

## Nursing III

### Section 1: Loss and Grief

Significance of loss dependant on:

1. Importance of the lost person, object or function
2. Degree of change required because of the loss
3. Persons beliefs and values
  - a. Culture
  - b. Socio economic status
    - i. Severe financial loss and personal loss can create an increased hardship
  - c. Spiritual beliefs
  - d. Support system
  - e. Cause of loss or death.
    - i. Trauma, accident, cancer, cardiac,
    - ii. A death beyond the control of the individual may be more acceptable.
  - f. Gender : males expected to be “strong”

Fear of losing control of oneself may be greater than the fear of death.

Loss: when a valued person, object or situation is changed or made inaccessible so that its value is diminished or removed.

Loss is an opportunity to grow

Stages of Loss

1. Repudiation (denial)
2. Recognition (anger)
  - a. Beginning to accept, hostile and angry
3. Reconciliation (Acceptance)

Factors that influence loss:

- Visibility of loss
- Duration
- Abruptness
- Extent of loss
- Financial impact
- Meaning of illness to patient
- How others affected
- External resources

Coping styles

- Emotional style – unfocused, act out, no problem solving skills
- Fearful
- Suspicious
- Hostile
- Avoiding

#### Recovery

- Value on object
- Degree of support from family/friends
- Cultural and economic support
- Past experience.

Patient work to get better: Give clear, concise, factual info include time, dimensions for all events

- Physical
- Monitor care : teach self care
- Composure: allow patient to verbalize fears and concerns.
- Decision making
- Collaboration

## Section 2 Physical Assessment Lab

### Health Physical

Roles of the Nurse: Obtains the health history and performs a physical assessment.

Purpose:

- Obtain baseline data of functional abilities
  - Supplement, confirm, or refute data obtained in the history
  - Obtain data to help establish a nursing diagnosis and plan of care.
  - Evaluate physiologic outcomes of care and the progress of the health problem
  - Make clinical judgement about the health status
  - Identify areas for health promotion and disease intervention
- Explain why and how the information will be used.
  - Technology : decrease errors
  - Health history is questions to provide a picture of the overall health status.
    - The person providing this history may not always be the patient (informant)
    - Biographical Data
      - Name, address, age, gender, marital status, occupation and ethnic origin.
    - Chief Complaint

- What brings the person to the attention of the health care provider. Use the patient's exact words.
- Present health concerns
  - History of the present illness – single most important factor. Includes the date and the manner of the illness. Includes self treatment, what makes it worse, what makes it better.
- Past history
  - Detailed summary of past health: immunization status, allergies to medications, Patient's last physical, chest x-ray, ECG, eye and hearing test, dental, pap smear, mammogram, digital rectal exam, Childhood illnesses, surgery, etc.
- Family history
  - Identify genetic, communicable or possible environmental origin. First order and second order (grandparents). Conditions: Cancer, Hypertension, heart disease, diabetes, epilepsy, mental illness, tuberculosis, kidney disease, arthritis, allergies, asthma, alcoholism, obesity. Can be recorded in a family tree format.
- Review of systems
  - Overall of general health as well as symptoms related to each body system. Negative as well as positive answers are recorded. Checklist can be used.
- Patient profile
  - More biological information is gathered. Information is highly personal. Events related to health, education, occupation, environment, lifestyle, physical or mental disability, self concept, sexuality, risk for abuse, stress.
- Uncomplicated health problems (ear ache) do not require in depth history.
- With children always start with the LEAST invasive steps

## Respiratory System

### Common Symptoms

1. Dyspnea – common to many cardiac and pulmonary disorders
  - a. Right ventricle is affected - has to pump blood through the lungs which may have greater resistance from disease process.
  - b. Neurologic or neuromuscular disorders
    - i. Myasthenia gravis, Guillain Barré syndrome, MD, post polio disease
  - c. Acute disease produces more severe dyspnea than chronic problems
  - d. Sudden dyspnea, especially in immobile patients can denote pulmonary embolism
2. Cough- reflex that protects the lungs from accumulation of secretions
  - a. Can be a diagnostic clue
    - i. Some disorders cause it and some suppress it
    - ii. Can be impaired by prolonged inactivity, weakness, paralysis, NG tube, depressed brain function

- iii. Coughing at night may be from onset of left sided heart failure, or bronchial asthma. (onset is important to document)
- 3. Sputum
  - a. Chronic bronchitis can cause gradually increasing sputum production
  - b. Foul smelling – lung abscess, bronchiectasis, or infection caused by fusospirochetal or other anaerobic organism.
- 4. Chest pain
  - a. May be associated with pulmonary or cardiac.
  - b. Can be sharp, stabbing, intermittent, dull, aching, and persistent.
  - c. Pain from pleuritic infection can be splinted by laying on the affected side to splint it.
  - d. Assess
    - i. Quality, intensity, and radiation of the pain
- 5. Wheezing
  - a. High pitched musical sound heard mainly on expiration (asthma) or inspiration (bronchitis).
- 6. Rhonchi
  - a. Low pitched continuous sounds heard over the lungs in partial airway obstructions.
- 7. Hemoptysis
  - a. Symptom of both pulmonary and cardiac disease
  - b. Onset usually sudden, may be intermittent or continuous
  - c. Most common causes
    - i. Pulmonary infection
    - ii. Carcinoma of the lungs
    - iii. Abnormalities of the heart or blood vessels
    - iv. Pulmonary artery or vein abnormalities
    - v. Pulmonary embolism or infarct.
  - d. Determine the source of the bleeding (could be gums, nasopharynx, lungs or stomach)

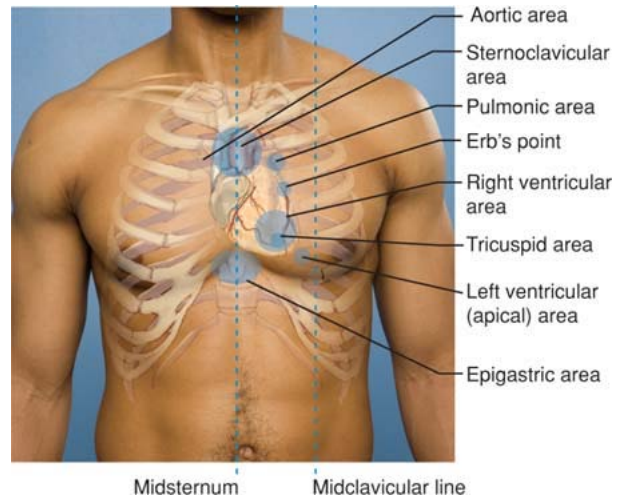
## Respiratory Assessment

- 1. General appearance may have clues to respiratory status
  - a. Clubbing of the fingers
    - i. Chronic hypoxic conditions; sponginess of nail bed and loss of nail bed angle
  - b. Cyanosis
    - i. Late indicator of hypoxia - 5 g/dl of unoxygenated blood.
    - ii. Anemic patients rarely manifest cyanosis
    - iii. Polycythemia patients may appear cyanotic even if adequately oxygenated
- 2. Thoracic Inspection
  - a. Provides information about musculoskeletal structure, nutritional status, and respiratory system.
  - b. Chest configuration: normal ratio of anteroposterior diameter to lateral diameter is 1:2
    - i. Barrel Chest – overinflation of the lungs ( emphysema)

- ii. Funnel Chest (pectus excavatum) : depressed lower portion of the sternum. May occur with rickets or Marfan's syndrome.
  - iii. Pigeon chest (pectus carinatum) : displacement of the sternum resulting in an increase in anteroposterior diameter. Can occur with Marfan's syndrome or severe kyphosis.
  - iv. Kyphoscoliosis: characterized by elevation of the scapula and corresponding S shaped spine. Limits lung expansion within the thorax. May occur with osteoporosis and other skeletal disorders that affect the thorax.
- c. Breathing pattern and rate : normal 12 to 18 breaths per minute.
- d. Thoracic palpation: Palpate the thorax for tenderness, masses, lesions, respiratory excursion and vocal fremitus: Palpate areas of tenderness LAST
- i. Respiratory excursion:
    - 1. Estimation of thoracic expansion. ( decreased expansion may indicate chronic fibrotic disease)
    - 2. Asymmetrical excursion – splinting of one side
  - ii. Tactile Fremitus
    - 1. Sound generated by the larynx travels distally along the bronchial tree to the set the chest wall in resonant motion. Detection of the vibration is called fremitus.
      - a. Normal fremitus is widely varied
      - b. More pronounced in men than women
    - 2. Patient is asked to say “ninety nine” over and over as you move your hands down the thorax. Corresponding areas of the thorax are compared.
      - a. Consolidation in a lobe will increase fremitus. Air in the pleural space does not conduct sound.
  - iii. Thoracic percussion
    - 1. Produces audible and tactile vibrations.
      - a. Flatness – extremely dull sound (very dense tissue ie muscle bone)
      - b. Dullness – thud like sound (dense tissue)
      - c. Resonance – hollow sound produced by lungs filled with air
      - d. Hyperresonance – not produced in the normal body: booming sound, heard over a emphysematous lung
      - e. Tympany – musical or drumlike – air filled stomach.
    - 2. Used to approximate size and location of certain structures within the thorax.
    - 3. Usually begins with the posterior thorax, ideally the patient is sitting.
    - 4. Dullness over the lung fields occur when filled with fluid.
    - 5. Diaphragmatic excursion
      - a. Normal resonance of the lungs stops at the diaphragm

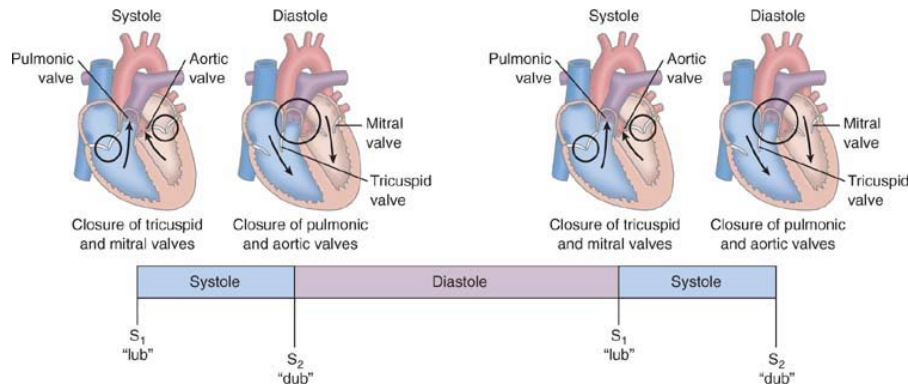


- v. Postural changes in BP
- c. Cardiac Output: look for
  - i. Reduced pulse pressure
  - ii. Hypotension
  - iii. Tachycardia
  - iv. Reduced urine output
  - v. Lethargy or disorientation
- d. Compensatory mechanism
  - i. Peripheral vasoconstriction
  - ii. Tachycardia
- e. Check pulse all arterial areas: temporal, carotid, brachial, radial, femoral , popliteal, dorsalis pedis.
- f. Right side heart function estimated by observing the pulsations of the jugular veins in the neck and the CVP.
  - i. Pulsations of the internal jugular veins are commonly assessed.
  - ii. Visible just above the clavicles adjacent to the sternocleidomastoid muscles.
    - External jugulars are frequently distended when the patient is laying down.
      1. Distention when the patient is above 45 degrees indicates an abnormal increase in the venous system.
      2. Bilateral JVD may indicate right sided heart failure.



- g. Heart inspection and palpation
  - i. Aortic area: second intercostal space to the right of the sternum. :
  - ii. Pulmonic area: second intercostal space to the left of the sternum
  - iii. Erb's point: third intercostal space to the left of the sternum
  - iv. Tricuspid area: Lower half of the sternum along the left parasternal area
  - v. Mitral valve: left fifth intercostal space at the midclavicular line
  - vi. Epigastric area: below the xiphoid process.
- h. Apical pulse palpation:
  - i. Normally only palpable in one intercostal space. If it is found in more than one, then there could be left ventricle enlargement.
- i. Heart sounds
  - i. Normal:
    1. S1=Tricuspid and mitral valve closing (systole), intensity increases during tachycardia and mitral stenosis

2. S2=Pulmonic and aortic valve closing (diastole), The aortic is louder, but the pulmonic slightly lags. In some patients the two can be distinguished, this patient is said to have a split S2. Normal split is



accentuated on inspiration but disappears on expiration.

ii. Abnormal:

1. S3=occurs during a period of rapid ventricular filling. (Lub-dub DUB) (gallop). Can be a normal finding in kids and adults up to age 35. In older adults it is a sign of serious pathophysiology, mainly overload.
2. S4=occurs late in diastole. Occurs just before S1, generated during atrial contraction as blood enters a noncompliant ventricle .(LUB lub-dub)
3. Murmurs : created by turbulent flow. Can be a critically narrowed valve, ventricular wall defect,
4. Friction rub: harsh grating sound. Caused by inflamed pericardial surfaces.

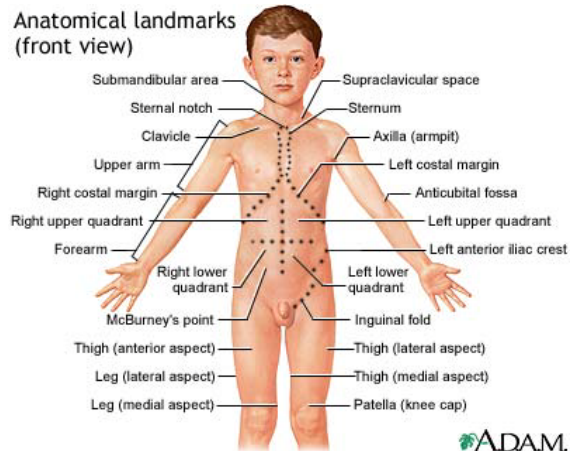
j. Buerger's Test (Arterial Adequacy Test)

- i. Client in the supine position
- ii. Client raises one arm or one leg about 1 foot
- iii. Move the leg or arm up and down briskly for 1 minute
- iv. Have client sit up and dangle the leg or arm
- v. Observe time it takes for original color to return to the arm or leg
  1. Delayed color (greater than 15 seconds) indicates arterial insufficiency.

Abdominal GI assessment

1. Common symptoms

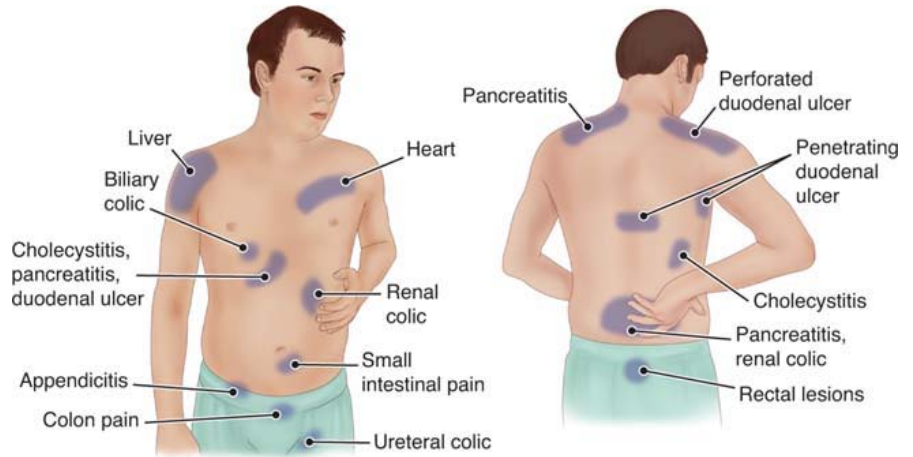
- a. Pain
  - i. Dyspepsia, gas, nausea, vomiting, diarrhea,



constipation, fecal incontinence, jaundice, and previous GI disorders.

ii. Pain is a major symptom

1. Character, duration, pattern, frequency, location, distribution and referred pain.



2. Dyspepsia: commonly called indigestion. Most common symptom

b. Bowel and stool characteristics.

i. Changes in bowel habits may signal colonic dysfunction or disease.

1. Diarrhea – abnormal increase in the frequency and liquidity of the stool
2. Normal stool: light to dark brown.

a. Drugs that may change color:

- i. Senna yellow
- ii. Bismuth, iron, charcoal black
- iii. Barium milky white

b. Stool test:

- i. Consistency, color, occult blood
- ii. Fecal urobilinogen, fecal fat, nitrogen, clostridium difficile, fecal leukocytes, parasites, pathogens, food residue and other substances.
- iii. Occult blood is the most common test.

1. Hemoccult II test (FOBT) – 72 hours prior to testing the patient should not eat red meats, aspirin, nonsteroidal anti-inflammatory drugs, turnips, and horseradish. These can cause a false positive.

3. Full gastrointestinal assessment begins with oral cavity inspection

4. Abdominal inspection, auscultation, palpation, and percussion

- a. Bowel sounds occur at about 5 to 35 per minute
- b. Borborygmi (stomach growling)
- c. Light palpation – identify tenderness and muscle resistance

- d. Deep palpation – identify masses
- 5. Final part of the assessment is the terminal parts of the GI tract, rectum, perianal region and the anus.
- 6. Laboratory studies
  - a. CBC
  - b. Complete metabolic panel
  - c. Prothrombin time/partial thromboplastin time
  - d. Triglycerides
  - e. Liver function tests
  - f. Amylase and lipase
  - g. Cancer studies
- 7. Urea breath test to detect Helicobacter Pylori.
- 8. Abdominal Ultrasonography- used to diagnose gallstones, enlarged ovary, ectopic pregnancy or appendicitis. Now also being used to detect colon diverticulitis.
  - a. Barium studies should be scheduled AFTER the ultrasonography. The barium will interfere with the sound waves.

#### Nervous System Assessment

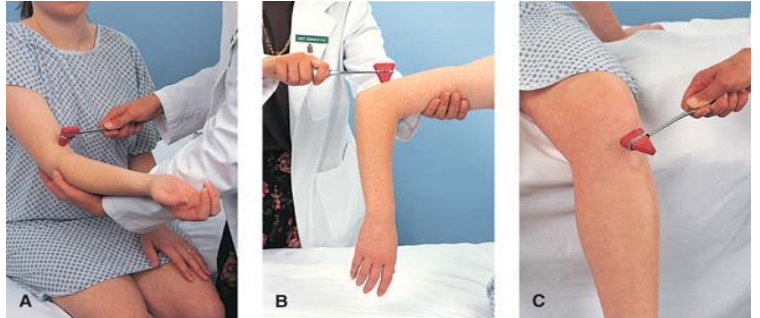
- 1. Health history – history of present illness.
  - a. Details about onset, character, severity, location, duration, and frequency of symptoms
- 2. Assessment areas
  - a. Mental status, cranial nerves, reflexes, motor function, sensory function
- 3. Common nervous system symptoms
  - a. Pain – subjective
    - i. Acute or chronic
      - 1. Acute can be associated with brain hemorrhage, spinal disk disease, or trigeminal neuralgia
      - 2. Chronic can be from degenerative and chronic neurologic conditions (like MS)
  - b. Seizures
    - i. Result of abnormal paroxysmal discharges in the cerebral cortex. – can be the first obvious sign of a brain lesion.
  - c. Dizziness and vertigo
    - i. Causes include viral syndromes, hot weather, roller coaster rides, middle ear infections, etc.
  - d. Visual disturbances
    - i. Range from decreased acuity to sudden blindness caused by glaucoma.
  - e. Muscle weakness
    - i. Common manifestation of neurologic disease. Usually coexists with other symptoms.

