CHAPTER 4 – CARDIOVASCULAR SYSTEM

First Nations and Inuit Health Branch (FNIHB) Clinical Practice Guidelines for Nurses in Primary Care.
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ASSESSMENT OF THE CARDIOVASCULAR SYSTEM

HISTORY OF PRESENT ILLNESS AND REVIEW OF SYSTEMS

The following characteristics of each symptom should be elicited and explored:

- Current situation (improving or deteriorating)
- Location
- Chronology
- Onset (sudden or gradual)
- Precipitating and aggravating factors
- Quality
- Radiation
- Severity
- Timing (frequency, duration)
- Relieving factors
- Associated symptoms
- Effects on daily activities
- Previous investigations and/or diagnosis of similar episodes
- Previous treatments
- Efficacy of previous treatments

CARDINAL SYMPTOMS

In addition to the general characteristics outlined above, additional characteristics of specific symptoms should be elicited, as follows.

Chest Pain

- Associated symptoms (for example, faintness, syncope, shortness of breath, diaphoresis, nausea, vomiting)
- Relation to effort, exercise, emotional state, meals, bending over

Shortness of Breath

- Relation to exercise (level ground, uphill, stairs)
- Relation to posture
- Orthopnea (number of pillows used for sleeping)
- Paroxysmal nocturnal dyspnea
- Associated swelling of ankles or recent weight gain

Fainting or Syncope

- Weakness, lightheadedness, loss of consciousness
- Associated symptoms (for example, pain, palpitations, shortness of breath, lightheadedness, nausea, sweating)
- Relation to postural changes, vertigo or neurologic symptoms

Palpitations

- Description: fast or slow, irregular or regular
- Relation to exercise
- Relation to anxiety or panic attack

Sputum

- Colour, amount
- Consistency (for example, frothy white, pink)

Cyanosis

- Observation of blue colour of the lips or fingers (under what circumstances, when first noted, recent change in this characteristic)

Extremities

- Site of edema (for example, in dependent body parts)
- Relation of edema to activity or time of day, (for example, relieved by rest, elevation of legs)
- Intermittent claudication (exercise-induced leg pain)
- Distance client can walk before onset of pain related to claudication
- Time needed to rest to relieve claudication
- Temperature of affected tissue (warm, cool or cold)
- Tingling
- Leg cramps or pain at rest
- Presence of varicose veins

MEDICAL HISTORY

- Increased cholesterol level
- Hypertension
- Coronary artery disease (angina)
- Myocardial infarction
- Cardiac murmurs
- Rheumatic fever
- Valvular heart disease
- Diabetes mellitus
- Thyroid disease
- Chronic renal disease
- Chronic obstructive pulmonary disease (COPD)
- Systemic lupus erythematosus
- Recent viral illness (for example, viral cardiomyopathy)
- Previous cardiac investigations (for example, echocardiogram, exercise stress test)

FAMILY HISTORY

- Diabetes mellitus
- Hypertension
- Coronary artery disease (ischemic) (especially in family members < 50 years of age)
- Myocardial infarction (especially in family members < 50 years of age)
- Sudden death from cardiac disease
- Hypercholesterolemia
- Hypertrophic cardiomyopathy

PERSONAL AND SOCIAL HISTORY

- Smoking
- Exposure to second-hand smoke
- Abdominal obesity (waist measurement > 100 cm [40 in] in men, > 90 cm [35 in] in women)
- Body mass index (BMI) > 25
- Lack of physical activity (< 20 minutes of vigorous activity 3 times a week, or < 30 minutes of moderate activity per day)
- High stress levels (personal or occupational)
- Chronic abuse of cocaine, amphetamines, anabolic steroids, solvents, ecstasy
- Alcohol abuse

PHYSICAL EXAMINATION

An examination of the cardiovascular system involves more than just examining the heart. The examination generally covers two systems: the central cardiovascular system (head, neck and precordium [anterior chest]) and the peripheral vascular system (extremities). Examination of the cardiovascular system must also include a full assessment of the lungs and neuromental status (for signs of confusion, irritability or altered level of consciousness).

VITAL SIGNS

- General appearance (for example, respiratory distress, acute pain, diaphoretic, unwell)
- Temperature
- Pulse (regular, regularly irregular or irregularly irregular)
- Respiratory rate
- Oxygen saturation
- Blood pressure (lying and standing, in both arms)
- Height, weight, BMI
- Waist circumference

HEAD AND NECK

- Central cyanosis
- Colour of lips
- Jugular venous pressure
- Carotid bruits

INSPECTION OF PRECORDIUM (ANTERIOR CHEST)

- Look for visible pulsations of the chest wall (point of maximal impulse)
- Look for accessory muscle use

PALPATION

- Location of apical beat (point of maximal impulse [PMI]) (in adults, normal apical beat is found in the 5th intercostal space, medial to the mid-clavicular line)
- Quality and intensity of apical beat (normal, diffuse, weak, forceful)
- Heave (abnormally forceful PMI)
- Thrill (a palpable murmur that feels like a purr)
- Identify and assess pulsations and thrills in aortic, pulmonic, mitral and tricuspid areas, along left and right sternal borders, in epigastrium and along left anterior axillary line

AUSCULTATION

- Listen to normal heart sounds before trying to identify murmurs and extra heart sounds
- Use diaphragm of stethoscope first, then bell of stethoscope, when listening to the heart
- Listen at apex, in aortic and pulmonic areas and along left sternal border
- Listen at bilateral carotid arteries for bruits

HEART SOUNDS

- Determine rate and rhythm
- Determine if there is an underlying rhythm or if rhythm is completely irregular (for example, regular, regularly irregular or irregularly irregular)
- Identify and describe intensity of first and second heart sounds
- Identify extra sounds (S_3, S_4, splitting of second sound, rubs)

MURMURS

- Timing (in relation to the cardiac cycle, for example, systolic or diastolic)
- Quality
- Intensity (loudness)
- Location where murmurs sound loudest
- Radiation
- Pitch

DIFFERENTIAL DIAGNOSIS OF ABNORMALITIES IN HEART SOUNDS

S_3 (Volume Overloaded Ventricle)

- May be normal in children and younger adults (< 30 years), however, some may not be and should have further assessment and investigations
- Left ventricular failure (due to systolic dysfunction, acute myocardial infarction)
- Mitral regurgitation (rapid ventricular filling)
- Right ventricular S_3 (due to tricuspid regurgitation, mitral stenosis, right ventricular failure)

To hear an example of S_3, refer to the 3M Heart and Lung Sounds page at: http://solutions.3m.com/wps/portal/3M/en_US/Littmann/stethoscope/education/heart-lung-sounds/.

S_4 (Pressure Overloaded Ventricle)

- Ischemia
- Ventricular hypertrophy from systemic hypertension, aortic stenosis, hypertrophic cardiomyopathy
- Right ventricular S_4 due to pulmonary hypertension or pulmonic stenosis

To hear an example of S_4, refer to the 3M Heart and Lung Sounds page at: http://solutions.3m.com/wps/portal/3M/en_US/Littmann/stethoscope/education/heart-lung-sounds/.

Other Extra Sounds

- Opening snaps (early diastolic sounds associated with mitral stenosis)
- Ejection clicks (associated with aortic stenosis, pulmonic stenosis)
- Mid-systolic clicks (mitral valve prolapse, tricuspid valve prolapse)
- Pericardial fraction rubs (pericarditis)

To hear an example of an ejection click and opening snap refer to the 3M Heart and Lung Sounds page at: http://solutions.3m.com/wps/portal/3M/en_US/Littmann/stethoscope/education/heart-lung-sounds/.

Systolic "Ejection" Murmurs (Diamond Shape, Crescendo-Decrescendo)

- Murmur gets louder and then quieter during one cardiac cycle
- Outflow obstruction (aortic stenosis, pulmonic stenosis, hypertrophic cardiomyopathy)
- High output or "flow" murmurs (accompanying anemia, pregnancy, thyrotoxicosis, fever, arteriovenous fistula and in young children)

To hear an example of a systolic murmur (early and late), refer to the 3M Heart and Lung Sounds page at: http://solutions.3m.com/wps/portal/3M/en_US/Littmann/stethoscope/education/heart-lung-sounds/.

Pansystolic Murmurs

- Mitral regurgitation
- Tricuspid regurgitation
- Ventricular septal defect

To hear an example of a pansystolic murmur, refer to the 3M Heart and Lung Sounds page at: http://solutions.3m.com/wps/portal/3M/en_US/Littmann/stethoscope/education/heart-lung-sounds/.

High-Pitched Diastolic Decrescendo Murmurs

- Aortic regurgitation
- Pulmonic regurgitation

Low-Pitched Diastolic Murmurs (Mid-Diastolic Rumbles)

- Mitral stenosis
- Tricuspid stenosis
- Continuous murmurs
- Patent ductus arteriosus
- Mammary souffle (goes away with pressure on stethoscope)
- Coronary arteriovenous fistula
- Venous hum (due to high flow in jugular veins, high cardiac output states)

To hear an example of a diastolic rumble, refer to the 3M Heart and Lung Sounds page at: http://solutions.3m.com/wps/portal/3M/en_US/Littmann/stethoscope/education/heart-lung-sounds/.

Bruits

- Carotid
- Abdominal
- Iliac
- Femoral

EXTREMITIES

Hands

- Colour of skin, nail beds
- Nicotine stains
- Clubbing of fingers
- Temperature
- Equality of pulses (brachial, radial)
- Synchrony of radial and femoral pulses
- Capillary refilling time

Legs

- Colour (pigmentation, discolouration), distribution of hair
- Temperature, texture
- Capillary refilling time
- Changes in foot colour with changes in leg position (for example, blanching with elevation, rubor with dependency)
- Ulcers (for example, venous stasis), varicose veins, pitting or nonpitting edema (check sacrum if client is bedridden)
- Presence and equality of pulses (femoral, popliteal, posterior tibial, dorsalis pedis)

OTHER ASSESSMENTS

For a client whose condition is not of an urgent nature, assess the following:

- Evidence of hypertensive or diabetic retinopathies (funduscopic exam)
- Colour, temperature, rashes, lesions, xanthoma of skin
- Abdominal bruits, enlargement of liver, tenderness in right upper quadrant of abdomen

DIFFERENTIAL DIAGNOSES OF CARDINAL CARDIOVASCULAR SYMPTOMS

CHEST PAIN

When assessing chest pain, it is important to consider and rule out serious causes of chest pain. *See Table 1, "Differential Diagnosis of Chest Pain."*

Table 1 – Differential Diagnosis of Chest Pain

	Differential Diagnosis of Chest Pain+				
Characteristic of Chest Pain	**Myocardial Infarction or Acute Coronary Syndrome**	**Angina**	**Aortic Dissection**[1]	**Pneumonia**	**Pulmonary Embolism***
Onset	Sudden, patient at rest	With exertion	Sudden	Gradual or sudden	Sudden
Location	Retrosternal, anterior chest	Retrosternal, anterior chest	Retrosternal, epigastric, back, posterior and/or anterior chest	Anterior, lateral and/or posterior lung field(s)	Retrosternal, anterior chest, lateral chest
Radiation	Left arm, left shoulder, neck, jaw, back, upper abdomen	Left arm, left shoulder, neck, jaw, back, upper abdomen	Anywhere in thorax or abdomen	Anterior chest, shoulder, neck	Variable
Duration	> 20 min	Usually <1–2 min	> 20 min	Hours	Variable
Intensity	Severe	Mild to moderate	Severe	Moderate	Absent or mild to moderate
Quality	Sensation of squeezing, pressure	Sensation of tightness, pressure	Tearing or sharp	Constant ache, with intermittent knife-like pain	Dull ache; knife-like pain may also be present
Relief	May be relief with sublingual nitroglycerin[2]	Rapid relief with rest and/ or sublingual nitroglycerin	None	None	None
Precipitating or aggravating factors	None may be obvious	Exertion, heavy meal, walking uphill against a cold wind, occasionally from lying down[3]	None	Increased pain with coughing or deep inspiration; recently ill with a cold	Immobilization, estrogen therapy, active cancer or cancer treatment;[4] none may be obvious; pain may be worse with deep inspiration or coughing
Associated signs and symptoms	Nausea, sweating, shortness of breath, anxiety, palpitations	Typically none	Hypotension, shock, syncope, myocardial infarction	Fever, cough, sputum, shortness of breath, malaise, weakness or fatigue	Shortness of breath, sweating, hemoptysis, leg pain (rare), leg swelling or pitting edema

+ other presentations are common among women (for example, epigastric pain or indigestion) and diabetics (for example, may have silent symptoms)

* Chest pain may be absent in pulmonary embolism

Table 1 – Differential Diagnosis of Chest Pain (Continued)

