Early Childhood Poisoning

- Heavy Metal Poisoning
  - Most common is lead ingestion
  - Mercury toxicity (less frequently)
- Lead Poisoning
  - Most common by peeling lead-based paint
  - Micro particles of lead contaminate bare soil
  - Can be inhaled or ingested
  - Affects renal, hematologic, and neurologic systems; developing brain and nervous systems are especially vulnerable
- Lead Poison Diagnosis
  - Rarely symptomatic
  - Venous blood sample of 10 mcg/dl
  - Screening for lead poisoning at ages 1 and 2 years
  - Chelation therapy with calcium disodium edetate (calcium EDTA) and succimer (DMSA)
- Prognosis
- Acetaminophen Poisoning
  - Acetaminophen toxicity is a relatively common occurrence, particularly in children, given that this drug is the most widely used analgesic-antipyretic medication taken by people in the United States and around the world.
  - Toxicity
    - Children who have acutely ingested 250 mg/kg or more of acetaminophen (APAP) pose significant concern for acetaminophen (APAP)-induced hepatotoxicity.
    - Patients who ingest more than 350 mg/kg develop severe hepatotoxicity, if they are not appropriately treated.
- Clinical Signs
  - Stage I (2-24 hrs.) N/V, sweating, pallor, anorexia.
  - Stage II (24-36 hrs.) latent period.
  - Stage III RUQ pain, jaundice, pruritis, steatorrhea, wt loss, anemia.
  - Stage IV SGOT drops and recovery
- Treatment/Prognosis
  - The proper medical use of the antidote N-acetylcysteine (Mucomyst) has significantly lowered the mortality rate. Tx of choice.
  - Use of charcoal controversial. Do not use with above.
  - Most patients do not have clinically significant sequelae if they are treated in a timely manner with antidotal therapy and appropriate supportive care.
  - In acute exposures, mortality and morbidity rates are lower in young children (≤5 y) than in older children, adolescents, and adults. The cause for this age-related difference is unclear.
  - Gastric lavage
- Hydrocarbon Ingestion
  - Gasoline, kerosene, lamp oil, furniture polish, lighter fluid, turpentine, paint thinner.
• Symptoms
• Gagging, choking, coughing
• N/V
• Alteration in sensorium, lethargy
• Weakness
• Tachypnea, cyanosis, retractions, grunting
• Danger of aspiration and pneumonia
• DO NOT INDUCE EMESIS
• Symptomatic tx of chemical pneumonia
• Corrosives
• Drain, toilet and oven cleaners
• Dishwasher detergent
• Batteries
• Bleach
• Symptoms
• Severe oropharyngeal burning.
• Swollen mucous membranes, edema of oropharyngeal structures posing threat of respiratory obstruction.
• Violent vomiting & hemoptysis
• Drooling
• Shock
• Anxiety and agitation
• Treatment
• Emesis contraindicated (redamages mucosa)
• DO NOT NEUTRALIZE (EXOTHERMIC REACTION)
• Maintain airway
• Parenteral analgesics
• NPO
• Tx of esophageal strictures in future.

Immunizations

Immunizations

Recommendations provided by:

• Advisory Committee on Immunization Practices (ACIP) of the Centers for Disease Control and Prevention (CDC)
• Committee on Infectious Diseases of the American Academy of Pediatrics (AAP)
• Current Recommendations
• http://www.cdc.gov/vaccines/recs/schedules/child-schedule.htm#hcp
• http://www.aap.org
• AAP Report of the Committee on Infectious Diseases (“The Red Book”)
• CDC Morbidity and Mortality Weekly Report (MMWR)
• VAERS and VIS
- Vaccine Adverse Event Reporting System (VAERS)
  - To report any adverse reactions after administration of any vaccine
- Vaccine information statements (VIS)
  - Information statements that must be given to parents before vaccines administered
  - Provide updated information for parent/guardian of child being vaccinated

**Infectious Diseases**

**Communicable diseases:**
- Incidence has declined with increase of immunizations
- Further decreased with use of antibiotics and antitoxins

**Nursing Assessment in Identification of Infection**
- Recent exposure to infectious agents
- Prodromal symptoms
- Immunization history
- History of having the disease
- Prevent Spread of Disease

**Primary prevention of the disease:**
- Immunization
- Control spread of disease to others:
  - Reduce risk of cross-transmission of organisms
  - Infection control policies
- Hand washing

**Caution for Compromised Children**
- Children with immunodeficiency:
  - Receiving steroid therapy
  - Other immunosuppressive therapies
  - Generalized malignancies
  - Immunologic disorder
  - Risk for complications from communicable diseases, especially varicella (chickenpox) and erythema infectiosum
  - Risk for viremia from varicella zoster virus (VZV)

**Erythema Infectiosum (Fifth Disease)**
- Agent: human parvovirus
- Rash in three stages:
  - “Slapped face” appearance disappears between 1 and 4 days
  - Maculopapular rash on extremities; lasts 7 days or more
  - Rash subsides but reappears if skin irritated or traumatized by heat, cold, friction, etc.