- f. Abnormal sensation
  - i. Central and peripheral nervous system.
- 4. Assessing consciousness and cognition
  - a. Specific observations of the patient's mental status, and intellectual functioning
  - b. Appearance, behavior, dress, grooming, posture, facial expressions.
  - c. Oriented to time place and date
  - d. Intellectual function can be tested with simple tests such as counting backwards, or serial sevens (100, 93, 86)
  - e. Thought content: clear, relevant and coherent?
  - f. Point to common objects and have the patient identify them
  - g. Emotional status: patient's affect (external manifestation of mood)
  - h. Language ability:
    - i. Ability to express oneself by speech or written language
    - ii. Receptive aphasia – loss of ability to comprehend written or spoken word
    - iii. Expressive aphasia – loss of ability to express by speech or writing
- 5. Motor System
  - a. Assessment of muscle size, tone, strength, coordination, and balance
    - i. Patient is told to walk across room.
  - b. Muscle strength
    - i. Flex or extend extremities against resistance. Compare one side of the bod with the other.
  - c. Balance and coordination
    - i. Lower leg coordination: have the patient run the heel down the anterior surface of the tibia of the other leg. *Ataxia is the incoordination of voluntary muscle action.*
    - ii. An injury in the cerebellum would affect coordination, smooth movement, and equilibrium.
- 6. Sensory System
  - a. More complex than the motor system.
  - b. Test for tactile sensation, superficial pain, temperature, vibration, and position sense (proprioception)
    - i. Tactile: lightly touching a cotton wisp to a corresponding area on each side of the body.
    - ii. Pain sensation is reserved for patients that cannot or do not sense touch.
    - iii. Vibration is tested with a tuning fork of 128 to 256 Hz The fork is placed against a bony prominence. Distal joints of the great toe, and proximal thumb joint are common test points.
    - iv. Three types of tactile discrimination are tested
      - 1. One and two point discrimination- the ability to sense whether one or two areas of skin are being stimulated
        - a. Fingertips 2.8mm
        - b. Palm 8-12mm

- c. Chest, forearm 40mm
  - d. Back 50-70mm
  - e. Upper arm, thigh 75mm
  - f. Toes 3-8mm
2. Stereognosis – recognizing objects by touching and manipulating them
  3. Extinction – the failure to perceive touch on one side of the body when two symmetrical areas are touched at once.

c. Reflexes Deep tendon reflexes - Reflex hammer used to elicit the reflex

- i. Biceps Reflex: strike bicep tendon over a slightly flexed elbow. (A)
- ii. Triceps Reflex: arm flexed at elbow and positioned in front of chest. Identify triceps tendon by palpating 1 to 2 inches above the elbow. A direct blow on the tendon normally produces contraction of the triceps, and extension of the elbow. (B)
- iii. Brachioradialis Reflex: rest patient's forearm. Gently strike hammer 2.5 to 5 cm above wrist in flexion and supination of the forearm.



- iv. Patellar Reflex: Strike patella tendon, just below the patella. Patient can be sitting or laying. Contractions of the quadriceps and knee extension are normal. (C)



- v. Achilles Reflex: dorsiflex at the ankle and hammer strikes the stretched Achilles tendon. Normally produces plantar flexion. (D)

d. Gerontologic considerations

- i. Loss of neurons occur with age
  1. Results in slower nerve conduction and response time.
  2. Brain weight is decreased, the ventricle size increases to maintain cranial volume.
  3. Cerebral blood flow and metabolism are reduced leading to slower mental functions.
  4. Myelin loss results in slower conduction
- ii. The reduced nerve input results in overall reduction in muscle bulk

- iii. Complaints of pain, such as abdominal discomfort or chest pain, may be more serious than the patient's perception and needs careful evaluation.

## Breast Assessment

### 1. Inspection

- a. Size and symmetry – slight variation is normal.
- b. **In female, the largest portion of glandular tissue is located in the upper outer quadrant of each breast. The same area is also where the majority of breast tumors are located.**
- c. Skin - color, venous pattern, thickening, edema
  - i. Erythema may indicate benign local inflammation or superficial lymphatic invasion by a neoplasm.
  - ii. Prominent venous pattern can signal increased blood supply required by a tumor.
  - iii. Edema and pitting may result from a neoplasm blocking lymphatic drainage, giving the skin an orange peel appearance (peau d'orange), a classic sign of advanced breast cancer.
- d. Patient raises both hands overhead, then place hands on waist and push in. Causes contraction of the pectoral muscles. Any dimpling or retraction during position changes suggest an underlying mass.
- e. Clavicular and axillary regions are inspected for swelling, discoloration, lesions or enlarged lymph nodes.



### 2. Palpation

- a. When the patient is supine, use a pillow behind the shoulders to help balance the breast on the chest wall.
- b. Palpation is done systematically using the flat part of the second, third and fourth fingertips, making dime size circles. Follow a concentric circle pattern.
- c. If the patient reports a lump, begin palpation on the "normal" breast to establish a baseline.
- d. Cysts are commonly found in menstruating woman, and are usually well defined and freely movable.
- e. Malignant tumors tend to be hard, poorly defined and nontender.
- f. On men, the nipple and areola are inspected for masses and nipple discharge.
  - i. Gynecomastia: firm enlargement of glandular tissue beneath and immediately surrounding the areola of the male.

### 3. Self Exam (BSE)

- a. Nurse plays a teaching role

- b. BSE is best performed after menses (day 5 to day7 counting the first day of menses as day1)
  - c. Woman in early 20s should begin BSE
4. Abnormal Findings
- a. Retraction signs
    - i. Skin dimpling, creasing, changes in contour
    - ii. May occur only in positional changes
  - b. Increased venous prominence
    - i. Unilateral localized increase associated with malignant tumors
    - ii. Normal with bilateral and symmetrical breast enlargement associated with pregnancy and lactation
  - c. Peau d'orange (edema)
    - i. Associated with inflammatory breast cancer
    - ii. Caused by interference with the lymphatic drainage
    - iii. Breast skin resembles orange peel
    - iv. Skin pores enlarge
    - v. May be noted on the areola
  - d. Nipple inversion
    - i. Normal if LONG STANDING
    - ii. Associated with fibrosis and malignancy if recent development
  - e. Acute mastitis (inflammation)
    - i. Associated with lactation but may occur at any age
    - ii. Nipple cracks or abrasions noted
    - iii. Breast skin reddened or warm to the touch
    - iv. Tenderness
  - f. Paget's Disease (malignancy of mammary ducts)
    - i. Early signs: erythema of nipple and areola
    - ii. Late signs: thickening, scaling and erosion of the nipple and areola

## Delegation

1. Transferring responsibility for the performance of an activity, while retaining accountability.
2. Team Nursing (today)
  - a. Need to know a lot about all the patients for delegation issues
  - b. Advantage: collaboration with others and team members can contribute their special expertise
3. Nursing is a knowledge based profession.
4. Essential skill for all RN's to know
  - a. Involves critical decision making skills
  - b. Must use good judgment
  - c. Must know the *Nurse Practice Act* (items that cannot be delegated)
5. Delegate repetitive and technical tasks ONLY to unlicensed personal.
  - a. They can do the tasks (collect data) but cannot interpret the data.
  - b. Cannot "monitor" anything.
  - c. Cannot do patient teaching.
  - d. UAPs are there to assist, not replace RNs
  - e. Need for competent, appropriately supervised UAPs
6. How to Delegate
  - a. Assess the situation
  - b. What are the goals of the task?
  - c. Is the patient stable?
  - d. Is there a complex situation?
  - e. Consider the competence of the UAP and the degree of supervision needed.
    - i. Delegate the best qualified UAP
  - f. Evaluate the task completed
7. Five Rights of Delegation
  - a. Right Task
    - i. Must conform to established guidelines. (Nurse Practice Act)
    - ii. They can only collect data
    - iii. Task does NOT require judgment
    - iv. Task does not require complex or multidimensional application of skill
  - b. Right Circumstance
    - i. Must consider patient's acuity level and stability
    - ii. Predictable results
    - iii. Repetitive task – standard,
  - c. Right Person
    - i. Must consider skill level for the task
    - ii. Verify UAPs level of competence
    - iii. Know the level of training
    - iv. Use your own judgement
  - d. Right Direction / communication

- i. Clear, concise description of task. (“ambulate this patient twice around the unit”)
  - ii. Include purpose of task
  - iii. Include time limits
  - iv. State exact expectations
  - v. State when to report back
  - vi. Give positive feedback
- e. Right supervision
  - i. RN must be available to intervene if needed
  - ii. Monitor performance of UAP and evaluate the outcomes
  - iii. Complete proper documentation
    - 1. UAP can document the tasks
- f. Improper Delegation
  - i. KNOW NURSE PRACTICE ACT
  - ii. Consider the Potential for Harm
    - 1. Is the patient going to have an adverse reaction?
  - iii. Complexity of care – multiple systems involved that requires more complex care and treatment.
  - iv. If the patient requires nursing judgement – do not delegate.
  - v. Need for problem solving – if new ways to perform task are needed – **do not delegate**: assist the UAP with the task.
  - vi. Unpredictability of outcome – if unknown how the patient will tolerate the task.
    - 1. Only designate if the patient has a well tolerated treatment
  - vii. Level of interaction: only the nurse can obtain information, teach patient, counsel patient, calm or reassure patient.

## Acidosis and Alkalosis

Disorder	Initial Event	Compensation
Respiratory acidosis	↓ pH, ↑ or normal $\text{HCO}_3^-$ , ↑ $\text{PaCO}_2$	↑ Renal acid excretion and ↑ serum $\text{HCO}_3^-$
Respiratory alkalosis	↑ pH, ↓ or normal $\text{HCO}_3^-$ , ↓ $\text{PaCO}_2$	↓ Renal acid excretion and ↓ serum $\text{HCO}_3^-$
Metabolic acidosis	↓ pH, ↓ $\text{HCO}_3^-$ , ↓ or normal $\text{PaCO}_2$	Hyperventilation with resulting ↓ $\text{PaCO}_2$ (conserves $\text{HCO}_3^-$ )
Metabolic alkalosis	↑ pH, ↑ $\text{HCO}_3^-$ , ↑ or normal $\text{PaCO}_2$	Hypoventilation with resulting ↑ $\text{PaCO}_2$

Acidosis: too much Acid

pH concentration of  $\text{H}^+$  ions

P=negative logarithm

*Run inversely*

pH 5=10 -5 .00001

pH7=10 -7 .0000001

Balance maintained by

1. Buffers
  - a. Chemicals that act like a sponge
  - b. Phosphate buffers (potassium phosphate : potassium and a buffer)
  - c. Protein, esp. Hgb (can pick up a hydrogen)
  - d. Bicarbonate/Carbonic acid
    - i.  $\text{CO}_2 + \text{H}_2\text{O} = \text{H}_2\text{CO}_3 = \text{H}^+ + \text{H}_2\text{CO}_3^-$
2. Lungs  $\text{CO}_2$  a volatile acid  $\text{H}_2\text{CO}_3$ 
  - a. Blow off and retain as needed : acts quickly
3. Kidneys
  - a. Secrete  $\text{H}^+$  ions for removal
  - b. Exchange Na for H ions and remove
  - c.  $\text{NH}_3$  becomes  $\text{NH}_4$

Acidosis: too much acid (Respiratory)

1.  $\text{CO}_2 + \text{H}_2\text{O} = \text{H}_2\text{CO}_3$  Carbonic Acid
1. Lungs control carbon dioxide

2. Low pH
3. High carbon dioxide
4. Normal bicarbonate

Acidosis: too much acid (Metabolic)

1. Ketoacids
2. Lactic Acids
  - a. Poor perfusion (not enough oxygen to tissue)
    - i. Patients in shock
    - ii. Septic shock
3. Ingestion acid (aspirin overdose)
4. Decreased elimination acids (kidney failure)

Acidosis: not enough base

1. Loss of intestinal base
  - a. Diarrhea, c-diff
  - b. If you losing things from the ass, your acidotic
  - c. Pancreatic problem

Alkalosis : not enough acid

2. Decreased CO<sub>2</sub>
3. Means less H<sub>2</sub>CO<sub>3</sub>
4. Respiratory : hyperventilation
5. Metabolic: excess vomiting, loss of HCL from stomach

Alkalosis: too much base

1. Ingesting bicarbonate
2. Diuretics: dump sodium, potassium, and hydrogen ions through the kidneys, the kidneys reabsorb excess bicarb ions.

Laboratory Values

1. pH 7.35 – 7.45
2. pO<sub>2</sub> above 90 (dissolved in blood, not a %)
3. pCO<sub>2</sub> 35-45
4. HCO<sub>3</sub> (bicarb) 22-26
5. Base excess +2 to -2

Pulse Oximeter

1. Light emitting and light receiving
2. % of Hgb carrying oxygen

In a respiratory acidosis, buffers come into play, the respiration increase, the renal system absorb and dump hydrogen ions, If this is not possible, decompensation occurs

CO<sub>2</sub> is a vasal dilator, symptoms include, headache, lethargy, nausea, vomiting

Disorientation

- Muscle weakness (from potassium imbalance)
- High bicarb levels
- Urine acidotic

Metabolic Acidosis

- Acids accumulate
- Buffers
- Respiratory (Kussmaul's respirations)
- Renal (acidic patient) *can take bicarb tablets*
- Symptoms: Fast respirations, acidic urine, weak flaccid muscles, lethargy, headache, confusion

Respiratory Alkalosis

- Buffers : excrete bicarb
- Urine : alkaline
- Respirations will slow
- Decompensation symptoms
  - Risk for cardiac dysrhythmias
  - Tachycardia
  - Anxiety sweating
  - Numbness, tingling
  - seizures

Metabolic Alkalosis

- buffers will try to soak up bicarb and release hydrogen ions
- Respirations : slow
- Renal: retain hydrogen ions, urine alkalotic
- Decompensation
  - Decreased LOC
  - Tetany
  - Convulsions
  - Dysrhythmias
  - Paralytic ileus
  - Tingling dizziness

Overview

1. Look at pt's history
2. Determine pH
3. Leaning in one direction?
4. Determine cause
5. Acidosis: is CO<sub>2</sub> the cause or bicarb?

# Diabetes Mellitus

## Definitions:

<b>CGMS</b>	A device that continuously monitors blood glucose levels (typically 72 hours)
<b>Sub Q insulin Pump</b>	Small device that delivers insulin on a 24 hour basis
<b>Diabetes mellitus</b>	A group of metabolic diseases. Results from defects in insulin secretion and/or actions
<b>Diabetic Ketoacidosis DKA</b>	Metabolic derangement of type 1, resulting from insulin deficiency, acidosis
<b>Fasting Plasma Glucose</b>	Blood glucose level after fasting for more than 8 hours
<b>Gestation diabetes</b>	Glucose intolerance during pregnancy
<b>Glycated hemoglobin</b>	A long term measure of glucose that is the result of glucose attachment to hemoglobin
<b>HNNS</b>	Disorder of type 2 results from insulin deficiency initiated by illness that raises insulin demand
<b>Insulin</b>	Hormone secreted by the beta cells of the islets of Langerhans of the pancreas
<b>Ketone</b>	Highly acidic substance formed when the liver breaks down free fatty acids in the absence of insulin
<b>Nephropathy</b>	Long term complication of diabetes in which the kidney is damaged
<b>Neuropathy</b>	Long term complication of diabetes in which nerve cells are damaged
<b>Retinopathy</b>	Microvascular system of the eye is damaged by long term diabetes
<b>Type 1 diabetes</b>	Diabetes from the absence of insulin production
<b>Type 2 diabetes</b>	Diabetes from the relative deficiency of insulin production and decreased insulin action

## Risk factors

- ❖ Family history
- ❖ Obesity: >20% over desired body weight or BMI
- ❖ Race/ethnicity: black, Hispanic
- ❖ Age: > 45
- ❖ Previously identified impaired fasting glucose or impaired glucose tolerance
- ❖ Hypertension > 140/90 mm Hg
- ❖ HDL cholesterol level <35 mg/dl and or triglyceride level >250 mg/dl
- ❖ History of gestational diabetes or delivery of babies over 9 lbs

## Foundation of diabetes management: Nutrition, meal planning, and weight control

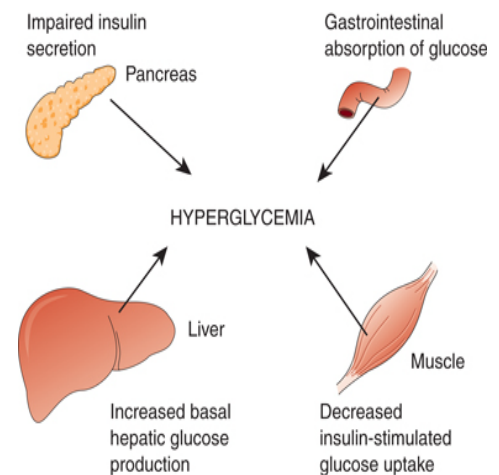
- Objective: control total caloric intake, maintain a reasonable body weight, control blood glucose levels, and normalize lipids and blood pressure to prevent heart disease.
- Success is often associated with reversal of hyperglycemia in type 2 diabetes.

Glucagon: pancreatic hormone

## Insulin transports and metabo

1. Type 1 diabetes
  - a. Characterized by the destruction of the pancreatic beta cells. This causes acute onset.
    - i. Results in unchecked glucose production by the liver and hyperglycemia.
    - ii. Unchecked glucose production by the liver
  - b. Effects 5 to 10% of people with the disease
  - c. Causes
    - i. Genetic susceptibility
    - ii. Autoimmune response
    - iii. Viruses and toxins (environmental)
  - d. Glucose derived from food cannot be stored in the liver.
  - e. Fat breakdown resulting in increased production of ketone bodies.
    - i. Ketones acids upset the acid base balance.

- ii. Resulting diabetic ketoacidosis (DKA) causes signs and symptoms of:
    1. Abdominal pain
    2. Nausea
    3. Vomiting
    4. Hyperventilation
    5. Fruity breath odor
    6. Decreased LOC
    7. Coma
    8. Death
  - f. Type 1: not inherited but a genetic predisposition combined with immunologic and possibly environmental (viral) factors
2. Type 2 diabetes
- a. Occurs more commonly among people older than 30
  - b. Main problems are insulin resistance and impaired insulin secretion
    - i. Insulin resistance: decreased tissue sensitivity to insulin
    - ii. In order to compensate, increased amounts of insulin must be secreted to maintain the glucose level at a normal, or slightly elevated level. (metabolic syndrome).
      1. Metabolic syndrome: hypertension, hypercholesterolemia, and abdominal obesity.
    - iii. If the beta cells cannot keep up with the increased demand, the glucose level rises and type 2 diabetes develops.
  - c. Type 2 diabetes is a slow progression and it may go undiscovered for years
  - d. Symptoms include:
    - i. Fatigue, irritability, polyuria, polydipsia, polyphagia, poorly healing skin wounds, vaginal infections, and blurred vision.
    - ii. Long term complications: eye disease, peripheral neuropathy, peripheral vascular disease.
  - e. Treatment
    - i. Diet and exercise
    - ii. Oral antidiabetic agents
    - iii. Insulin
  - f. Type 2 Risk Factors: family history of diabetes, obesity, race/ethnicity, age greater than 45 years, previous identified impaired fasting glucose or impaired glucose tolerance, hypertension  $\geq 140/90$ , HDL  $\leq 35$  and/or triglycerides  $\geq 250$ , history of gestational diabetes or babies over 9 pounds
  - g. Clinical Manifestations
    - i. "Three Ps"
      1. Polyuria (from osmotic diuresis)
      2. Polydipsia
      3. Polyphagia (eating)
    - ii. Fatigue, weakness, vision changes, tingling or numbness in hands or feet, dry skin, skin lesions or wounds that are slow to heal, recurrent infections



- h. Type 1 may have sudden weight loss, nausea, vomiting, and abdominal pain if DKA has developed
  - i. Diagnostic Findings
    - i. Fasting blood glucose (FPG) 126 mg/dL or more
    - ii. Random glucose exceeding 200 mg/dL
    - iii. Gerontologic considerations: age-related elevation of blood glucose
3. Dietary Management Goals
    - a. Provide optimal nutrition; all essential food constituents
    - b. Meet energy needs
    - c. Achieve and maintain a reasonable weight
    - d. Prevent wide fluctuations of blood glucose levels
    - e. Decrease serum lipids, if elevated
  4. Role of the Nurse
    - a. Be knowledgeable about dietary management
    - b. Educate
      - i. Monitoring
      - ii. Medications
      - iii. Diet/nutrition
      - iv. Exercise/activity
      - v. Foot care
    - c. Communicate important information to the dietician or other management specialists
    - d. Reinforce patient understanding
    - e. Support dietary and lifestyle changes
    - f. Daily adjustment in therapy for patient
  5. SICK DAY
    - a. Managing diabetes when Ill
    - b. Do not eliminate insulin dose if nausea and vomiting occur
    - c. Take usual dose
    - d. Try to consume frequent small portion carbohydrates
    - e. Assess blood glucose and ketones every 3-4 hours
  6. Meal Planning (normally done by a dietitian)
    - a. Consider food preferences, lifestyle, usual eating times, and cultural/ethnic background
    - b. Review diet history and need for weight loss, gain, or maintenance
    - c. Caloric requirements and calorie distribution throughout the day
      - i. Based on age, gender, height/weight, activity
    - d. Carbohydrates: 50–60% carbohydrates, emphasize whole grains (break down 100% rapidly into glucose)
    - e. Fat: 20–30%, with >10% from saturated fat and < 300 mg cholesterol
    - f. Fiber
  7. Glycemic Index
    - a. Describes how much a food increases blood glucose
    - b. Combine starchy food with protein and fat containing food slows absorption, and glycemic response
    - c. Raw or whole foods tend to have lower response than cooked, chopped, or pureed foods
    - d. Eat whole fruits rather than juices; decreases glycemic response due to fiber-slowing absorption

