Nursing IV Monday Lecture Notes

Medical Surgical Unit

Acute Respiratory Failure

1. Respiratory failure: PaO2 less than 50mm Hg (hypoxia), PaCO2 > 50mm (normal 35-45), and a pH less than 7.35
    a. Restlessness, fatigue, headache, dyspnea, air hunger, tachycardia, increased blood pressure, confusion, use of accessory muscles
2. Apnea not readily reversible
3. Nursing management
    a. Maintain mechanical ventilation; monitor LOC, blood gasses, SaO2, vital signs, turning schedule, mouth and skin care, range of motion exercises.

Chronic Respiratory Failure

Acute Respiratory Distress Syndrome

1. Severe form of acute lung injury
2. Characterized by sudden and progressive pulmonary edema. (increasing bilateral infiltrates on chest x-ray),
3. Hypoxia unresponsive to oxygen, regardless of peep.
4. Occurs as a result of **diffuse alveolar damage**.
5. Dyspnea, arterial hypoxemia, cardiogenic pulmonary edema, increased alveolar dead space.
6. Clinical manifestations
    a. ARDS is an acute event
        i. Develops in 4 to 48 hours
    b. Closely resembles severe hemodynamic pulmonary edema.
    c. Acute phase marked by rapid onset of severe dyspnea 12-48 hours after the initiating event.
    d. Increased alveolar dead space, “stiff lungs”
7. Assessment
    a. Plasma brain natriuretic peptide (BNP) levels
        i. This is helpful to distinguish ARDS from a cardiac event
    b. Echocardiography, and pulmonary artery catheterization (the definitive test)
8. Nutrition
    a. ARDS patients require 35-45 kcal/kg/day
9. Nursing management

a. Oxygen, nebulizer treatments, physiotherapy, intubation, tracheostomy, suctioning, bronchoscopy
b. Positioning is important : oxygenation in patients with ARDS is sometimes improved by placing the patient in the prone position

10. Ventilator considerations
    a. Sedatives so the patient does not "buck" the ventilator
        i. Lorazepam (Ativan), versed, Precedex, propofol (Diprivan)
    b. Paralytics
        i. Pancuronium, vecuronium, atracurium, rocuronium
11. Major cause of death is multiple organ failure

Mechanical Ventilation *Brunner 655-660*

1. Indications: PaO2 <50 mm Hg with FiO2 >0.60
    a. PaO2 >50 mm Hg with pH <7.25
    b. Vital capacity < 2 times tidal volume
    c. Respiratory rate >35/min (Adult)
2. Classification of ventilators
    a. Negative Pressure (iron lung)
    b. Positive Pressure (most common)
    c. CPAP – continuos positive pressure
    d. BiPAP – mask or nasal
    e. ET tube up to 2 Weeks, Trach after that.
    f. Lingo
        i. RR = Respiratory Rate on expiration. VT=Tidal volume 10-15ml/kg (amount expelled Peak flow rate: when air enters lung
        ii. Inspiratory Pressure limit O2=% of oxygen delivered with each breath
        iii. FiO2= fractional measure of inspired O2=70%, FiO2=70
        iv. PEEP=Positive end-expiratory pressure (very important)
        v. BPM-Breath per minute Set rate=mandatory rate delivered by vent
        vi. Total Rate=spontaneous AND set breaths.
3. Volume cycled ventilator : most commonly used
4. Ventilator Modes (breath initiation)
    a. Assist control Ventilation [A/C]
        i. Delivers a preset tidal volume and rate. If the patient initiates, then the machine delivers the set volume. (more chance of bucking)
    b. Intermittent mandatory ventilation [IMV]
        i. Combination of mechanically assisted breaths and spontaneous breaths. Delivers preset interval and **tidal volume** (breath out) regardless of patient effort. It lowers mean airway pressure to decrease barotrauma.
    c. Synchronized intermittent mandatory ventilation [SIMV]

Know

        i. Delivers preset volume and rate, but it the patient breaths spontaneously, it provides no assistance.
    d. Pressure support ventilation [PSV]
        i. Applies a pressure plateau to airway throughout patient triggered respiration. Pressure is reduced over time as patient's strength is increased.
    e. Airway Pressure release ventilation [APRV]
        i. Time triggered, pressure limited, time cycled. Allows unrestricted, spontaneous breathing throughout the ventilatory cycle.
    f. Proportional assist ventilation [PAV]
        i. Provides partial ventilatory support. Ventilator provides pressure in proportion to the patient's effort. The ventilator synchronizes the ventilation with the patient's, providing "additional muscle"
5. Goal: Adjust ventilator so the patient is comfortable and breathes "in sync" with the machine.
6. Initial setting
    a. Tidal volume 10 to 15 ml/kg
    b. Record peak inspiratory pressure
    c. Record minute volume.
    d. Sensitivity – 2-cm H2O inspiratory force should trigger ventilation
7. Suctioning: 10-15 seconds, 100% O2 prior to suctioning (button on vent)
    a. Can cause sinus bradycardia (atropine)
8. Nursing Process
    a. Evaluate the patient's psychological status
        i. How are they coping, how is the family coping
    b. In depth focus on the respiratory system
        i. Vitals, respiratory rate, volume, quality, breath sounds, pattern
        ii. Potential evidence of hypoxia
    c. Two major interventions:
        i. Pulmonary auscultation
        ii. Interpretation of arterial blood gasses
    d. Potential complications
        i. Barotrauma
        ii. Pulmonary infection
        iii. Sepsis
    e. Goals : achievement of optimal gas exchange
    f. Risk for DVT, PE, skin breakdown
        i. Close monitoring, turn & reposition, Neuro checks, ABGs
9. Weaning Process
    a. Patient assessment
    b. Trial by decreasing support
    c. If HR > by 20, they go back on the vent
    d. If BP systolic > by 20, they go back on the vent
    e. Cardiac dysrhythmia, they go back on the vent