**Chickenpox**
- Agent: varicella zoster virus
- VZG also causes herpes zoster (shingles)
- VZIG treatment for children at risk
Transmission: direct contact, droplet, and contaminated objects
Incubation: 2 to 3 weeks
Communicability: 1 day before eruption until all lesions crusted

Fifth Disease
Roseola
Agent: human herpes virus type 6
Incubation: 5-15 days
Persistent high fever for 3-4 days; otherwise appears well
After fever subsides, rash appears
Rash first on trunk, then face and extremities

Roseola
Rubeola (Measles)
Agent: virus
Source: secretions; droplet transmission
Incubation period: 10-20 days; communicability from 4 days before to 5 days after appearance of rash
Koplik spots appear 2 days before rash

Mumps
Agent: paramyxovirus
Transmitted via droplet or direct contact
Incubation period: 14-21 days
Fever, headache, malaise, followed by parotitis
May cause orchitis and meningoencephalitis

Mumps
Pertussis (Whooping Cough)
Agent: Bordetella pertussis
Transmission: droplet or direct contact
Incubation period: 6-20 days
Short rapid coughs followed by crowing or “whoop” sound
Complications: pneumonia (usual cause of death)

Pertussis
Rubella (German Measles)
Agent: rubella virus
Transmission: direct contact or indirect contact with article freshly contaminated with nasopharyngeal secretions, blood, stool, or urine
Incubation period: 14-21 days
Complications: rare; greatest danger is teratogenic effect on fetus

Scarlet Fever
Agent: group A β-hemolytic streptococci
Transmission: droplet or direct contact
Incubation period: 1-7 days
Complications: carditis, peritonsillar abscess, glomerulonephritis
Meningitis

Intracranial Infections
CNS has limited response to injury
Difficult to distinguish etiology by looking at clinical manifestations
Laboratory studies required to identify causative agent
Inflammation can affect meninges, brain, or spinal cord

Meningitis

Bacterial Meningitis
Acute inflammation of CNS (95% of all cases >2 mo. Age)
Decreased incidence following use of Hib vaccine (meningococcal vaccine)
Can be caused by various bacterial agents:
  - *Streptococcus pneumoniae*
  - β-Hemolytic streptococci
  - *Escherichia coli*

Transmission of Bacterial Meningitis
Droplet infection from nasopharyngeal secretions
Appears as extension of other bacterial infection through vascular dissemination
Organisms then spread through cerebrospinal fluid

Clinical Manifestations
Fever, chills, headache, vomiting associated with alterations in sensorium.
May have seizures.
Extremely irritable.
May have photophobia, hallucinations
May lead to coma
Under 2 years:
  - Poor feeding
  - Vomiting
  - Bulging fontanel
  - High pitched cry
  - Frequently seizures
  - Neonates
  - Nonspecific and difficult to diagnose
  - Diarrhea
  - Poor muscle tone
  - Poor feeding/suck
  - Weak cry
  - Hypothermia or fever
  - Apnea
  - Bulging fontanel may be a late sign
  - Positive Brudzinski

With pt supine, passive flexion of neck causes involuntary bending of both knees and hips in pt >2 yrs.
Kernig’s sign
With pt supine and hips flexed, passive straightening of leg at knee causes active resistance and back pain in pts > 2 yrs.
Diagnostics: lumbar puncture is definitive diagnostic test
Therapeutic management – antibiotics, hydration, isolation, ventilation, decrease ICP, manage shock, control seizures, control temp, tx complications.
Nursing care management – quiet environment, sl. elevate HOB, VS, neuro checks, I&O, family support.
Prognosis – 10-15% fatal.