	Differential Diagnosis of Chest Pain+			
Characteristic of Chest Pain	**Pericarditis**	**Chest Wall Pain (for example, costochondritis)**	**Esophageal, Gastric or Duodenal Disorder**	**Stress or Emotional Disorder**
Onset	Gradual or sudden	Gradual or sudden	Gradual or sudden	Gradual or sudden
Location	Retrosternal, anterior chest	Anterior, lateral and/or posterior chest wall	Retrosternal, epigastric, left chest, left or right upper quadrant	Variable; anterior chest, left chest
Radiation	Variable: shoulder tip, neck	Arm, shoulder, neck, back, abdomen	May be felt in back or arm	Usually none
Duration	Hours to days	Minutes or hours	Minutes or hours	Minutes or hours
Intensity	Usually moderate, but may be severe	Mild to moderate	Moderate	Mild to moderate
Quality	Sharp	Dull ache; sharp pain may also be present	Burning (usually), tightness	Achy, stabbing
Relief	Sitting up and leaning forward often helps; other changes in position may alter the pain	Rest, mild analgesics	Antacids, milk, sitting up or standing up	Rest, relaxation, distraction
Precipitating or aggravating factors	Previous infection of upper respiratory tract; pain worse with deep inspiration or coughing	History of unaccustomed physical work; pain worse with arm action	Certain foods, a large meal, bending over; pain may awaken person from sleep and may occur when stomach is empty	Stressful situations, fatigue
Associated signs and symptoms	Symptoms of infection of upper respiratory tract may be present; malaise; usually occurs in younger adults	Localized chest-wall tenderness, tender costochondral area	Regurgitation of acid in mouth, belching, difficulty swallowing, sticking sensation when food swallowed, cough (rare); test of stool for occult blood may be positive	Tightness in neck and shoulder(s), headaches, reduced appetite, mild weight loss, fatigue, sleep disturbance, palpitations, dizziness, hyperventilation symptoms

+ other presentations are common among women (for example, epigastric pain or indigestion) and diabetics (for example, may have silent symptoms)

* Chest pain may be absent in pulmonary embolism

DYSPNEA

Cardiac Causes

- Congestive heart failure (right, left or biventricular)
- Coronary artery disease
- Myocardial infarction (recent or past history)
- Cardiomyopathy
- Valvular dysfunction
- Left ventricular hypertrophy
- Asymmetric septal hypertrophy
- Pericarditis
- Arrhythmias

Pulmonary Causes

- COPD/obstructive lung disease
- Asthma
- Restrictive lung disorders, such as pulmonary fibrosis
- Hereditary lung disorders
- Pneumothorax
- Primary pulmonary hypertension
- Pulmonary embolus
- Pneumonia

Mixed Cardiac and Pulmonary Causes

- COPD with pulmonary hypertension and cor pulmonale
- Deconditioning
- Chronic pulmonary emboli
- Trauma

Noncardiac or Nonpulmonary Causes

- Metabolic conditions (for example, acidosis)
- Pain
- Neuromuscular disorders
- Otorhinolaryngeal disorders

Functional Causes

- Anxiety
- Panic disorders
- Hyperventilation

FAINTNESS AND SYNCOPE

Faintness (pre-syncope) is characterized by transient symptoms of lack of strength associated with an impending sense of loss of consciousness. Syncope is characterized by transient symptoms of generalized weakness associated with loss of consciousness and loss of muscle tone. Symptoms are due to a temporary impairment of cerebral function and are usually precipitated by a reduction in cerebral perfusion.

Vascular Causes

- Vasovagal hypotension (common faint)
- Postural hypotension
- Cerebrovascular disease (transient ischemic attack, stroke, vertebral-basilar insufficiency, carotid insufficiency) is an uncommon cause of syncope

Neurological Causes

- Seizure
- Head trauma

Cardiac Causes

- Abnormally slow heart rate and rhythm
- Abnormally rapid heart rate and rhythm
- Reduced cardiac output
- Acute blood loss (gastrointestinal hemorrhage) causing hypotension
- Valvular heart disease (aortic or pulmonic stenosis, hypertrophic cardiomyopathy)
- Pulmonary hypertension

Other Causes

- Hypoglycemia
- Hyperventilation (syncope rare, faintness common)
- Hypoxia

PALPITATIONS

Primary Arrhythmic Causes

- Sinus tachycardia or arrhythmia
- Premature supraventricular or ventricular ectopic contractions
- Bradycardia-tachycardia syndrome ("sick sinus syndrome")
- Supraventricular tachycardia
- Multifocal atrial tachycardia
- Atrial fibrillation, flutter or tachycardia
- Atrioventricular nodal re-entrant tachycardia
- Atrioventricular reciprocating tachycardia (Wolff-Parkinson-White syndrome)
- Accelerated junctional rhythm
- Ventricular tachycardia
- Bradycardia due to advanced atrioventricular block or sinus node dysfunction

Extracardiac Causes

- Changes in contractility, heart rate or stroke volume
- Fever
- Hypovolemia
- Anemia
- Hypoglycemia
- Pulmonary disease
- Pheochromocytoma
- Thyrotoxicosis
- Vasovagal episodes

Drug-Related Causes

- Vasodilators
- Substance abuse (for example, cocaine, alcohol, tobacco, caffeine)
- Digoxin
- Phenothiazine
- Theophylline
- $Beta_2$-agonists
- Antiarrhythmics

Psychiatric Causes

- Panic attack
- Hyperventilation

Other Cardiac Causes

- Changes in contractility or stroke volume
- Valvular disease such as aortic insufficiency or stenosis
- Atrial or ventricular septal defect
- Congestive heart failure
- Cardiomyopathy
- Congenital heart disease
- Pericarditis
- Pacemaker-mediated tachycardia
- Pacemaker syndrome

LEG EDEMA

See Table 2, "Differential Diagnosis of Leg Edema."

Table 2 – Differential Diagnosis of Leg Edema

Mechanism	Disease or Syndrome	Usual Clinical Features
Increased capillary pressure Obstruction of inferior vena cava	Thrombosis, malignancy	Bilateral, severe (may be mild if partial obstruction)
Deep venous obstruction in leg	Thrombosis, extrinsic compression	Unilateral, mild
Reduced venous channels or venous valve incompetence	Coronary bypass grafting, stroke, varicosities	Unilateral or bilateral, mild
Right atrial hypertension	Left ventricular dysfunction	Bilateral
	Pulmonary disease	Bilateral
	Valve disease	Bilateral
	Renal dysfunction	Bilateral, mild
Reduced lymphatic clearance (lymphatic obstruction)	Lymphadenopathy, filariasis	Unilateral or bilateral
Decreased capillary oncotic pressure (hypoalbuminemia)	Severe malnutrition; liver, renal, gastrointestinal disease	Bilateral, mild or severe, generalized, poor prognosis
Increased capillary permeability	Calcium channel blockers	Bilateral, mild
	Idiopathic cyclic edema	Bilateral, mild, premenstrual female

COMMON PROBLEMS OF THE CARDIOVASCULAR SYSTEM

AORTIC ANEURYSM, ABDOMINAL (PULSATILE ABDOMINAL MASS)

A pulsatile abdominal mass is considered and treated as an abdominal aortic aneurysm until proven otherwise. It may be asymptomatic and discovered by accident.

An aortic aneurysm is a dilation of a specific part of the aorta. This may be in the thorax or in the abdomen.

RISK FACTORS[5]

- Age over 60 years
- Smoking
- Male
- Family history of aortic aneurysm
- History of atherosclerosis (for example, intermittent claudication)
- Hypertension

HISTORY

If an aneurysm is leaking:

- Sudden onset of pain in mid-abdomen or back (or both)
- Sudden weakness and faintness

PHYSICAL FINDINGS

- Pulse rapid and weak
- Blood pressure low-normal to low
- Blood pressure may drop with change in posture
- Pulsating mid- or upper-abdominal mass

If an aneurysm has ruptured:

- Shock (hypovolemia)
- In severe distress, client may be unconscious
- Pulse diminished or absent
- Blood pressure low or cannot be determined
- A pulsating abdominal or flank mass may be palpable
- Subcutaneous bruising may be present
- Death usually occurs

MANAGEMENT OF ASYMPTOMATIC CLIENT

Goals of Treatment

- Identify and monitor the asymptomatic abdominal aneurysm

Appropriate Consultation

Consult a physician when an asymptomatic aortic aneursym is suspected or detected.

Monitoring and Follow-Up

- Annual follow-up by physician
- Annual abdominal ultrasonography to measure size

Referral

Referral for vascular surgery (depending on size of the aneurysm) will usually be done by a physician.

MANAGEMENT OF SYMPTOMATIC CLIENT

This is a medical emergency.

Goals of Treatment

- Replace blood loss

Appropriate Consultation

Consult a physician immediately after intravenous access is established and oxygen is started.

Adjuvant Therapy

- Oxygen via non-rebreather mask; titrate flow to keep oxygen saturation ≥ 90%
- IV (16- to 18-gauge) with normal saline (or lactated Ringer's solution)
- Insert the needle into the largest vein available
- Start a second IV line for rapid fluid replacement if client is in shock (*See section "Shock" in Chapter 14, "General Emergencies and Major Trauma"*)
- Insert a nasogastric tube, if upon consultation a physician supports its use (because paralytic ileus is common)
- Insert a urinary catheter (optional unless transfer delayed)

Nonpharmacologic Interventions

- Bed rest
- Maintain "nothing-by-mouth" order

Monitoring and Follow-Up

- Monitor ABC (airway, breathing and circulation) and vital signs closely, including oxygen saturation
- Aim for pulse < 100 bpm and systolic blood pressure (BP) > 100 mm Hg
- Monitor urinary output

Referral

Medevac as soon as possible.

ARRHYTHMIAS

Abnormal heart rhythm. The following are the most common types.

SINUS BRADYCARDIA

Heart rate < 60 bpm; impulse originates in sinoatrial (SA) node.

BRADYCARDIA

Multiple causes which may include high grade atrioventricular blocks; junctional escape rhythms; heightened vagal tone; hyperkalemia.

SINUS TACHYCARDIA

Heart rate > 100–160 bpm; impulse originates in SA node.

SUPRAVENTRICULAR TACHYDYSRHYTHMIAS

Heart rate > 100 bpm; impulse originates above the ventricles. There are two major types:

- *Atrioventricular (AV) nodal re-entrant tachycardia* is intranodal re-entry by means of fast and slow conduction pathways within the AV junction
- *Orthodromic AV re-entrant tachycardia* is tachycardia across accessory pathways associated with pre-excitation

ATRIAL FIBRILLATION

Chaotic electrical activity caused by rapid discharges from numerous ectopic foci in the atria. Atrial rate is difficult to count. There are two types of atrial fibrillation:

- *Paroxysmal atrial fibrillation* occurs in people who usually have normal sinus rhythm
- *Chronic atrial fibrillation* occurs in people who have a permanent fibrillation rather than brief episodes of symptoms

PREMATURE VENTRICULAR CONTRACTIONS[6]

Extra impulses that form within the purkinje fibres and result in an extra heart beat. They are very common and usually benign.

PREDISPOSING FACTORS

Predisposing Factors for Bradycardia

- Increased vagal tone
- Decreased sympathetic drive
- Ischemia to sinoatrial node
- Drugs: digoxin, beta-blockers (atenolol, metoprolol, propranolol), calcium channel blockers, amiodarone and other antiarrythmic drugs
- Alcohol consumption
- Athletic activity (normal variant in athletes)
- Injury or other insult (normal body response)
- Atrial enlargement
- Acute myocardial infarction
- Congestive heart failure
- Rheumatic heat disease
- Hypertensive heart disease
- Hypothermia
- Electrolyte abnormality
- Acidosis
- Infection

Predisposing Factors for Tachycardia

- Decreased vagal tone
- Increased sympathetic tone
- Myocardial infarction
- Drugs (caffeine, nicotine, illicit drugs)[7]

Predisposing Factors for Supraventricular Tachycardia

- Digoxin toxicity
- Catecholamines

Predisposing Factors for Atrial Fibrillation

- Myocardial ischemia
- Thyrotoxicosis

Predisposing Factors for Premature Ventricular Contractions

- The prevalence of PVC is higher in:[8]
  - Men than women
  - African Americans than Whites
  - Patients with organic disease
- Prevalence increases with age
- Prevalence increases with hypokalemia, hypomagnesemia, hypertension and in those with a faster sinus rate
- Stress
- Fatigue

HISTORY

- Symptoms may not be present; however, client may note irregular heartbeat
- Palpitations
- Chest discomfort
- Shortness of breath
- Dizziness
- Diaphoresis
- Weakness
- Syncope
- Nausea
- Drug history (with particular attention to those that affect heart rate)

PHYSICAL FINDINGS

- *Sinus bradycardia*: pulse regular but decreased (< 60 beats per minute [bpm]); blood pressure normal or low; sinus rhythm on ECG (rate < 60 bpm)
- *Sinus tachycardia*: pulse regular but increased (> 100 bpm); systolic blood pressure may be normal, low or elevated; sinus rhythm on ECG (rate >100 bpm)
- *Atrial fibrillation*: pulse irregularly irregular, rate variable; systolic blood pressure variable; narrow complex ECG (absent p waves, rate variable)
- *Atrial flutter*: atrial waves present as “sawtooths” most commonly at 300 bpm; may be a regular block present such as a 2:1 atrial ventricular block so the ventricular rate is regular at 150 bpm

- *Supraventricular tachycardia*: pulse regular and fast; blood pressure normal to low; regular narrow complex tachycardia on ECG (rate so rapid it is hard to see P waves)
- *Premature ventricular contractions and premature atrial contractions*: pulse regular with occasional extra beat and pauses; blood pressure unaffected; ECG: sinus rhythm with extra narrow or wide complex beats

DIFFERENTIAL DIAGNOSIS

- Multifocal atrial tachycardia
- Sinus tachycardia with multiple premature atrial contractions
- Atrial flutter
- Ventricular tachycardia
- Atrioventricular block
- Wolff-Parkinson-White syndrome

COMPLICATIONS

- Heart failure
- Myocardial infarction
- Cerebrovascular accident, stroke
- Thromboembolism
- Life-threatening arrhythmia

DIAGNOSTIC TESTS

- Obtain ECG
- 24-hour Holter monitoring (must be ordered by a physician)
- Determine level of thyroid-stimulating hormone (TSH)
- Check digoxin level if patient is on digoxin
- Check electrolytes including magnesium
- Obtain complete blood count
- Determine international normalized ratio (INR), partial thromboplastin time (PTT)

MANAGEMENT

Goals of Treatment

- Treat underlying condition
- Relieve symptoms
- Prevent recurrence
- Prevent complications

Appropriate Consultation

Consult a physician if client has abnormal ECG pattern, new or refractory atrial fibrillation, suspicion of Wolff-Parkinson-White or "sick sinus" syndrome.