- e. Adding food with sugars may produce lower response if eaten with foods that are more slowly absorbed
8. Insulin Therapy : blood glucose monitoring
- a. Insulin : hormone produced by the pancreas
  - b. Some monitors are calibrated for blood glucose and some for plasma glucose, plasma is 10-15% higher

c. Hemoglobin A1C – percentage of hemoglobin with a sugar molecule (normal 4 – 5.6%) :

Measures Long term average blood glucose levels. (3 month period)

A1c(%)	Mean blood sugar (mg/dl)
6	135
7	170
8	205
9	240
10	275
11	310
12	345

- d. Categories of insulin
  - i. Rapid-acting (Onset 15, peak 30-60min)
    - 1. Lispro (Humalog)
    - 2. Aspart (Novolog)
    - 3. Glulisine (Apidra)
  - ii. Short-acting (Onset30-60min)
    - 1. Regular (Humalog R, Novolin R, Iletin II Regular)
  - iii. Intermediate-acting( Onset 2-4, Peak 4-12hrs)
    - 1. NPH (neutral protamine hagedorn)
    - 2. Humulin N, Iletin II NPH, Novolin L (lente), Novolin N (NPH)
  - iv. Very long-acting
    - 1. Glargine (Lantus)
    - 2. Detemir (Levemir)
  - v. Inhaled insulin

9. Primary goal of treatment

- a. Controlling blood glucose levels
- b. Preventing acute and long term complications
- c. Help diabetic patients to develop self care management and skills

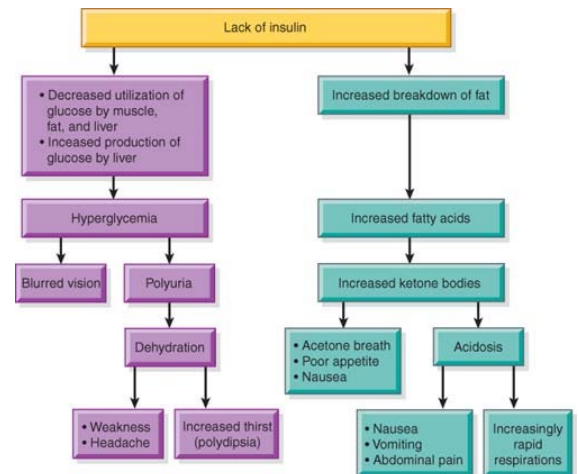
10. Acute Complications of Diabetes

- a. Hypoglycemia
  - i. Abnormally low blood glucose level (below 50–60 mg/dL)
  - ii. Causes include too much insulin or oral hypoglycemic agents, too little food, and excessive physical activity
  - iii. manifestations
  - iv. Adrenergic symptoms: sweating, tremors, tachycardia, palpitations, nervousness, hunger
  - v. Central nervous system symptoms: inability to concentrate, headache, confusion, memory lapses, slurred speech, numbness of lips and tongue, irrational or combative behavior, double vision, drowsiness
  - vi. Severe hypoglycemia may cause disorientation, seizures, and loss of consciousness
  - vii. Assessment
    - 1. Onset is abrupt and may be unexpected
    - 2. Symptoms vary from person to person
    - 3. Symptoms also vary related to the rapidly of decrease in blood glucose and usual blood glucose range

4. Decreased adrenergic response may affect symptoms in persons who have had diabetes for many years probably related to autonomic neuropathy
- viii. Treatment
1. **Treatment must be immediate**
  2. **Give 15 g of fast-acting, concentrated carbohydrate**
  3. 3 or 4 glucose tablets
  4. 4–6 ounces of juice or regular soda (not diet soda)
  5. 6–10 hard candies
  6. 2–3 teaspoons of honey
  7. Retest blood glucose in 15 minutes, retreat if >70 mg/dL or if symptoms persist more than 10–15 minutes and testing is not possible.
  8. Provide a snack with protein and carbohydrate unless the patient plans to eat a meal within 30–60 minutes.
- ix. Emergency measures if the patient is unable to swallow or is unconscious
1. Glucagon 1mg IM or SQ
  2. 25-50ml of 50% dextrose (D50) IV

b. Diabetic Ketoacidosis (DKA) (type 1 diabetes)

- i. Caused by an absence of or inadequate amount of insulin resulting in abnormal metabolism of carbohydrate, protein, and fat
- ii. Clinical features
- iii. Hyperglycemia
- iv. Dehydration, electrolyte loss
- v. Acidosis
- vi. Manifestations include polyuria, polydipsia, blurred vision, weakness, headache, anorexia, abdominal pain, nausea vomiting, acetone breath, hyperventilation with Kussmaul respirations, and mental status changes



- vii. Assessment of DKA
1. Blood glucose levels vary from 300–800 mg/dL
  2. Severity of DKA is not related to blood glucose level
  3. Ketoacidosis is reflected in low serum bicarbonate and low pH; low PCO<sub>2</sub> reflects respiratory compensation
  4. Ketone bodies in blood and urine
  5. Electrolytes vary according to water loss and level of hydration
- viii. Treatment of DKA
1. Rehydration with IV fluid
  2. IV continuous infusion of regular insulin
  3. Reverse acidosis and restore electrolyte balance
  4. **Note: rehydration leads to increased plasma volume and decreased K<sup>+</sup>, insulin enhances the movement of K<sup>+</sup> from extracellular fluid into cells**
  5. **Monitor**
  6. Blood glucose and renal function/UO
  7. EKG and electrolyte levels—**Potassium**

## 8. VS, lung assessments, signs of fluid overload



### Nursing Alert

Because a patient's serum potassium level may drop quickly as a result of rehydration and insulin treatment, potassium replacement must begin once potassium levels drop to normal.

- c. Hyperglycemic Hyperosmolar Nonketotic Syndrome (type 2 Diabetes)
  - i. Hyperosmolality and hyperglycemia occur due to lack of effective insulin. Ketosis is minimal or absent.
  - ii. Hyperglycemia causes osmotic diuresis with loss of water and electrolytes; hypernatremia, and increased osmolality occur.
  - iii. Manifestations include hypotension, profound dehydration, tachycardia, and variable neurologic signs due to cerebral dehydration.
  - iv. High mortality.
  - v. Treatment
    - 1. Rehydration
    - 2. Insulin administration
    - 3. Monitor fluid volume and electrolyte status
  - vi. Prevention
    - 1. BGSM
    - 2. Diagnosis and management of diabetes
    - 3. Assess and promote self-care management skills

## 11. Nursing Process

- a. Assess the primary presenting problem
- b. In addition, assess needs related to diabetes
- c. Patient knowledge of diabetes and diabetes care skills
- d. Blood glucose levels
- e. Skin assessment
- f. Preventative health measures
- g. Teaching Patients self care
  - i. Assess knowledge and adherence to plan of care.
  - ii. Provide basic information about diabetes, its cause and symptoms, and acute and chronic complications and their treatment.
  - iii. Teach self-care activities to prevent long-term complications including foot care, eye care, and risk-factor management.
  - iv. Include family in plan of care.
  - v. Provide information, encourage health promotion activities, and recommended health screenings.

- h. Diabetes mellitus is a chronic illness that requires a lifetime of special self-management behaviors. Because MNT, physical activity, and physical and emotional stress affect diabetic control, patients must learn to balance a multitude of factors

12. Insulin Treatment

- a. Insulin varies according to three main characteristics
- i. Time course of action
    1. Based on onset, peak and duration of action
    2. Human insulin has shorter duration because animal insulin triggers an immune response.
    3. Rapid acting has a shorter duration (called regular insulin)
      - a. Only insulin for IV use
    4. Intermediate acting (called NPH) appear white and cloudy
    5. Very long acting (peakless) are used as a basal insulin. This CANNOT be mixed with other insulin because it has a pH of 4 and will precipitate other insulin
  - ii. Species or source
    1. In the past, all insulins were obtained from beef (cow) and pork (pig) pancreases. DNA technology has replaced the need for animal sources.

Time Course	Agent	Onset	Peak	Duration	Indications
Rapid-acting	Lispro (Humalog) Aspart (Novolog) Glulisine (Apidra)	10–15 min 5–15 min 5–15 min	1 h 40–50 min 30–60 min	2–4 h 2–4 h 2 h	Used for rapid reduction of glucose level, to treat postprandial hyperglycemia, and/or to prevent nocturnal hypoglycemia
Short-acting	Regular (Humalog R, Novolin R, Iletin II Regular)	½–1 h	2–3 h	4–6 h	Usually administered 20–30 min before a meal; may be taken alone or in combination with longer-acting insulin
Intermediate-acting	NPH (neutral protamine Hagedorn) (Humulin N, Iletin II Lente, Iletin II NPH, Novolin L [Lente], Novolin N [NPH])	2–4 h 3–4 h	4–12 h 4–12 h	16–20 h 16–20 h	Usually taken after food
Very long-acting	Glargine (Lantus) Detemir (Levemir)	1 h	Continuous (no peak)	24 h	Used for basal dose

- b. Complications of insulin therapy
- i. Local Allergic Reactions. A local allergic reaction may appear at the injection site 1 to 2 hours after the insulin administration. These reactions, which usually occur during the beginning stages of therapy and disappear with continued use of insulin, are becoming rare because of the increased use of human insulins. A antihistamine can be taken 1 hour before the injection.
  - ii. Systemic Allergic Reactions. Systemic allergic reactions to insulin are rare. When they do occur, there is an immediate local skin reaction that gradually spreads into generalized urticaria (hives). The treatment is desensitization, with small doses of insulin administered in gradually increasing amounts using a desensitization kit.
  - iii. Insulin Lipodystrophy. Lipodystrophy refers to a localized reaction, in the form of either lipoatrophy or lipohypertrophy, occurring at the site of insulin injections. Lipoatrophy is loss of subcutaneous fat; it appears as slight dimpling or more serious pitting of subcutaneous fat. The use of human insulin has almost eliminated this disfiguring complication.
  - iv. Lipohypertrophy, the development of fibrofatty masses at the injection site, is caused by the repeated use of an injection site. If insulin is injected into scarred areas, absorption

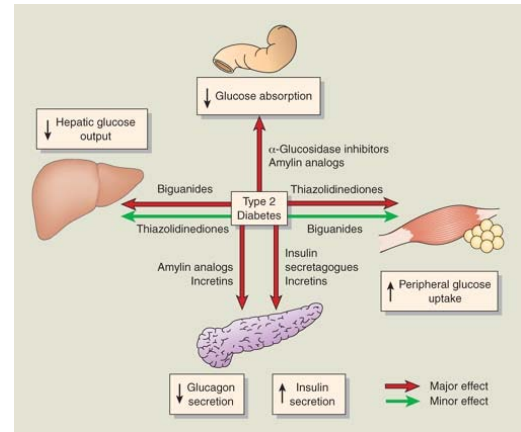
may be delayed. Patients should avoid injecting insulin into these areas until the hypertrophy disappears.

- v. Resistance to Injected Insulin. Most patients have some degree of insulin resistance at one time or another. The most common cause is obesity, which can be overcome by weight loss. Clinical insulin resistance has been defined as a daily insulin requirement of 200 units or more. In most patients with diabetes who take insulin, immune antibodies develop and bind the insulin, thereby decreasing the insulin available for use. All animal insulins, and human insulins to a lesser degree, cause antibody production in humans.

### 13. Oral medications

Generic (Trade) Name	Action/Indications	Side Effects	Implications
<b>First-Generation Sulfonylureas</b>			
Acetohexamide (Dymelor) Chlorpropamide (Diabinese) Tolazamide (Tolinase) Tolbutamide (Orinase)	Used infrequently in U.S. today Used in type 2 diabetes to control blood glucose levels <b>Stimulate beta cells of the pancreas to secrete insulin;</b> may improve binding between insulin and insulin receptors or increase the number of insulin receptors	Hypoglycemia Mild GI symptoms Weight gain Drugdrug interactions (NSAIDs, warfarin, sulfonamides) Sulfa allergy Skin reactions	Monitor patient for hypoglycemia Monitor blood glucose and urine ketone levels to assess effectiveness of therapy Patients at high risk for hypoglycemia: advanced age, renal insufficiency <b>When taken with beta-adrenergic blocking agents may mask usual warning signs and symptoms of hypoglycemia</b> Instruct patients to avoid use of alcohol Check for interactions with other medications
<b>Second-Generation Sulfonylureas</b>			
Glipizide (Glucotrol, Glucotrol XL) Glyburide (Micronase, Glynase, Dia-Beta) Glimepiride (Amaryl)	<b>Stimulate beta cells of the pancreas to secrete insulin;</b> may improve binding between insulin and insulin receptors or increase the number of insulin receptors Used in type 2 diabetes to control blood glucose levels Have more potent effects than first-generation sulfonylureas May be used in combination with metformin or insulin to improve glucose control	Hypoglycemia Mild GI symptoms Weight gain Drugdrug interactions (NSAIDs, warfarin, sulfonamides) Sulfa allergy	Monitor patient for hypoglycemia Monitor blood glucose and urine ketone levels to assess effectiveness of therapy Patients at high risk for hypoglycemia: advanced age, renal insufficiency When taken with beta-adrenergic blocking agents, may mask usual warning signs and symptoms of hypoglycemia Instruct patients to avoid use of alcohol
<b>Biguanides</b>			
Metformin (Glucophage, Glucophage XL, Fortamet) Metformin with glyburide (Glucovance)	<b>Inhibit production of glucose by the liver</b> <b>Increase body tissues' sensitivity to insulin</b> Decrease hepatic synthesis of cholesterol Used in type 2 diabetes to control blood glucose levels	Lactic acidosis Hypoglycemia if metformin is used in combination with insulin or other antidiabetic agents Drugdrug interaction GI disturbances Contraindicated in patients with impaired renal or liver function, respiratory insufficiency, severe infection, or alcohol abuse	Monitor for lactic acidosis and hypoglycemia Monitor renal function <b>Patients taking metformin are at increased risk of acute renal failure and lactic acidosis with use of iodinated contrast material for diagnostic studies; metformin should be stopped 48 h prior to and for 48 h after use of contrast agent or until renal function is evaluated and normal</b> Check for interactions with other medications
<b>Alpha-Glucosidase Inhibitors</b>			
Acarbose (Precose) Miglitol (Glyset)	<b>Delay absorption of complex carbohydrates in the intestine and slow entry of glucose into systemic circulation</b> Do not increase insulin secretion Used in type 2 diabetes to control blood glucose levels Can be used alone or in combination with sulfonylureas, metformin, or insulin to improve glucose control	Hypoglycemia (risk increased if used with insulin or other antidiabetic agents) GI side effects (abdominal discomfort or distention, diarrhea, flatulence) Drugdrug interactions	<b>Must be taken with first bite of food to be effective</b> Monitor for GI side effects (diarrhea, abdominal distention) Monitor for blood glucose levels to assess effectiveness of therapy <b>Monitor liver function</b> studies every 3 mo for 1 y, then periodically Contraindicated in patients with GI or renal dysfunction, or cirrhosis <b>Alert: Hypoglycemia must be treated with glucose, not sucrose</b>
<b>Non-Sulfonylurea Insulin Secretagogues</b>			
Repaglinide (Prandin) categorized as a meglitinide Nateglinide (Starlix) categorized as a D-phenylalanine derivative	<b>Stimulate pancreas to secrete insulin</b> Used in type 2 diabetes to control blood glucose levels Can be used alone or in combination with metformin or thiazolidinediones to improve glucose control	Hypoglycemia/weight gain less likely than sulfonylureas Drugdrug interactions (with ketoconazole, fluconazole, erythromycin, rifampin, isoniazid)	Monitor blood glucose levels to assess effectiveness of therapy Has rapid action and short half-life Should be taken only if able to eat a meal immediately Teach patients symptoms of hypoglycemia Monitor patients with impaired liver function and renal impairment Has no effect on plasma lipids Is taken before each meal Check for interactions with other medications
<b>Thiazolidinediones (or glitazones)</b>			
Pioglitazone (Actos) Rosiglitazone (Avandia)	Sensitize body tissue to insulin; <b>stimulate insulin receptor sites to lower blood glucose and improve action of insulin</b> May be used alone or in combination with sulfonylurea, metformin, or insulin <b>Increases sensitivity at receptor site</b>	Hypoglycemia (risk increased with use of insulin or other antidiabetic agents) Anemia Weight gain, edema <b>Decrease effectiveness of oral contraceptives</b> Possible liver dysfunction Drugdrug interactions Hyperlipidemia (has variable effect on lipids; pioglitazone may be preferred choice in patients with lipid abnormalities) Impaired platelet function	Monitor blood glucose levels to assess effectiveness of therapy Monitor liver function tests Arrange dietary teaching to establish weight control program <b>Instruct patient taking oral contraceptives about increased risk of pregnancy</b>
<b>Dipeptidyl Peptidase-4 (DPP-4) Inhibitor</b>			
Sitagliptin (Januvia) Vildagliptin (Galvus)	<b>Increase and prolongs the action of incretin, a hormone that increases insulin release and decreases glucagon levels,</b> with the result of improved glucose control	Upper respiratory infection Stuffy or runny nose and sore throat Headache Stomach discomfort and diarrhea Hypoglycemia, if used with sulfonylurea	<b>Usually administered once a day</b> Used alone or with other oral antidiabetic agents Instruct patient about signs and symptoms of hypoglycemia and other adverse effects to report Monitor renal function

- a. Action sites of hypoglycemic agents and mechanisms of lowering blood glucose in type 2 diabetes. The incretins are the dipeptidyl peptidase-4 (DPP-4) inhibitors and glucagon-like peptide-1 (GLP-1) agonists
- b. For patients with newly diagnosed type 2 diabetes, emphasis is placed on meal planning and exercise.
- c. Plan in depth and continuing education
- d. Assess readiness to learn
  - i. Patients and their families may go through a process similar to grieving.



#### 14. Nursing Diagnoses

- Risk for fluid volume deficit related to polyuria and dehydration
- Fluid and electrolyte imbalance related to fluid loss or shifts
- Deficient knowledge about diabetes self-care skills or information
- Anxiety related to loss of control, fear of inability to manage diabetes, misinformation related to diabetes, fear of diabetes complications

#### **Collaborative Problems/Potential Complications**

Based on assessment data, potential complications may include the following:

- Fluid overload, pulmonary edema, and heart failure
- Hypokalemia
- Hyperglycemia and ketoacidosis
- Hypoglycemia
- Cerebral edema

#### 15. Interventions

- a. Maintaining fluid and electrolyte balance
  - i. I&O, IVs as proscribed
  - ii. Vitals hourly, LOC, Edema, cardiac status
- b. Monitor for
  - i. Fluid overload
  - ii. Hypokalemia
  - iii. Cerebral edema
- c. Teach patient self-care

#### 16. Expected outcomes

- a. Achieve fluid and electrolyte balance
  - i. Demonstrates intake and output
  - ii. Exhibits electrolytes within normal limits
  - iii. Vitals stable, resolution of orthostatic hypotension and tachycardia
- b. Demonstrates knowledge about DKA and HHNS
- c. Absence of complications

#### 17. Long term complications of Diabetes

- a. Macrovascular complications
  - i. MI is twice as likely in men
  - ii. Blood vessels walls thicken, sclerose and become occluded with plaque.