Spinal Tap/Lumbar Puncture
Sequelae most often seen in those < 2 mo.
Purpura of the Lower Extremities

Nonbacterial Meningitis (Aseptic Meningitis) (Viral Meningitis)
Causative agents are principally viruses especially enterovirus.
Frequently associated with other diseases:
Measles, mumps, herpes, leukemia
Onset is abrupt or gradual
Manifestations: headache, fever, malaise
Diagnosis and treatment - supportive
Prognosis - good

Tuberculosis Meningitis
Causes
Tuberculous meningitis is caused by *Mycobacterium tuberculosis*, the bacteria that cause tuberculosis. The bacteria spread to the brain from another site in the body.
Risk factors include a history of:
- AIDS
- Excessive alcohol use
- Pulmonary tuberculosis
- Weakened immune system

Tuberculous meningitis is a very rare disorder in the U.S.
Symptoms
The symptoms usually begin gradually, and may include:
- Fever and chills
- Mental status changes
- Nausea and vomiting
- Sensitivity to light (photophobia)
- Severe headache
- Stiff neck (meningismus)
Other symptoms that can occur with this disease:
- Agitation
- Bulging fontanelles
- Decreased consciousness
Poor feeding or irritability in children
Unusual posture, with the head and neck arched backwards (*opisthotonos*)

**Pulmonary tuberculosis**

**Seizures**

Physical examination will usually show:
- Fast heart rate
- Fever
- Mental status changes
- Stiff neck

Tests
- **Biopsy** of the brain or meninges
- **Blood culture**
- **Chest x-ray**
- CSF examination for cell count, glucose, and protein
- **CT scan of the head**
- Gram stain, other special stains, and **culture of CSF**
- Polymerase chain reaction (PCR) of CSF
- Skin test for tuberculosis (**PPD**)

Treatment involves several antitubercular drugs at the same time, as it does for **pulmonary tuberculosis**. Treatment sometimes must begin if the diagnosis is only suspected, not proved, in order to save a person's life.

- Treatment usually lasts for at least 12 months. **Systemic** steroids may also be used.

**Outlook (Prognosis)**

- Tuberculous meningitis is life-threatening if untreated. Long-term follow-up is needed to detect repeated infections (recurrences).
- Possible Complications
  - Brain damage
  - Build-up of fluid between the skull and brain (**subdural effusion**)
  - Hearing loss
  - **Hydrocephalus**
  - **Seizures**

**Prevention**

- In areas where tuberculosis is more common, the BCG vaccine may help prevent severe forms of tuberculosis, such as meningitis, in very young children.

- Treating people who have evidence of a non-active (dormant) tuberculosis infection can prevent the spread of tuberculosis. A dormant infection can be detected by a positive PPD.

**Otitis Media**

- **Otitis Media (OM)**
- Pathophysiology and etiology – inflammation of middle ear.
- **Diagnostics** – assessment of TM by pneumatic otoscopy.
• Therapeutic management
  – Pharmacologic – antibiotics, analgesics, fever control
  – Surgical – tympanostomy tubes
• Nursing considerations – feeding positions, breast feeding for 6 mo., secondhand smoke, tonsilar hypertrophy, frequent URI’s, PCV vaccine, no pacifier after 6 mo, if rupture no water in ear until healed, finish antibiotics.

Child Abuse
• Child Maltreatment
  • Intentional physical abuse (22%) or neglect (54%)
  • Emotional abuse (4%) or neglect
  • Sexual abuse of children (8%)
• Types of Neglect
  • Physical neglect:
    • Deprivation of food, clothing, shelter, supervision, medical care, and education
  • Emotional neglect:
    • Lack of affection, attention, and emotional nurturance
  • Emotional abuse:
    • Destroy or impair child’s self-esteem
• Physical Abuse
  • Deliberate infliction of physical injury on a child
• Factors Predisposing to Physical Abuse
  • Parental characteristics
  • Social isolation, poor support systems
  • Parental low self-esteem and less adequate maternal functioning
• Child Characteristics Predisposing to Physical Abuse
  • Compatibility between child’s and parent’s temperament and parent’s ability to deal with behavioral style
  • Removing child victim from home may place other siblings at risk for abuse
• Environmental Characteristics
  • Chronic stress
  • Divorce, poverty, unemployment, poor housing, substance abuse, frequent relocation, crowded living conditions
  • Child abuse can occur in any socioeconomic population
• Sexual Abuse
  • Defined as: “the use, persuasion, or coercion of any child to engage in sexually explicit conduct or simulation of such conduct for producing visual depiction of such conduct, or rape, molestation, prostitution, or incest with children”
• Characteristics of Abusers and Victims
  • Typical abuser is male whom the victim knows, but may be ANYONE
  • All socioeconomic backgrounds
• Nursing Care of the Maltreated Child
• Identify abusive situations as early as possible
• History pertaining to the incident
• Evidence of maltreatment:
  • Pattern or combination of indicators that arouse suspicion and further investigation
  • Protect child from further abuse
• Munchausen Syndrome by Proxy
  • Caregiver fabricates signs and symptoms of illness in child (the proxy) to gain attention from medical staff
  • Child may undergo needless and painful procedures and treatments; 10% cases may be fatal to the child