Nonpharmacologic Interventions

Client education

- Teach client some relaxation techniques
- Teach client and family members the signs of hemodynamic compromise, including rapid heart rate, unexplained weight gain, worsening dyspnea on exertion, decreased exercise tolerance
- Teach client about long-term medication and its side effects
- Identify and remove any contributing factors (for example, reduce caffeinated beverages)

Pharmacologic Interventions

Initial treatment prescribed only by a physician.

Selection of treatment modality should be based on underlying pathophysiology.

Chronic atrial fibrillation is associated with stroke and clients should be offered treatment with anticoagulants such as warfarin (Coumadin) if the benefits outweigh the increased risks.

Monitoring and Follow-Up

- For clients taking antiarrhythmic agents, liver enzyme levels should be measured during first 4–8 weeks of therapy
- Clients with risk factors for cardiac complications of therapy should undergo ECG during first weeks of therapy and every 3–6 months thereafter
- Clients taking digoxin should be monitored carefully for toxic effects
- Evaluate INR on a regular basis to monitor therapeutic response to warfarin

Referral

Medevac clients with hemodynamic instability.

ARTERIAL PERIPHERAL VASCULAR DISEASE, CHRONIC

Decreased blood flow to one or more extremities, primarily lower, leading to ischemia of the leg muscles.

CAUSES

- Atherosclerotic narrowing of the aorta and larger arteries supplying the lower limb

RISK FACTORS

- Major: smoking, diabetes, hyperchromocysteinemia
- Minor: hypertension, hyperlipidemia, obesity, family history, sedentary lifestyle, male gender

HISTORY

- Symptoms initially intermittent, reversible, reproducible
- Intermittent claudication: pain, ache, cramp located in calf, instep, buttock, hip or thigh (rarely in an arm)
- Pain precipitated by exercise
- Discomfort quickly and consistently relieved with rest (in 2–5 minutes)
- Distance client can walk before experiencing claudication (should be documented)
- As disease progresses, symptoms occur with less effort and last longer
- With advanced disease, foot pain occurs at night (nocturnal pain)
- Nocturnal pain relieved by placing the leg into a dependent position or by standing on a cold floor
- With severe disease the involved area becomes chronically ischemic, and pain is present during rest
- Impotence may occur
- Associated vascular disease of other target organs may be present (angina, previous stroke or transient ischemic attacks)

PHYSICAL FINDINGS

- Hypertension
- Ischemic skin changes in foot and distal limb may be present (thin, fragile skin; loss of hair on distal leg; shiny and atrophic skin; leg muscle atrophy)
- Arterial ulcers on toes or feet
- Toenails may be hypertrophic
- Rubor of foot with dependency, blanching of foot with elevation
- Capillary refilling time slowed (> 2 seconds)
- Peripheral pulses decreased or absent
- Pulsating abdominal mass may be present (aortic aneurysm)
- Arterial bruits may be present (abdominal aortic, iliac, femoral, popliteal)

DIFFERENTIAL DIAGNOSIS

- Acute arterial occlusion
- Raynaud's disease (Raynaud's phenomenon)
- Venous stasis (for example, varicose veins)
- Osteoarthritis
- Neurogenic claudication due to lumbar spinal stenosis or spinal radiculopathy
- Popliteal entrapment (for example, tumour, Baker's cyst)

COMPLICATIONS

- Ischemic ulcer
- Infection of ischemic ulcer
- Loss of distal ischemic limb
- Acute arterial occlusion

DIAGNOSTIC TESTS

- Ankle-brachial index (ABI < 0.90 is abnormal)
- Arterial doppler
- Arteriography (gold standard for diagnosis)

MANAGEMENT

Goals of Treatment

- Slow progression of disease
- Identify, modify and treat risk factors (diabetes mellitus, hypertension, hyperlipidemia)
- Promote formation of collateral circulation
- Prevent complications

Appropriate Consultation

Consult a physician immediately if any of the following are present: ischemic ulcer, pain at rest, nocturnal pain, recent transient ischemic attack and/or pulsatile abdominal mass.

Nonpharmacologic Interventions

Client education

- Recommend strongly that client stop smoking
- Recommend weight loss (if appropriate)
- Recommend daily exercise to improve fitness and exercise tolerance of the leg muscles, which will also help to improve collateral circulation (walking is the best exercise)
- Recommend that client elevate the head of the bed (using 5- to 8-cm [2- to 3-in] wooden blocks)
- Recommend that client keep feet and legs cool while sleeping

- To reduce skin irritation, client should put sheepskin or bubble pads on the bed
- Teach proper foot care: avoid clipping nails too close to the skin, avoid tight-fitting shoes, keep feet dry and protected from injury (no slippers or bare feet, even in the house)
- For diabetic clients, teach proper foot care to a family member, if possible, so that this person can carry out the necessary tasks; alternatively, have the client attend a clinic on a monthly basis for care of nails and feet

Pharmacologic Interventions

Consult a physician regarding an antiplatelet agent (ASA or clopidogrel) and analgesia if there are significant symptoms at time of diagnosis.

Monitoring and Follow-Up

- Recommend follow-up every 6 months to identify new symptoms or changes in existing symptoms
- Advise client to return to the clinic if a leg/foot injury occurs, no matter how small
- With conservative therapy 60–80% will improve, 20–30% will stay the same, 5–10% deteriorate, 5% will require intervention within 5 years, and < 4% will require amputation

Referral

Refer to a physician as soon as feasible if there is evidence of advanced disease (for example, intolerable pain, pain at rest, nocturnal pain, foot ulcers and impending gangrene). A consult with a vascular surgeon may be necessary. All patients with risk factors and symptoms consistent with intermittent claudication should be assessed by a physician for appropriate investigations.

ATRIAL FIBRILLATION

Atrial fibrillation is a cardiac arrhythmia in which chaotic electrical activity replaces the orderly activation sequence of normal sinus rhythm.

ASSOCIATED CONDITIONS[9]

- Hypertensive heart disease
- Valvular or rheumatic heart disease
- Coronary artery disease
- Acute myocardial infarction
- Pulmonary embolus
- Cardiomyopathy
- Congestive heart failure
- Infiltrative heart disease
- Pericarditis
- Hyperthyroidism (uncontrolled)
- Obstructive pulmonary disease (for example, COPD)
- Pulmonary hypertension
- Obesity

HISTORY

- Palpitations
- Lightheadedness
- Poor exercise capacity
- Fatigue
- Dyspnea
- Angina
- Syncope or near syncope
- Stroke
- Arterial embolization

PHYSICAL FINDINGS

Do a complete cardiovascular and respiratory examination. Also assess the eyes for lid lag (hyperthyroid sign) and the neck for thyroid enlargement and elevated jugular venous pressure (JVP). The pulse should be irregularly irregular and is usually tachycardic. The client may exhibit signs of heart failure and/or hypotension.

DIFFERENTIAL DIAGNOSIS

- Multifocal atrial tachycardia
- Sinus tachycardia with frequent atrial premature beats
- Atrial flutter

COMPLICATIONS

- Embolic stroke
- Peripheral arterial embolization
- Complications of pharmacologic therapy, including bradycardic arrhythmias
- Inherent risk of bleeding with anticoagulation

DIAGNOSTIC TESTS

For asymptomatic people:

- ECG (rapid, irregular, narrow QRS complexes with no P waves)
- Thyroid-stimulating hormone (TSH)
- International normalized ratio (INR) and partial thromboplastin time (PTT)
- Chest x-ray if there are concerns about heart failure or cardiomegaly
- Echocardiogram
- Exercise tolerance test

MANAGEMENT

Goals of Treatment

- Search for and treat all predisposing factors (*See section "Associated Conditions" in this chapter*)
- Reduce symptoms
- Prevent complications

Appropriate Consultation

Consult a physician for a symptomatic client as soon as possible.

NONPHARMACOLOGIC INTERVENTIONS

Client education

- Ensure that client understands disease process and prognosis
- Teach client signs and symptoms of complications that require immediate follow-up (rapid heart rate, palpitations, edema, shortness of breath on exertion, chest pain)
- Recommend avoidance of alcohol, caffeine
- Recommend smoking cessation (if applicable)
- Counsel client to avoid sleep deprivation

Pharmacologic Interventions

Drug therapy is directed at controlling the ventricular rate, and in some cases of new onset atrial fibrillation, converting the client to sinus rhythm. A wide variety of medications are used, such as beta-blockers (for example, atenolol, metoprolol) or calcium channel blockers (for example, diltiazem, verapamil). These medications must be prescribed by a physician.

In clients with chronic atrial fibrillation, long-term anticoagulation may be advised to prevent thromboembolic complications, depending on the age of the patient and other comorbid conditions such as hypertension. Warfarin is usually used for anticoagulation, but in some patients an antiplatelet agent such as ASA may be sufficient or preferable. Before any cardioversion with drugs or electricity, a client must be adequately anticoagulated to prevent a thromboembolic event.

Counsel client about appropriate medication use, including side effects.

Monitoring and Follow-Up

- Clients with stable atrial fibrillation should be followed up regularly to assess for symptoms and signs of recurrence, complications, compliance with therapy and side effects of medication
- Clients on anticoagulation must have INR levels monitored regularly (target INR level is usually 2–3; frequency of INR monitoring is individual, depending on response to drug)

Referral

Refer the stable symptomatic client to a physician for thorough evaluation and initiation of therapy as soon as possible.

Medevac clients who are hemodynamically unstable. Electrical cardioversion in hospital is sometimes necessary if symptoms are severe.

CONGESTIVE HEART FAILURE

A clinical syndrome caused by an accumulation of fluid peripherally (right ventricular failure) or in the lungs (left ventricular failure), or both, from inadequate functioning of the heart. Congestive heart failure is a complication of an underlying disease process.

Systolic heart failure (the more common form) is due to impaired systolic pumping action of the heart. Diastolic heart failure occurs when the systolic function is normal but the filling of the heart is impaired.

NEW YORK HEART ASSOCIATION (NYHA) FUNCTIONAL CLASSIFICATION OF CHRONIC HEART FAILURE[10]

Clients with cardiac dysfunction (ejection fraction < 40%, grade II-IV left ventricular dysfunction or dilatation) and

Class I

- No symptoms
- No undue fatigue or shortness of breath with ordinary physical activity, such as walking or climbing stairs

Class II

- Symptoms of heart failure occur with ordinary physical activity
- Undue fatigue or shortness of breath with emotional stress or on walking or climbing stairs rapidly, walking uphill, walking or climbing stairs after meals or walking in cold air or wind

Class III

- Symptoms of heart failure with less than ordinary physical activity
- Undue fatigue and shortness of breath on walking one or two blocks on level ground, or climbing more than one flight of stairs in normal conditions

Class IV

- Symptoms of heart failure at rest
- Inability to carry out any physical activity without fatigue, discomfort or shortness of breath

CAUSES[10]

- Drugs[11]
  - Negative inotropic medications (for example, beta-blockers, calcium channel blockers, antiarrhythmics)
  - Drugs that cause sodium and water retention (for example, NSAIDs, corticosteroids, thiazolidinediones)
  - Cardiotoxic drugs (for example, alcohol, cocaine, certain chemotherapeutic agents)
- Myocardial ischemia or infarction (60–70%)
- Myocarditis
- Hereditary hypertrophic, hypertrophic obstructive, restrictive or peripartum cardiomyopathy
- Infiltrative diseases (for example, hemochromatosis)
- Valvular disease
- Substance abuse
- Connective tissue disease
- HIV infection
- Hypertension

CAUSES OF EXACERBATIONS

Increased Myocardial Demand

- Arrhythmias
- Infection or fever
- Anemia
- Hyperthyroidism
- Hypertension
- Pregnancy
- Stress (physical, environmental or emotional)

Compliance and Lifestyle

- Inadequate or improper medication intake
- Dietary indiscretion (for example, excess consumption of salt or water in a client with underlying cardiac or renal disease)
- Heavy alcohol consumption

Salt and Water Retention

- Medications: nonsteroidal anti-inflammatory drugs (NSAIDs), steroids
- Renal disease

Decreased Pump Function of the Ventricles

- Negative inotropic medications: beta-blockers, calcium channel blockers, antiarrhythmics, certain chemotherapeutic agents
- Arrhythmias
- Myocardial ischemia or infarction (60–70%)
- Valvular heart disease
- Hereditary hypertrophic cardiomyopathy
- Pulmonary embolism
- Radiation treatment
- Chronic obstructive pulmonary disease
- Pulmonary hypertension
- Infiltrative diseases (for example, hemochromatosis)

HISTORY

- Shortness of breath (initially induced by exercise)
- Later progression to orthopnea, paroxysmal nocturnal dyspnea and dyspnea at rest
- Nocturnal angina symptoms
- Chronic, nonproductive cough, worse at night or when lying down