10. Comprehensive Nursing Care Plan
    a. Promoting gas exchange
        i. Suction the patient whenever PaO2 decreases below a set level
        ii. Turn and reposition patient every 2 hours
    b. Promoting effective airway clearance
        i. Suction the patient as needed
    c. Preventing trauma and infection
        i. Enforce strict aseptic technique and hand hygiene
        ii. Monitor and record cuff pressure
    d. Promoting optimal level of mobility
        i. Patient in chair
    e. Promoting optimal communication
        i. Use nonverbal, board
    f. Promoting coping ability
        i. Maintain sedation level
        ii. Education: explain procedures and status of patient
        iii. Educate family members
11. Complications of Vents
    a. Cardiac
        i. During the inspiration, heart and great vessels are compressed: decreased venus return, therefore decrease in cardiac output and thereby decrease tissue perfusion. Puts the patient at greater risk for cardiac dysrhythmias
    b. Barotrauma / pneumothorax
        i. Prevention: Turn and reposition, chest PT, head of bed elevated (this also decreases the risk of gastric secretion complications)
            1. Gastric secretion risk also decreased with H2 histamine blockers (Pepcid)
    c. Pulmonary Infection: similar to barotrauma.
12. Labs (ABG) need to tell lab that they are on the vent/oxygen/how long.
    a. pH 7.35-7.45
    b. PaCO***2*** 35-45 mmHG
    c. HCO***3 -*** 22-26 mEq/L
    d. PaO***2*** 80-100 mmHg
    e. SaO***2*** greater than 95% (pulse ox)
    f. BE (base excess) -3.0-+3.0
13. Compensation
    a. When both the PaCO2 and HCO3- are abnormal, one reflects a primary disorder and the other reflects a compensatory response.
    b. Determine what has caused the change in pH : that is the primary disorder. The other abnormal value is compensating for the primary abnormality.
    c. Non-compensation: an alteration of either pCO2 or HCO3-.
    d. Partial compensation: an alteration of both PaCO2 and HCO3-.

e. Complete (full) compensation: an alteration of both PaCo2 and HCO3-, but the pH is normal: ph of 7.35-7.40 acidosis and 7.40-7.45 alkalosis.

Medications

1. Fentanyl
2. Versed
3. Diprivan
    a. Used a lot in surgery with general anesthesia
    b. Drug of choice for vented patients
4. Ativan
5. Modified Ramsay Sedation Scale (see study guide supplement)
    a. Used with adult population
    b. Used to titrate medications
    c. Monitor vital signs closely (breath sounds, turn and reposition)
    d. Neuro checks
    e. Monitor arterial blood gasses

SHOCK

-widespread perfusion to the cells is inadequate to deliver oxygen and nutrients to support vital organs and cellular function. (Decreased Tissue Perfusion)

- MAP (mean arterial pressure) = cardiac output x peripheral resistance. Normal >65
    - To calculate approx MAP: (Pulse Pressure/3) + diastolic pressure
    - (2 x diastolic + systolic) / 3
- Pathophysiology
    - Cellular changes result in a decrease in adequate blood supply, decreased oxygen, decreased energy to the cell. (anaerobic)
    - Decrease in ATP, body will eventually eat it's muscle tone (glucogenesis)
    - Vascular Responses : vasodilation and a vasoconstriction.
    - Blood Pressure regulation: Cardiac output will eventually deplete

Stages of shock

1. Compensatory (Chart on 315 Brunner)
    a. SNS causes vasoconstriction
    b. BP within normal limits
    c. Clinical signs : HR > 100, Resp >20, Skin cold, clammy, Urinary output decreased, confused, respiratory alkalosis (compensatory)
        i. Serum sodium and blood glucose are elevated (aldosterone and catecholamine release)
        ii. Pulse pressure within normal limits **(30-40 mm Hg)**

d. Nurse must systematically assess the patient at risk for shock to recognize the subtle clinical signs of the compensatory stage BEFORE the BP drops.

2. Progressive
    a. Mechanism that regulates BP can no longer compensate, MAP falls below normal
        i. BP < 90 or decrease of 40 systolic below patient's norm.
        ii. Vasoconstriction continues
    b. Mechanical ventilation may be needed, respirations are rapid and shallow
    c. BNP can be used to assess ventricular function in patients in shock states
    d. When the MAP falls below 70, glomerular filtration cannot be maintained, changes in renal function occur.
        i. Increase in BUN and serum creatinine levels
    e. Decreased blood flow to the liver: AST and ALT and bilirubin levels elevated
    f. DIC may occur as a cause or complication of shock
3. Irreversible Stage
    a. Usually not diagnosed until death occurs, treatment same as progressive
    b. At this point organ damage is so severe the patient will not survive.

Shock Management

1. Early identification and timely treatment: identify and treat the underlying cause
    a. The sequence of events for different types of shock will vary.
2. Support the respiratory system
    a. Oxygenation is first line treatment
3. Fluid replacement to restore intravascular volume
    a. IV crystalloids commonly used: 0.9% sodium chloride, Lactated ringers
        i. Lactated ringers contains lactate ion that is converted by the body to bicarbonate, which helps buffer acidosis that occurs with shock
    b. Colloids (usually albumin, a plasma protein) expand intravascular volume by exerting oncotic pressure, pulling fluid into the intravascular space.
        i. Synthetic colloids (Hetastarch, and dextran) can be used but dextran may interfere with platelet aggregation and is not indicated if hemorrhage is the cause of hypovolemic shock.
    c. Blood transfusion : the need for blood is based on the patient's oxygenation needs
    d. Complications can occur when fluid is replaced too rapidly: the nurse must monitor the patient closely.
        i. Cardiovascular overload, pulmonary edema (watch for SOB, Crackles, JVD, elevated BP)
        ii. Monitor hemodynamic pressures, vital signs, arterial blood gases, serum lactate levels, hemoglobin and hematocrit levels, and I&Os. Temp. should be monitored closely (hypothermia from cold fluids)
4. Vasoactive medications : should be administered through a central line (brunner p321)
5. Nutritional support (3000 cal per day needed)

a. Administration of glutamine
b. H2 blockers or proton pump inhibitors.

