surface area; normal is 25-40 litres/minute/meter^2

cardiac output: volume of blood pumped out of the ventricle in one minute normal is 5-6 litres/minute

contractility: refers to the ability of the heart to develop tension and/or shorten

automaticity: the ability of the heart to transmit an electrical impulse

heart failure: pathophysiological state in which the heart is unable to pump an adequate supply of blood for the metabolic needs of the body, provided there is adequate venous return to the heart

myocardial ischemia: a condition of oxygen deprivation to myocardial tissue accompanied by inadequate removal of metabolites secondary to tissue hypoperfusion; does not occur until the oxygen demand of myocardial tissue exceeds the oxygen supply

myocardial infarction: irreversible cellular injury and necrosis to a given region of the myocardium as a consequence of a prolonged reduction in coronary blood flow below a critical level

stroke volume: amount of blood pumped out of the ventricle during one contraction; normal is 65-86 ml/systolic period

Acronym List

ACE Angiotensin-converting enzyme

AMI Acute myocardial infarction

AV Atri-ventricular

BMI Body Mass Index

BP Blood pressure

CPK Creatine phosphokinase

CPK-MB Creatine phosphokinase myocardial band

EDV End diastolic volume

HF Heart failure

LAD Left anterior descending

LCA Left coronary artery

LCXA Left circumflex artery

LDH Lactate dehydrogenase

LMS Left main stem

MI Myocardial infarction

PTCA Percutaneous transluminal angioplasty

RCA Right coronary artery

SA Sino-atrial

SGOT Serum glutamic oxaloacetic transaminase

TPA Tissue type plasminogen activator

Answers to Learning Activities

Learning Activity #1—Case Study: A Person Suffering from a Myocardial Infarction

1. Answer

Risk Factor	Rationale and Associated Pathophysiology
Nonmodifiable	
Age	• increased incidence of MI with age • atherosclerosis build up over years
Sex	• males are more susceptible to MI at earlier age than females • males usually have more progressed atherosclerosis than fema up until females reach menopause where incidence equals. Female hormones protect women up until that time.
Family History	• father died at age 74 which is not very significant two brothers presented with cardiac symptoms

Risk Factor	Rationale and Associated Pathophysiology
Modifiable	
Weight gain (20 lbs)	• patient weighs 102 kg. • increase in weight or obesity causes an increase in cardiac workload and oxygen demand • weight gain is also associated with increased levels of LDLS, tendency to hypertension, and glucose intolerance
Smoking (second hand)	• the risk of second hand smoke is controversial and depends upon amount of smoke exposure • causes adrenergic stimulation • carbon monozide rather than oxygen is bound to hemoglobin therefore stimulating hyperplasia of the smooth muscle layer • causes peripheral and coronary vasoconstriction • associated with decreased HDL levels
Diet	• no specific diet followed -- "patient eats well" • ? increased intake of fats, cholesterol, sugar, salt and total calories
Exercise	• although patient is getting some exercise by walking his dog it does not indicate the level of exercise he achieves • exercise can help increase levels of HDL rather than LDL • there is a decreased incidence of CAD with well-conditioned persons
Stress	• patient feels responsible for employees • increase in stress causes the release of catecholamines • increase in circulating catecholamines leads to an increase in cardiac workload, thereby stressing the myocardium • see sympathetic nervous system stimulation

2. Answer

	Angina	**MI**
Location of pain	Substernal or across chest	Same as angina
Radiation of pain	Neck, jaw, arms, back	Same as angina
Nature of pain	Dull, heavy discomfort, etc. - pressure or squeezing	Same as angina but more intense
Duration	3-8 min. rarely longer	> 30 mins.
Other symptoms	None usually	Perspiration, weakness, nausea and vomiting, plae grey color
Precipitating factors	Extremes in weather, exertion, stress, meals. Does not usually occur at rest.	Often none
Factors giving relief	Stopping physical activity, reducing stress, Nitroglycerine	Nitroglycerine may give incomplete, or no relief.

3. Answer

Thrombolytics

- are "clot busters" that actually dissolve clots and therefore improve blood flow through the coronary arteries. The most common thrombolytics include streptokinase and t-PA. Not all patients meet the criteria to receive thrombolytics. Some new clot busting drugs are presently being tested.

Cardizem

- is a calcium channel blocker. It dilates coronary arteries and peripheral arterioles, therefore reducing total peripheral resistance (afterload), and decreasing blood pressure. Cardizem is also used to inhibit coronary artery spasm.
- also used to decrease heart rate.

Captopril

- is a angiotensin-converting enzyme (ACE) inhibitor. It inhibits the action of the renin-angiotensin-aldosterone system. Inhibition of ACE causes:

1. a decrease in vascular tone (therefore decreases blood pressure)
2. inhibits aldosterone release (therefore reduces sodium & water reabsorption, and leads to a decrease in blood pressure
3. an increase in renin activity (decreases blood pressure). Captopril has been reported to improve survival in postmyocardial patients with left ventricular dysfunction

4. Answer

Pericarditis. The pain Mr. Zabou is experiencing may be caused by the rubbing together of the pericardial sac lining and the outer lining of the heart.

5. Answer

1. Ausculation of heart sounds may identify a pericardial friction rub (squeaking sound) caused by the rubbing together of the heart wall linings.
2. Pain on deep inspiration due to the increased intrathoracic pressure.

6. (a) Answer

An elevated ST segment is indicative of cardiac injury.

(b) Answer

The artery which must be occluded is the right coronary artery (RCA).