- Ankle edema
- Recent weight gain
- Nocturia
- Chronic fatigue
- Palpitations
- Symptoms of intercurrent illness (for example, pneumonia)
- Anxiety may aggravate condition
- Alterations of mental status in elderly clients may be present as chronic heart failure progresses

PHYSICAL FINDINGS

- There is a broad range in severity of findings
- Heart rate elevated
- Respiratory rate increased
- Hypoxia may be present
- Blood pressure may be normal, elevated or low
- Weight increased (reflecting fluid retention)
- Minimal to extreme distress when client lies down
- Jugular venous distention may be present
- Jugular venous pressure elevated (> 3 cm)
- Edema may be present (pedal, ankle or tibial; sacral if bedridden)
- Hepatomegaly
- Hepatojugular reflux
- Ascites (rare)
- Lung bases may be dull (pleural effusion) bilaterally, but only rarely. More commonly congestive heart failure causes a unilateral pleural effusion
- Fine crackles in the bases of lungs
- S_3, S_4 or gallop rhythm may be present; murmurs may be present if there is associated valvular dysfunction

DIFFERENTIAL DIAGNOSIS

- See section *"Causes of Exacerbations"*
- Acute exacerbation of chronic obstructive pulmonary disease (COPD) or asthma
- Other causes of edema (renal disease, liver disease, local venous stasis, lymphedema)
- Pulmonary embolism

COMPLICATIONS

- Arrhythmias
- Hepatomegaly
- Ascites
- Acute pulmonary edema
- Respiratory failure, respiratory arrest
- Hypokalemia from use of diuretics
- Angina or myocardial infarction
- Decreased renal function, decreased renal clearance of drugs that are renally eliminated (digoxin toxicity if the dosage is not adjusted)
- Pulmonary embolism
- Side effects of medication

DIAGNOSTIC TESTS

Do the following diagnostic tests only if the person is not ill enough to require hospitalization and if not conducted within the past 3 months:

- Perform ECG and compare with any previous tracings (look for signs of ischemia [depression or elevation of ST segment, inversion of T wave], atrial fibrillation, bradycardia)
- Complete blood count
- Blood glucose level
- Lipids
- Thyroid function
- Liver function
- Ferritin level
- Creatinine level
- Electrolyte levels
- Digoxin level (if applicable)
- Chest x-ray (for cardiomegaly, pulmonary edema, pleural effusions), if available
- Echocardiogram
- Exercise tolerance test

MANAGEMENT

MANAGEMENT OF CHRONIC HEART FAILURE

Goals of Treatment

- Control symptoms
- Identify and manage underlying cause
- Limit risk factors that precipitate or aggravate condition
- Prevent progression
- Improve quality of life and survival

Because there is a broad range of severity *(see section "NYHA Functional Classification of Chronic Heart Failure" in this chapter)*, assessment of severity will help guide management. Definitive and precise medical management depends on whether the failure is due to systolic or diastolic dysfunction and the underlying or precipitating cause (for example, atrial fibrillation).

Appropriate Consultation

Consult a physician as soon as possible.

Adjuvant Therapy

- Pneumococcal vaccine every 5 years
- Influenza vaccine annually

Nonpharmacologic Interventions

Client education

- Ensure client understands disease process and outcome (progressive, can be controlled but not cured)
- Recommend dietary modifications: reduce sodium (to 2 g/day), increase dietary potassium (if renal function has been adequate in the past), reduce fat and cholesterol
- Recommend reduction in fluid intake to 1.5–2 L/day
- Recommend restriction of alcohol use
- Recommend weight loss, if applicable
- Recommend that client monitor weight at home, and see the nurse if he or she gains more than 1.5 kg (3 lb) in a day
- Recommend rest after meals
- Encourage client to start an exercise program (walking) to improve exercise tolerance
- Stress the importance of long-term follow-up (every 3–6 months when stable)
- Teach clients taking digoxin to monitor pulse

Pharmacologic Interventions

Several classes of drugs are used to manage congestive heart failure (Table 3). Medications used depend on symptom level *(see section "NYHA Functional Classification of Chronic Heart Failure" in this chapter)*, left ventricular ejection fraction and individual client variables. All of these medications must be prescribed by a physician.

Table 3 – Drugs to manage congestive heart failure[11]

Evidence-based drugs and oral doses as shown in large clinical trials		
Drug	**Start dose**	**Target dose**
ACE inhibitor		
Captopril	6.25 mg to 12.5 mg tid	25 mg to 50 mg tid
Enalapril	1.25 mg to 2.5 mg bid	10 mg bid
Ramipril	1.25 mg to 2.5 mg bid	5 mg bid*
Lisinopril	2.5 mg to 5 mg od	20 mg to 35 mg od
Beta-blocker		
Carvedilol	3.125 mg bid	25 mg bid
Bisoprolol	1.25 mg od	10 mg od
Metoprolol CR/XL†	12.5 mg to 25 mg od	200 mg od
ARB		
Candesartan	4 mg od	32 mg od
Valsartan	40 mg bid	160 mg bid
Aldosterone antagonist		
Spironolactone	12.5 mg od	50 mg od
Eplerenone†	25 mg od	50 mg od
Vasodilator		
Isosorbide dinitrate	20 mg tid	40 mg tid
Hydralazine	37.5 mg tid	75 mg tid

* The Healing and Early Afterload Reducing Therapy (HEART) trial (165) showed that 10 mg once a day (od) was effective for attenuating left ventricular remodelling.

† Not available in Canada.

ACE Angiotensin-converting enzyme; ARB Angiotensin receptor blocker; bid Twice a day; CR/XL Controlled release/extended release; tid Three times a day

All patients with a left ventricular ejection fraction ≤ 40% should receive an ACE inhibitor and a beta-blocker. They are first-line therapy for all patients with heart failure (NYHA class II-IV), unless contraindicated or not tolerated.[11]

ACE inhibitors (Table 3) reduce symptoms, improve quality of life, slow disease progression of heart failure, reduce hospitalizations and decrease mortality in patients with heart failure. When used in combination with an ACE inhibitor, beta-blockers reduce hospitalizations and decrease mortality.

ARBs are alternatives for clients who cannot tolerate ACE inhibitors.

Loop diuretics (for example, furosemide) are used to control symptoms of congestive heart failure. Once congestion has resolved the dose should be reduced to a level that results in stable symptom control (monitor weight, serum potassium and renal function).

Aldosterone antagonists (for example, spironolactone) has been shown to reduce mortality in people with severe heart failure receiving optimal therapy (for example, ACE inhibitors, ARBs, beta-blockers and/or diuretics).[12,13] Monitor serum potassium closely in patients receiving spironolactone in combination with ACE inhibitors and/or ARBs because all of these drugs cause potassium retention.

The combination of hydralazine plus oral isosorbide dinitrate (a nitrate) improves symptoms and may reduce mortality. It is generally used only in African Americans and in those unable to tolerate standard therapy.[11]

Nitrates in all forms (topical and oral) may be used as an adjunct to the above therapies. They can improve symptoms and exercise tolerance and are useful for clients who have a component of myocardial ischemia. A nitrate-free period of 10–12 hours per day is required to prevent loss of effect.

Cardiac glycosides (for example, digoxin) are used to control ventricular rate in clients with atrial fibrillation. They can also improve symptoms and exercise tolerance and reduce hospital admissions, especially in those with severe ventricular dysfunction.

Anticoagulation is strongly recommended for all clients with heart failure and associated atrial fibrillation.

Counsel client about appropriate use of medications (dose, frequency, compliance, side effects).

Long-Term Monitoring and Follow-Up

- Review cardiac and respiratory systems for symptoms and signs
- Weigh client and chart weight every visit
- Monitor blood pressure, renal function and serum potassium
- Review current medications for use, dosage, frequency, compliance, side effects, drugs with sodium-retaining effects (for example, NSAIDs)
- Instruct client to return to clinic if symptoms worsen or chest pain develops
- Laboratory tests every 3–6 months: complete blood count, creatinine level, electrolyte levels, uric acid level (if taking a thiazide diuretic), urinalysis for proteinuria, digoxin level
- Annual screening of blood glucose and lipids, TSH

Referral

Refer client to a physician for a thorough evaluation and tailoring of drug therapy regimen.

MANAGEMENT OF ACUTE DECOMPENSATED HEART FAILURE

Appropriate Consultation

Consult a physician immediately.

Adjuvant Therapy

- Oxygen via non-rebreather mask; titrate flow to keep oxygen saturation ≥ 90%
- Start intravenous (IV) therapy with normal saline to keep vein open

Nonpharmacologic Interventions

Bed rest with head elevated and legs hanging down (unless client is hypotensive).

Pharmacologic Interventions

Fluid removal with IV loop diuretics can relieve symptoms and improve oxygenation status.[14]

Diuretics:

furosemide (Lasix), 40–80 mg IV

The dose may have to be higher in a person on an oral maintenance dose. It is reasonable to administer an initial dose that is equivalent to the client's usual maintenance dose.[14] Adjust the diuretic dose according to client's response (monitor urine output). Look for improvement in respiratory status.

Nitrates can relieve congestive symptoms in patients without hypotension. Intravenous nitroglycerin is preferred because of its rapid onset of action and the ability to rapidly titrate the dose,[14] but may be impractical to administer in the nursing station. If it is used, it is to be ordered by a physician. For this reason nitroglycerin patches (or ointment) may be used. Nitroglycerin is rapidly absorbed after application of a patch (steady state is achieved within 30 minutes and is maintained for 24 hours). The plasma concentration decreases to zero within 2 hours of removal.[15]

nitroglycerin (Nitro-Dur) transdermal patches, 0.2- 0.8 mg/h

Sublingual nitroglycerin may be useful for rapid relief of chest pain:

sublingual nitroglycerin, 0.4 mg

Monitoring and Follow-Up

- Monitor vital signs, pulse oximetry (if available)
- Airway, breathing and circulation (ABC)
- Level of consciousness
- Listen to heart and lung sounds
- Record intake and urinary output
- Monitor response to therapy

Referral

Medevac as soon as possible.

DEEP VEIN THROMBOSIS

Acute formation of a blood clot or thrombus within a vein resulting in obstruction of venous return.

CAUSES

Unknown, but the triad of venous stasis, injury to vessel intima and altered blood coagulability are central to the process.

Risk Factors

- Prolonged bed rest for any reason
- Recent prolonged air travel
- Paralysis
- Malignant disease
- Childbirth
- Pregnancy
- Drugs
  - Oral and transdermal contraceptives
  - Hormone replacement therapy
  - Tamoxifen
  - Bevacizumab (Avastin), an anticancer agent
- Trauma
- Major surgery
- Infection after orthopedic surgery
- Acute myocardial infarction
- Stroke
- Old age (related to decreased activity)
- Hypercoagulable states (for example, chronic infection, autoimmune disease, primary blood diseases like antithrombin III deficiency)

HISTORY

- Symptoms may be subtle, variable or vague
- Usually occurs in leg or deep pelvic veins (popliteal, femoral, iliac)
- Presence of one or more risk factors (*see "Risk Factors" in this section*)
- Recent leg injury
- Leg pain may be mild or absent
- Pain described as a dull ache or tightness, rarely severe
- Leg discomfort worse when walking
- Swelling of lower leg
- Fever

Symptoms may be absent or minimal until shortness of breath and other pulmonary complaints appear because of embolism to the lungs. The risk of pulmonary emboli is low when only the calf veins are involved but increases to 40% when the thigh veins are involved.

PHYSICAL FINDINGS

- Variable; dependent on size and location of clot and severity of venous obstruction
- Heart rate may be elevated
- Minimal to moderate distress
- Difficulty walking
- Minimal to marked swelling of lower leg
- Redness of affected calf or leg may be present
- Superficial leg veins may be distended
- Mild to moderate calf tenderness: flexion of the ankle may increase pain
- Localized warmth may be present
- Peripheral pulses (compare sides for symmetry)

DIFFERENTIAL DIAGNOSIS

– Calf-muscle strain
– Trauma with hematoma
– Thrombophlebitis
– Cellulitis
– Ruptured Baker's cyst (popliteal cyst)

COMPLICATIONS

– Pulmonary embolism
– Chronic venous insufficiency

DIAGNOSTIC TESTS

– D-Dimer can be helpful to rule out diagnosis
– EKG if pulmonary embolus is suspected
– CBC, renal function, INR and PTT
– Ultrasound of lower limb(s)
– CT scan or V/Q scan if pulmonary embolus is suspected

MANAGEMENT

Goals of Treatment

– Early detection
– Prevent complications

Appropriate Consultation

Consult a physician immediately if you have any suspicion of this disorder.

Nonpharmacologic Interventions for Acute Symptoms

– Bed rest
– Elevation of leg above level of the heart
– Monitor vital signs closely

Nonpharmacologic Interventions Over the Long Term

Client education

– Counsel client about appropriate use of medications (dose, frequency, side effects)
– Recommend prophylactic use of anti-embolic stockings
– Recommend avoidance of restrictive clothing around knees and ankles (for example, socks, garters)
– Ensure that bedridden clients are turned and repositioned frequently (q2h)
– Recommend active or passive leg exercises while in bed

Pharmacologic Interventions

Consult a physician regarding initial anticoagulation with a low molecular weight heparin (LMWH). Doses for treatment of existing DVT may differ from those for prevention of DVT.

enoxaparin (Lovenox), 1 mg/kg twice daily SC or 1.5 mg/kg once daily SC for treatment of acute DVT

Administration should be alternated between the left and right front abdominal wall, towards the sides.

Dosage reductions may be necessary for patients with impaired kidney function.