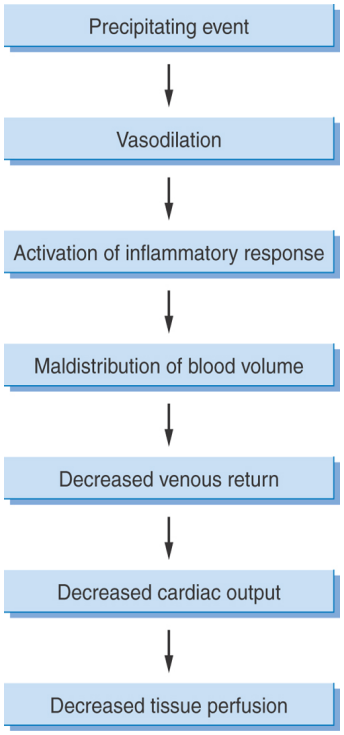
6. Hypovolemic shock : most common type : Decreased Blood Volume
    a. Occurs with 15% - 30% reduction in intravascular volume (about 750-1500 ml of blood in a 70kg person)
    b. Elderly patient: dehydration may be the cause
    c. Treatment
        i. Restore intravascular volume
        ii. Insulin if dehydration secondary to hyperglycemia
        iii. Modified Trendelenburg
        iv. Desmopressin (DDAVP) for diabetes insipidus: decreased vasopressin in the body and therefore increased urine output
7. Cardiogenic Shock:
    a. Coronary cardiogenic shock is more common than non-coronary, and is seen most often with an MI, specifically, anterior wall MI because of the potential damage to the left ventricle. (pump is impaired)
    b. Non-coronary cardiogenic shock is related to conditions that stress the myocardium: severe hypoxia, acidosis, hypoglycemia, hypocalcemia, tension Pneumothorax, cardiomyopathies, valvular damage, cardiac tamponade, dysrhythmias.
    c. Treatment:
        i. Oxygen first line
        ii. Pain control
            1. Morphine: drug of choice
                a. Pain relief, dilates blood vessels reducing cardiac workload by decreasing preload and afterload. Also decreases anxiety.
        iii. **Vasoactive meds** – multiple strategies: improve contractility, decrease preload and afterload, stabilize rate and rhythm.
            1. Used when fluid therapy does not maintain MAP
            2. Given through a central line.
            3. Titrated through the patient's response (hemodynamic monitoring through an invasive catheter)
            4. Most common: dobutamine, nitroglycerin, and dopamine.
            5. Dobutamine: increase strength of contraction, improves stroke volume, and cardiac output
            6. Epinephrine: increase contractility, rate
            7. Nitroglycerin: venous dilator – reduces preload and at higher doses causes arterial vasodilation and thereby reduces afterload.
            8. Dopamine: sympathomimetic agent with varying effects dependant on dosage. 2 – 8 micrograms/kg/min improve contractility (low dose for renal (2micrograms/kg/min). Higher than 8 micrograms/kg/min cause vasoconstriction which increases afterload this increases cardiac workload. (high doses(10mcg/kg/min) used to increase cardiac output)

a. With severe metabolic acidosis, dopamine is not as effective.

9. Vasopressors (neo-synephrine) – increase blood pressure by constriction of blood vessels. (P322)
10. Anti arrhythmics
    a. Atropine
    b. Amiotarone.

d. Nursing Management
    i. Monitor hemodynamic and cardiac status.
    ii. Look for adventitious breath sounds, changes in cardiac rhythm and other abnormal assessment findings.
    iii. IMPORTANT: monitor for decreased BP after admin morphine and nitro
    iv. IMPORTANT: neurologic assessment after admin of thrombolytic therapy.
    v. Check the neurovascular status of the lower extremities frequently when patient is on intra-aortic balloon counterpulsation.

8. Circulatory Shock: when blood pools in peripheral blood vessels. [decreased venous return]
    a. Varied mechanism can lead to three sub-types
        i. Septic Shock: most common – caused by widespread infection
            1. Reduced by using strict infection control practices
            2. Early stage BP may be within normal limits, or the patient may be hypotensive, but responsive to fluids
            3. Signs of hypermetabolism include increased serum glucose and insulin resistance.
            4. GOALS: identify and treat early sepsis within 6 hours to optimize outcome.
            5. Confusion may be the first sign in elderly patients
            6. Identify the source of sepsis
            7. SIRS – systemic inflammatory response syndrome: unknown source



        ii. Neurogenic shock
            1. IMPORTANT: elevate and maintain the head of bed at least 30 degrees to prevent neurogenic shock when patient receives spinal or epidural anesthesia. (brunner p331) Muscle tone effected. (Fracture above T-6)
            2. Restore sympathetic tone
        iii. Anaphylactic shock
            1. Assess all patients for allergies or previous reactions to antigens

9. Multiple organ dysfunction syndrome (MODS)
    a. Frequently occurs toward the end of the continuum of septic shock
    b. Insidious onset

c. Organ failure usually begins with the lung.
d. Patient experiences progressive dyspnea and respiratory failure requiring intubation and mechanical ventilation.
e. Management
    i. Prevention is the top priority in managing MODS
    ii. Supporting the patient
    iii. Motoring organ perfusion
    iv. Providing information to the patient
    v. Support the family
        1. Anxiety, support coping, patient and family education, communication, end-of-life issues, grief processes

Disseminated Intravascular Coagulation

-Not a disease but a sign of an underlying condition. Triggered by sepsis, trauma, cancer, shock, abruptio placentae, toxins, or allergic reactions, Severity is variable; may be life threatening

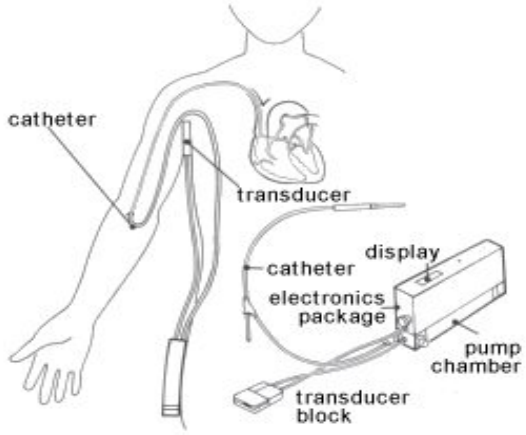
1. Initiates the process of coagulation within vasculature.
    a. Massive amount of tiny clots form in the microcirculation.
    b. As the platelets and clotting factors are consumed to form microthrombi, coagulation fails. Therefore a paradoxical result of excessive clotting is bleeding.
    c. Mortality rate can exceed 80% in patients who develop severe DIC with ischemic thrombosis and frank hemorrhage.
        i. Patients with frank DIC may bleed from mucous membranes, venipuncture sites, and the GI and urinary tracts.
    d. Treat underlying cause
    e. Treat ischemia
    f. Replace fluids, correct electrolyte imbalances
    g. Xigris for 96 hours (thrombolytic enzyme)
    h. Heparin for thrombosis process