**Child Passenger Safety**

Safety Seat Law CA
Step 1 REAR-FACING SEATS
• Newborns and infants up to 20-35 pounds.
• Babies must ride rear-facing until one year of age AND at least 20 pounds in the backseat. (AAP recommends until 2).
• Seat can never be forward facing.

Step 2 Front Facing Seats
Children over one year of age AND at least 20 pounds face forward only. (AAP recommends until age 2).
• Most can be converted to a belt positioning booster after child reaches 40 pounds.

Forward-Facing Convertible Seat

**Discipline**

- Discipline
- Limit setting
- Distraction and redirection
- Positive reinforcement
- Provide transition time
- Guidance that helps child learn to understand and care for self and to get along with others.
- Constructive, consistent.
- Pick your battles.
- Avoid temptations and put breakables away.
- Don’t ask open ended questions.

**Respiratory**

- General Aspects of Respiratory Infections
- Upper respiratory tract:
  - Nose, pharynx
- Lower respiratory tract:
  - Bronchi and bronchioles
Croup syndromes:
  - Infections of epiglottis, larynx

Infectious Agents

Viruses:
  - Respiratory syncytial virus (RSV)

Others:
  - Group A β-hemolytic streptococci
  - Staphylococci
  - Chlamydia trachomatis, mycoplasma organisms, pneumococci
  - Haemophilus influenzae

Age
  - Infants <6 months: maternal antibodies
  - 3-6 months: infection rate increases
  - Toddler and preschool ages: high rate of viral infections
  - >5 years: increase in mycoplasmal pneumonia and β-streptococcal infections
  - Increased immunity with age

Size
  - Diameter of airways
  - Distance between structures is shorter, allowing organisms to rapidly move down
  - Short eustachian tubes

Resistance
  - Immune system
  - Allergies, asthma
  - Cardiac anomalies
  - Cystic fibrosis
  - Daycare
  - Immunizations

Seasonal Variations
  - Most common during winter and spring
  - Mycoplasmal infections more common in fall and winter
  - Asthmatic bronchitis more frequent in cold weather
  - RSV season considered winter and spring

Clinical Manifestations of Respiratory Infections
  - Vary with age
  - Generalized signs and symptoms and local manifestations differ in young children:
    • Fever
    • Anorexia, vomiting, diarrhea, abdominal pain
    • Cough, sore throat, nasal blockage or discharge
    • Respiratory sounds

Nursing Interventions for Respiratory Infections
Croup Syndromes
- Ease respiratory effort
- Fever management
- Promote rest and comfort
- Infection control
- Promote hydration and nutrition

Family support and teaching

Characterized by hoarseness, “barking” cough, inspiratory stridor, and varying degrees of respiratory distress

Croup syndromes affect larynx, trachea, and bronchi:
- Epiglottitis, laryngitis, laryngotracheobronchitis (LTB), tracheitis

Acute Epiglottitis
- Clinical manifestations:
  - Sore throat, pain, tripod positioning, retractions
  - Inspiratory stridor, mild hypoxia, distress

Therapeutic management:
- Potential for respiratory obstruction

Nursing considerations
- Prevention: Hib vaccine

Acute LTB
- LTB = laryngotracheobronchitis
- Most common of the croup syndromes
- Generally affects children <5 years
- Organisms responsible:
  - RSV, parainfluenza virus, Mycoplasma pneumoniae, influenza A and B

Manifestations of LTB
- Inspiratory stridor
- Suprasternal retractions
- Barking or seal-like cough
- Increasing respiratory distress and hypoxia

Can progress to respiratory acidosis, respiratory failure, and death

Stridor

Therapeutic Management of LTB
- Airway management
- Maintain hydration (PO or IV)
- High humidity with cool mist
- Nebulizer treatments:
  - Epinephrine
  - Steroids
  - Acute Spasmodic Laryngitis