- iii. Coronary artery disease, cerebrovascular disease and peripheral vascular disease are the three main types
- b. Diabetic retinopathy
  - i. Leading cause of blindness among ages 20 to 74 in the US
  - ii. Caused by changes in the small blood vessels in the retina.
- c. Nephropathy
  - i. Renal disease, common complication of diabetes.
  - ii. Decreased renal clearance change insulin needs
  - iii. Urine should be checked for presence of microalbumin.
    - 1. End stage needs dialysis
- d. Peripheral Neuropathy
  - i. Affects the distal portions of nerves, especially the lower extremities.
  - ii. Initial symptoms include paresthesias (prickling, tingling, or heightened sensation)
  - iii. Decreased deep tendon reflexes and vibratory sensation.
- e. Foot and Let Problems
  - i. 50-75% of lower extremity amputations are performed on people with diabetes. Half are preventable.
  - ii. Neuropathy, Peripheral vascular disease, and immunocompromise complications (infections) of diabetes contribute.
    - 1. Typically, a diabetic foot ulcer develops which the patient cannot feel, this becomes infected and wound healing is delayed due to the lack of circulation. Amputation becomes necessary to prevent the spread of the infection.

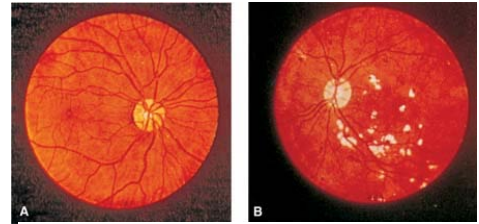


Fig. B shows waxy looking retinal lesions, microaneurysms of vessels and hemorrhages



## Pharmacology

Diabetics who undergo surgery

Increase stress

## Oxygen Deprivation and Acute Respiratory Problems

Lungs: lower respiratory system

Needs to be airtight

Adequate tissue exchange depends on the ventilation / perfusion ratio

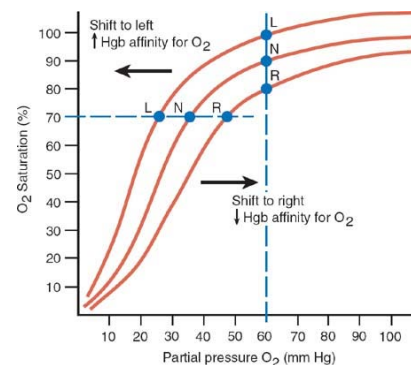
1. Normal V/Q
  - a. Equal (V=P) 1:2
2. Low V/Q (shunt)
  - a. Low ventilation perfusion, perfusion exceeds ventilation
    - i. Blood bypasses alveoli and gas exchange does not occur
3. High v/q (dead space)
  - a. Ventilation exceeds perfusion
    - i. Alveoli do not have an adequate blood supply
4. Silent unit (pneumothorax, or acute respiratory distress)
  - a. No exchange or minimal

*Hypoxia: deficiency of oxygen*

*Hypoxemia: decreased oxygen concentration in blood, measured by PaO<sub>2</sub>*

Oxyhemoglobin dissociation curve : *relationship between the partial pressure of oxygen (PaO<sub>2</sub>) and the percentage of saturation of oxygen (SaO<sub>2</sub>).*

1. An increase in carbon dioxide, hydrogen ion concentration, temperature, and 2,3-diphosphoglycerate shifts the curve to the right so that less oxygen is picked up in the lungs but more oxygen is released to the tissues, if PaO<sub>2</sub> is unchanged.
2. A decrease in these factors causes the curve to shift to the left, making the bond between oxygen and hemoglobin stronger. If the PaO<sub>2</sub> is still unchanged, more oxygen is picked up in the lungs, but less oxygen is given up to the tissues.
3. There is a distinct advantage to the patient for two reasons
  - a. If the PaO<sub>2</sub> decreases from 100 to 80mm Hg as a result of lung disease or heart disease, the hemoglobin of the arterial blood remains almost maximally saturated and the tissues do not suffer.
  - b. When the arterial blood passes into tissue capillaries and is exposed to the tissue tension of oxygen (about 40mm Hg), hemoglobin gives up large quantities of oxygen to the tissues.
4. Normal PaO<sub>2</sub> is 80 to 100 and SaO<sub>2</sub> is 95-98%: there is a 15% margin of excess oxygen available to the tissues.
  - a. If a patient had a normal hemoglobin of 15mg/dl but had a PaO<sub>2</sub> of 40mmHg (SaO<sub>2</sub> 75%), there is adequate oxygen for the tissue , but no reserve for stresses that increase demand.



**Table 21-1 Lung Volumes and Lung Capacities**

Term	Symbol	Description	Normal Value*	Significance
Lung Volumes				
Tidal volume	VT or TV	The volume of air inhaled and exhaled with each breath	500 mL or 510 mL/kg	The tidal volume may not vary even with severe disease.,
Inspiratory reserve volume	IRV	The maximum volume of air that can be inhaled after a normal inhalation	3000 mL	
Expiratory reserve volume	ERV	The maximum volume of air that can be exhaled forcibly after a normal exhalation	1100 mL	Expiratory reserve volume is decreased with restrictive conditions, such as obesity, ascites, pregnancy.
Residual volume	RV	The volume of air remaining in the lungs after a maximum exhalation	1200 mL	Residual volume may be increased with obstructive disease.
Lung Capacities				
Vital capacity	VC	The maximum volume of air exhaled from the point of maximum inspiration $VC = TV + IRV + ERV$	4600 mL	A decrease in vital capacity may be found in neuromuscular disease, generalized fatigue, atelectasis, pulmonary edema, COPD, and obesity.
Inspiratory capacity	IC	The maximum volume of air inhaled after normal expiration $IC = TV + IRV$	3500 mL	A decrease in inspiratory capacity may indicate restrictive disease. May also be decreased in obesity.
Functional residual capacity	FRC	The volume of air remaining in the lungs after a normal expiration $FRV = ERV + RV$	2300 mL	Functional residual capacity may be increased with COPD and decreased in ARDS and obesity.
Total lung capacity	TLC	The volume of air in the lungs after a maximum inspiration $TLC = TV + IRV + ERV + RV$	5800 mL	Total lung capacity may be decreased with restrictive disease (atelectasis, pneumonia) and increased in COPD.

**Breath sounds:**

Crackles: Course crackles : early  
 Fine crackles: late sound

Rhonchi: also known as son ores  
 Pleural Friction Rub

**Diagnostic evaluations**

- Ventilation-Perfusion scan
- Pulmonary Function Tests
- Pulse Oximetry
- Peripheral Vascular studies
- ABG's
- Cultures
- Sputum Studies

### Imaging Studies

- Chest X-Ray
- Computed Tomography
- Magnetic Resonance Imaging
- Pulmonary Angiography
  - Thromboembolic disease (emboli)
- Lung Scan (radioisotope procedure)
  - V/Q Scan, gallium scan, PET

### Endoscopic Procedures : review pages 510

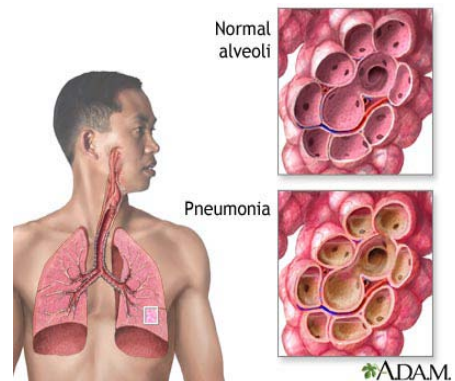
- Bronchoscopy
  - Ensure that gag reflex has returned before giving them anything PO
  - Head of bed elevated
- Thoracoscopy
  - Diagnostic test of looking in the pleural cavity
- Thoracentesis
  - Removing air or fluid from the lungs
  - Assess for pallor, cyanosis, respiratory distress (post procedure)

### Nursing interventions for diagnostic procedures

1. Signed consent form
2. No food or drink for 6 hours prior
3. Explain the procedure
4. Remove dentures and other oral prostheses.
5. After the procedure, nothing by mouth until the gag reflex returns

### Pneumonia

- Inflammatory process
- Fluid build up
- Most common cause of death in the US from infectious disease
- Seventh leading cause of death in the US for all ages  
70,000 deaths per year
- Patterns of pneumonia
  - Lobar
  - Broncho: more common, may start in many areas at the same time.
- Caused by various microorganisms
  - Bacterial: most common strepto coccus
  - Viral: mitoplasm
  - Fungal: aspergillus
- Classifications
  - Community acquired (CAP)
  - Nosocomial / Hospital (HAP)
    - Ecoli
  - Immunocompromised Host



- TB
    - PCP pneumo cystis carcinoma
    - More apt for pneumonia
  - Aspiration
- Risk Factors
  - Mechanical ventilation (potential for aspiration)
  - Elderly
  - Chronic disease
  - Cigarette smoking
  - Immobility
  - Poor nutritional status
- Clinical manifestations
  - Crackles
  - Chills
  - Rising Fever
  - Pleuritic Chest pain
  - Tachypnea
  - SOB
  - Tires easily
  - Orthopnea
  - Purulent, blood tinged, rust colored sputum
  - Dullness on percussion
  - Poor appetite
  - Diaphoretic
- Nursing Interventions
  - Strict handwashing
  - Hydration
  - Vital signs
  - Incentive spirometry
  - Turn, cough, deep breath
  - Chest PT
  - Promote rest
  - Promote proper nutrition
  - Oxygen therapy
- Medications
  - Antitussives : expectorants
  - Bronchodilators
    - Rinse mouth after steroids to prevent thrush
  - Antihistamines
    - Benadryl : relaxes smooth muscles
  - Nasal decongestants
  - Antibiotics: penicillin (Levaquin)
  - Tylenol for fever
- Nursing diagnosis
  - Ineffective airway clearance
  - Activity Intolerance
  - Risk for fluid volume deficit
  - Knowledge deficit

## Pleural Conditions

- Pleurisy
  - Inflammation of both parietal and visceral layers
    - Occurs with pneumonias
  - Pleural effusions
    - Collection of fluid in the pleural space
  - Empyema

- Pusy fluid

Pulmonary Embolism: Obstruction of the pulmonary artery or one of its branches (P582 brunner)

*Defined by the degree of hemodynamic instability rather than the percentage of pulmonary vascular occlusion*

- Blood clot/thrombus
- Air
- Fat
  - Shortness of breath and petechiae
- Risk factors
  - Venous stress
  - Trauma
  - Surgery
  - Pregnancy
  - Age
  - Obesity
  - Oral contraceptive use
  - Constrictive clothing
  - Hypercoagulability (tumor)
- Clinical Manifestations
  - Sudden onset of dyspnea (#1)
  - Pleuritic chest pain (#2)
  - Apprehension
  - Feeling of impending doom
  - Death commonly occurs within 1 hour after onset
  - Diaphoresis
  - Petechiae over chest and axillae
  - Low grade fever
  - Cough
  - Crackles
  - Pleural friction rub
  - Tachycardia
  - Hemoptysis
  - tachypnea
- Assessment
  - Chest x ray, ABG, V/Q scan.
  - Chest x ray is usually normal, but may show infiltrates, atelectasis, elevation of the diaphragm on the effected side, or pleural effusion.
  - Pulmonary angiography is the best diagnostic tool
  - ECG
- Treatment
  - Anticoagulation therapy
    - Heparin, warfarin sodium
  - Thrombolytic therapy
    - Urokinase, streptokinase, alteplase
    - Used in patients who are severely compromised
    - Major side effect: bleeding
    - Contraindicated if patient had a CVA in the past 2 months
      - active bleeding, or surgery in the last 10 days
    - Prior to therapy, INR, partial thromboplastin time (PTT), hematocrit, and platelet counts are obtained.
    - After thrombolytic therapy, anticoagulant therapy begins.
- Nursing management
  - Passive and active range of motion
  - Identify the patients that are at high risk and maintain a high degree of suspicion in all patients
  - Ambulation

- Antiembolism and pneumatic compression stockings
- Avoid constricting clothing
- Prevent pressure under the popliteal space
- Anti-coagulation therapy
  - Heparin : reversing agent: Protamine sulphate
- Education

#### Acute laryngotracheobronchitis (LBT)

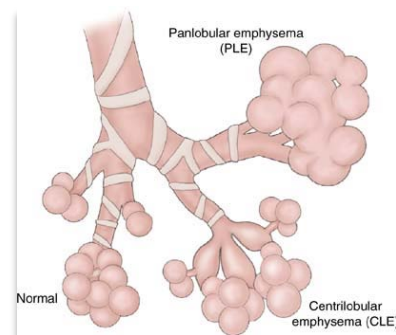
- Most common croup syndrome
- Affects children less than five years of age
- Inflammation of the mucosa lining of the larynx and trachea
- Clinical manifestations
  - Barking or seal-like cough
  - Acute stridor
  - Hypoxia
  - Gradual onset of low-grade fever
  - Hoarseness
  - Restlessness
  - Rapid respirations
- Treatment
  - Maintain a patent airway
  - High humidity with cool mist
  - Corticosteroids
  - Racemic epinephrine
    - Ribavirin : antiviral given through an aerosol
      - Usually given in a croup tent.
      - Improvement usually in 15 minutes
  - Encourage PO fluids unless respiratory rate greater than
  - Prednisone

# Chronic Respiratory Problems COPD

## Emphysema

- **Three primary symptoms: Chronic Cough, Sputum Production, and Dyspnea on exertion**
- Disease of the alveoli. The elastic balloon-like sacs that form the alveoli become permanently stretched, making them larger than normal
- END STAGE process from slow disease progression
- Increased CO<sub>2</sub> Levels (elimination is impaired)
- Asthma and other diseases must be ruled out (Asthma: primary differential diagnosis)
- Inflammation of the lung
  1. Pulmonary capillary bed size reduced, resistance to pulmonary blood flow is increased and therefore the right side of the heart has to work harder.
  2. Cor pulmonale is a complication (right side heart failure)
    - Congestion, dependant edema, distended neck veins, pain in the region of the liver.
- Starts with bronchitis
- Four stages
  1. New onset FEV<sub>1</sub>/FVC < 70%, and an FEV<sub>1</sub> =>80% predicted
    - With or without symptoms
  2. FEV<sub>1</sub>/FVC <70% and FEV<sub>1</sub> 50-80%
    - Shortness of breath upon exertion
  3. FEV<sub>1</sub>/FVC less than 70% and FEV<sub>1</sub> <30-50% predicted
    - severe symptoms SOB, reduced exercise ability
  4. FEV<sub>1</sub>/FVC <70% and FEV<sub>1</sub> <30-50% AND signs and symptoms of chronic respiratory failure.
- #1 reason is smoking
- Genetic factor
  1. Alpha1 antitrypsin enzyme plasma protein – without this enzyme there can be destruction of the alveoli
- Environmental factors
- Types
  1. Centrilobular : bronchials and most of the alveoli
    - Mainly takes place in the center of the secondary lobule, preserving the peripheral portion of the acinus (termination point in the bronchioli)
    - Derangement of the Vent/perfusion ratio
      - Chronic hypoxia, hypercapnia, polycythemia and episodes of right sided heart failure.
      - Leads to central cyanosis and respiratory failure
    - Peripheral edema
  2. Panlobular: effects the entire lobe (worse)

FEV<sub>1</sub> = Forced Expiratory Volume (1 second)  
FVC = Forced Vital Capacity  
FEV<sub>1</sub>/FVC normal 75-80% in adults



- Destruction of the bronchiole, alveolar duct, and alveolus, but with little inflammatory disease
    - Hyperinflated chest, dyspnea on exertion, and weight loss occur
    - Expiration becomes active and requires muscular effort
  - 3. Some people can have both
- Clinical manifestations
 

<ol style="list-style-type: none"> <li>1. Decreased FEV</li> <li>2. Barrel chest</li> <li>3. DOE</li> <li>4. Orthopnea</li> <li>5. Diaphragmatic breathing</li> <li>6. Increased cough</li> <li>7. Purulent sputum</li> <li>8. Anorexia</li> <li>14. clubbing of fingers</li> <li>15. weak pulse</li> </ol>	<ol style="list-style-type: none"> <li>9. Weakness</li> <li>10. Inactivity</li> <li>11. Diminished breath sounds and prolonged expiration and crackles</li> <li>12. JVD</li> <li>13. hypoxemia</li> <li>16. skin color normal to flushed</li> </ol>
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#### Diagnostic Findings

- Polycythemia
- Pulmonary function tests
- ABG's
- Chest X-ray

#### Nursing Management

- Position sitting up, leaning forward
  - Improve breathing patterns: training in diaphragmatic breathing, pursed lip breathing
- Chest PT
- Encourage frequent rest periods
- Medications as ordered
  - Bronchodilators (key), corticosteroids, other meds
- **Exacerbated COPD: an event of an acute change in patient's baseline dyspnea, cough, sputum production.**
  - Primary cause treated
  - Corticosteroids, antibiotics, oxygen therapy, intensive respiratory intervention
  - Hospitalization if patient does not respond.
  - Outcome related to onset of respiratory acidosis, presence of comorbidities, and need for noninvasive or invasive positive pressure ventilatory support.
- Administer oxygen at low flow
  - Goal: PaO<sub>2</sub> at least 60mm Hg and SaO<sub>2</sub> at least 90%
- Encourage fluids: 3,000ml per day if not contraindicated
- Adequate nutrition/Increase calories and protein to meet increased energy requirements
- Promote deep breathing exercises
- Nutritional therapy
- Address sexual concerns
- Monitor and manage complications
  - Pulse ox, O<sub>2</sub>, Vitals, signs and symptoms
- Teaching self care
  - Familiarity of medications, side effects
  - Family members educated to notice signs and symptoms

- Setting realistic short and long term goals.
- Avoiding heat and cold
- Lifestyle of moderate activity, ideally in an environment of minimal shifts in temp and humidity.

#### Surgical intervention

- Bullectomy : removal of an airspace that is not involved in respiration.
  - These airspaces increase the chance of a pneumothorax
- Lung reduction
- Lung transplant

#### Chronic Bronchitis

- Inflammation of bronchi with hypertrophy and hypersecretion(increased number) of the goblet cells and eradication of cilia
- Abnormal inflammatory response
- Alveoli adjacent to bronchioles become damaged resulting in altered function of the alveoli macrophages.
  - Damaged macrophages can no longer effectively destroy bacteria
    - Patient becomes more susceptible
- Diagnostic findings
  - Presence of cough and sputum production for 3 months in 2 consecutive years
  - Chest X ray
  - FEV forced expiratory volume
  - Low vital capacity
  - H & H
- Treatment
  - 3000 ml fluid per day
  - Chest PT : before meals & prior to HS
  - Oxygen
  - Ambulation
  - Suction if needed
  - Cough and deep breath
  - Incentive spirometry
  - Nutrition
    - Soft foods better
    - Stay away from dairy products

#### Medications for COPD

- **Medications (bronchodilators), oxygen**
  - Beta<sub>2</sub> Agonists – dilates smooth muscles of lungs, resulting in bronchodilation
    - Beta<sub>1</sub> agonists act primarily on heart.
    - Beta agonists that act on both are called non-selective bronchodilators
  - Short acting Beta<sub>2</sub> – onset is several minutes and duration is 2-6 hours
  - Beta<sub>2</sub> drugs: (beta blockers will inhibit effects)
    - Albuterol (Proventil, Ventolin, VoSpire) : short acting
    - Arformoterol (Brován): prescribed with short and intermediate drugs fail to control the symptoms.
    - Formoterol
    - Levalbuterol (Xopenex) isomer of albuterol
    - Pirbuterol (Maxair): short acting onset 5min.