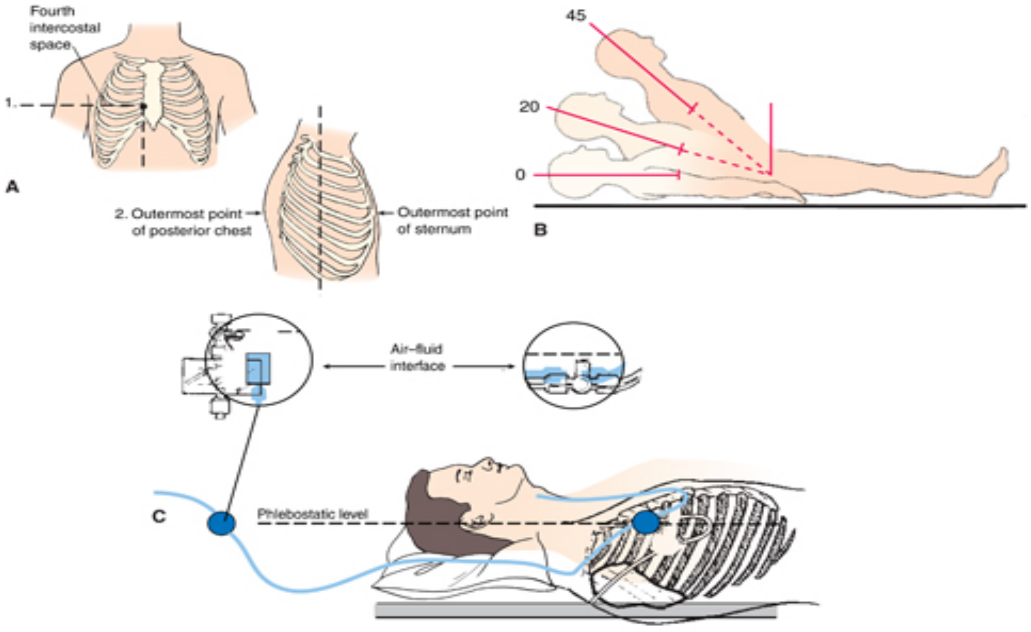
Hemodynamic Monitoring : how is the heart functioning

1. Determines cardiovascular assessment
2. Primary purpose of invasive monitoring is the early detection, identification, and treatment of life-threatening conditions, such as heart failure.
    a. Can also be used to monitor certain meds
3. Types
    a. CVP: central venous pressure (2-6mm Hg)
        i. Measurement of pressure in the Vena Cava, or right atrium.
        ii. If greater than 6mm Hg = heart failure
        iii. If less than 2mm Hg = hypovolemia

b. Pulmonary artery pressure
    i. Use “Allan Test” to determine arterial blood flow in extremity
c. Intra-arterial BP monitoring

4. Complications of invasive pressure monitoring
    a. Pneumothorax
    b. Infection
        i. Temp, > HR, >breathing, diaphoretic, shock signs
    c. Air embolism
5. CVP inserted under sterile technique
    a. Through antecubital vein, into vena cava, right atrium
    b. Need pressure bag on an IV bag at 300
        i. Will instill 3-5 ml of saline into the catheter.





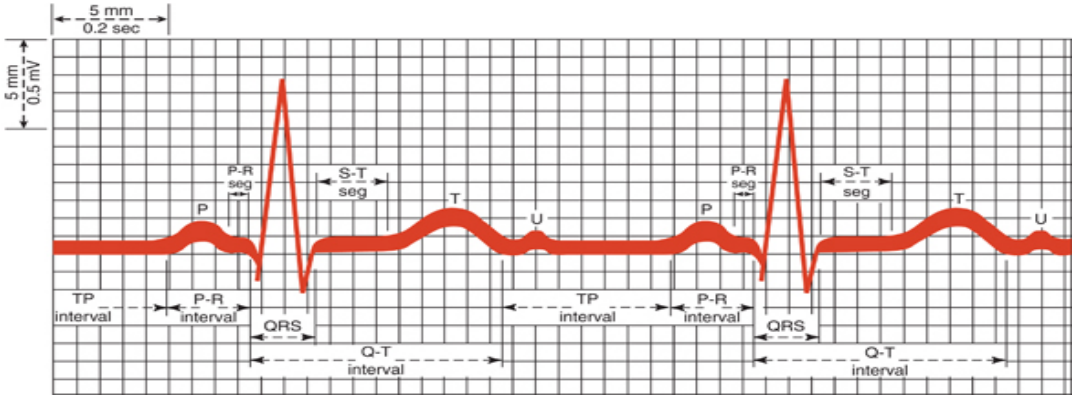
Stop-cock on transducer needs to be at the phlebostatic level. (zero point)

Cardiac dysrhythmias

-Dysrhythmias: disorders of the formation or conduction of the electrical impulses in the heart

- These disorders can cause disturbances of
    - Rate

- Rhythm
- Both



25mm per second

0.04 per little box, 0.20 per big box

1500 little boxes per minute, 300

R – R = ventricular rhythm

P – P = atrial rhythm

Impulse vs Conductivity

1. Alteration of the impulses
    a. Tach or Brady
2. Conduction
    a. Heart blocks

A. Atrial flutter – usually a regular rate
    a. Hypothyroid, chronic CHR are at higher risk for A – flutter
    b. Treated with adenosine: then rapid flush then raise arm
    c. Decreased cardiac output – decreased oxygen to tissues
        i. Ischemia, shock

B. Atrial Fibrillation
    a. Increased chance of blood clots
    b. Unstable hemodynamically
    c. Caused by alcohol, electrolyte imbalance, drugs (cocaine)
        i. Electrocution, burns (cause electrolyte imbalance)
    d. Treatment
        i. Cardio conversion – synchronized
        ii. Amiodarone, verapamil, digitalis, quinidine, warfarin

C. Premature Ventricular Contraction – impulse starts in and conducts through the ventricle
    a. Dig Toxicity, acidosis, sleep apnea are more apt for PVCs

b. Amiodarone

D. Ventricular Tachycardia
   a. Treat the cause
   b. Amiodarone is the drug of choice.
   c. Faster than 100 BPM

E. Ventricular Fibrillation
   a. Most common dysrhythmia for cardiac arrest
   b. Rapid, disorganized, ventricular rhythm
   c. CPR until defibrillator is available

F. Asystole
   a. CPR, Intubate, IV Access
   b. After 2 minutes of CPR, Epi, Vasopressin, Atropine

G. First Degree AV Block
   a. Conduction slowed through the AV node
   b. No treatment if not symptomatic

H. Second Degree AV Block
   a. Some impulses do not make it through the AV node

I. Third Degree AV Block
   a. No impulses make it through the AV node
   b. Atropine
   c. Pacemaker

J. Nursing Process: Care of patient with dysrhythmia
   a. Assessment
      i. Apical HR, vitals, LOC, rate and rhythm, heart sounds, breath sounds
      ii. Check for JVD
      iii. Brunner P 728 for medication list
         1. Amiodarone, Betapace, atropine, Lopressor

K. Implanted Transvenous pacemaker
   a. ECG on-demand pacing
   b. Complications
      i. Infection, bleeding or hematoma formation, dislocation of the lead, skeletal muscle or phrenic nerve stimulation, cardiac tamponade, malfunction
      ii. Pain at pacemaker site & increased WBCs

L. Defibrillation
   a. Biphasic – current goes back to the paddles

M.