Antiplatelet drugs (ASA, clopidogrel) and NSAIDs increase the risk of bleeding in patients receiving low molecular weight heparins.

LMWH do not cross the placenta and there is no evidence of teratogenicity or risk of fetal bleeding. These drugs are preferred for the treatment of DVT in pregnant women.[16]

Long-term anticoagulant therapy is recommended for 3–6 months as prophylaxis against recurrence in nonpregnant patients. The duration of anticoagulation depends on a variety of factors (for example, cause, presence of pulmonary embolus and whether this is a first occurrence or recurrence).

Monitoring and Follow-Up

Acute symptoms:

– Observe client for shortness of breath or unexplained tachycardia (signs of pulmonary embolism)

Long-term:

– Follow up every 3–6 months once stable
– Review prevention strategies, medication use, side effects
– Regular INR to monitor warfarin (Coumadin) therapy

Referral

Medevac the acutely symptomatic client as soon as possible.

DYSLIPIDEMIA (HYPERLIPIDEMIA)

Elevation in serum lipoproteins are a major risk factor for coronary artery disease. The two main lipids in blood are cholesterol and triglyceride. Cholesterol is transported in the blood as a component of high-density lipoprotein (HDL), low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL).

Triglyceride is found in VLDL particles. Moderate hypertriglyceridemia (1.7–5 mmol/L) is part of the "metabolic syndrome" of insulin resistance, elevated triglycerides, elevated LDL and low HDL. The metabolic syndrome is associated with a marked increase in cardiovascular risk. High triglyceride levels (>11 mmol/L) increase the risk for pancreatitis.

Dyslipidemia is one of the primary causes of atherosclerotic plaque. Up to 75% of clients with coronary artery disease have dyslipidemia. Normalization of lipid values lowers the rate of symptomatic coronary artery disease and improves overall survival. Dyslipidemia is strongly associated with recurrence of symptomatic coronary artery disease.

CAUSES

Primary Hyperlipidemia

Primary (genetic) single-gene disorders are transmitted by simple dominant or recessive mechanism.

Secondary Hyperlipidemia

Secondary hyperlipidemia occurs as part of a constellation of abnormalities in certain metabolic pathways.

- Diet
- Diabetes
- Hypothyroidism
- Pregnancy
- Obesity (high triglycerides)
- Excess alcohol intake (high triglycerides)
- Liver disease (cholestatic, acute hepatitis)
- Renal disease (nephrotic syndrome, uremia)
- Medications (for example, thiazide diuretics, some beta-blockers, oral contraceptives, corticosteroids, antiretroviral therapy)

HISTORY

- Ask about risk factors and possible causes of secondary hyperlipidemia
- Previously identified hypercholesterolemia (total cholesterol > 5.2 mmol/L)
- Previously identified low levels of HDL cholesterol (< 0.9 mmol/L)
- Smoking
- Hypertension: blood pressure (BP) of 140/90 mm Hg confirmed on repeated determinations or while client is taking antihypertensive medication
- Antecedent cardiovascular disease or family history of premature myocardial infarction (in people < 55 years of age)
- Endocrine disease (diabetes mellitus or secondary causes, including hypothyroidism, renal disease or medications) (note: clients > 35 years of age with diabetes have a very high risk of cardiovascular disease)
- Men > 45 years of age are at greater risk
- Postmenopausal women (> 55 years of age) and younger women with artificial menopause and no hormonal replacement are at greater risk

PHYSICAL FINDINGS

- BP may be elevated
- Arcus corneae (significant in a younger person)
- Retinopathies (seen on funduscopy)
- Xanthomas (lipid deposits that occur as skin eruptions or as lumps on tendons)
- Arterial bruits may develop if atherosclerosis is present
- Peripheral pulses may be diminished if atherosclerosis is present
- Abdominal obesity (waist measurement > 100 cm [40 in] in men, > 90 cm [35 in] in women)
- Body mass index (BMI) > 25

COMPLICATIONS

- Coronary artery disease (for example, angina, myocardial infarction)
- Cerebrovascular disease
- Peripheral arterial disease
- Pancreatitis (hypertriglyceridemia)

DIAGNOSTIC TESTS[17]

Guidelines for Lipid Screening

Fasting lipid profile (total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides) is suggested for the following groups.

Every 5 years

- Asymptomatic men > 40 years of age and women > 50 years of age or postmenopausal with no other risk factors

Every 1–3 years

- Any adult with the following risk factors:
  - diabetes, cigarette smoking, hypertension, obesity (body mass index > 27 kg/m^2), family history of premature coronary artery disease, clinical signs of hyperlipidemia (for example, xanthomas), evidence of atherosclerosis (clinical evidence of coronary artery disease, peripheral vascular disease or carotid vessel disease [transient ischemic attacks, carotid bruits, carotid plaques on ultrasound, previous cerebrovascular accident]), rheumatoid arthritis, systemic lupus erythematosis, psoriasis, HIV infection on highly active antiretroviral therapy, estimated glomerular filtration rate of less than 60 mL/min/1.73 m^2 or erectile dysfunction
- Any child with a family history of hypercholesterolemia or chylomicronemia

Frequency of screening for the above groups is based on clinical judgement regarding risk. More frequent screening may be needed to monitor levels of those under treatment.

Lipid test results should be interpreted in light of other risk factors for coronary artery disease. A cardiovascular risk assessment like the Framingham Risk Score helps one to determine an estimated 10-year risk of cardiovascular disease for an individual. It considers family history, age, HDL-C, total cholesterol, systolic blood pressure, whether patient's hypertension is treated or not, smoking status, sex and diabetes. *See pages 575 to 577 of the "Canadian Cholesterol Guidelines 2009" for how to estimate 10-year risk of cardiovascular disease* (available at: http://www.ccs.ca/download/consensus_conference/consensus_conference_archives/2009_Dyslipidemia-Guidelines.pdf).

A person at high risk has a 10-year risk score of greater than or equal to 20%, at moderate risk a risk score of 10–19% and at low risk a risk score less than 10%.

MANAGEMENT

Goals of Treatment

- Decrease cardiovascular disease by modifying serum cholesterol according to client's level of risk
- Prevent pancreatitis from severe hypertriglyceridemia

Nonpharmacologic Interventions[18]

- Dietary modification (decrease saturated fats [substitute unsaturated fats for saturated and trans fats]; increase consumption of fruits and vegetables; decrease consumption of sodium and simple sugars)
- Weight reduction and maintenance
- Smoking cessation
- Increased physical activity (daily exercise)
- Stress management

Optimal control of other diseases related to the development of heart disease:

- For hypertension, target BP: systolic < 140 mm Hg, diastolic < 90 mm Hg (130/80 mm Hg for clients with diabetes, < 125/75 if diabetic with nephropathy)
- For diabetes mellitus, aim for optimal, realistic blood glucose level

Pharmacologic Interventions[19]

See Table 4 for drugs and recommended dosage ranges.[20]

Table 4 – Lipid-lowering Medications

Generic name	Trade name (manufacturer)	Recommended dose range (daily)
Statins		
Atrovastatin	Lipitor (Pfizer Canada Inc)	10 mg – 80 mg
Fluvastatin	Lescol (Novartis Pharmaceuticals Canada Inc)	20 mg – 80 mg
Lovastatin	Mevacor (Merck Frosst Canada Ltd)	20 mg – 80 mg
Pravastatin	Pravachol (Bristol-Myers Squibb Canada)	10 mg – 40 mg
Rosuvastatin	Crestor (AstraZeneca Canada)	5 mg – 40 mg
Simvastatin	Zocor (Merck Frosst Canada Ltd)	10 mg – 80 mg*
Bile acid and/or cholesterol absorption inhibitors		
Cholestyramine	Questran (Bristol-Myers Squibb, USA)	2 g – 24 g
Colestipol	Colestid (Pfizer Canada Inc)	5 g – 30 g
Ezetimibe	Ezetrol (Merck Frosst/Schering Pharmaceuticals Canada)	10 mg
Fibrates		
Bezafibrate	Bezalip (Actavis Group PTC EHF, Iceland)	400 mg
Fenofibrate†	Lipidil Micro/Supra/EZ (Fournier Pharma Inc, Canada)	48 mg – 200 mg
Gemfibrozil†‡	Lopid (Pfizer Canada Inc)	600 mg – 1200 mg
Niacin		
Nicotinic acid	Generic crystalline niacina	1 g – 3 g
	Niaspan (Oryx Pharmaceuticals Inc, Canada)	0.5 g – 2 g

* Increased myopathy on 80 mg.
† Reduce dose or avoid in renal impairment.
‡ Should not be used with a statin because of an increased risk of rhabdomyolysis.

A baseline FBS, TSH, ALT, AST, creatinine, creatinine kinase should be done to identify other causes of dyslipidemia and monitor potential side effects prior to starting treatment.

If clients experience unexplained muscle pain or tenderness, advise them to discontinue statins immediately and to see a nurse for a serum creatine kinase (CK) measurement. Contact the physician with CK level for suggestions on continued therapy.

Monitoring and Follow-Up

Check the response to treatment within 6 weeks (blood tests should be carried out early – see below) and, if the results are satisfactory, continue follow-up at regular intervals thereafter (every 3–12 months).

Monitor liver function (ALT,AST), creatinine kinase, complete blood count and serum creatinine at 3, 6 and 12 months after initiation of lipid-lowering drugs and semi-annually thereafter, or with any changes in therapy. Clients on niacin also need a FBS and glycosylated hemoglobin done every 6–12 months.

Frequency of testing to monitor treatment of dyslipidemia:

Patients on diet therapy only:

Initiation: Every 3–6 months to 1 year

Maintenance: Every 6–12 months

Patients on diet and drug therapy:

Initiation of drug therapy: Every 6–8 weeks to 6 months, depending on severity

Maintenance: Every 3 months in the first year, every 6–12 months thereafter

Referral

Refer all clients diagnosed with hyperlipidemia to a physician so that they can be evaluated for risk of coronary artery disease and to determine whether lipid-lowering medications are needed.

PREVENTION[21]

Primary prevention is aimed at identifying dyslipidemia before complications occur. Target levels of LDL cholesterol are based on individual cardiovascular risk factors. Generally, any client with

LDL cholesterol > 5 mmol/L, even in the absence of other risk factors, is considered at high risk for developing cardiovascular disease. *See section "Guidelines for Lipid Screening" in this chapter to calculate a risk score.*

For anyone at high risk (10-year risk score of ≥ 20%), or men > 45 years and women > 50 years with diabetes, or anyone with evidence of coronary artery disease, peripheral vascular disease and/or atherosclerosis:

Target: LDL cholesterol < 2 mmol/L or a 50% reduction from baseline LDL cholesterol

For those at high risk, pharmacologic interventions should be started as soon as possible, in conjunction with healthy lifestyle changes (for example, smoking cessation, diet with reduced saturated fats and refined sugars, weight reduction and maintenance, exercise, stress management).

For clients at moderate risk (10-year risk score of 10–19%), LDL-C > 3.5 mmol/L, TC/HDL-C > 5.0:

Target: LDL cholesterol < 2 mmol/L or a 50% reduction in LDL cholesterol from baseline

For those at moderate risk, healthy lifestyle changes should be promoted; if target lipid levels are not achieved in 3 months, drug therapy should be started, after consultation with a physician.

For clients at low risk (10-year risk score of < 10%)

Target LDL cholesterol is a 50% reduction from baseline

For those at low risk, healthy lifestyle changes should be promoted; if target lipid levels are not achieved in 6 months, drug therapy should be considered in consultation with a physician.

All risk factors should be treated, if possible, by nonpharmacologic means.

Secondary prevention is directed at reducing the impact of dyslipidemia on people with previous cardiovascular disease. These targets are aimed specifically at high-risk clients and are more stringent than those recommended for the general population.

Target: LDL cholesterol < 2 mmol/L

HYPERTENSION

Persistently elevated blood pressure from increased peripheral arterial resistance related to salt or water retention or endogenous pressure activity.

CAUSES

Cause of essential hypertension (which accounts for 90% of cases of hypertension) is unknown.

Risk Factors for Primary (Essential) Hypertension

- Heredity
- Obesity
- High salt intake
- Smoking
- High alcohol consumption
- Chronic stress
- Age
- Hyperlipidemia

Risk Factors for Secondary Hypertension (10% of Cases)

- Renal disease
- Polycystic kidneys
- Renal vascular disease
- Pregnancy
- Hyperthyroidism
- Cushing's syndrome
- Primary hyperaldosteronism
- Pheochromocytoma
- Coarctation of aorta
- Chronic alcohol abus
- Drugs:
  - NSAIDs
  - Corticosteroids
  - Oral contraceptives, sex hormones, anabolic steroids
  - Cyclosporine, tacrolimus (to prevent transplant rejection)
  - Erythropoietin
  - Monoamine oxidase inhibitors (for depression)
  - Midodrine
  - Stimulants including cocaine

HISTORY

- Presence of one of the risk factors *(see section "Risk Factors for Primary (Essential) Hypertension" in this chapter)*
- Client usually > 35 years of age
- Condition usually discovered only on routine screening of blood pressure; all adults over 21 years of age should be screened at every office visit[22]

- Usually asymptomatic
- Headache on rising in the morning, gradually subsiding during the day (rare)
- Fatigue
- Transient ischemic attack
- Nausea or vomiting
- Altered level of consciousness
- Palpitations
- Angina
- Symptoms of cardiac failure
- Epistaxis

PHYSICAL FINDINGS[23]

When assessing for hypertension, if the systolic BP is ≥ 140 mm Hg and/or the diastolic BP is ≥ 90 mm Hg take 2 more readings during the same visit. Discard the first reading and average the last two.