AKA spasmodic croup, midnight croup
- Paroxysmal attacks of laryngeal obstruction
  - Occur chiefly at night
  - Inflammation: mild or absent
  - Most often affects children ages 1-3 years

- Therapeutic management
- Bacterial Tracheitis
  - Infection of the mucosa of the upper trachea
  - Distinct entity with features of croup and epiglottitis
  - Clinical manifestations similar to LTB
  - May be complication of LTB
  - Thick, purulent secretions result in respiratory distress

- Therapeutic Management of Bacterial Tracheitis
  - Humidified oxygen
  - Antipyretics
  - Antibiotics
  - May require intubation

- Infections of the Lower Airways
  - Considered the “reactive” portion of the lower respiratory tract
  - Includes bronchi and bronchioles
  - Cartilaginous support not fully developed until adolescence
  - Constriction of airways

- Bronchitis
  - Also known as tracheobronchitis

- Definitions
- Causative agents
- Clinical manifestations

- Bronchiolitis and RSV
  - Definitions
  - RSV = respiratory syncytial virus
  - Pathophysiology
  - Diagnostics
  - Therapeutic management
  - Prevention of RSV: prophylaxis
  - Nursing considerations

- Foreign Body Aspiration
  - Risk among children
  - Diagnostic evaluation
  - Therapeutic management
  - Nursing considerations

- Cystic Fibrosis (CF)
Exocrine gland dysfunction that produces multisystem involvement
Most common lethal genetic illness among Caucasian children
Approximately 3% U.S. Caucasian population are symptom-free carriers
Autosomal recessive trait
Inherits defective gene from both parents, with an overall incidence of 1:4
Pathophysiology of CF
  ◦ Characterized by several unrelated clinical features
  ◦ Increased Viscosity of Mucous Gland Secretion
  ◦ Results in mechanical obstruction
  ◦ Thick inspissated mucoprotein accumulates, dilates, precipitates, coagulates to form concretions in glands and ducts
  ◦ Respiratory tract and pancreas are predominantly affected
  ◦ Increased Sweat Electrolytes
Basis of the most reliable diagnostic procedure: sweat chloride test
  ◦ Sodium and chloride will be 2-5 times greater than in the controls
Other Factors in CF
  ◦ Increased organic-enzymatic constituents of saliva
  ◦ Abnormalities of the autonomic nervous system
Respiratory Manifestations of CF
  ◦ Present in almost all CF patients but onset and extent are variable
  ◦ Stagnation of mucus and bacterial colonization result in destruction of lung tissue
  ◦ Tenacious secretions are difficult to expectorate, obstruct bronchi and bronchioles
  Decreased $O_2$-$CO_2$ exchange
  ◦ Results in hypoxia, hypercapnia, acidosis
  ◦ Compression of pulmonary blood vessels and progressive lung dysfunction lead to pulmonary hypertension, cor pulmonale, respiratory failure, and death
Infectious Pathogens
  ◦ *Pseudomonas aeruginosa*
  ◦ *Burkholderia cepacia*
  ◦ *Staphylococcus aureus*
  ◦ *Haemophilus influenzae*
  ◦ *Escherichia coli*
  ◦ *Klebsiella pneumoniae*
Respiratory Progression
  ◦ Gradual progression follows chronic infection
  ◦ Bronchial epithelium is destroyed
  ◦ Infection spreads to peribronchial tissues weakening bronchial walls
  ◦ Peribronchial fibrosis
  ◦ Decreased $O_2$-$CO_2$ exchange
  ◦ Further Respiratory Progression
- Chronic hypoxemia causes contraction and hypertrophy of muscle fibers in pulmonary arteries and arterioles
- Pulmonary hypertension
- Cor pulmonale
- Pneumothorax
- Hemoptysis

- Gastrointestinal (GI) Tract
  - Thick secretions block ducts → cystic dilation → degeneration → diffuse fibrosis
  - Prevents pancreatic enzymes from reaching duodenum
  - Impaired digestion and absorption of fat: steatorrhea
  - Impaired digestion and absorption of protein: azotorrhea
  - Endocrine function of pancreas initially stays unchanged
  - Eventually pancreatic fibrosis occurs; may result in diabetes mellitus
  - Focal biliary obstruction results in multilobular biliary cirrhosis
  - Impaired salivation