Increased Intracranial Pressure

P 1587, 1864-1869, 1923-1924, 1977-1978

1. Pathophysiology : change in pressure due to increased fluids, or solids in the intracranial vault

a. Increase 80% brain, 10% blood, 10% csf
    i. Brain, Blood, CSF
    ii. Maintain SAO2 at 93% and higher
b. Monroe/Kelly hypothesis: if one is altered, the other needs to compensate.
c. Normal pressure 0-10 mm Hg, top normal 15mm Hg

2. Etiology
    a. Brain Trauma
    b. Brain tumor
    c. Intracranial Hemorrhage
3. Signs and symptoms *Change in LOC is the first sign ABC then ABG (confusion or increased drowsiness has neurological significance)*
    a. Restlessness
    b. Irritability
    c. Decrease in LOC
    d. Hyperventilation
    e. Pupil Changes
    f. Late signs
        i. Posturing
            1. Decorticate: hands in toward body: legs internally rotated, arms adducted
            2. Decerebrate: Wrists and fingers flexed, forearms pronated, elbows extended, arms adducted. Feet plantar flexed.
        ii. Increased BP, Decreased pulse, Decreased respiration
        iii. Temp increase
    g. Cushing's Response: occurs when cerebral blood flow is significantly decreased.
        i. Widening of pulse pressure
        ii. Slowing of the heart
        iii. Increase in systolic BP
        iv. CUSHINGS TRIAD: Bradycardia, Hypertension, Bradypnea
    h. Diabetes insipidus from decreased secretion of antidiuretic hormone
4. ICP Monitoring
    a. Ventriculostomy: sensor placed into the ICP for constant monitoring (some meds)
        i. Can remove fluid
        ii. Inserted into the lateral ventricle.
        iii. Biggest complication: infection
        iv. Can collapse ventricle
    b. Subarachnoid bolt – less invasive
        i. Complications include infection & blockage by clot.
5. Treatment
    a. Mannitol: osmotic diuretic
        i. Pulls water out, reduces pressure
        ii. Crosses blood/brain barrier easily

b. Hypertonic Saline
c. Glucocorticoids : Decreases inflammation (Solu-Medrol , Decadron(meningitis))
d. Hyperventilation : push toward alkalosis want PACO2 between 26 and 30
e. Barbiturates: sedation -
f. Therapeutic hypothermia
g. Decompressive Craniectomy
h. Removal of CSF

6. Nursing Diagnosis
    a. Ineffective airway clearance related to diminished protective reflexes
    b. Ineffective breathing patterns related to neurological dysfunction
    c. Ineffective cerebral tissue perfusion related to effects of increased ICP
    d. Deficient fluid volume related to fluid restriction
    e. Risk for infection related to ICP monitoring system
7. Interventions
    a. Suction airway secretions
        i. Coughing is discouraged!
    b. Elevating the head of bed to aid in clearing secretions and improve venous drainage of brain.

Seizures

Sudden, abnormal, and excessive electrical discharges from the brain that can change motor or autonomic function, consciousness, or sensation.

1. Partial: Begin on one part of the brain
    a. Begin on one side of cerebral cortex
    b. Types
        i. Simple partial seizures
        ii. Complex partial seizures
2. Generalized
    a. Both hemispheres involved
    b. Convulsive or non-convulsive (generally 1-2 minutes)
3. Etiology
    a. Idiopathic: no cause identified
    b. Acquired
        i. Underlying neurological disorder
        ii. Brain injury
            1. Alcohol withdrawal, head injury, DKA, renal failure, Low Ca, NA, sugar.
            2. Brain tumors
            3. Allergies
4. Signs and symptoms
    a. Aura

i. Visual distortion, odor, sound

b. Partial seizures

i. Automatisms – nor responsive to consciousness
ii. Maintains consciousness
iii. Usually < 1 minute
iv. Paresthesias
v. Visual disturbances

c. Complex partial

i. Loss of consciousness

d. Generalized Seizures

i. Absence (petit Mal)
    1. Staring
ii. Tonic clonic
    1. May have aura
    2. Usually lose consciousness
    3. Rigidity followed by muscle contraction and relaxation
    4. Incontinence
    5. Postictal period

5. Diagnosis
    a. Diagnostic assessment to determine type
        i. Frequency
        ii. Severity factors that precipitate
    b. EEG
    c. Look for underlying cause
    d. Medical history
        i. Injuries
        ii. Pregnancies or births
        iii. Illness
6. Therapeutic interventions
    a. Correct cause
    b. Anticonvulsant medication
    c. Surgical resection
7. Emergency Care: seizure
    a. Pad side rails
    b. Prevent injury
    c. Monitor airway
    d. Do not restrain
    e. Turn on side to prevent aspiration
    f. Suction PRN
    g. Observe and document
8. Status epilepticus
    a. 30 minutes of seizure activity

b. Therapeutic interventions
    i. Ensure airway and oxygenation
    ii. Administer IV diazepam, or Ativan, Fosphenytoin.

9. Antiseizure medications *P1885 Table 61-4*
    a. Carbamazepine (Tegretol)
    b. Clonazepam (Klonopin)
    c. Ethosuximide (Zarontin)
    d. Felbamate (Felbatol)
    e. Gabapentin (Neurontin)
    f. Lamotrigine (Lamictal)
    g. Levetiracetam (Luminal)
    h. **Phenytoin (Dilantin) –** *gum problems (gingival hyperplasia) good oral care is critical*
    i. Primidone (Mysoline)
    j. Tiagabine (Gabitril)
    k. Topiramate (Topamax)
    l. Valproate (Depakote)
    m. Zonisamide (Zonegran)
10. Epilepsy
    a. Unprovoked, recurring seizures
    b. Primary or secondary
    c. Dx EEG
11. Long term anti seizure medications are at risk for osteoporosis
    a. High incidence, new onset in aging patients
    b. Cerebrovascular disease leading cause of seizures in elderly.
    c. Financial considerations of medications.
    d. Use one pharmacy (they know the patients)
12. Diagnosis
    a. Risk for injury
    b. Risk for ineffective management of therapeutic regimen
    c. Fear related to possibility of a seizure
    d. Deficient knowledge related to epilepsy and its control.
13. Expected outcomes
    a. Sustain no injury during seizure activity
    b. Indicates a decreased fear
    c. Displays effective individual coping
    d. Exhibits knowledge and understanding of epilepsy (identifies the side effects of meds, avoids factors that may cause seizure, follows healthy lifestyle.)
14. Nursing Management
    a. MAJOR RESPONSIBILITY: observe and record the sequence of signs
        i. Ie. Circumstances, aura, first action, type or part of body, pupils, presence or absence of involuntary motor activity, incontinence, duration of each phase, unconsciousness, post seizure actions.