Hypertension can be diagnosed immediately if there is evidence of urgency or emergency:

- Asymptomatic diastolic BP ≥ 130 mm Hg
- Hypertension compromising vital organ function (encephalopathy, left ventricular failure, acute myocardial ischemia)
- Hypertension and an acute aortic dissection. Evidence of malignant hypertension (sufficient elevation of blood pressure to cause papilledema and other manifestations of vascular damage, such as retinal hemorrhages, bulging optic disks, mental status changes)

Hypertension can be diagnosed in 2 visits within 1 month if the BP is ≥ 180/110 mm Hg *or* BP is 140–179/90–109 mm Hg with target organ damage, diabetes or chronic kidney disease.

Target organ damage can be initially established on the basis of a history of angina, myocardial infarction, transient ischemic attacks, cerebrovascular accident, peripheral ateriovascular insufficiency (claudication) or renal insufficiency.

Hypertension can be diagnosed in 3 visits if the average across the visits is a systolic BP of ≥ 160 mm Hg or a diastolic BP of ≥ 100 mm Hg. Hypertension can be diagnosed in 5 visits if the average across the visits is a systolic BP of ≥ 140 mm Hg or a diastolic BP of ≥ 90 mm Hg and there is no target organ dysfunction. If blood pressure is still elevated on the third visit, baseline diagnostic investigations should be done.

Other Findings (Target Organ Damage)

- Ocular funduscopic exam may reveal retinal changes
- Augmented second aortic heart sound
- Enlarged heart (left ventricular hypertrophy)
- Bruits (carotid, abdominal aortic, renal and femoral)

Differential Diagnosis

- Essential hypertension
- Secondary hypertension

General Clues to Secondary Hypertension

- Severity of high blood pressure: severe hypertension is more likely secondary to a specific underlying cause
- Speed of onset: if hypertension develops rapidly, it should be considered secondary until proven otherwise
- Age at onset: rapid onset in people younger than 25 years or older than 55 years should suggest secondary hypertension
- Female sex: the presence of hypertension in a young woman in whom an abdominal bruit is heard suggests stenosis of the renal arteries

COMPLICATIONS

- Congestive heart failure
- Angina
- Myocardial infarction
- Stroke or transient ischemic attacks
- Hypertensive crisis
- Kidney disease
- Retinal disease
- Peripheral disease
- Complications related to therapy (for example, thiazide diuretics increase risk of gout)
- Poor response to therapy

DIAGNOSTIC TESTS

- Urinalysis (routine and for microalbuminuria in diabetic clients)
- Obtain complete blood count
- Determine blood glucose; total, LDL and HDL cholesterol; and triglyceride levels (while fasting)
- Determine creatinine and electrolyte levels
- Obtain baseline electrocardiogram (ECG) and chest x-ray (if available) if > 50 years of age

MANAGEMENT

Goals of Treatment

– Decrease morbidity and mortality associated with high blood pressure
– Optimal control of blood pressure with an effective, well-tolerated treatment regimen

Target BP levels are:[24]

– 140/90 mm Hg in the general population
– 130/80 mm Hg in clients with diabetes or renal dysfunction

Appropriate Consultation

Consult a physician if there is a need to treat hypertension with medications.

Nonpharmacologic Interventions[25]

Lifestyle modifications are first-line therapy for mild elevation of blood pressure.

Client education

– Ensure that client understands disease process and prognosis
– Encourage client to lose weight if appropriate (aim for a loss of 10% of current body weight, or at least 4.5 kg [10 lb]); maintain BMI of 20–25
– Encourage client to achieve and maintain a waist circumference < 102 cm for men and < 88 cm for women
– Recommend dietary modifications (for example, avoid high-salt foods, avoid adding salt in cooking or at table; adhere to diet high in fresh fruits and vegetables, high in soluble fibre and low fat dairy products, low in saturated fats)
– Recommend smoking cessation
– Recommend restriction of alcohol consumption (0–2 drinks per day – ≤ 14 drinks per week for men and ≤ 9 for women)
– Recommend regular exercise, especially if sedentary (30–60 minutes of moderate physical activity on most days, for example, a brisk walk 4–5 times a week)
– Counsel client about appropriate use of medications (dose, frequency, side effects and importance of compliance)
– Ask client to return to clinic if any unusual symptoms occur or there is a change in status

Pharmacologic Interventions[26]

All pharmacotherapy must be initiated only after consultation with a physician.

Pharmacotherapy should be initiated in the following instance:

– Diastolic BP ≥ 100 mm Hg or systolic BP ≥ 160 mm Hg in the client who does not have cardiovascular disease, cardiovascular risk factors or target organ damage

Pharmacotherapy should be strongly considered in the following instance:

– Diastolic BP ≥ 90 mm Hg or systolic BP ≥ 140 mm Hg in the client with cardiovascular disease, cardiovascular risk factors or target organ damage

The following classes of drugs are used in the treatment of hypertension:

– Beta-blockers
– Angiotensin-converting enzyme (ACE) inhibitors
– Angiotensin receptor blockers (ARBs)
– Diuretics
– Calcium channel blockers (CCBs)
– Alpha blockers (not commonly used)

The majority of patients will require combination therapy to achieve recommended blood pressure goals. Many antihypertensive medications are available in combination products that can simplify the regimen and may improve compliance.[27] Consult a pharmacist to discuss availability of specific combinations. Depending on the clinical situation, these drugs can be used in combination. The selection of agent is made on the basis of the client's age, coexisting medical conditions and risk factors.

Hypertension in pregnancy requires assessment and treatment by a physician. Not all of the above drugs are safe in pregnancy.

Hypertension Treatment Strategies According to Comorbidities

The Canadian Hypertension Education Program (CHEP) Web site offers recommendations for the management of hypertension in the presence of comorbidities (such as ischemic heart disease, non-diabetic chronic kidney disease or diabetes mellitus).[27] Information can be accessed at: http://hypertension.ca/chep/wp-content/uploads/2010/04/FullRecommendations2010.pdf.

Monitoring and Follow-Up

For clients on nonpharmacolgic therapy, follow up every 3–4 months.

For clients on antihypertensive drug therapy, follow up every month until 2 successive blood pressure readings are at target level. Once blood pressure has reached target level, follow up every 3–6 months.

More frequent follow-up is recommended for clients with symptomatic hypertension, severe hypertension, antihypertensive drug intolerance or target organ damage.

Routine Follow-up Assessment Related to Hypertension

Determine history related to the following:

- Headaches
- Dizziness
- Angina
- Congestive heart failure
- Transient ischemic attack
- Stroke
- Nausea and vomiting
- Vision changes
- Medication compliance
- Drug side effects

The physical examination should include the following:

- Blood pressure (supine and standing)
- Funduscopy (dilated)
- Neck examination (carotid artery for bruits, JVP [jugular venous pressure] for congestive heart failure)
- Cardiovascular examination
- Respiratory examination
- ECG (annually)
- Chest x-ray (annually) if cardiomegaly documented
- Ophthalmologic exam (if funduscopic changes have been documented)
- Blood work q3-6 months: complete blood count, blood glucose level, creatinine level, electrolyte levels, uric acid level (if client is taking thiazide diuretics)
- Urinalysis (for protein)

Referral

Arrange follow-up with physician at least yearly if the client's hypertension is stable or as soon as possible if poorly controlled.

Repeat physician consultation is necessary for chronically hypertensive clients if any of the following situations apply:

- Client is not responding to therapy
- Target organ damage caused by poorly controlled blood pressure
- Signs and symptoms of complications

PREVENTION[28]

Maintaining a healthy body weight (BMI 18.5–24.9 kg/m^2 and a waist circumference < 102 cm for men and < 88 cm for women is recommended.

ISCHEMIC HEART DISEASE, INCLUDING ANGINA

Ischemic heart disease (IHD) is a symptom complex that is a result of an imbalance between oxygen supply and demand in the myocardium.

SPECTRUM OF IHD

The spectrum of ischemic heart diseases ranges from asymptomatic disease to sudden death from myocardial infarction or arrhythmia.

ANGINA PECTORIS

The result of myocardial ischemia, which occurs when the cardiac workload and myocardial oxygen demands exceed the ability of the coronary arteries to supply oxygenated blood. It is the main clinical expression of coronary artery disease (subintimal deposition of atheromas in the large- and medium-sized arteries serving the heart).

CHRONIC STABLE ANGINA

Most often due to a fixed stenosis caused by an atheroma. It is characterized by a predictable pattern of pressure sensation in the anterior chest precipitated by exertion, emotion and eating. Typically is of brief duration < 10–15 minutes and is relieved by rest or nitroglycerin. Alternative presentations should be considered particularly in women and diabetics (for example, different types of chest pain or primary complaint of episodic shortness of breath).

UNSTABLE ANGINA

A syndrome of acute plaque rupture with incomplete or transient vessel occlusion. A client with new onset of undiagnosed ischemic pain is considered to have unstable angina. In a previously diagnosed client, it is characterized by an accelerating pattern of pain (for example, increased frequency, severity or duration [more than 30 minutes, occurring with less exertion, occurring at rest or decreased response to current treatment]). It can also be present if never diagnosed previously. Pain on presentation at the clinic in anyone with a history of recent onset angina or anginal symptoms at rest, and anyone with known heart disease and an increase or change in anginal pattern and ECG changes may be unstable angina. However, ECG changes do not need to present in unstable angina, particularly if the person is pain free when the ECG is done. Anyone presenting with acute chest pain should be treated as potentially having a myocardial infarction until it is ruled out.

MYOCARDIAL INFARCTION

A syndrome of acute plaque rupture and thrombosis with total coronary occlusion resulting in myocardial necrosis. *For details, see section "Emergencies of the Cardiovascular System" later in this chapter.*

Causes

– The main pathogenetic mechanisms are atherosclerosis and thrombus formation
– A less common cause is coronary artery vasospasm

Risk Factors

See Table 5, "Gender-Specific Risk Factors for Cardiac Disease."

Table 5 – Gender-Specific Risk Factors for Cardiac Disease

Risk Factor	Women	Men
CAD = coronary artery disease, HDL = high-density lipoprotein, LDL = low-density lipoprotein, MI = myocardial infarction		
Family history	Premature MI in parent increases risk 2.8 times	Premature MI in parent increases risk 3 to 5 times
Obesity	3 times greater risk of CAD	2 times greater risk of CAD
Smoking	MI occurs 19 years earlier than in nonsmokers	MI occurs 7 years earlier than in nonsmokers
Lipids	High levels of triglycerides and low levels of HDL cholesterol are better predictors of CAD	High levels of total cholesterol and high levels of HDL cholesterol are better predictors of CAD
Hypertension	Higher prevalence in older women	Higher prevalence in middle age
Diabetes	Risk of CAD increases 7 times	Risk of CAD increases 3 times
Menopause	Increases LDL, decreases HDL cholesterol	
Male:female ratio = 2:1 for all age groups, 8:1 for age under 40 years and 1:1 for age over 70 years.		
Peak incidence of symptomatic IHD is age 50–60 years for men and 60–70 years for women		

Other associated risk factors:

– Sedentary lifestyle
– Hyperhomocysteinemia
– Hormone replacement therapy (in some women)
– Chronic autoimmune diseases (for example, lupus)

Clinical Presentation in Women

Women with coronary artery disease or coronary heart disease experience more noncardiac chest pain than men. They are more likely to have atypical manifestations such as:

– Chest pain at rest or during sleep or with mental stress
– A prodrome of fatigue and a nonspecific feeling of unwellness for weeks before a cardiac event
– Chronic fatigue, dizziness, ankle swelling, nausea, indigestion, change in chronic headache pattern and hot and flushed feelings
– Pain in the back, neck or throat
– Shortness of breath, fatigue, flushing, nausea, jaw pain and abdominal pain, which occur over hours rather than minutes
– No Q waves on ECG during myocardial infarction
– Nondiagnostic, reversible ST segment elevations or T wave abnormalities

Young premenopausal women who have had a myocardial infarction have significantly higher mortality rates than men who have had a myocardial infarction at the same age. One possible explanation for this may be the difference in primary prevention strategies applied to women; management of early symptoms is often less aggressive.

History

Chest pain described as tightness, pressure or aching that is typically located in the substernal area, may radiate to neck, jaw and/or one or both shoulders/arms. Duration is brief – < 10–15 minutes. Precipitated by exercise or emotional stress or eating. Typically relieved by rest and/or nitroglycerin.

Dyspnea or fatigue may present as "chest pain equivalents," especially in post menopausal women.

Associated Symptoms

- Dyspnea
- Nausea or vomiting
- Sweating
- Weakness
- Palpitations

Physical Findings

- Diaphoresis
- Apprehension
- Oxygen saturation (usually normal unless complications present)
- Blood pressure (may be elevated if hypertension)
- Tachycardia – assess for arrhythmias
- Arterial bruits (carotid, aortic, femoral arteries)
- S_4 gallop
- Murmurs (aortic stenosis)
- Hypertrophic cardiomyopathy
- ECG changes (elevation or depression of ST segment, inversion of T wave)

Physical findings are transient in stable angina and disappear when the pain resolves. People with stable angina are usually seen in a clinic after an attack because of the mild, short, episodic nature of the discomfort. After an episode there are usually no significant physical findings and no ECG changes. There may be signs of underlying atherosclerotic disease (for example, arterial bruits, heart murmurs, hypertension).