- Salmeterol (Serevent): asthma prophylaxis
- **Anticholinergics** : block the parasympathetic nervous system
  - Ipratropium (Atrovent, Combivent) MDI drug of choice for treating bronchospasm caused by COPD: onset in minutes, peak 1-2 hr
  - Tiotropium (Spiriva) : long duration : admin: DPI
- **Inhaled corticosteroids** (most effective for long term control of asthma)
  - Work by increasing the synthesis and release of inflammatory mediators such as histamine, leukotriene, cytokines and prostaglandins
  - Beconase AQ, Pulmicort, Aerobid, Flovent, Asmanex, Azmacort
  - Prednisone :taper up dose
  - Side effects:
    - Immune system depression
    - Fluid retention
    - High blood sugars
    - Insomnia
    - Delayed wound healing
    - Euphoria or depression
    - Osteoporosis
    - Thinning of the skin
- **Mast Cell Stabilizers** : Prevent the release of histamines
  - Cromolyn (Intal) : effect may not be noted for 2-4 weeks
  - Tilade : similar to Intal, very bitter taste
- **Leukotriene Modifiers** : reduce the inflammatory component of asthma
  - Blocks leukotriene receptors in the airway.
  - Accolate: Asthma prophylaxis anti-inflammatory
  - Singulair
  - Zflo : Inhibits lipoxygenase, 1<sup>st</sup> enzyme in leukotriene pathway
- **Methylxanthines** : now rarely prescribed
  - potent, can be used as a rescue drug
  - A patient who is toxic will be anorexic
    - Confusion, headache, restlessness, cardiac dysrhythmias
    - Food will decrease the GI irritation
    - No caffeine, interferes with the absorption and increase the serum levels
  - Modest bronchodilators, related to caffeine
  - Theophylline: relaxes bronchial smooth muscles and suppresses airway responsiveness to stimuli that cause bronchospasm.
  - Aminophylline, Dyphylline
  - Smokers will require higher doses
- Epinephrine
- Antibiotics: higher risk for pneumonia

#### Complications

- Respiratory infections/empirical therapy
  - Standing order if there are oncoming symptoms
- Hypoxemia
  - Restlessness

- Tachycardia
- Cyanosis (late stage)
- Respiratory acidosis
- Cardiac dysrhythmias
  - Low oxygen
  - Meds
- Cor Pulmonale
  - Right ventricle in heart enlarges (Right Side heart failure)
  - Most frequent cause: COPD
  - Pulmonary arterial hypertension
    - Peripheral edema
    - Ascites
    - JVD
    - Crackles
    - Heart murmur

#### Clinical manifestations


- Edema
- Distended Neck Veins
- Enlarged Liver
- Ascites
- Pleural Effusion
- Increased levels of CO<sub>2</sub>

#### Occupational Lung Diseases

- Silicosis : restrictive lung disease
  - Chronic fibrotic pulmonary disease
    - Nodular lesions
  - Caused by silica dust
    - Glass manufacturing, pottery, mining
  - Presents with SOB, Fever, cough
  - Rapidly progressive
- Asbestosis : restrictive lung disease
  - Diffuse pulmonary fibrosis
  - Inhaled asbestos
  - Found in shingles, cement, some paints
  - Tiny fibers are inhaled and they get into the lungs
  - Usually go into lung failure
- Coal workers pneumoconiosis : black lung disease
  - Variety of respiratory diseases
  - Coal dust gets into the alveoli
    - Macrophages attack it, but repeated exposure
  - Chronic cough, very productive black sputum
  - Painful

#### Chest Tumors

- Most common cause is inhaled carcinogens (cig 90%)
  - Arises from a single transformed epithelial cell in the tracheobronchial airways, a carcinogen binds to and damages the cell's DNA.
    - Small cell carcinoma 15-20%
    - Non small cell carcinoma 80% of tumors

- Clinical manifestations
  - Can develop insidiously and asymptomatic until late in its course
  - Signs and symptoms depend on location
  - Most frequent symptom is a cough, or chronic cough.
    - May start as a dry, persistent cough
    - When obstruction occurs, it may produce sputum due to infection
    -  Nursing Alert: Cough that changes in characteristic should arouse the suspicion of lung cancer
  - Recurring fever – response to infection
  - Mediastinum tumors
    - Symptoms result from pressure of the mass against organs
    - Distention of neck veins or chest wall
- Assessment and diagnostic findings
  - Chest X-ray : pulmonary density, pulmonary nodule (coin lesion), atelectasis
  - CT scan – identify small nodules
  - Fiberoptic bronchoscopy (commonly used)
  - Fine needle aspiration
  - PET and MRI scans
  - Pre-operative pulmonary function tests: V/Q scans, exercise testing
- Medical management: surgery, radiation, and chemotherapy
  - Chemotherapy used to alter tumor growth patterns
    - May provide pain relief, but it **does not usually cure the disease or prolong life to any great degree.**
      - Valuable in reducing pressure symptoms of lung cancer, and in treating brain, spinal cord, and pericardial metastasis.
- Treatment related complications:
  - Respiratory failure from surgical lung resection
  - Radiation therapy may diminish cardiopulmonary function
  - Chemo, especially in combination with radiation can cause pneumonitis.
- Manage symptoms
  - Airway
    - Deep breathing, chest PT, directed cough, suctioning and bronchoscopy
    - Patient education
- Reducing Fatigue (major symptom, and reduces quality of life)
  - Psychological support
    - Resources for the patient and family
    - Informed decision about treatment options
    - End of life treatment options

**Nursing diagnosis related to respiratory diseases:**

1. Impaired gas exchange and airway clearance due to chronic inhalation of toxins
2. Impaired gas exchange related to ventilation-perfusion inequality
3. Ineffective airway clearance related to bronchoconstriction, increased mucus production, ineffective cough, bronchopulmonary infection, and other complications.
4. Ineffective breathing pattern related to shortness of breath, mucus, bronchoconstriction, and airway irritants
5. Self care deficits related to fatigue secondary to increased work of breathing and insufficient ventilation and oxygenation.

## 6. Activity intolerance due to fatigue, hypoxemia, and ineffective breathing patterns

*Cor Pulonale: enlargement of the right ventricle of the heart as a response to increased resistance or high blood pressure in the lungs.*

*Polycythemia : excessive red blood cell production: body compensation for decreased oxygen*

## Tuberculosis

### Mycobacterial infection

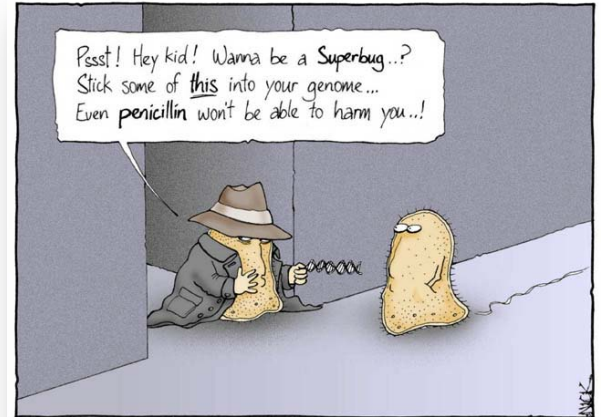
*Mycobacteria: aerobic organism that have a waxy coating of mycolic acid over their cell surface. This coating is 40% of the cell weight and protects the bacteria.*

1. M. Tuberculosis bacteria: acquired through the respiratory route (airborne droplets, but airborne precautions). Bacteria can remain alive outside the body for several hours.
2. Most primary infections occur in the upper lobes where the body's immune response isolates them by creating a wall of macrophages around them. The macrophages ingest them, but the mycobacteria can grow inside the macrophages.
3. Acute respiratory infection develops and lasts a few weeks.
4. Eventually granulomatous lesions called tubercles are formed in the lungs.
5. Tissue within the tubercles necrosis, causing cavities within the lung. (cheesy like)
6. Mantoux test : 4mm –no reaction, 10mm +Reaction
7. Sputum test (Acid Fast)
8. Acid fast organism, travels to the alveoli and multiplies

### Pharmacology

1. Drug therapy is 6 to 12 months long, patients who develop multidrug resistant infections may require therapy for as long as 2 years.
2. Two to seven antimycobacterial drugs are given concurrently. Combination drugs are needed because mycobacteria grow slowly and develop resistance during treatment
3. Mycobacterial drugs are used extensively for preventing the disease in addition to treating the infection
4. Signs of hepatotoxicity must be monitored.
5. Antituberculosis agents (antimycobacterial) First Line
  - a. Isoniazid (INH) : mycolic acid inhibitor
    - i. Metabolized by the liver
    - ii. Inhibits synthesis of mycolic acid
    - iii. Monitor for jaundice, fatigue, elevated hepatic enzymes
  - b. Rifampin (Rifadin, Rimactane) : Inhibits RNA synthesis
    - i. Broad spectrum antibiotic
    - ii. 50% of people get flu-like symptoms (hypersensitivity)
    - iii. Increases the metabolism of other drugs (birth control)
    - iv. Always used in combination
    - v. Rifapentine (Priftin) and Rifabutin (Mycobutin) use the same mechanism
  - c. Pyrazinamide (PZA) : Inhibits mycolic acid
    - i. Usually taken in combination with rifampin and INH for 8 weeks
    - ii. Causes some dose related hepatotoxicity is about 15% of patients
    - iii. Monitor Uric acid levels
  - d. Ethambutol (Myambutol): mechanism not well understood
    - i. Sometimes works against multidrug resistant infections

- ii. Adverse effect: optic neuritis – affects visual acuity and the ability to distinguish red from green. Effect is dose related and usually reverses when therapy is stopped.
6. Second line antituberculosis drugs, when resistance develops
- a. Aminoglycosides: Amikin, Kantrex, streptomycin
    - i. Streptomycin was the first clinically effective drug used to treat TB
    - ii. Drug is given IM and can produce sterile abscesses at the injection site.
  - b. Aminosalicic acid
  - c. Capreomycin
    - i. Only for multidrug resistant TB
    - ii. Must be injected deep into large muscle mass.
  - d. Cycloserine
    - i. Blocks mycobacterial cell wall synthesis
    - ii. Also given to treat UTIs that are unresponsive to safer antibiotics.
  - e. Ethionamide
    - i. Given several times per day
    - ii. May act by inhibiting mycobacterial protein synthesis
  - f. Fluoroquinolones: Cipro and Floxin
    - i. Widely used to treat bacterial infections: concern for resistant strains
    - ii. Safest second line drug.



It was on a short-cut through the hospital kitchens that Albert was first approached by a member of the Antibiotic Resistance.

## Thoracic Surgery

### Preoperative Care:

- Objective – ascertain the patient’s functional reserve to determine if the patient is likely to survive and recover from surgery.
- Ensure the patient is in the optimal condition possible for surgery

### Preoperative management

### Assessment and diagnostic findings

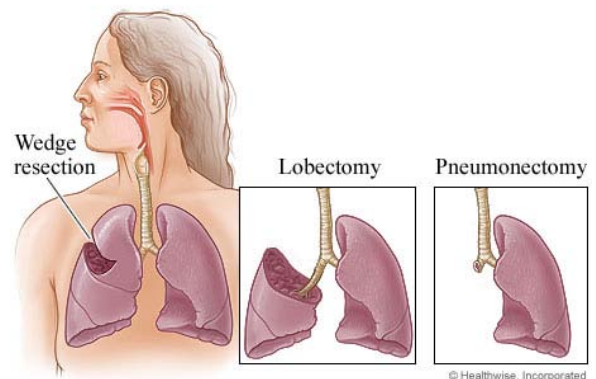
1. Signs and symptoms
2. Smoking history
3. Cardiopulmonary tolerance while resting, eating, bathing, and walking
4. Breathing pattern, how much exertion until dyspnea
5. Orthopnea
6. General physiological status
7. Other medical conditions
8. Manage pain control before surgery

### Patient Education

1. Informed Patient
  - a. Coughing techniques (tell them before surgery)
  - b. Blood product administration possible
  - c. What to expect (chest tubes, water seal, pain meds, what they must do)
2. Relieving anxiety
  - a. Address patient’s fears
  - b. Correct misconceptions
  - c. Teach how to use the PCA before surgery

### Postoperative

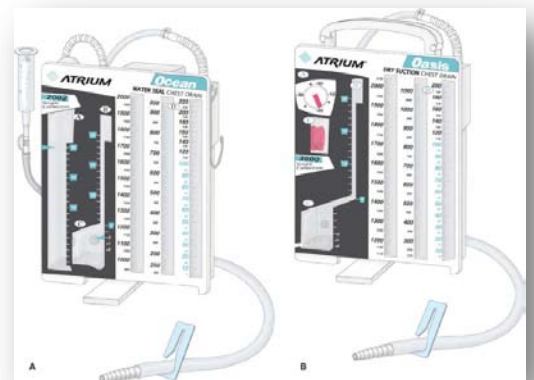
1. Vitals, oxygen sat, LOC, checked frequently
2. After the patient is conscious and vitals are stable, the head can be elevated 30-40 degrees
3. After a pneumonectomy, the patient is turned from the back to the operative side and should not be completely turned to the unoperated side. This allows fluid left in the space to consolidate
4. A patient with a lobectomy can be turned to either side “good lung up”
5. A patient with a segmental resection usually is not turned onto the operative side.
6. Assess for dyspnea, cyanosis, acute chest pain.
7. Increased temp, or white blood cell count may indicate infection
8. Increased pulse and pallor may indicate internal hemorrhage.
9. Dressings are assessed for fresh bleeding



### Chest Drainage: after thoracic surgery, chest drainage tubes and a closed drainage system are used.

1. Pathological substances collect in the pleural space : fibrin, clotted blood, liquids, and gasses
  - a. Normally only about 20ml of fluid exists in this “space”
2. Chest Tubes:

- a. Small bore catheters (7 Fr to 12 Fr)
    - i. Have a one way valve to prevent air from moving back
    - ii. Inserted through a small skin incision
  - b. Large bore catheters (up to 40 Fr)
    - i. Usually connected to a chest drainage system to collect any pleural fluid and monitor for air leaks.
  - c. Wet suction system:
    - i. Turn on suction and increase until a slow, steady bubbling appears in the suction control chamber
  - d. Dry suction control system:
    - i. Set suction at 20cm H<sub>2</sub>O
  - e. Mark the drainage from the collection chamber with tape, mark date/time at drainage levels.
  - f. If tubing disconnects: cut off the contaminated tip of the chest tube and tubing
    - i. Insert a sterile connector in the cut ends and re-attach to the drainage system
  - g. Tiding: Increase in water level when patient inhales and a return to the baseline when the patient exhales
  - h. Dry suction with a one way mechanical valve.
    - i. Collection chamber with a one way mechanical valve
    - ii. Can be setup quickly in an emergency situation
    - iii. Position is not a factor and therefore it is good during patient transportation
    - iv. Cannot detect an increase in intrathoracic pressure .
  - i. When assisting removal, have the patient perform a gentle Valsalva maneuver, or to breath quietly. The chest tube is then removed quickly and small bandage is applied and made airtight with petrolatum gauze covered by a 4x4 pad and sealed with nonporous tape.
  - j. Links: Atrium Drainage systems
    - i. Wet  
<http://www.youtube.com/watch?v=WVH-elcllee8&feature=related>
    - ii. Dry  
<http://www.youtube.com/watch?v=GWxKZbKAxe8&feature=related>
3. Monitor heart rate and rhythm because episodes of major dysrhythmias are common after thoracic and cardiac surgery.
  4. Immediate post op
    - a. Arterial line may be present for monitoring blood gasses and blood pressure
  5. Postop FEV<sub>1</sub> volume vs the Preop FEV<sub>1</sub> of more than 2L or more than 70% of predicted value indicates a good lung reserve. Postop FEV<sub>1</sub> of less than 40% of predicted value, places them at risk of respiratory failure, and death.
  6. Vital signs are monitored every 15 minutes for the first 2 hours.
  7. Breathing techniques taught pre-op are used to expand the alveoli
  8. Nasotracheal suctioning may be needed to stimulate a deep cough: <120 mm H<sub>2</sub>O
  9. Patient should be positions “good lung up”, or on back.
  10. Nursing Diagnosis
    - a. Ineffective airway clearance related to lung impairment, anesthesia, and pain.



- b. Acute pain related to incision, drainage tubes, and the surgical procedure
- c. Anxiety related to outcomes of surgery, pain, technology

## Chest Trauma

### Blunt Trauma

1. More common than penetrating trauma
2. Often difficult to identify the extent of the damage because symptoms can be generalized
3. Most common cause is motor vehicle accidents
  - a. Acceleration
    - i. Patient thrown into something
    - ii. Something thrown at patient
  - b. Deceleration
    - i. MVA
4. Often life threatening : pathological states:
  - a. Hypoxia from disruption of the airway, injury to the lung, rib cage, or respiratory muscles
  - b. Hypovolemia from massive fluid loss from great vessels, cardiac rupture, or hemothorax
  - c. Cardiac failure from cardiac tamponade, cardiac contusion, or increased intrathoracic pressure.
5. Time is critical
  - a. Time elapsed since injury
  - b. Mechanism of injury
  - c. LOC
  - d. Specific injuries
  - e. Blood loss
  - f. Recent drug or alcohol use
  - g. Prehospital treatment
6. Assessment
  - a. Assessment for airway obstruction
  - b. Tension or open pneumothorax
  - c. Flail chest
  - d. Cardiac tamponade.
  - e. Secondary injuries
    - i. Simple pneumothorax, hemothorax, pulmonary contusion, traumatic aortic rupture, tracheobronchial disruption, esophageal perforation, traumatic diaphragmatic injury and penetrating wounds to the mediastinum.
  - f. Assess the chest for symmetrical movement, breath sounds, entrance and exit wounds, impaled objects, tracheal deviation, JVD, subcutaneous emphysema, paradoxical chest movement
  - g. Chest wall for burns, petechiae, and lacerations
  - h. Thorax is palpated for tenderness and crepitus.
7. Initial workup
  - a. Chest x ray, ct scan, complete blood count, clotting studies, type and crossmatch, electrolytes, O2 sat, ABG, and ECG.
  - b. Patient is completely undressed to avoid missing any injuries that may complicate care.
  - c. Even if the initial x ray is clear, changes may appear in 1 to 2 days.
8. Treatment
  - a. Maintaining the airway and providing adequate oxygenation

- b. In mild pulmonary contusions, adequate hydration via IV fluids, although oral intake is important to mobilize secretions.
- c. Pain is managed by intercostal nerve blocks or opioids
- d. Antibiotics are usually prescribed due to the lung injury and its vulnerability to infection.
- e. Indwelling catheter to monitor urine output
- f. Shock is treated with colloid solutions, crystalloids, or blood as indicated by condition

#### Pneumothorax

1. Assess trachea for alignment (simple vs tension)
  - a. In a tension pneumothorax, the effected side is hyperresonant
  - b. Air hunger, agitation, increasing hypoxemia, central cyanosis, hypotension, tachycardia, and profuse diaphoresis
2. Medical management
  - a. A small chest tube (28 Fr) is inserted in the second intercostal space, midclavicular (this is the thinnest part of the chest wall, and has the lowest danger of contacting the thoracic nerve, and leaves a less noticeable scar.
  - b. 20mm H2O suction is applied
  - c. If an excessive amount of blood is collected, than an autotransfusion may be done