b. Patient safety

15. Planning and goals: prevention of injury (primary), control of seizures.

Traumatic Brain Injury

1. Trauma
    a. Hemorrhage
    b. Contusion
    c. Laceration
2. Can cause
    a. Cerebral edema
    b. Hyperemia
    c. Hydrocephalus
    d. Brain herniation
    e. Death
3. Etiology
    a. MVA most common
    b. Falls
    c. Assaults
    d. Sports related injuries
4. Mechanism of injury
    a. Acceleration
    b. Deceleration
    c. Acceleration-deceleration
    d. Rotational
5. Types of Injury
    a. Concussion
    b. Contusion
    c. Hematoma
    d. Subdural – fast change of velocity usual cause
    e. Epidural – between dura matter and skull
6. Diagnosis is done with a CT or MRI
    a. CT is quicker
    b. Treatment is based on the rate of growth.
    c. Neuropsychological testing
7. Therapeutic Intervention
    a. Surgical Removal of hematoma
    b. Control ICP
        i. ICP monitoring
        ii. Osmotic diuretic (mannitol)
        iii. Mechanical hyperventilation
    c. Therapeutic coma
    d. Three
        i. ACUTE – ABCs
        ii. Subacute – in hospital (rehab)
        iii. Chronic – long term, placement or rehabilitation

8. Complications of TBI
    a. Brain herniation
    b. Diabetes insipidus (inadequate release of ADH due to damaged pituitary)
    c. Acute hydrocephalus
    d. Labile vital signs
    e. Posttraumatic syndrome
    f. Seizures
    g. Brain infections
    h. Cognitive and personality changes
9. Nursing Diagnosis
    a. Ineffective airway clearance and impaired gas exchange related to brain injury
    b. Ineffective cerebral tissue perfusion related to increased ICP, decreased CPP
    c. Deficient fluid volume related to decreased LOC and hormonal dysfunction
    d. Imbalanced nutrition, less than body requirements related to increased metabolic demands, fluid restrictions, and inadequate intake.
    e. Risk for injury related to seizures, disorientation, brain damage
    f. Disturbed thought process

Brain Tumors

1918, 1933-1947, 1881-1889 (Brunner). London 1688-1689

1. Gliomas – most common
2. Meningiomas
    a. Usually benign
3. Acoustic neuromas
    a. Always on the eighth cranial nerve
4. Pituitary adenomas and angiomas
    a. 10-15% of all tumors
    b. 83% on cerebellum (balance and motion)
5. Signs and symptoms
    a. Seizures
    b. Motor and sensory deficits
    c. Headaches
    d. Visual disturbances
    e. Vomiting
    f. Hormone disturbances
        i. Papilledema (edema of the optic disk)
6. Diagnosis
    a. MRI, CT, Angiogram, MRA, Hormone levels
7. Therapeutic interventions
    a. Surgical removal
    b. Radiation therapy
    c. Chemotherapy
    d. Symptom control
        i. Anticonvulsants
        ii. Dexamethasone or prednisone

iii. Mannitol

8. Nursing Diagnosis
    a. Disturbed thought processes
    b. Self care deficit
    c. Pain
    d. Sensory perceptual disturbance
    e. Impaired physical mobility
    f. Risk for injury
9. Intracranial Surgery
    a. Indications
        i. Hematoma
        ii. Tumor
        iii. AV malformation
        iv. Trauma
        v. Seizures
10. Types of Surgery
    a. Craniotomy
    b. Craniectomy
    c. Cranioplasty
11. Preoperative Care
    a. Patient education
    b. Anxiety control
    c. ICU visit (preplan their visit)
12. Postoperative Care
    a. Nursing diagnosis
        i. Risk for ineffective cerebral tissue perfusion
        ii. Risk for infection
        iii. Body image disturbance
        iv. Deficient knowledge

Spinal Cord injury

1. Complications
    a. Infection
    b. DVT & skin breakdown
    c. Orthostatic hypotension
    d. Depression and substance abuse
    e. Autonomic dysreflexia
2. Injury see page 1936 table 63-3
    a. Deficit may appear later.
3. Diagnosis
    a. X-Ray
    b. CT scan
    c. MRI
    d. EMG later on (Electro Myogram) –determines the level of injury
4. Emergency Management
    a. ABCs then ABGs
    b. Gastrointestinal

c. Genitourinary
d. Immobilization

5. Surgical management
    a. Stabilize spine
        i. Halo
        ii. Rods
        iii. Corset
        iv. Brace
        v. Body cast
6. Spinal Shock
    a. Occurs immediately after the injury
    b. Peripheral vasodilation
        i. Normal to low heart rate
    c. Pulmonary related problems are the most common cause of mortality
        i. More prone to infections
        ii. The higher up the injury the greater the respiratory threat
        iii. Accessory muscles to breath.
        iv. C-4 and higher usually needs a mechanical ventilator
    d. SNS disruption
        i. Vasodilation
        ii. Hypotension
        iii. Bradycardia
        iv. Hypothermia
        v. Urine and feces retention
    e. Autonomic dysreflexia: Life threatening emergency in spinal cord injured patients that causes a hypertensive emergency.
        i. Usually from an injury above T-6
        ii. Bowel and bladder may cause alterations
        iii. Hemorrhage , stroke, seizure and arrest
        iv. Sudden and severe pounding headache, profuse sweating, nasal congestion, piloerection (goose bumps), bradycardia, hypertension.
        v. Diaphoresis and flushing above the injury with pallor or coolness below the level of the injury
        vi. Dramatic drop of pulse with dramatic increase in BP
    f. Look at stability to swallow and deficit
    g. Muscles for atrophy
    h. Diagnosis
        i. Self care deficit
        ii. Risk for impaired skin integrity
        iii. Risk for ineffective role-performance
        iv. Risk for sexual dysfunction
        v. Anxiety

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Congestive Heart Failure

1. Congestive Heart Failure
    a. Cardiac output is inadequate to support the body's needs
    b. 90% of infants with uncorrected heart defects develop CHF – first 6-12 months
    c. Manifestations : subtle at first
        i. Tires easily (especially during feeding)
        ii. Weight loss, or lack of normal gain, irritability, frequent infections, diaphoresis.
        iii. Older children: exercise intolerance, dyspnea, abdominal pain or distention, peripheral edema
    d. Progression leads to tachypnea, tachycardia, pallor, cyanosis, nasal flaring, cough, crackles. Periorbital and facial edema and hepatomegaly (fluid excess).
    e. Cardiomegaly : enlargement of the heart by hypertrophy of its walls.
    f. Goals of medical management are to make the heart work more efficiently to remove excess fluid.
        i. Diuretics are given along with inotropic and afterload reducing medications.
        ii. Digoxin: increase cardiac contractility
        iii. Furosemide: rapid diuretic
        iv. Thiazides: diuretics (monitor potassium)
        v. Spironolactone: potassium sparing diuretic
        vi. Propranolol: increases contractility
        vii. Carvedilol: Improves left ventricle function, promotes vasodilation of systemic circulation for chronic heart failure and dilated cardiomyopathy.
    g. Nursing assessment
        i. Assess vital signs, behavior patterns, cardiac function, respiratory function, and fluid status.
        ii. History of previous hospitalizations
        iii. Developmental assessment
    h. Nursing diagnosis
        i. Decreased cardiac output related to cardiac anomaly (primary)
    i. Implementation
        i. Group assessment and interventions together to ensure the child has uninterrupted rest each hour.
        ii. Feeding no longer than 20 to 30 minutes. Frequent small feedings are best
        iii. Position baby in baby seat at 45 degrees decreases venous return and lowers metabolic demand.
2. Digoxin + Inotrope : increases force, slows HR
    a. Before giving: baseline vitals, quality of peripheral pulses, clinical symptoms, ECG, check serum electrolytes, hepatic function, and renal function. Assess hydration status and hydrate if hypovolemic.