Differential Diagnosis

- Chest-wall pain (costochondritis)
- Other musculoskeletal discomfort (rib fracture)
- Peptic ulcer disease
- Gastroesophageal reflux
- Esophageal spasm
- Indigestion
- Anxiety attack
- Pulmonary emboli
- Pericarditis
- Aortic dissection
- Pneumothorax (spontaneous)
- Pneumonia

Complications

- Unstable angina
- Future myocardial infarction

Diagnostic Tests

- ECG tracing – compare with previous one, if available; look for signs of ischemia (depression of ST segment, ST segment elevation, inversion of T wave, new changes)
- Obtain complete blood count, fasting blood glucose, creatinine, electrolytes and fasting cholesterol panel and TSH if this is the first presentation or it has not been done recently

MANAGEMENT

Management of Stable Angina

Goals of Treatment

- Decrease or prevent recurrence of pain
- Identify and manage cardiac risk factors to slow progression of disease
- Improve exercise tolerance
- Minimize the risk of nonfatal MI and cardiovascular death

Appropriate Consultation

Consult a physician as soon as possible if you suspect this diagnosis.

Nonpharmacologic Interventions

– Ensure that client understands disease process
– Encourage client to make lifestyle changes (for example, dietary modifications to reduce saturated fat and cholesterol, salt use and alcohol use)
– Encourage client to reduce weight, stop smoking, avoid strenuous exercise but increase moderate exercise (for example, walking, cycling, swimming)

Pharmacologic Interventions

For acute symptom relief:

nitroglycerin, 0.4 mg sublingual (SL) spray q5min X 3 doses prn

If pain not relieved with 3 doses proceed to treat as possible acute myocardial infarction *(See "Myocardial Infarction" under the section "Emergencies of the Cardiovascular System").*

For longer-term symptom prophylaxis:

Beta-blockers, calcium channel blockers and long-acting nitrate preparations are used to prevent recurrent attacks. The choice of agent depends on the presence of other comorbid illnesses. A combination of agents may be used to control symptoms.[29]

Beta-blockers relieve anginal symptoms by decreasing the heart rate and contractility and reducing blood pressure.[29] They should be used cautiously in clients with diabetes if there is a concern about possible hypoglycemic episodes (they impair awareness of hyperglycemia). They should also be used with caution in patients with bronchospastic disease but are sometimes used in patients who are not receiving beta-agonists. The goal is achievement of a resting heart rate of 50–60 beats/minute.

Oral, spray or transdermal nitrates can be used for prophylaxis (acute attack or prior to activities known to exacerbate angina) or chronic therapy. Nitrate tolerance is known to occur with continuous use. With any nitrate preparation it is essential to ensure a 10–12 hour[30] nitrate-free interval to prevent loss of effect over time. The presenting symptoms will influence the timing of the nitrate-free period, for example, a primarily exertional angina will be treated with a daytime nitrate dose while congested heart failure and nocturnal angina may be managed with an evening nitrate dose. Nitroglycerin preparations can be used in combination with beta-blockers or calcium channel blockers.[31]

Calcium channel blockers (CCB) (for example, diltiazem, verapamil) are used for treatment of angina, especially when there are contraindications to the above therapies, or if nitrates or beta-blockers are not adequate. CCBs are not usually a first-line therapy but may be a treatment of choice for clients with coronary arterial spasm.[31]

ACE inhibitors (ACEI) (for example, ramipril) reduce the risk of death in patients with stable heart disease including chronic stable angina.[32]

All clients with angina should receive secondary prophylaxis with an antiplatelet agent (for example, ASA 81 mg/day) and a statin[30] *(see section "Dislipidemia (Hyperlipidemia)" in this chapter).*

Monitoring and Follow-Up

– Follow up every 6 months once client's symptoms are stable
– Monitor symptoms and identify any changes, especially increases
– Monitor weight and smoking
– Monitor blood pressure and pulse
– Obtain regular blood work as directed
– Monitor adherence and response to long-term lifestyle modifications and medications (for example, beta-blockers)

Referral

Refer all previously undiagnosed clients and any clients whose symptoms are not controlled on current therapy to a physician for a thorough evaluation. Once the condition has been stabilized, the client should be assessed by a physician at least twice annually.

Management of Unstable Angina

Anyone presenting with chest pain should be treated as possibly having an acute myocardial infarction.

Goals of Treatment

– To relieve chest pain and other symptoms of myocardial ischemia
– To prevent further myocardial injury
– To reduce the severity of or eliminate episodes of ischemia

Appropriate Consultation

Consult a physician as soon possible.

Adjuvant Therapy

If chest pain is present at the time of presentation:

– Oxygen may be administered by nasal canula; titrate flow to keep oxygen saturation ≥ 94%[33]
– Start intravenous (IV) therapy with normal saline to keep vein open

Nonpharmacologic Interventions

Bed rest for clients experiencing pain on presentation.

Pharmacologic Interventions

nitroglycerin sublingual spray (0.4 mg) q5min X 3 doses prn

If the client is hypotensive or has bradycardia on presentation, do not give nitroglycerin without first consulting a physician. If pain is not relieved, treat as myocardial infarction *(see "Myocardial Infarction" under the section "Emergencies of the Cardiovascular System" later in this chapter).*

If no contraindications, give:

Uncoated ASA 162–325 mg as soon as possible (chew and swallow)[34]

Consult a physician regarding initial anticoagulation with a therapeutic dose of low molecular weight heparin while awaiting transfer.

Monitoring and Follow-Up

Continue to closely monitor pain, vital signs (including oxygen saturation), heart and lung sounds and ECG results.

Referral

Medevac as soon as possible.

Revascularization procedures such as coronary angioplasty, stenting or bypass surgery may be indicated for any client who continues to have significant symptoms despite medical therapy.

Prevention

Prevention of morbidity and mortality from vascular disease requires recognition and management of modifiable risk factors. Primary prevention involves management of risk factors before the patient suffers a vascular event such as a stroke, myocardial infarction or amputation. Secondary prevention involves management of risks after the patient suffered a vascular event.[35]

Primary Prevention

The Web site Hypertension.ca, Hypertension Canada presents information from the Canadian Hypertension Society (CHS), the Canadian Hypertension Education Program (CHEP) and Blood Pressure Canada (BPC). In Part 2 of the recommendations from CHEP, you will find information on modifiable lifestyle factors for the prevention of vascular events through the prevention of specifically hypertension. A summary of other risk factors and preventive measures are presented below.

Nutritional strategies including:

– Fat intake: < 30% of total caloric intake with < 10% of calories from saturated fats
– Omega-3 fatty acid rich foods: 2 or more servings of either fish (for example, salmon) or omega -3 rich plant foods (for example, flaxseed, canola oil, soybean oil or nuts)
– 7–8 servings of fruits and vegetables daily
– Whole grain and high-fibre foods
– Low salt intake:[36]
  – Adults age 50 or less: 1500 mg (65 mmol) per day
  – Adults 51–70 years: 1300 mg (57 mmol) per day
  – Adults > 70 years: 1200 mg (52 mmol) per day

Alcohol: 2 or fewer drinks per day, not exceeding 14 standard drinks per week for men and 9 standard drinks per week for women.[36]

Weight management: BMI within normal range of 18.5–24.9 kg/m^2 and waist circumference less than 102 cm for men and 88 cm for women.[36]

Exercise program: Most guidelines recommend 30–60 minutes of moderate intensity exercise (such as walking, jogging, cycling or swimming) 4–7 days a week.[37]

Smoking cessation counselling; minimize exposure to second-hand smoke.

Oral Contraceptives: Use the lowest effective dose of estrogen and progesterone to prevent pregnancy. Avoid use in women who smoke, those with uncontrolled hypertension and/or a history of stroke, ischemic heart disease or venous thromboembolism.[38]

Secondary Prevention[39]

Blood Pressure: Achieve and maintain a BP of < 140/90 mm Hg (130/80 mm Hg for clients with diabetes).

Diabetes: For type 1 or 2, maintain tight glycemic control (hemoglobin $HbA_{1c} \leq 7\%$).

Lipids:

- Achieve and maintain optimal lipid level for age, sex and cardiovascular risk status
- Target LDL cholesterol < 2 mmol/L

PERICARDITIS, ACUTE

An inflammation of the pericardium surrounding the heart muscle. It may occur with or without an effusion. The most common type is idiopathic or nonspecific pericarditis.

CAUSES

- Idiopathic (unknown)
- Viral infection (for example, coxsackievirus, ECHO virus, adenovirus, Epstein-Barr virus, mumps)
- Bacterial infection (for example, *Streptococcus pneumoniae* and other *Streptococcus* species or *Staphylococcus* species. Isolated gram-negative bacteria include *Proteus* species, *Escherichia coli*, *Pseudomonas* species, *Klebsiella* species, *Salmonella* species, *Shigella*, *Neisseria meningitidis* and *Haemophilus influenza*, *Nocardia* species)
- Fungal infection (for example, aspergillosis, candidiasis, histoplasmosis)
- Mycobacterial infection (for example, *Mycobacterium tuberculosis*)
- Parasites: protozoa
- Neoplasm: breast, lung, lymphoma, renal cell, melanoma
- Drug-induced: isoniazid, phenytoin, procainamide, hydralazine, among others
- Connective-tissue disease: systemic lupus erythematosus, rheumatoid arthritis, scleroderma, acute rheumatic fever
- Radiation therapy
- Post-myocardial infarction (Dressler's syndrome)
- Chest trauma
- Uremia
- Hypothyroidism (myxedema)
- Aortic dissection
- Sarcoidosis
- Pancreatitis
- Inflammatory bowel disease
- AIDS

HISTORY

- Chest pain, typically sharp; retrosternal with radiation to the trapezial ridge
- Pain, frequently sudden in onset
- Pain reduced by leaning forward and sitting up
- Splinted breathing
- Odynophagia
- Fever
- Myalgias
- Anorexia

PHYSICAL FINDINGS

- Temperature may be elevated (low-grade fever)
- Respiration fast and shallow
- Sinus tachycardia
- A narrow pulse pressure (hypotension-elevated neck veins may indicate a restrictive pericarditis that requires urgent decompression)
- Pulsus paradoxus – weakening or disappearance of the radial, brachial or femoral pulse during inspiration (urgent decompression may be required if the difference in arterial pressure is greater than 10 mm Hg between the first Korotkoff sounds heard only during expiration and the Korotkoff sounds first heard throughout inspiration and expiration)
- Electrocardiogram (ECG) initially diffuse ST segment elevation +/- depressed PR segment, 2–5 days later ST isoelectric with T wave flattening and inversion
- Anxiety
- Mild distress
- Flushing
- Splinted breathing
- Shortness of breath (only in cases of pericardial tamponade or constrictive pericarditis)
- Pericardial friction rub
- Localized lung crackles may be present

DIFFERENTIAL DIAGNOSIS

- Acute myocardial infarction
- Pneumonia with pleurisy
- Pulmonary emboli
- Aortic dissection
- Pneumothorax
- Mediastinal emphysema
- Cholecystitis
- Pancreatitis

COMPLICATIONS

- Pericardial tamponade
- Recurrence of pericarditis
- Noncompressive effusion
- Chronic constrictive pericarditis
- Atrial arrhythmia

DIAGNOSTIC TESTS

- ECG
- Chest x-ray (if available), to rule out complications such as pleural effusion or enlarged heart

MANAGEMENT

Goals of Treatment

- Prevent complications
- Identify underlying treatable causes

Appropriate Consultation

Consult a physician if you suspect this diagnosis.

The otherwise healthy client can often be safely treated on an outpatient basis.

Nonpharmacologic Interventions

Client education

- Ensure that client understands disease process and prognosis
- Counsel client about appropriate medication use and side effects
- Recommend avoidance of heavy physical labour
- Teach client about symptoms and signs of complications, and instruct client to report any that occur
- Stress the importance of follow-up

Pharmacologic Interventions

Drugs are mainly used in cases of idiopathic pericarditis. In other cases, underlying causes must be treated appropriately.

NSAIDs[40]: acetylsalicylic acid (ASA), 650 mg q4-6h initially, then taper the dose over three to four weeks (to reduce the likelihood of recurrence)

or

ibuprofen (Motrin), 400–800 mg, q6-8h initially, then taper the dose

In some clients, the condition becomes refractory and corticosteroids or pericardiectomy may be required.

Monitoring and Follow-Up

- Follow up in 2 or 3 days, to make sure no complications develop, and then again in 2 weeks
- Repeat ECG and chest x-ray should be considered at about 4 weeks
- In most clients complete resolution occurs after 2 weeks of therapy
- 15% of clients will have at least one recurrence within the first few months

VENOUS INSUFFICIENCY, CHRONIC

Impairment of the venous system that inhibits normal return of blood from the legs to the heart.

CAUSES

Incompetent valves in veins of the legs.

Risk Factors

- Familial predisposition
- Prolonged standing
- Pregnancy
- Obesity
- Constricting garments worn over a long period of time

HISTORY

- Dull aching heaviness or fatigue in legs, often occurring at the end of the day and relieved by elevation of the legs
- Mild edema at end of day
- Cramps in legs at night
- Itching may be present (due to stasis dermatitis)
- Stasis dermatitis, brownish red discolouration

PHYSICAL FINDINGS

- Dilated, tortuous, elongated varicose veins in foot, lower leg, medial thigh or behind knee
- Varicose veins seen more readily when person is standing
- Skin changes may be present (erythema, brownish pigmentation, flaking and scaling, skin breakdown)
- Venous ulcers may be present on medial side of lower leg just above medial malleolus or on medial aspect of ankle
- Edema of foot and ankle may be present
- Dilated veins easily palpable when person is standing

DIFFERENTIAL DIAGNOSIS

- Chronic occlusive arterial disease with arterial ulcers
- Orthopedic problems

COMPLICATIONS

- Stasis dermatitis
- Cellulitis
- Stasis ulcer
- Thrombophlebitis
- Deep vein thrombosis (if deep veins involved)

DIAGNOSTIC TESTS

None.