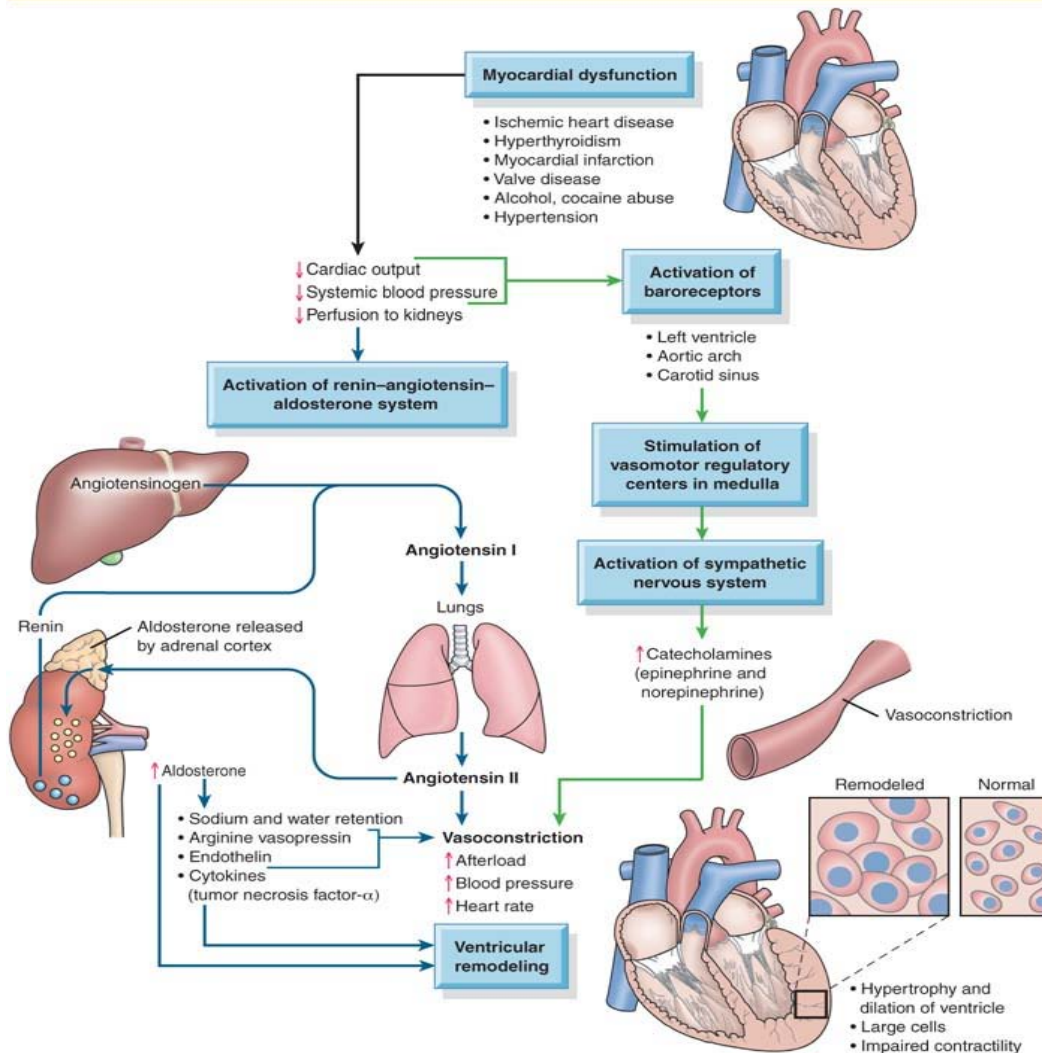
Heart Disease: Abnormal cardiac function  
Vasoconstriction

Pathology of Heart failure

1. Systolic Failure
  - a. Weakened heart muscle
  - b. Most common cause of heart failure
  - c. Results in the inability of the heart to pump blood
  - d. Defect in the ability of the ventricle to contract and push out blood
  - e. Hallmark : decrease in the left ventricle ejection fraction (EF)
2. Diastolic Failure
  - a. Impaired ability of the ventricles to relax and fill during diastole
  - b. Usually the result of left ventricular hypertrophy (big)
    - i. Stretched from chronic hypertension
  - c. Stiff, non compliant heart muscle
3. Mixed systolic and diastolic failure
  - a. Heart becomes dilated
  - b. Cardio myopathy
    - i. Condition where there is poor systolic function, compromised by a dilated ventricle.

ii. Poor function from weakened heart muscle

**Physiology** ■■■ **Pathophysiology**



4. Compensatory mechanisms: insidious problem (slow, progressive changes)

- a. Sympathetic nervous system
  - i. Least effective system
  - ii. Increases oxygen demand
- b. Neurohormonal response
  - i. Kidneys cannot tolerate decreased blood flow
  - ii. Decrease renin
  - iii. Renin gets converted to angiotensin I
  - iv. Angiotensin I gets converted to angiotensin II
  - v. Angiotensin II causes aldosterone to get released
  - vi. Aldosterone causes sodium and water retention
  - vii. Vasoconstriction
  - viii. Causes blood pressure increase.
- c. Dilation
  - i. Enlargement in the chambers of the heart

- ii. Over time the heart chamber pressure becomes elevated due to fluid overload and Chronic HTN
    - iii. Muscle fibers stretch
    - iv. Initially this mechanism functions well, but when the heart muscles become overstretched, they cannot maintain an adequate cardiac output.
  - d. Anti Diuretic Hormone released by brain
    - i. Causes fluid retention
    - ii. Temporarily maintains cardiac output.
  - e. Hypertrophy
    - i. Increase of the size of the muscle mass and cardiac wall thickness
      - 1. Overworked and overstrained
    - ii. Poor contractibility
    - iii. Requires more oxygen
    - iv. Heart becomes ischemic
    - v. Ventricular dysrhythmias develop
  - f. Counterregulatory mechanisms
    - i. Body's ability to maintain balance
    - ii. B type Natriuretic peptides (BNP)
      - 1. Promote venous and arterial dilation
      - 2. Hormones produced by the heart muscle
      - 3. Improves preload and afterload
      - 4. Assists and enhances diuresis, by increasing GFR glomerular filtration rate
  - g. Cardiac compensation: when these compensation mechanisms are working
    - i. Cardiac decompensation: when they are not working.
- 5. Types of Heart Failure
  - a. Left sided heart failure
    - i. Results in left ventricular dysfunction
    - ii. Blood backs up into the atrium and then into the pulmonary veins
    - iii. Increased pulmonary pressure : causes fluid accumulation from the veins into pulmonary capillary beds then into the interstitial and into the alveoli
    - iv. L for Lung
    - v. Signs: Left ventricular heaves, Pulsus alternans, increased HR, decreased PaO<sub>2</sub>, slight increase in PaCO<sub>2</sub>, crackles, pleural effusion, changes in mental status, restless, confused
    - vi. Symptoms: weakness, fatigue, anxiety, depression, dyspnea, nocturia, dry hacking cough
  - b. Right side failure
    - i. Causes a backup of blood into the right atrium, backs up into the rest of the body.
    - ii. Venous distention (JVD)
    - iii. Hepatomegalia (from fluid congestion)
    - iv. Splenomegalia
    - v. Peripheral edema
    - vi. Right sided heart failure is usually caused by left sided failure
    - vii. Signs: Right ventricular heaves, murmurs, JVD, edema, weight gain, ascites, anasarca ( massive generalized body edema), hepatomegalia

- viii. Symptoms: fatigue, anxiety, depression, right upper quadrant pain, anorexia, gi bloating, nausea
  - c. Acute Decompensated heart failure
    - i. Pulmonary edema (lung alveoli filled with serosanguiness fluid)
- 6. Diagnostic Studies
  - a. Look for BNP (B type natriuretic peptides)
- 7. Nursing and collaborative management (Acute Heart Failure)
  - a. Improve quality of life
  - b. Decrease intravascular volume
    - i. Lasix
    - ii. Improves afterload
    - iii. Hemodialysis
  - c. Decreasing venous return
    - i. Decrease preload
    - ii. Get them up, dangle legs off the bed
    - iii. IV nitroglycerine (vasodilation)
  - d. Decreasing afterload
    - i. Decrease afterload (resistance against arteries)
    - ii. Decreases strain of left ventricle
    - iii. IV Nipride (potent vasodilator) – drug of choice to decrease afterload
      - 1. Give slow (at proscribed rate)
  - e. Increasing gas exchange and oxygenation
  - f. Improving cardiac function
    - i. Digoxin (drug of choice)
      - 1. Increases contractibility
        - a. Thereby increases cardiac output
      - 2. Increases myocardial oxygen consumption
  - g. Protect target organs (kidney)
  - h. Reducing anxiety
    - i. Morphine sulphate
      - 1. Calms patient
      - 2. Relieve dyspnea
      - 3. Reduces preload and afterload
      - 4. Dilates pulmonary arteries
  - i. Collaborative care: chronic heart failure
    - i. Drug therapy
    - ii. Nutritional therapy
    - iii. Mobilize edemous fluid
    - iv. Reduce preload
  - j. Vasodilation
    - i. Improve ejection fraction
    - ii. Reduce pre-load
    - iii. Control cardiac function
    - iv. Reduce size of heart
    - v. Drugs
      - 1. ACE inhibitors
        - a. Lotensin
        - b. Vasotec

- c. Nitrates
  - i. Act on smooth muscles
- d. Synthetic BNP (b-type natriuretic peptides)
- e. Beta blockers – block effects of the sympathetic nervous system
- f. Positive inotropes
  - i. Digitalis
    - 1. Increases force of cardiac contraction
    - 2. Increases stroke volume
    - 3. Improves cardiac contractibility
    - 4. Normal Dig levels = 0.5 – 1.1
    - 5. Potassium Levels = 3.5 – 5.0
      - a. Watch potassium level, if the potassium gets too low, increases changes of going into dig toxicity.
    - 6. Dig toxicity
      - a. Anorexia, nausea, vomiting
      - b. Blurred vision, colored vision, visual halos around dark objects
      - c. Fatigue, drowsiness
      - d. Dysrhythmias, bradycardia, tachycardia, apical radial pulse deficit.
      - e. Take an apical pulse, hold if HR is under 60
- g. Nutrition
  - i. Low salt diet
  - ii. Weight, first thing in the morning (same time, place, scale)
    - 1. 3 pounds in three days, call MD

1. Assessing for Heart Failure
  - a. Fatigue
  - b. Decreased activity tolerance
  - c. Dependent edema
  - d. Weight gain
2. Cardiovascular symptoms
  - a. Third heart sound (S3)
  - b. Apical impulse enlarged with left lateral displacement
  - c. Pallor and cyanosis
  - d. JVD
3. Respiratory (left sided failure)
  - a. Dyspnea on exertion
  - b. Pulmonary crackles that do not clear with cough
  - c. Paroxysmal nocturnal dyspnea
  - d. Cough on exertion or when supine
  - e. Increased pulmonary resistance
  - f. Pulmonary edema (common cause is acute left ventricular failure)

- i. Tachypnea, SOB
  - ii. Severe dyspnea
  - iii. Accessory muscles
  - iv. Orthopnea
  - v. Wheezing and coughing
  - vi. Pink frothy sputum (typical for left sided heart failure)
- 4. Cerebrovascular
  - a. Unexplained confusion or altered mental status
  - b. Lightheadedness
- 5. Renal
  - a. Oliguria and decreased frequency during the day
  - b. Nocturia
- 6. Risk factors for heart failure
  - a. Advancing age
  - b. Coronary artery disease
  - c. Hypertension (biggest risk factor)
  - d. Diabetes
  - e. Cigarette smoking
  - f. Obesity
  - g. High cholesterol
- 7. Gastrointestinal
  - a. Anorexia and nausea
  - b. Enlarged liver
  - c. Ascites
  - d. Hepatojugular reflux

## Cardiac Medications

### Nitroglycerin    **Antianginal**    **vasodilator**

ORGANIC NITRATES MAY BE USED TO TERMINATE OR PREVENT ANGINA EPISODES. THE ORGANIC NITRATES RELIEVE ANGINA BY DILATING VEINS AND CORONARY ARTERIES. THE SHORT-ACTING NITRATES ARE DRUGS OF CHOICE FOR TERMINATING ACUTE ANGINA PAIN. THE LONG-ACTING AGENTS ARE USED TO REDUCE THE FREQUENCY OF ANGINA EPISODES; HOWEVER, TOLERANCE CAN LIMIT THEIR USE.

1. Route: sublingual, lingual spray, PO, IV, transmucosal, transdermal, topical, ext. release
  - a. Sublingual – reaches peak plasma levels in minutes.
  - b. Long acting should not be suddenly discontinued – vasospasm may occur
2. Indications and uses
  - a. Chest pain : CP unrelieved after 2-3 doses may indicate MI
  - b. Also used to control hypotension induction during anesthesia
  - c. CHF, acute pulmonary edema, acute MI, severe HTN,
  - d. Off label use: uterine relaxant to aid in placenta extraction
3. Mechanism:
  - a. Forms nitric oxide at smooth muscles
  - b. Triggers cascade – release calcium ions in smooth muscle.
  - c. Relaxes both arterial and venous smooth muscle
  - d. Venous dilation reduces amount of blood returning to heart –reduced preload
    - i. Myocardial oxygen demand reduced.
  - e. Arterial dilation of the coronary arteries increases blood supply to myocardium
    - i. Partially responsible for therapeutic effects
4. Pharmacokinetics
  - a. Rapid absorption
  - b. Hepatic metabolism
  - c. Renal excretion
  - d. Onset
    - i. Sublingual 1-3 minutes
    - ii. Buccal 2-5 minutes
    - iii. Transderm 40-60 minutes
  - e. Duration
    - i. Sublingual 30-60 minutes
    - ii. Buccal 2 hours
    - iii. Transderm 18-24 hours
5. Adverse effects
  - a. Flushing of face
  - b. Throbbing, transient headache
  - c. Orthostatic hypotension and syncope
  - d. Anaphylaxis (rare)
6. Contraindications
  - a. Preexisting hypotension, shock, head injury and increased intracranial pressure
  - b. Pericardial tamponade
  - c. Sustained release not given to glaucoma patients
7. Interaction
  - a. Viagra (can cause severe hypotension)
8. Overdose treated with iv normal saline
9. Nursing responsibilities

- a. Assess BP and HR prior to administration
    - i. Hold in systolic <100
  - b. Assess chest pain (location, duration, quality)
  - c. Evaluate effectiveness
  - d. Monitor for adverse effects
10. Similar (same class)
- a. Amyl nitrate
  - b. Isosorbide

**Atenolol**      **Antianginal**      **Beta-adrenergic antagonists**

BETA-ADRENERGIC ANTAGONISTS ARE OFTEN DRUGS OF CHOICE FOR STABLE ANGINA. BETA-ADRENERGIC BLOCKERS RELIEVE ANGINA PAIN BY DECREASING THE OXYGEN DEMANDS ON THE HEART: LOWERING BLOOD PRESSURE, SLOWING HEART RATE, AND REDUCING CONTRACTILITY. THEY ARE DRUGS OF CHOICE FOR PREVENTING ACUTE ANGINA EPISODES.

1. Most frequently prescribed drug
2. Treats : heart failure, HTN, stable angina, MI
3. Route: PO only
4. Mechanism: selectively blocks beta<sub>1</sub>-adrenergic receptors in the heart.
  - a. Slows the heart rate
  - b. Reduces contractility
  - c. Therefore lowers myocardial oxygen demand.
5. NOT METABOLIZED
6. Onset 1 hour
7. Duration 6-7 hour half life
8. Adverse effects
  - a. Bradycardia and hypotension
  - b. Fatigue, weakness, dizziness
  - c. Nausea and vomiting
9. Contradictions
  - a. Patients with severe bradycardia
  - b. Advanced AV block
  - c. Severe hypotension
  - d. Patients using alcohol and or severe depression
10. Drug interactions
  - a. Anticholinergics (like Benadryl) may decrease absorption from the GI tract
  - b. **Used with digoxin or other antidysrhythmic agents may cause AV heart block.**
11. Overdose
  - a. Atropine or isoproterenol can be used to reverse hypotension and bradycardia.
12. Nursing Responsibilities
  - a. Assess BP and HR before administration
  - b. Monitor apical pulse, BP, Resp, and peripheral circulation throughout dosage adjustment period.
  - c. Assess pulmonary status for bronchospasm and bronchoconstriction
  - d. Hold dose for BP<100 or HR<60
  - e. Monitor diabetic patients for hypoglycemia
  - f. Monitor hypertensive patients with CHF for signs of impending heart failure
    - i. Dyspnea on exertion, night cough, edema, orthopnea, JVD
  - g. Discontinue if patient develops depression
  - h. Discontinue slowly (over 1 to 2 weeks)

## Calcium Channel Blockers

CALCIUM CHANNEL BLOCKERS ARE EFFECTIVE AT REDUCING MYOCARDIAL OXYGEN DEMAND AND TREATING STABLE AND VASOSPASTIC ANGINA. CALCIUM CHANNEL BLOCKERS RELIEVE ANGINA BY DILATING THE CORONARY VESSELS AND REDUCING THE WORKLOAD ON THE HEART. THEY ARE DRUGS OF CHOICE FOR TREATING VASOSPASTIC ANGINA, AND ARE EFFECTIVE ALTERNATIVES FOR STABLE ANGINA WHEN BETA BLOCKERS ARE CONTRAINDICATED.

1. Relaxes smooth arteriolar muscles – reduces blood pressure
2. Some slow cardiac conduction velocity through AV node – decreases HR
3. Dilates coronary arteries
4. Uses
  - a. For stable exertional angina patients who cannot tolerate beta blockers
  - b. For persistent symptoms – can be used along with nitrates or beta-blockers
5. Adverse effect
  - a. Not generally serious, related to vasodilatation
    - i. Headache, dizziness, edema of ankles and feet
  - b. Use cautiously in patients on meds that slow AV conduction
    - i. May cause partial or complete heart block
    - ii. Particularly digoxin or beta adrenergic blockers.

## Angiotensin – converting enzyme (ACE)

1. Angiotensin I converted to Angiotensin II by ACE (angiotensin convertin enzyme)
2. ACE also breaks down bradykinin (mediator of inflammation and pain)
3. Angiotensin II acts directly on vascular smooth muscles to cause vasoconstriction
  - a. **Rapid – instantaneous increase in blood pressure**
  - b. Effect is much greater on the arterioles
  - c. **Arterioles serving the kidneys are very sensitive to vasopressor action**
  - d. Activates the sympathetic nervous system causing norepinephrine release
  - e. Adrenal medulla responds by releasing epinephrine
  - f. Increases sympathetic outflow from the brain.
  - g. Alteration of cardiovascular structure: **cardiac remodeling**
    - i. Causes hypertrophy of myocardial cells and promotes collagen deposits
      1. Scar like tissue in the heart
  - h. Causes the kidney nephron to increase  $\text{Na}^+$  absorption and  $\text{K}^+$  and  $\text{H}^+$  excretion
    - i. Also causes additional amounts of aldosterone to be produced with also increase  $\text{Na}$  absorption and  $\text{K}$  and  $\text{H}$  excretion
    - ii. Causes the body to retain water, thus increasing blood volume and BP
    - iii. The action on the kidney develops slowly and may be responsible for sustained elevated BP and gradual decline of renal function.
4. Multiple points for drug interaction
  - a. ACE inhibitors
    - i. Detected in venom pit vipers 1960s
    - ii. Block conversion of angiotensin I to angiotensin II
    - iii. Causes the BP to decrease due to less sympathetic activation
    - iv. Slow the progression of heart failure
    - v. Benefit to recent MI patients
    - vi. Prophylaxis for patients with a high risk of a cardiovascular event
    - vii. Delay or prevent progression of renal disease
    - viii. Drugs
      1. Lotensin, Capoten, Vasotec, Monopril, Prinivil, Zestril, Univasc, Aceon, Accupril, Altace, Mavik.

2. Route PO
3. Absorption : 25-30% GI track
4. Not Metabolized
5. Excretion: renal
6. Onset 1hour, peak 6-8 hours
7. Duration 24 hours, half life 12 hours
8. Use in extreme caution in patients with hyperkalemia (dysrhythmias)
9. Interaction: interact with NSAIDs, decreases antihypertensive action
- ix. Adverse effects : headache, dry cough, dizziness, orthostatic hypertension, rash
  1. Angioedema, acute renal failure first dose phenomenon
- x. Nursing responsibilities
  1. Complete history
  2. Notify prescriber if patient has a history of kidney disease
  3. Monitor BP before and 30min to 1 hour after
    - a. Withhold if BP <90
  4. Evaluate effect of the drug
  5. Discontinue if evidence of angioedema of extremities (face, lips, tongue, glottis and larynx)
  6. **Monitor sodium and potassium levels for hyponatremia and hyperkalemia.**
  7. Monitor kidney function tests
  8. If patient is receiving lithium, monitor level frequently.
- b. Angiotensin receptor blockers
 

ARBs PREVENT ANGIOTENSIN II FROM REACHING THE AT<sub>1</sub> RECEPTOR, THUS CAUSING MANY OF THE SAME BIOLOGIC EFFECTS AS THE ACE INHIBITORS. THEY ARE APPROVED TO TREAT HTN, HEART FAILURE, AND ACUTE MI, AND THE PROPHYLAXIS OF CVA AND MI. ADVERSE EFFECTS ARE USUALLY MINOR AND TRANSIENT.