- Clinical Manifestations of CF
  - Pancreatic enzyme deficiency
  - Progressive chronic obstructive pulmonary disease (COPD) associated with infection
  - Sweat gland dysfunction
  - Failure to thrive
  - Increased weight loss despite increased appetite
  - Gradual respiratory deterioration

- Presentation of CF
  - Wheezing respiration; dry, nonproductive cough
  - Generalized obstructive emphysema
  - Patchy atelectasis
  - Cyanosis
  - Clubbing of fingers and toes
  - Repeated bronchitis and pneumonia
  - Meconium ileus
  - Distal intestinal obstruction syndrome
  - Excretion of undigested food in stool—increased bulk, frothy, and foul
  - Wasting of tissues
  - Prolapse of the rectum
  - Delayed puberty in females
  - Sterility in males
  - Parents report children taste “salty”
  - Dehydration
  - Hyponatremic or hypochloremic alkalosis
  - Hypoalbuminemia
Diagnostic Evaluation of CF
- Quantitative sweat chloride test
- Chest x-ray
- Pulmonary function tests (PFTs)
- Stool fat and/or enzyme analysis
- Barium enema

Treatment Goals for CF
- Prevent or minimize pulmonary complications
- Adequate nutrition for growth
- Assist in adapting to chronic illness
- Respiratory Management of CF
  - CPT
  - Bronchodilator medication
  - Forced expiration
  - Aggressive treatment of pulmonary infections
  - Home IV antibiotic therapy
  - Aerosolized antibiotics

Pneumothorax
Hemoptysis
Nasal polyps
Steroid use; NSAIDs
Transplantation
GI Management of CF
- Replacement of pancreatic enzymes
- High-protein, high-calorie diet, as much as 150% RDA
- Intestinal obstruction
- Reduction of rectal prolapse
- Salt supplementation

Prognosis of CF
- Estimated life expectancy for child born with CF in 2003 is 40-50 years

Maximize health potential:
- Nutrition
- Prevention and early aggressive treatment of infection
- Pulmonary hygiene

New research—hope for the future:
- Gene therapy
- Bilateral lung transplants
- Improved pharmacologic agents

Family Support for the Child with CF
- Coping with emotional needs of child and family
Child requires treatments multiple times each day
- Frequent hospitalization
- Implications of genetic transmission of disease

Nursing Diagnoses for CF
- Name some nursing diagnoses
- “Feels like you are breathing through a small straw all the time”

**Rheumatic Fever (RF) and Rheumatic Heart Disease (RHD)**

- **RF**
  - Inflammatory disease occurs after group A β-hemolytic streptococcal pharyngitis
  - Infrequently seen in United States; big problem in Third World
  - Self-limiting
    - Affects joints, skin, brain, serous surfaces, and heart

- **RHD**
  - Most common complication of RF
  - Damage to valves as result of RF

- **Clinical Manifestations of RF**
  - Carditis
  - Polyarthritis
  - Erythema marginatum
  - Subcutaneous nodules
  - Erythema Marginatum

- **Prevention of RHD**

- **Treatment of streptococcal tonsillitis and pharyngitis:**
  - Penicillin G IM X 1
  - Penicillin V PO q 10 days
  - Sulfa PO q 10 days
  - Erythromycin (if allergic to above) PO q 10 days

- **Treatment of recurrent RF:**
  - Same as above

- **Nursing Diagnoses for RF**
- Name some nursing diagnoses
- Pt teaching?

**GU**

- **UTI**
- **Causes:**
  - *Escherichia coli* most common pathogen
  - Streptococci
  - *Staphylococcus saprophyticus*
  - Occasionally fungal and parasitic pathogens

- **Classification of UTI**
- **Upper tract:** involves renal parenchyma, pelvis, and ureters
Typically causes fever, chills, flank pain

• Lower tract: involves lower urinary tract
  – Usually no systemic manifestations
• Lower tract:
  – Cystitis
  – Urethritis
• Upper tract:
  – Pyelonephritis
  – Vesicoureteral reflux (VUR)
  – Glomerulonephritis

• Types of UTIs
  – Recurrent: repeated episodes
  – Persistent: bacteriuria despite antibiotics
  – Febrile: typically indicates pyelonephritis
  – Urosepsis: bacterial illness, urinary pathogens in blood
• Recurrent & Persistent
  – Recurrent is reinfection in person whose prior infection was successfully eradicated
  – Recurrent occurs because original infection not adequately eradicated
  – Unresolved bacteriuria: bacteria resistant or drug discontinued before bacteriuria is completely eradicated
  – Bacterial persistence: resistance developed or foreign body in urinary system serves as harbor and anchor for bacteria to survive despite therapy