i. Apical pulse for one minute: withhold if bradycardic
ii. Observe for dig toxicity

Congenital Heart Disease

- Defect in the heart or great vessels, or persistence of fetal structure after birth.
- Occurs in approximately 1% of all pregnancies and 1 in 170 live births
- Most occur during the first 8 weeks of gestation
    - Fetal exposure to drugs such as phenytoin, lithium and alcohol
    - Maternal viral infections of Rubella and Coxsackie B5
    - Diabetes mellitus and hypercalcemia
    - Mother of increased age and antepartal bleeding
    - Genetic factors
    - Chromosomal abnormalities: 22q11 most frequent site
        - Turner Syndrome
        - Noonan Syndrome
        - Marfan syndrome
        - DiGeorge syndrome
- Nursing Diagnosis
    - Delayed growth and development related to effects of physical disability
    - Ineffective therapeutic regimen management related to complexity of therapeutic regimen
    - Imbalanced nutrition: less than body requirements related to chronic illness and tiring while feeding.
    - Activity intolerance related to poor cardiac output
    - Caregiver role strain (parent) related to 24-hour responsibility for child's care

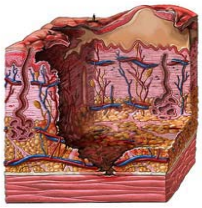
1. Congenial heart defects categorized by pathophysiology and hemodynamics. : presence of a heart murmur is often the first indicator
    a. Increased pulmonary blood flow
        i. Tachypnea, tachycardia, murmur, CHF, poor weight gain, diaphoresis, periorbital edema, frequent respiratory infections
    b. Decreased pulmonary blood flow
        i. Cyanosis, hypercyanotic spells, poor weight gain, polycythemia
    c. Obstructed systemic blood flow
        i. Diminished pulses, poor color, delayed cap refill, decreased urine output, CHF with pulmonary edema
    d. Mixed defects
        i. Cyanosis, poor weight gain, pulmonary congestion, CHF may occur

BEERS High risk for elderly

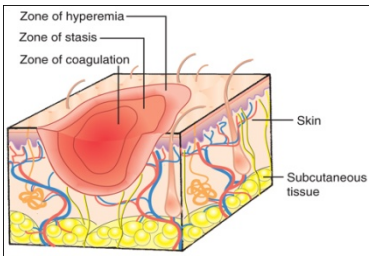
Antihistamines, benzodiazepines, NSAIDs, Digitalis, Tricyclic antidepressants.

Burn Injuries

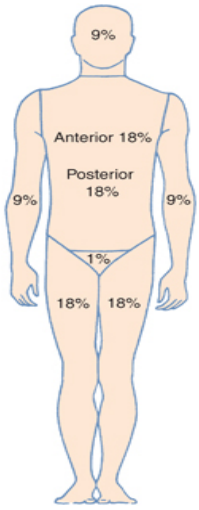
1. Third leading cause of death in children age 1 to 9 years old (most in home)
2. Elderly are also at high risk
3. Classification of Burns
    a. Superficial partial thickness (1st Degree)
        i. Epidermis
        ii. Red, blanches with pressure, dry, usually minimal or no edema.
        iii. Possible blisters (do not pop)
    b. Deep Partial thickness (2nd Degree)
        i. Edema (blisters)
        ii. Mottled red appearance, surface usually weeping
        iii. Very painful
    c. Full Thickness (3rd Degree)
        i. Epidermis, dermis, subcutanious, muscle.
        ii. Dry, pale white, leathery, broken skin, sometimes fat exposed.


3rd degree burn

4. Zones
    a. Zone of coagulation (zone of death)
    b. Zone of statis (comprimised blood supply)
    c. Zone of hyperemia (sustains least damage)



5. Factors to consider in determing burn depth
    a. How did the injury occur
        i. Causitive agent (thermal, chemical, electrical, radiation)
    b. Temperature of the agent
    c. Thickness of the skin
    d. Burn injury usually results in energy transferred from a heat source to the body.
6. Area Measurement: Rule of 9s (most commonly Used)
    a. Lund and Bowder method – most accurate
        i. Revised 72 hours later : demarcation is seen more clearly



7. Types of burns
    a. Thermal
    b. Chemical (acids, ammonia)
        i. Contact time
    c. Electrical
        i. Type of current, amount, pathway, how long they touched it, area of burn.
    d. Radiation
8. Physiologic changes.
    a. Burns less than 25% TBSA (total body surface area) produce a primarily local response

b. Burns more than 25% may produce a local and systemic response and are considered major burns.
c. Systemic response includes release of cytokines and other mediators intot he systemic circulation
d. Fluid shifts and shock result in tissue hyoperfusion and organ hypofunction
    i. Hemodynamic instability results from loss of capilary integrity
    ii. Sodium and potasium shifts
    iii. Fluid resusitation is extremely important

9. Effects of major burn injury
    a. Fluid and electrolyte shiftsw
    b. Cardiovascular effects
        i. Hypovolemic : hypotissue perfusion
            1. Dysruptive shock (hypovolemic and septic, and/or neurogenic)
        ii. Fluids to keep systolic pressure above 100
        iii. Peripheral vaso constriction (decreased cardiac injury)
    c. Pulmonary injury
        i. Upper airway
        ii. Inhalation below the glottis
            1. Enclosed space, burn of face, singed nasal hair, hoarseness,
        iii. Carbon monoxide poisoning
        iv. Restrictive deficits
        v. Chest X-ray, ABG, Pulse Ox
    d. Renal and GI alterations
        i. Myoglobulin excreted
        ii. 20% or higher burns needs a nasogastric tube (higher risk of nausea, stress ulcers, risk of aspiration)
    e. Immunologic alterations
        i. Needs meticulous skin care to prevent infection
    f. Thermoregulation
        i. First they are hypothermic, than core body temperature goes up.
            1. Due to inflammatory response, lack of cooling ability, inability to regulate