  - i. Inhibit AT<sub>1</sub> receptor and are used to treat hypertension and heart failure
  - ii. Cause vasodilatation, decreased peripheral resistance and a decreased BP
  - iii. Drugs: atacand, teveten, avapro, cozaar, benicar, micardis, diovan
  - iv. Adverse effects: same as ACE inhibitors
  - v. Metabolism: hepatic
  - vi. Excretion: 35% renal 60% in feces
  - vii. Peak onset 6 hours, half life 1.5 - 2 (losartan), 6-9 Hr (active metabolite)
  - viii. Contraindications: pregnancy and lactation
  - ix. Interactions: same as ACE inhibitors
    1. Patient should avoid OTC meds for colds, sinus fever and rash.
- c. Aldosterone antagonists
- d. Renin inhibitors

#### Anticoagulants

COAGULATION IS REGULATED BY A BALANCE OF PROCOAGULANTS AND ANTICOAGULANTS CIRCULATING IN THE BLOOD. THROMBOEMBOLIC DISORDERS RESULT WHEN A THROMBUS OR EMBOLUS FORMS IN A VEIN OR ARTERY. DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM ARE TWO PRIMARY DISORDERS CAUSED BY ABNORMAL CLOTTING.

1. Anticoagulants and antiplatelet agents prevent the formation of clots, thrombolytics dissolve existing clots, and hemostatics promote the formation of clots. Clotting factors may be administered to patients with hemophilia to lessen the risk of bleeding.

2. Venous thromboembolism (VTE) -blood flow through veins is very slow and coagulation factors accumulate.
  - a. Manifestations
    - i. DVT and Pulmonary embolism
3. Parenteral Anticoagulants
  - a. Heparin : most commonly used anticoagulant
    - i. Indirectly inhibits thrombin
    - ii. Natural chemical found in the liver and lining of blood vessels
    - iii. Binds to antithrombin III (AT-III inactivates thrombin)
      1. Produces a structural change in AT-III that makes it 1000 times more effective in inhibiting thrombin, factor Xa, factor IXa and other substances involved in coagulation.
    - iv. Monitoring of anticoagulation critical
      1. PTT is the conventional measurement
        - a. Should be between 1.5 to 2 times the patient's baseline with normal values ranging from 60 to 18 seconds.
        - b. Elevated PTT indicates the patient is at risk for bleeding
        - c. Value under 60 seconds indicate a need for higher doses
  - b. Low molecular weight heparins
    - i. Given sub-Q
    - ii. Unable to bind to thrombin, more selective for factor Xa inhibition
    - iii. Several advantages over heparin
      1. Less binding to proteins and macrophages - less frequent lab monitoring
      2. Less likely to cause thrombocytopenia (not enough platelets)
      3. **Dosage based on weight, PTT unaffected by Factor Xa**
        - a. Predictable plasma levels
      4. Xa target values 0.5 to 1 unit/ml
  - c. Drugs similar to heparin
    - i. Fragmin : smaller chains compared to heparin
    - ii. Lovenox:
    - iii. Innohep: for acute symptomatic DVT
4. Direct thrombin inhibitors: anticoagulants derived from medical leeches
  - a. Hirudin class drugs
  - b. Lepirudin (hirudin class drug)
    - i. Protein
    - ii. IV as a 15 to 20 second bolus or by continuous infusion
    - iii. Monitor PTT - ratio of 1.5 - 2.5 is optimum
    - iv. Distribution: extracellular fluids
    - v. Metabolism: blood
    - vi. Excretion: renal 50%
    - vii. Duration: unknown
  - c. Nursing responsibilities
    - i. Monitor baseline laboratory tests
      1. PT, INR, aPTT
    - ii. Withhold if PTT is greater than 2.5
    - iii. No specific antidote for an overdose.
    - iv. Monitor PTT 4 hours after start of therapy

- v. NO oral anticoagulants until lepirudin dose has been reduced and aPTT is just above 1.5
- d. Drugs similar to lepirudin
  - i. Acova, Novastan, Angiomax, Iprivask,

### Oral Anticoagulants

Warfarin (Coumadin) is the most widely used drug for oral anticoagulants

Warfarin is a frequently prescribed anticoagulant for the long-term prevention of thromboembolic disease. The anticoagulant activity of warfarin is monitored through prothrombin (PT) testing. Many drug–drug interactions are possible and therapy requires careful monitoring to prevent abnormal bleeding events.

1. PT is the standard laboratory test to monitor warfarin
  - a. Normal PT range is 12 to 15 seconds.
  - b. During therapeutic treatment, PT should increase 1.5 to 2 times the patients baseline
  - c. PT time is also reported as an INR value, INR values of 2.5 are considered therapeutic for most indications.
2. Warfarin is in a class by itself.
3. Vitamin K antagonist
4. Used for long term prophylaxis of thromboembolisms
5. Takes several days to reach optimum therapeutic effect
6. Mechanism of action
  - a. Inhibits two enzymes involved in formation of activated vitamin K
  - b. Vitamin K is required for synthesis of clotting factors II, VII, IX and X
  - c. Inhibits new clotting factors, but does nothing to those that already exist
  - d. Takes 3 to 4 days for existing clotting factors to fall
7. Metabolism: Hepatic
8. Excretion: renal
9. Duration: 3 to 5 days.
10. Contraindications
  - a. Patients with recent trauma, bleeding disorders, intracranial hemorrhage, severe HTN
  - b. Cannot take while pregnant
11. Drug interactions
  - a. Use with other anticoagulants may cause harm
    - i. this includes aspirin, NSAIDS, and antiplatelet drugs
12. Treatment of overdose:
  - a. Vitamin K

### Beta Blockers

BETA<sub>1</sub>-ADRENERGIC ANTAGONISTS ARE SELECTIVE FOR RECEPTORS IN CARDIAC MUSCLE. THIS RESULTS IN THESE DRUGS HAVING FEWER ADVERSE EFFECTS THAN THE NONSELECTIVE AGENTS. THE PRIMARY INDICATIONS ARE THE SAME AS THOSE FOR THE NONSELECTIVE BETABLOCKERS: HTN, ANGINA, AND MI. A FEW ARE USED FOR THEIR ANTIDYSRHYTHMIC ACTIVITY AND FOR THEIR ABILITY TO LOWER INTRAOCULAR PRESSURE IN PATIENTS WITH GLAUCOMA.

-note: a few beta blockers have beta-antagonist, AND beta-agonist activity. The low level of beta agonist is called intrinsic sympathomimetic activity (ISA). These may have fewer adverse effects in patients with bradycardia, CHF of poor pulmonary function.

1. Have potential to
  - a. Reduce heart rate, decrease force of contraction, and slow conduction velocity through AV node.

- b. Effects are minor while at rest, but during stress, they prevent normal sympathetic stimulation of the heart. (may hide some signs of shock like rapid heart rate)
- 2. Primary use: Treatment of Hypertension
- 3. Mechanism
  - a. Hypotensive effect via decreased cardiac output through decreased contractility
  - b. Antagonize release of renin by kidney, lowering BP
- 4. Reduce acute chest pain by reducing cardiac work load.
- 5. Reduced cardiac conduction effects some dysrhythmias.
- 6. Some non-selective beta blockers reduce intraocular pressure when given as drops
  - a. Timolol
- 7. Treatment with diabetics must be closely monitored
  - a. Patient can develop hypoglycemia and the beta blocker hides the warning sign of tachycardia
- 8. Drugs (selective Beta1)
  - a. Acebutolol (Sectral)
  - b. Atenolol (tenormin)
  - c. Betaxolol
  - d. Bisoprolol
  - e. Esmolol
  - f. Metoprolol
  - g. Nebivolol
- 9. Drugs (Alpha 1 and Beta Blockers)
  - a. Inderal Antihypertensive, antidysrhythmic
    - i. Non selective beta adrenergic blocker
    - ii. Treats HTN, Angina pectoris, dysrhythmias, migraine prophylaxis, MI Prophylaxis
    - iii. Metabolism Hepatic
    - iv. Excretion Renal
    - v. Onset PO 1-2 hours, IV-immediate
    - vi. Half life 3-5 hours
    - vii. Adverse effects: nausea, diarrhea
    - viii. Contradictions: bradycardia, cardiogenic shock, severe heart failure
    - ix. Drug interactions: Use with calcium channel blockers may produce bradycardia
    - x. Use with drugs that slow conduction through AV node may produce bradycardia
    - xi. Use with ethanol or antacids will slow absorption
  - b. Nursing responsibilities
    - i. Monitor vital signs especially apical pulse
    - ii. Assess for COPD, asthma, and allergies : can cause bronchoconstriction
    - iii. Evaluate response to HTN
    - iv. Check labs for Kidney, liver, hematology and cardiac function
    - v. Monitor I&O
    - vi. Assess for cold, painful, or tender hands or feet (impaired circulation)
- 10. Drugs (Beta1 antagonist - selective for receptors in myocardium)
  - a. Used to treat HTN
  - b. Little effect on beta 2 (bronchial smooth muscles)
    - i. Safe with COPD patients
  - c. Use cautiously with heart failure
  - d. Overdose treated with an anticholinergic (atropine, isoproterenol)
    - i. Plasma volume expanders

- e. Digoxin or diuretics can be used if heart failure develops
  - f. Drugs : Lopressor (metoprolol)
    - i. Acebutolol, atenolol, betaxolol, bisoprolol, esmolol
11. Patient goals on beta1 blockers
- a. Exhibit a decreased blood pressure with few side effects
  - b. Report a decrease in urinary symptoms such as difficulty voiding
12. Key concepts
- a. Beta blockers block the effects of norepinephrine
  - b. Used to treat hypertension and prostatic hyperplasia (enlargement)
  - c. Beta1 used to treat HTN and cardiovascular disorders

### Calcium Channel Blockers

Calcium channels facilitate contraction in cardiac and smooth muscles. Contraction of cardiac and smooth muscles requires an influx of calcium ions through calcium channels and the release of calcium ions from the sarcoplasmic reticula. The increase in intracellular calcium removes the inhibition of actin and myosin, allowing the filaments to slide and contraction to occur

Blocking calcium channels has significant physiological effects on the heart and vascular smooth muscle. Blockade of calcium channels has three important physiological effects. In vascular tissue, blockade prevents contraction of smooth muscle, resulting in the vasodilation of arterioles, including those in the coronary arteries. In the heart, cardiac conduction is slowed and the force of myocardial contraction is diminished

1. Types of Calcium ion channels
  - a. L type (most important) - these bind calcium channel blockers
    - i. On the sarcolemma of cardiac and smooth muscles
      1. Extracellular - receptor to bind CCBs
      2. Central portion - pore where calcium ions travel
      3. Intracellular - second messenger initiates cascade to perform cells function
    - ii. Regulated by depolarization across sarcolemma
    - iii. L type are slow, allowing sustained Ca<sup>+</sup> flow
    - iv. CCB changes the shape of the channel
      1. Shape change prevents Ca<sup>+</sup> from flowing through the channel
  - b. T-Type Present in smooth muscles and SA node
    - i. No current T type selective blockers
  - c. N-type Present throughout the nervous system
    - i. Controls neurotransmitter release at synapses
    - ii. Transmission of pain impulses in the spinal chord
    - iii. None are currently approved
2. CCBs DO NOT EFFECT serum calcium levels
3. CCBs have a significant effect on peripheral resistance
  - a. Prevent contraction of peripheral arterioles
  - b. Vasodilation
  - c. Decrease in blood pressure
  - d. Afterload is reduced - lower myocardial oxygen demand
    - i. Less chest pain
  - e. Veins are not affected - NO AFFECT ON PRELOAD

- f. Reduce the force of contraction
- g. Slows the speed of conduction
  - i. Slows the SA node and the conduction through the AV node
- 4. Classified as dihydropyridines and nondihydropyridines
  - a. Dihydropyridines
    - i. Largest class of CCBs
      - 1. Norvasc, Plendil, DynaCirc, Cardene, Adalat, Procardia, Nisocor
    - ii. Bind reversibly to closed type calcium channels to make them unresponsive to depolarization
    - iii. Primarily used for vasodilation effect (mainly affect vascular smooth muscles)
    - iv. Used in:
      - 1. Hypertension (Norvasc, Plendil, DynaCirc, Cardene, Cargene SR, Procardia)
      - 2. Chronic stable angina (Norvasc, Cardene Procardia)
      - 3. Vasospastic Angina (Procardia, Procardia SL, Adalat)
      - 4. Subarachnoid Hemorrhage (Nimotop)
    - v. Dosage must be adjusted in patients with significant hepatic problems
    - vi. In high doses they lose their selectivity and also effect the heart
    - vii. Side effects: flushed skin, headache, dizzy, peripheral edema, nausea
      - 1. May cause reflex tachycardia (transient)
    - viii. Overdose treated with dopamine or dobutamine
    - ix. Nursing Obligations
      - 1. Monitor BP before and 30min to 1 hour after
      - 2. Monitor glucose in diabetic patients
      - 3. Report gingival hyperplasia (rare but serious reaction)
        - a. Swelling and bleeding gums
  - b. Nondihydropyridines
    - i. Bind to different subunits of the L-type calcium channels
      - 1. Bind to open type calcium channels causing them to close
      - 2. Delay re-activation, slowing re-polarization
    - ii. Cardizem, verapamil
    - iii. More effective with cardiac muscle
      - 1. Slow the speed of myocardial conduction
    - iv. Also have the vasodilation effect on smooth muscle
    - v. Can suppress certain dysrhythmias (atrial flutter, or vibration)
    - vi. Used in :
      - 1. Chronic angina (cardizem, cardizem SR, Cerapamil)
      - 2. Hypertension (Cardizem SR, Verapamil)
      - 3. Dysrhythmias (Cardizem, verapamil)
    - vii. Keep the patient in recumbent position for at least 1 hour after a dose to avoid transient asymptomatic hypotension (IV infusion only)

Nursing Diagnosis related to blocking medications

- Ineffective health maintenance related to uncontrolled blood pressure
- Deficient knowledge (calcium channel blocker) related to purpose, precautions, and adverse effects of drug
- Decreased cardiac output related to disease process
- Ineffective tissue perfusion (cardiopulmonary) related to drug therapy

## Cardiac Drugs

### Drugs for Dyslipidemias (high lipids)

Derived from fungi, known as statins (Lovastatin, Pravastatin and Simvastatin are natural)

Can reduce LDL 20-40% and reduce cardiac events 25-30%

#### 1. Statins

- a. All statins are given orally
- b. Patients who develop unexplained muscle or joint pain during statin therapy to report this to their prescriber
  - i. CK enzyme (muscle) is elevated then statin should be discontinued
- c. Lovastatin should be administered at night (short half life)

#### d. Drugs:

##### i. Lipitor                                      Antihyperlipidemic                      HMG-CoA reductase inhibitor statin

1. Used to treat hypercholesterolemia
2. Causes the liver to produce less cholesterol and it responds by creating more LDL receptors, removing LDL from the blood
3. Given PO
4. Only 30% reaches bloodstream
5. Metabolized Hepatic
6. Excretion : Biliary
7. Onset : 2 weeks for lipid lowering effect
8. Adverse reaction: headache, intestinal cramps, diarrhea, constipation
  - a. Serious adverse affect : rhabdomyolysis - breakdown of muscle fiber
9. Contraindications/precautions
  - a. Patients with reduced hepatic function
  - b. Interacts with Digoxin (increases levels by 20%)
  - c. Grapefruit juice inhibits the metabolism of statins allowing them to reach high serum levels
10. Similar drugs: Zocor, Mevacor, Pravachol, Crestor

#### 2. Bile Acid Sequestrants - often combined with statins

- a. Cholesterol in bile acid is recycled.
- b. Sequestrants can cause a 20% decrease in cholesterol
- c. Tend to cause more adverse effects than statins
  - i. Not absorbed into systemic circulation, their effects are limited to the GI and can cause abdominal pain, anorexia, bloating, steatorrhea (excess fat in feces), and constipation.

#### d. Drugs:

##### i. Questran                                      Antihyperlipidemic                      Bile Acid sequestrant

1. Comes as a powder and mixed with 60 to 180ml of water, non carb bev, highly liquid soup, or pulpy fruits to avoid esophageal irritation.
2. If not taken with enough fluid, or not completely swallowed, it can cause swelling in the throat, or esophagus and cause an obstruction.
3. Binds to bile acids forming an insoluble complex that holds the cholesterol and is excreted with the feces.
4. Contraindicated if serum triglycerides rise above 400 mg/dl
5. May block absorption of iron and fat soluble vitamins.

## Angina Pectoris

### 1. Types

- a. Stable angina: predictable in frequency, intensity and duration - relieved by rest
- b. Vasospastic angina (Prinzmetal's): decreased myocardial blood flow is caused by spasms of the coronary arteries. - the vessels may or may not have atherosclerotic plaque.
  - i. Cause is vasoconstriction
  - ii. Often occurs during periods of rest
- c. Silent angina: ischemia occurs in the absence of pain.
  - i. Associated with a high risk for acute MI and sudden death
- d. Unstable angina: episodes occur suddenly, have added intensity, and occur during periods of rest. Type of acute coronary syndrome in which an atherosclerotic plaque within a coronary artery ruptures.
  - i. A thrombus builds on the displaced plaque and the artery becomes at risk of occlusion

### 2. Angina Pain: parallels the signs and symptoms of MI

- a. Important to differentiate the differences.

### 3. Nonpharmacological therapy

- a. Limit alcohol consumption
- b. Eliminate food high in cholesterol or saturated fat
- c. Keep blood cholesterol within normal range
- d. Keep BP in normal range
- e. Keep blood glucose in normal range
- f. Exercise regularly and maintain optimal weight
- g. Do not smoke

### 4. Pharmacologic therapy

- a. Two categories: terminate the angina episode, and prevent or reduce episodes
- b. Three primary classes of drugs: rapid acting nitrates, calcium channel blockers, and beta blockers.
  - i. [Nitrates](#) are used for terminating an acute episode
    1. Dilates veins and arteries reducing the amount of blood returning to the heart and decreasing the workload
    2. **While it does dilate the coronary arteries, it is no longer considered the primary mechanism of nitrate action. (Adams P 592)**
    3. Tolerance is common with long acting nitrates
    4. Common side effect is flushing of the face and headache
    5. Reflex tachycardia from the baroreceptor reacting the sudden drop in BP is rare, but an undesired side effect.
  - ii. Blockers are used for long term prophylaxis
  - iii. New class of drug called ranolazine (Ranexa) - 2006
    1. Partial fatty-acid oxidation inhibitors
      - a. Exact mechanism unknown
    2. May shift cardiac cells to use primarily glucose instead of fatty acids
    3. Approved for chronic angina that does not respond to other meds
  - iv. Other antianginal agents
    1. [Atenolol](#)

Myocardial Infarction : early diagnosis increases chances of survival.