MANAGEMENT

Goals of Treatment

- Facilitate venous return
- Prevent complications

Nonpharmacologic Interventions

Client education

- Teach client proper skin hygiene and care of lesions
- Recommend support hose or support stockings (compression stockings should exert a minimum of 20–30 mm Hg pressure at the ankle to be effective for moderate to severe varicose veins)[41]
- Recommend elevation of legs above the level of the hip when sitting
- Recommend avoidance of prolonged standing (client should sit with legs elevated whenever possible and should avoid crossing legs)
- Recommend avoidance of restrictive clothing around the knees (for example, knee socks, garters)
- Recommend weight loss (if appropriate)
- Recommend smoking cessation (if appropriate)
- Instruct client to return to clinic if signs of skin breakdown or skin irritation occur, or if a vein becomes sore and tender
- Instruct client to do leg exercises qid in bed to prevent deep vein thrombosis

Monitoring and Follow-Up

Arrange follow-up in 1 month to assess adherence to and efficacy of interventions.

Referral

Refer to a physician if condition does not improve with conservative treatment or if complications arise.

EMERGENCIES OF THE CARDIOVASCULAR SYSTEM

ACUTE ARTERIAL OCCLUSION OF A MAJOR PERIPHERAL ARTERY

Sudden occlusion of a peripheral artery with resultant acute ischemia in the distal limb.

CAUSES

- Thrombus
- Embolus
- Trauma
- Idiopathic
- Predisposing factors: peripheral vascular disease, atrial fibrillation, recent myocardial infarction, prosthetic heart valve, oral contraceptive use, history of TIA/stroke, antiphospholipid syndrome or other hypercoagulable states

HISTORY

- Sudden onset of severe pain in distal part of a limb
- Paresthesia, coldness and pallor in distal limb follow later
- Previous symptoms of intermittent claudication may be present
- History of cardiac disease may be present

PHYSICAL FINDINGS

- Heart rate elevated, pulse may be irregular
- Respiratory rate normal or increased
- Blood pressure normal or increased
- Anxious, in acute distress
- Signs of longstanding peripheral vascular disease in the opposite limb
- Colour of limb normal initially, becomes pale later
- Skin temperature may be normal initially, becomes cool or cold later
- Peripheral pulses lower than in opposite limb or absent altogether
- Cutaneous sensation decreased or absent
- Tenderness in calf on dorsiflexion of foot
- Arterial bruits may be present (aortic, iliac, femoral, popliteal)

The 6 Ps of acute arterial occlusion are: pain, pallor, polar (cold), pulseless, paresthesia and paralysis.

DIFFERENTIAL DIAGNOSIS

- Compartment syndrome if trauma has been involved

COMPLICATIONS

- Ischemic muscular contracture
- Loss of limb

MANAGEMENT

Goals of Treatment

- Improve oxygenation of the limb
- Prevent injury to or loss of limb

Appropriate Consultation

Consult a physician immediately.

Nonpharmacologic Interventions

- Bed rest
- Prevent injury to limb: handle carefully, protect from pressure or injury
- Do not elevate ischemic limb (keep horizontal or slightly dependent)

Adjuvant Therapy

- Start IV therapy with normal saline to keep vein open
- Oxygen may be needed if there is evidence of underlying cardiorespiratory compromise

Pharmacologic Interventions

Analgesia for pain:

morphine, 2–5 mg IV prn (maximum 10 mg/h or 10 mg IM)

Consult a physician regarding initial anticoagulation before transfer.

Monitoring and Follow-Up

Monitor vital signs, general condition, cardiac and respiratory status frequently.

Referral

Medevac as soon as possible. There is only a 4- to 6-hour window of opportunity to perform surgical intervention to save limb from irreparable damage.

MYOCARDIAL INFARCTION

Interruption of blood flow through the coronary arteries, resulting in ischemic injury and necrosis of a portion of the myocardium. As many as 15% to 25% of cases are silent or atypical in presentation.

CAUSES

- Acute thrombosis within a coronary artery with underlying atherosclerosis
- Coronary artery spasm

Risk Factors

- Smoking
- Family history of heart disease
- Hypertension
- Dyslipidemia
- Obesity
- Diabetes mellitus
- Sedentary lifestyle
- Cocaine use[42]

HISTORY

- Acute retrosternal chest pain (heaviness, aching, squeezing, typical pain of myocardial infarction)
- Pain may radiate into left arm, neck, fingers, shoulders, epigastrium, right chest, right upper quadrant, right arm or upper back
- Pain usually occurs at rest, with gradual or sudden onset, and can be precipitated by stress
- Pain not relieved by nitroglycerin
- Pain lasts longer than 30 minutes
- Shortness of breath
- Nausea and vomiting
- Diaphoresis
- Weakness
- Loss of consciousness may occur

In women, myocardial infarction tends to present atypically as shortness of breath, fatigue, flushing, nausea, jaw pain and abdominal pain, with these symptoms occurring over hours rather than minutes.

PHYSICAL FINDINGS

- Respiration rapid and shallow
- Pulse variable (rapid or slow, regular or irregular, full volume, "thready")
- Blood pressure increased, decreased or normal
- Oxygen saturation may be abnormal if client is in shock or has congestive heart failure
- Acute distress
- Pale
- Diaphoresis
- Cyanosis (central or peripheral, or both)
- Client may be unconscious
- Skin may be cool and clammy
- Lungs are usually clear; crackles present if congestive heart failure develops
- S1, S2 normal; S_3 and/or S_4, murmurs, pericardial friction rub may be present if there are complications

DIFFERENTIAL DIAGNOSIS

- Peptic ulcer disease
- Esophageal spasm or esophagitis
- Gallbladder disease
- Large pulmonary embolism
- Indigestion
- Pancreatitis
- Acute anxiety attack
- Acute pericarditis
- Dissecting aortic aneurysm
- Spontaneous pneumothorax

COMPLICATIONS

- Arrhythmias and conductive disturbances
- Hypotension
- Congestive heart failure
- Pericarditis
- Thromboembolism
- Cardiogenic shock
- Cardiac arrest
- Rupture of the heart
- Death

Young premenopausal women who have had a myocardial infarction have significantly higher mortality rates than men who have had a myocardial infarction at the same age. One possible explanation for this may be the difference in primary prevention strategies applied to women. Management of early symptoms is often less aggressive.

DIAGNOSTIC TESTS

- Obtain a 12-lead electrocardiogram (ECG) tracing; compare with a previous tracing, if available
- Identify new changes if possible; check for Q waves, elevation of ST segment and inversion of T wave (signs of myocardial infarction)
- Check for depression of ST segment, inversion of T wave (angina)

During myocardial infarction in women, Q waves may not be present on ECG. Women are more likely than men to demonstrate nondiagnostic, reversible ST segment elevations or T wave abnormalities.

- If the patient has continuing pain, repeat 12-lead ECG twice more at 30-minute intervals, noting any evolving changes
- Blood may need to be drawn for baseline cardiac enzymes (CK, troponin) before transferring client

MANAGEMENT

Goals of Treatment

- Improve oxygenation of myocardium
- Prevent complications
- Keep infarct from extending

Appropriate Consultation

Consult a physician urgently.

Adjuvant Therapy

– Oxygen via nasal prongs; titrate flow to keep oxygen saturation ≥ 94%[43]
– Start intravenous (IV) therapy with normal saline to keep vein open

Nonpharmacologic Interventions

– Bed rest with head elevated (unless hypotensive)
– Offer support and reassurance to reduce anxiety

Pharmacologic Interventions

Nitrates:

sublingual nitroglycerin 0.4 mg spray prn but only if systolic blood pressure (BP) > 100 mm Hg

Observe response and monitor severity of pain; if pain not relieved, repeat:

0.4 mg q5min for another 2 doses, but only if systolic BP remains > 100 mm Hg

Nitroglycerin can cause headache, hypotension and tachycardia. Nitrates are contraindicated in patients who have taken sildenafil (Viagra, Revatio), tadalafil (Cialis) or vardenafil (Levitra).

Then give:

uncoated acetylsalicylic acid (ASA), 162–325 mg[44,45] stat PO chewed, unless contraindicated (for example, allergy to ASA or NSAIDs, active peptic ulcer)

Patients hypersensitive to ASA or with major gastrointestinal intolerance to ASA should receive clopidogrel on physician consult.

If pain unrelieved by nitrates, administer analgesia:

morphine, 2–5 mg IV; repeat dose only under the direction of a physician

Other pharmacologic measures, as prescribed by a physician:

Beta-blockers are routinely used unless contraindicated (see below). Initial oral dose (example):

metoprolol 50 mg PO BID (range 50–200 mg bid)

Oral administration is preferred. IV administration is associated with an increased risk of cardiogenic shock and is not warranted unless there is ongoing pain at rest especially with tachycardia or hypertension in the absence of contraindications.

Beta-blockers should not be used if heart rate is < 60 bpm, systolic BP is < 100 mm Hg, congestive heart failure or atrioventricular (AV) block is present, or if there is a history of asthma. Use of beta-blockers is not recommended in patients with cocaine-associated myocardial infarction.[42]

Other drugs may be ordered by a physician. Access to a cardiac monitor or defibrillator will influence therapeutic choices.

Ultimately, a thrombolytic medication (for example, streptokinase, tissue plasminogen activator [tPA]) might be required, if it can be given within the first few hours of the onset of chest pain. Tenecteplase is generally the easiest to administer. Clients treated with these agents also need LMWH (for example, enoxaparin) or unfractionated heparin.[45,46]

Monitoring and Follow-Up

– Monitor vital signs (including pulse oximetry)
– Repeat ECG (to check for arrhythmias)
– Monitor lungs and heart sounds frequently for signs of heart failure

Referral

Medevac as soon as possible.

PULMONARY EDEMA

Accumulation of fluid within the lungs that interferes with ventilation and oxygenation.

CAUSES

Acute left-heart failure, with or without right-heart failure *(see "Differential Diagnosis").* Adult respiratory distress syndrome or non-cardiogenic pulmonary edema can occur with severe infections, malignancies and with some medications.

HISTORY

– Severe shortness of breath
– Orthopnea, paroxysmal nocturnal dyspnea (left ventricular failure)
– Fluid retention peripherally and weight gain (right heart failure) may also be present
– Cough productive of frothy pink sputum may be present

PHYSICAL FINDINGS

- Pulse rapid and may be "thready" or weak
- Respiratory rate elevated
- Blood pressure normal, elevated or decreased
- Acute respiratory distress
- Diaphoresis
- Central cyanosis may be present
- Peripheral cyanosis with cool, mottled extremities
- Swelling of ankles may be present
- Jugular venous pressure (JVP) may be elevated
- Hepatojugular reflux and hepatomegaly may be present
- Peripheral pitting edema may be present
- Crackles and wheezes in lower half of lung fields
- S_3 gallop rhythm in the heart

DIFFERENTIAL DIAGNOSIS

- Chronic congestive heart failure
- Acute myocardial infarction
- Acute pulmonary embolism
- Atrial fibrillation
- Valvular heart disease
- Adult respiratory distress syndrome
- Acute exacerbation of COPD

COMPLICATIONS

- Dependent on underlying disease process
- Angina
- Hypotension, shock
- Respiratory failure

DIAGNOSTIC TESTS

- Obtain ECG: look for signs of myocardial ischemia or infarction
- CBC, electrolytes, renal function

MANAGEMENT

Goals of Treatment

- Improve oxygenation
- Promote diuresis of accumulated fluids
- Reduce venous return to the heart
- Treat any reversible precipitants (for example, cardiac ischemia, hypertension, arrhythmia)

Appropriate Consultation

Consult a physician immediately.

Adjuvant Therapy

- Oxygen via non-rebreather mask; titrate flow to keep oxygen saturation ≥ 90%
- Start IV therapy with normal saline to keep vein open

Nonpharmacologic Interventions

- Bed rest with head elevated
- Insert an indwelling urinary catheter (monitor input and output)

Pharmacologic Interventions

IV loop diuretics:

furosemide (Lasix), 40–80 mg IV push

The dose may have to be higher in persons on an oral maintenance dose. It is reasonable to administer an initial dose that is equivalent to the client's usual maintenance dose.[14] Adjust the diuretic dose according to client's response (monitor urine output). Look for improvement in respiratory status.

To reduce venous return and workload on the heart, the physician may order nitrates. All forms of nitrates are effective.

Nitrates:

sublingual nitroglycerin 0.4 mg spray prn

or

transdermal nitroglycerin 0.2 mg/hour patch

but only if systolic blood pressure (BP) > 100 mm Hg.

Nitroglycerin can cause headache, hypotension and tachycardia. Nitrates are contraindicated in patients who have taken sildenafil (Viagra, Revatio), tadalafil (Cialis) or vardenafil (Levitra).

Monitoring and Follow-up

- Monitor vital signs (watch for hypotension) and ABCs (airway, breathing and circulation) frequently, including oxygen saturation
- Monitor urine output hourly (if not diuresing, the client requires more IV diuretics)

Referral

Medevac as soon as possible.

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