10. Phases of burn injury
    a. Emergent or resuscitative phase
        i. Onset of injury to completion of fluid resuscitation
        ii. Prevent injury to rescuer
        iii. Stop injury: extinguish flames, cool the burn, irrigate chemical burn
        iv. ABCs
        v. Start oxygen, large bore IVs, Remove restrictive objects and cover the wound, assessment: surveying all body systems, and obtain a history of the incident and pertinent patient history.
        vi. Pt brought to the ER : 10% full thickness – burn center

1. Fluid resuscitation is begun
2. Foley catheter is inserted: renal perfusion, color of urine, glycourine (high levels of sugar in urine)

vii. Electrical burns – ECG
viii. Address pain: only IV medication only, Morphine drug of choice.
ix. Psychosocial consideration and emotional support needed for patient and fam.

b. Acute or intermediate phase
   i. 48-72 hours after injury
   ii. Continue assessment and maintain respiratory and circulatory support
      1. Fluid shifts as capillaries try to regain their integrity
      2. Patient at higher risk of CHF
      3. Fever – result of the body trying to regulate itself: maintain body temperature between 99-101 when hypothermic, then temp develops and cooling will be necessary.
c. Rehab phase
   i. Up to two years after discharge
   ii. Focus is upon wound healing, psychosocial support, self-image, lifestyle, and restoring maximal functional abilities so the patient can have the best quality life.
   iii. Reconstructive surgery

11. Management of shock: fluid resuscitation
    a. Maintain BP above 100 systolic, urine outputs of 30-50 ml/hr. Maintain serum sodium at near normal levels. Need CVP line
    b. Hypertonic saline (3% NS) 4ml/kg/hr
    c. Parkland Baxter Formula
       i. 2- 4 (ml/kg) x %TBSA) ½ fluid in first 8 hours, rest over next 16 hours
    d. Fluid and Electrolyte shifts (emergent phase)
       i. Generalized dehydration
       ii. Reduced blood volume and hemoconcentration
       iii. Decreased urine output
       iv. Trauma causes release of potassium into extracellular fluid: hyponatremia
12. Burn Wound Care
    a. Wound cleaning
       i. Hydrotherapy : Room 80-85 degrees Water 100 degrees
    b. Topical agents
       i. Silver containing topicals are highly effective.: watch for leukopenia (decreased WBC)
    c. Wound debridement
       i. Natural – separates naturally (sometimes occur with topical agents)
       ii. Mechanical debridement
          1. Removal of necrotic eschar (black necrotic area) with scalpel
       iii. Chemical – silver

iv. Surgical debridement
    1. Operative procedure of excising the injured area.

d. Wound dressing, dressing changes, and skin grafting
    i. Silverlon – cannot be used on the face
    ii. Mepitel – goes over the burn, another dressing goes overtop
    iii. CEA – cultured

13. Pain management
    a. Burn pain: most severe forms of acute pain
    b. Breakthrough – episodic and severe (activity)
    c. Background – resting – intense
    d. Procedural
    e. Drugs
        i. Morphine, Versed, Percocet,
    f. Non pharmacological measures
14. Nutritional support
    a. Burn injuries produce profound metabolic abnormalities. Patients with burns have great nutritional needs related to stress response, hypermetabolism, and wound healing.
    b. Goal of nutritional support is to promote a state of nitrogen balance and match nutrient utilization.
    c. Nutritional support is based on patient's preburn status and % of TBSA burned.
    d. Enteral route is preferred. Jejunal feedings are frequently used to maintain nutritional status with lower risk of aspiration in a patient with poor appetite, weakness, or other problems.

Transfusion

1. Infusion of blood products :
    a. Is it needed? H&H
2. Whole Blood
    a. Used to treat shock, low blood volumes, low hematocrit and hemoglobin, hemorrhage.
    b. Only 1% is platelets, need 180 donors for 1 bag of platelets.
3. Donation: every 56 days.
4. Demand is most in summer and holidays.
5. Packed RBCs – most common
    a. Treats anemia, reduce risk of volume overload.
    b. Can be refrigerated for 42 days.
    c. About 220ml in a bag. Transfused in less than 4 hours.
6. Fresh frozen plasma
    a. Used to restore plasma volume, treat some bleeding problems
    b. Thawed in the lab and good for 24 hours
7. Platelets
    a. Maintain normal coagulability of blood

b. Used to treat some bleeding disorders, and to compensate when marrow can not produce enough
c. Platelets: 80-60ml/pack: usually 4-6 packs are pooled for transfusions, infuses as quickly as the pt tolerates.
d. Steroids increase production of blood.

8. Cryoprecipitate
    a. 10-15ml bag, usually 10 bags pooled.
9. Type undergoes type and crossmatch
    a. Blood type, Rh, and antigens
    b. Antigen: substance that prompt the generation of antibodies and can cause an immune response.
10. Alternatives
    a. Volume builders
        i. Crystalloids
        ii. Artificial crystalloids: Dextran
            1. Can cause bleed problems or allergic reactions
            2. They only replace volume
11. Risks
    a. Disease Transmission
        i. Hepatitis B 1:140,000
        ii. Hepatitis C 1:225,000
        iii. Hepatitis A 1:1 million
        iv. HIV 1:1.5 million
        v. Syphilis 1:1 million
    b. Bacterial contamination
    c. Acute or delayed transfusion reactions
        i. Mismatched ABO 1:35,000
        ii. Incompatible death rate 1:600,000
    d. Circulatory overload
12. Infusion
    a. Each unit of blood currently undergoes tests for nine diseases
    b. Bacterial contamination is very rare, but may occur at any point
        i. Refrigeration helps prevent bacterial growth
    c. Transfusion reactions
        i. Allergic reactions, incompatibilities, anaphylactic response to plasma proteins
    d. Some risks specific to massive transfusion (more than 1 unit in 24 hours)
        i. Hypothermia
        ii. Hemodilution
        iii. Platelet dysfunction
        iv. Electrolyte problems (Calcium-low) starts in fingers
            1. Iron overload
13. Noninfectious serious hazards

a. Mis-transfusion and ABO/Rh incompatibility

14. Administration
    a. Assess transfusion history
        i. Previous transfusions, allergies and reactions
        ii. Type of transfusion reaction, manifestations.
    b. Blood must be transfused within 30 minutes.
        i. Positive patient identification
        ii. Appropriateness of blood component
        iii. Blood product inspection
        iv. Verification of donor-recipient compatibility
        v. Verification of product expiration date

A can get A and O

B can get B and O

O can get O