THE EARLY DIAGNOSIS OF MI ALLOWS FOR MORE AGGRESSIVE PHARMACOTHERAPY AND DECREASES PATIENT MORTALITY. EARLY PHARMACOTHERAPY MAY INCLUDE THROMBOLYTICS, ASPIRIN, BETA-BLOCKERS, AND ANTIDYSRHYTHMICS

1. Primary cause of an MI is CAD, atherosclerotic plaque in the endothelial wall of one or more branches of the coronary arteries.
  - a. Once blood flow stops to a section of the heart, it begins to use anaerobic metabolism after 8 to 10 seconds. Lactic acid accumulates, causing acidosis ischemia develops, contractility and normal conduction. The patient experiences heart failure.
    - i. Monocytes will begin to die in about 20 minutes
    - ii. The necrosis of heart tissue releases “marker” enzymes, such as creatine phosphokinase (CK) or cardiac specific troponin (cTn) which is measured in the blood to confirm the MI
  - b. Treatment of acute MI
    - i. In arrest: restart the heart & restore BP with vasopressors
    - ii. Restore blood supply to damaged areas with thrombolytics
    - iii. Reduce myocardial oxygen demand with nitrates, beta blockers, or CCBs
    - iv. Control or prevent MI associated dysrhythmias with amiodarone, beta blockers or other antidysrhythmics
    - v. Manage MI pain with narcotic analgesics
2. Thrombolytic agents: Accelerate the process of clot removal by converting plasminogen to plasmin that digests fibrin.
  - a. Contraindicated for patients with recent surgery or trauma, internal bleeding, active peptic ulcer, postpartum, intracranial hemorrhage (hx of), severe liver disease, thrombocytopenia.
  - b. Drugs
    - i. Streptokinase: first drug in this class
    - ii. Tenecteplase (TKN-tPa) (Alteplase)
      1. More fibrin specific, and drug of choice for cardiac thrombosis
      2. Replaced urokinase in clearing clotted central lines
      3. Metabolism: hepatic
      4. Excretion: renal
      5. Onset: immediate
      6. Duration 3h, half life <10 minutes
      7. Overdose: administration of blood products or hemostatics
  - c. Nursing responsibilities
    - i. Health history
    - ii. Prior to treatment: assess coagulation tests including
      1. aPTT, bleeding time, PT, and INR, Baseline Hct, Hgb, and platelet counts following drug administration to detect blood loss
      2. assess for signs of bleeding every 15 minutes for first hour than 30
      3. Avoid invasive procedures, IM injections, or physical manipulation
      4. Keep patient in bed
      5. Check vitals frequently
      6. Draw ABG using radial artery and hold for 30 minutes
  - d. Similar drugs: Reteplase, Streptokinase, TNKase
3. Aspirin

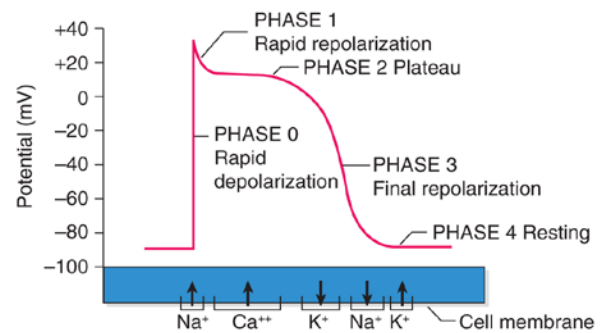
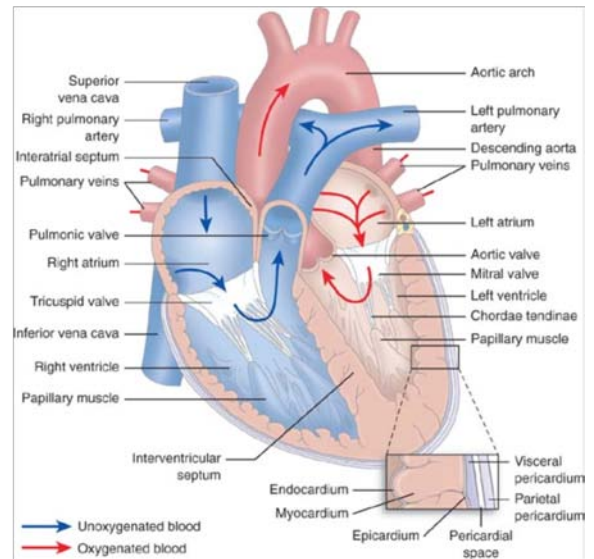
- a. 160 to 325 mg given as soon as an MI is suspected
- 4. ADP receptor blockers
  - a. Plavix and Ticlid - antiplatelet agents
- 5. Glycoprotein IIb/IIIa Inhibitors
  - a. Interferes with IIb/IIIa receptor on platelet
- 6. Anticoagulants
  - a. [Heparin](#), for 48 hours then switched to [Warfarin](#) (coumadin)
  - b. Alternative is a low molecular weight heparin ([lovanox](#))
- 7. Restore coronary and cerebral blood flow
  - a. Vassopressors (vasopressin or antidiuretic hormone)
  - b. Sympathomimetics (epinephrine)
- 8. Prevent recurrence of V-Fib
  - a. Amiodarone (Cordarone) now the drug of choice

## Assessment of Cardiovascular Function

### Coronary Vascular Disorders

#### Overview:

1. Three layers
  - a. Endocardium
  - b. Myocardium
  - c. Epicardium
2. Four chambers
3. Four valves
4. Depolarization : electrical activation of cells caused by influx of sodium into cells while potassium exits
5. Repolarization : return of cell to resting state caused by re-entry of potassium into cell while sodium exits.
6. Action Potential
7. Cardiac Output
  - a. The amount of blood pumped out of the left ventricle during a given period
  - b. In a resting adult, it's about 5 l/min
  - c. Can rise to 20 l/min
8. Stroke volume- the amount of blood ejected from the ventricle
9. Pulse pressure: Reflection of the stroke volume, ejection velocity, and systemic resistance.
  - a. Normal 30mm-40mm Hg
  - b. Increased in anxiety, exercise, bradycardia
  - c. Decreased in shock, HF, hypovolemia, mitral regurgitation, or obstruction to blood flow
  - d. Less than 30 signifies a serious reduction in cardiac output
10. Orthostatic hypotension, three most common causes with cardiac patients
  - a. Reduced fluid volume
  - b. Inadequate vasoconstrictor mechanism
  - c. Insufficient autonomic effect on vasoconstriction
  - d. Procedure
    - i. Patient supine and flat for 10 minutes prior to test
    - ii. Patient sits on side of bed, legs dangling
    - iii. 1 to 3 minutes should elapse after each postural change before measuring BP
    - iv. If patient exhibits any signs or symptoms, return them to the supine position
    - v. Both HR and BP are recorded.
    - vi. HR Normal change - 5 to 20 bpm
    - vii. BP normal : unchanged systolic pressure, or slight decrease 10mmHg systolic and slight increase of 5mm Hg diastolic.



**Cardiac Assessments** A diagnosis of an MI is made by evaluation of History, physical exam, 12-lead ECG and result of laboratory tests that measure serum cardiac markers.

1. Health History
  - a. Can the patient recognize his own cardiac symptoms?
    - i. Often the symptoms go unrecognized

1. Lack of knowledge
  2. Denial
  3. Attribute problem to something benign
  - ii. Speaking with family members may assist with information
  - iii. Medications: include over the counter and vitamins
    1. Is the patient independent in taking meds?
    2. Are side effects reported?
    3. Are doses ever forgotten or skipped, or does the patient ever decide to stop taking them.
  - b. Bowl habits
  - c. Eating habits: who normally shops for groceries or prepares meals
  - d. BMI (weight in kg/height in meters squared), waist measurement, lab results, glucose, lipoproteins
2. Physical Exam
- a. Common symptoms
    - i. Chest pain
      1. Quantity, quality, events leading up to pain
      2. The location is not well correlated with the cause
    - ii. SOB
    - iii. Peripheral edema
    - iv. Palpitations
    - v. Vital fatigue, or vital exhaustion
    - vi. Dizziness, syncope, or change in LOC
  - b. Acute Coronary syndrome
    - i. Prodromal symptoms up to a month prior to incident, often attributed to something benign
      1. Fatigue, SOB, sleep disturbances, anxiety, fleeting chest discomfort
      2. Only 50% of people with ACS experience chest symptoms. The remainder may have upper back, shoulder, arm or neck pain.
      3. At least four symptoms herald the onset of ACS
      4. Neuropathies in the elderly and diabetics may prevent them from feeling these.
    - ii. 12 lead and serum lab values determine If the patient with ACS symptoms has unstable angina, a non-ST segment elevation MI, or an ST segment elevation MI.
1. Laboratory Tests (Cardiac Related)
- a. Cardiac biomarker analysis
    - i. CK isoenzymes (CK-MB), and proteins (myoglobin, triponin T, and troponin I)
    - ii. Substances end up in the circulatory system from damaged tissue
  - b. Blood Chemistry, Hematology, and coagulation studies
    - i. Lipid Profile
      1. Cholesterol, triglycerides, and lipoproteins measure a persons risk
      2. Lipoproteins are referred to as low density lipoproteins (LDLs) and high density lipoproteins (HDLs)
        - a. The risk of CAD increases as the ratio of LDL to HDL increases
        - b. Of the ratio of total cholesterol (LDL+HDL) to HDL increases
      3. Cholesterol: normal <200 mg/dl
        - a. Required for hormone synthesis and cell membrane formation.
      4. LDLs : normal <160mg/dl

- a. Primary transporters of cholesterol and triglycerides into the cell
  - b. Harmful effect: deposition of these substances on the walls of the arteries.
  - c. Goal for lipid management is a reduction to <70 mg/dl
- 5. HDLs: normal 35-70 men, 35-85 women
  - a. Protective action
  - b. Transport cholesterol away from the tissue and cells of arterial wall to the liver for excretion.
  - c. Inverse relationship between the level of HDLs and the risk of CAD
  - d. Goal for lipid management is HDL >40 mg/dl
- 6. Triglycerides: normal 100 - 200 mg/dl
  - a. Composed of free fatty acids and glycerol
  - b. Stored in adipose tissue and are a source of energy
  - c. Increase after meals and are effected by stress
  - d. Diabetes, alcohol use and obesity can elevate
- 7. Brain (B-type) [natriuretic](#) peptide
  - a. Neurohormone that helps regulate BP and fluid volume.
  - b. Secreted from ventricles in response to increased preload with elevated ventricular pressure
  - c. Elevations in BNP can occur from pulmonary embolism, MI, and ventricular hypertrophy
  - d. Level > 100 pg/ml is suggestive of an MI
- 8. C-reactive protein
  - a. Protein produced by the liver in response to systemic inflammation
  - b. Inflammation plays a role in the progression of atherosclerosis
  - c. Increased hs-CRP (>3 mg/dl) may be at greatest risk for CVD
- 9. Homocysteine
  - a. Levels increase the ability to make a diagnosis
  - b. Homocysteine is an amino acid, is linked to the development of atherosclerosis because it can damage the endothelial lining of the arteries and promote thrombus production.
  - c. Elevated level is thought to identify a risk for stroke, CAD, PVD
  - d. Optimal <12 umol/l
  - e. High risk >12 umol/l
- 10. Chest X-ray
  - a. Determine the size, contour and position of the heart.
  - b. Reveals cardiac and pericardial calcifications and physiologic alterations.
  - c. Can't diagnose MI, but can diagnose complications
- 11. Electrocardiography
  - a. Graphic representation of the electrical activity of the heart
  - b. Used to diagnose dysrhythmias, conduction abnormalities, chamber enlargement, and myocardial ischemia, injury, and infarction.
- 12. Continuous ECG monitoring

- a. Detects abnormalities in HR and rhythm
  - b. Changes in the ST segment
13. Cardiac stress testing
- a. Normally coronary arteries dilate to four times their normal diameter to increased metabolic demands
  - b. Stress test can determine:
    - i. Presence of CAD
    - ii. Cause of chest pain
    - iii. Functional capacity of the heart after an MI or surgery
    - iv. Effectiveness of antianginal or antiarrhythmic medication.
    - v. Dysrhythmias that occur during physical exercise
    - vi. Specific goals for fitness programs
14. Echocardiography
- a. Ultrasound test used to measure the ejection fraction
  - b. Measure pericardial effusions
  - c. Chamber size
  - d. Etiology of heart murmurs
  - e. Evaluate the function of the valves
  - f. Wall motion
15. Radionuclide imaging
- a. Non-invasive evaluation of coronary artery perfusion
  - b. To detect ischemia and infarction
  - c. Assess left ventricle function
  - d. Uses Thallium 201, and technetium 99
    - i. Give off gamma rays as they decay
    - ii. Energy emitted can be detected
    - iii. 1 dimensional view from 3 points
  - e. Single photon emission computed tomography provides three dimensional images.
16. Myocardial perfusion imaging
- a. Thallium 201 is injected into the iv 1-2 minutes before the end of the stress test and an image is immediately taken.
  - b. It is taken up more slowly in damaged myocardial cells.
  - c. Resting images are taken 3 hours later to differentiate between ischemic cells and infarcted cells
17. Cardiac catheterization
- a. Invasive diagnostic procedure
  - b. Radiopaque arterial and venous catheters are introduced into selected blood vessels of the right and left sides of the heart.
  - c. Pressure and oxygen saturation levels in the chambers are measured.
  - d. Used to diagnose CAD, assess coronary artery patency, determine the extent of atherosclerosis

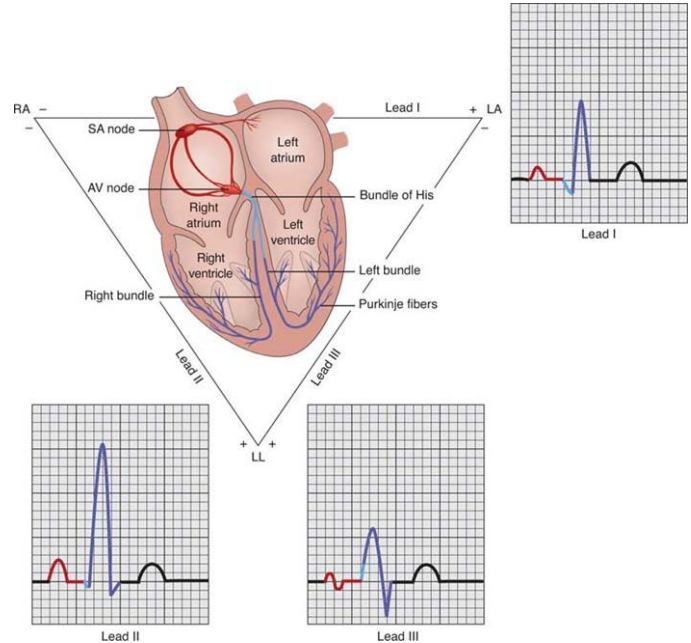
## Cardiac Dysrhythmias

-disorders of the conduction or formation of an electrical impulse  
-diagnosed by analyzing the electrocardiographic waveform (ECG)

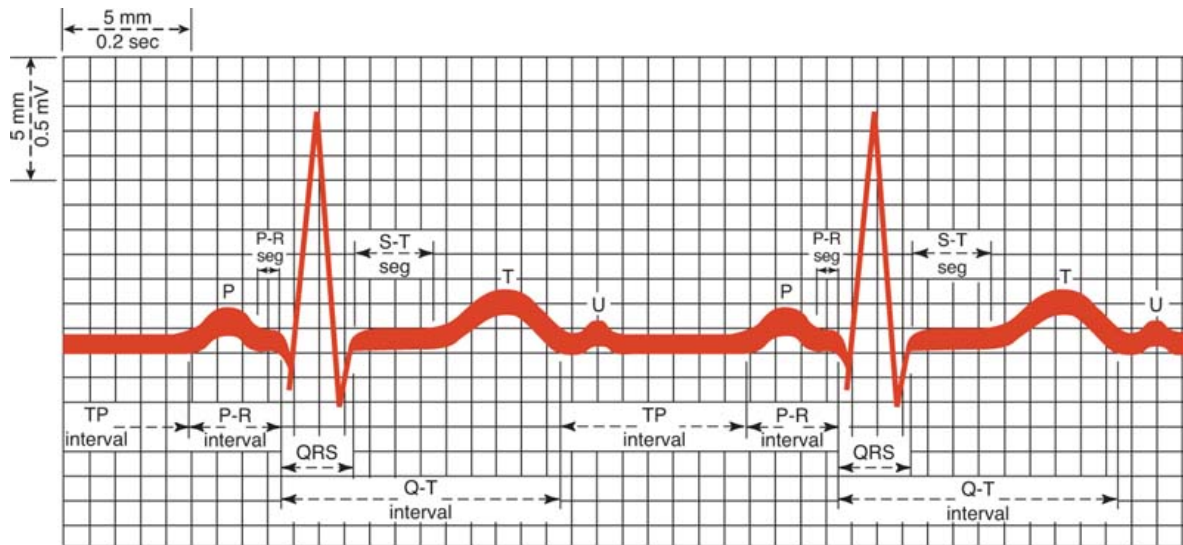
- Electrical stimulation - depolarization
- Contraction - systole
- Electrical relaxation - repolarization
- Mechanical relaxation - diastole

### Electrocardiograph

1. **P wave** - atrial depolarization
  - a. 2.5mm or less in height
  - b. **0.11 seconds or less**
2. **QRS complex** - ventricular depolarization
  - a. Q- first negative deflection
    - i. <.04 seconds
    - ii. <25% of R amplitude
  - b. R - first positive deflection after Q
  - c. S - first negative deflection after R
  - d. When QRS <5mm in height, small letters are used (qrs)
  - e. When QRS >5mm, capital letters are used (QRS)
  - f. **QRS is < 0.12 seconds** in duration
3. **T wave** - ventricular repolarization
  - a. Follows QRS usually in same direction as QRS
4. **U wave** - thought to represent repolarization of the purkinje fibers but is sometimes seen in patients with hypokalemia, hypertension, or heart disease.
  - a. If present, U wave follows the T wave
  - b. Usually smaller than the P wave
5. **PR interval**
  - a. Measure from beginning of the P to the beginning of the QRS
  - b. Time from SA node through atrial depolarization to conduction through AV node
  - c. **0.12 to 0.20 seconds**. (3 small boxes to 5 boxes) (each box = .04 seconds)
6. **ST segment**
  - a. From the end of the QRS complex to the beginning of the T wave.
  - b. Normally isoelectric (baseline)
  - c. If it is below the isoelectric line, may be a sign of cardiac ischemia
7. **QT interval**
  - a. Total time for ventricular depolarization through repolarization
  - b. Varies with HR, gender, and age
  - c. Usually between 0.32 and 0.40 seconds when HR is 65-95 bpm
  - d. If the QT is prolonged, the patient may get torsades de pointes (lethal rhythm)
    - i. This is a type of ventricular tachycardia (polymorphic)
8. **TP interval**
  - a. End of the T wave to the beginning of the next P
  - b. Isoelectric line : compare the ST segment to this line



9. PP interval
  - a. Used to determine ATRIAL rhythm and atrial rate.
10. RR interval
  - a. Used to determine VENTRICULAR rhythm and ventricular rate.



### Analyzing the ECG Strip

- DETERMINE THE VENTRICULAR RATE.
- DETERMINE THE VENTRICULAR RHYTHM.
- DETERMINE QRS DURATION.
- DETERMINE WHETHER THE QRS DURATION IS CONSISTENT THROUGHOUT THE STRIP. IF NOT, IDENTIFY OTHER DURATION.
- IDENTIFY QRS SHAPE; IF NOT CONSISTENT, THEN IDENTIFY OTHER SHAPES.
- IDENTIFY P WAVES; IS THERE A P IN FRONT OF EVERY QRS?
- IDENTIFY P-WAVE SHAPE; IDENTIFY WHETHER IT IS CONSISTENT OR NOT.
- DETERMINE THE ATRIAL RATE.
- DETERMINE THE ATRIAL RHYTHM.
- DETERMINE EACH PR INTERVAL.
- DETERMINE IF THE PR INTERVALS ARE CONSISTENT, IRREGULAR BUT WITH A PATTERN TO THE IRREGULARITY, OR JUST IRREGULAR.
- DETERMINE HOW MANY P WAVES FOR EACH QRS (P:QRS RATIO).

1. Normal sinus rhythm
  - a. Ventricular and atrial rate 60 - 100
  - b. Rhythm = regular
  - c. QRS - normal, but can be regularly abnormal
  - d. P wave normal and constant, always in front of the QRS
    - i. Interval .12 to .20 seconds
  - e. P:QRS ratio 1:1
2. Types of dysrhythmias
  - a. Sinus node dysrhythmias
    - i. Sinus bradycardia - SA node creates impulses < 60 per minute
      1. Causes :
        - a. Lower metabolic needs
        - b. Vagal stimulation

- c. Medications (blockers)
  - d. Idiopathic SA node dysfunction
  - e. Increased intracranial pressure
  - f. MI (inferior wall)
- ii. Sinus tachycardia - rate >100 but usually less than 120
  - 1. Causes:
    - a. Physiologic or psychological stress, shock
    - b. Medications that stimulate sympathetic system
    - c. Enhanced automaticity of SA node
    - d. Autonomic dysfunction
- iii. Sinus Arrhythmia - SA node fires irregularly
  - 1. Usually increases with inspiration and decreases with expiration
  - 2. Non respiratory causes: heart disease, valvular disease
  - 3. Rate 60-100 irregular timed P waves
- iv.