• Etiology and Pathophysiology of UTI
• Physiologic and mechanical defense mechanisms maintain sterility:
  – Emptying bladder
  – Normal antibacterial properties of urine and tract
  – Ureterovesical junction competence
  – Peristaltic activity

Alteration of defense mechanisms increases risk of UTI
• Organisms usually introduced via ascending route from urethra
• Less common routes:
  – Bloodstream
  – Lymphatic system

• Clinical Manifestations of UTI
• Symptoms:
  – Dysuria
  – Frequent urination (>q 2 hr)
  – Urgency
  – Suprapubic discomfort or pressure
  – Bed wetting after potty trained
  – Urine may contain visible blood or sediment (cloudy appearance)
  – Flank pain, chills, and fever indicate infection of upper tract (pyelonephritis)
• Pediatric Manifestations of UTI
  – Frequency
  – Fever in some cases
  – Odiferous urine
  – Blood or blood-tinged urine
  – Sometimes NO symptoms except generalized sepsis
  – Pediatric patients with significant bacteriuria may have no symptoms or nonspecific symptoms like fatigue or anorexia

• Diagnostic Studies of UTI
  – Dipstick
  – Microscopic urinalysis
  – Culture

• Clean-catch specimen is preferred
• U-bag for collection from child
• Specimen obtained by catheterization or suprapubic needle aspiration has more accurate results
  – May be necessary when clean-catch cannot be obtained

• Diagnostic Studies of UTI
  – Sensitivity testing determines susceptibility to antibiotics

• Imaging studies for suspected obstruction
  – IVP or abdominal CT

• Collaborative Care for UTI Drug Therapy: Antibiotics
  – Uncomplicated cystitis: short-term course of antibiotics
  – Complicated UTIs: long-term treatment
  – Teach proper hygiene (wipe front to back), no bubble bath or harsh soaps.
  – Collaborative Care for UTI Drug Therapy: Antibiotics (cont’d)
  – Trimethoprim-sulfamethoxazole (TMP-SMX) or nitrofurantoin
  – Amoxicillin
  – Cephalexin
  – Others:
    – Gentamycin, carbenicillin
    – Pyridium (OTC)
  – Combination agents (e.g., Urised) used to relieve pain
    – Preparations with methylene blue tint

• Second most common bacterial disease
• Account for more than 8 million office visits per year
• Results in >100,000 people hospitalized annually
• >15% patients who develop gram-negative bacteria DIE
• 1/3 of gram-negative infections originate in urinary tract

  **Glomerulonephritis**

• Symptoms
  – Generalized edema due to decreased glomerular filtration
    – Begins with periorbital area
– Progresses to lower extremities and then to ascites
– Hypertension due to increased extracellular fluid
– Oliguria
– Hematuria
  • Bleeding in upper urinary tract, resulting in tea or cola colored urine
– Proteinuria
  • Increased amount of protein = increased severity of renal disease

• Acute Poststreptococcal Glomerulonephritis (APSGN)
  – A noninfectious renal disease
    • Autoimmune
  – Onset 5-12 days after OTHER type of infection
  – Often group A β-hemolytic streptococci
  – Most common in 6-7 year olds
  – Uncommon in children <2 years old
  – Can occur at any age
  – Nursing Management of APSGN
  – Manage edema:
    • Daily weights
    • Accurate I&O
    • Daily abdominal girth
  – Nutrition:
    • Low sodium, low to moderate protein
  – Susceptibility to infections
  – Bed rest is not necessary
  – Prognosis
    – 95%—rapid improvement to complete recovery
    – 5% to 15%—chronic glomerulonephritis
    – 1%—irreversible damage

• Nephrotic Syndrome
  – Most common presentation of glomerular injury in children
  – Characteristics:
    • Proteinuria
    • Hypoalbuminemia
    • Hyperlipidemia
    • Edema
    • Massive urinary protein loss

Types of Nephrotic Syndrome
  – Primary or Minimal change nephrotic syndrome (MCNS)
    • Also known as:
      • Idiopathic nephrosis
      • Nil disease
      • Uncomplicated nephrosis
• Childhood nephrosis
  • Minimal lesion nephrosis
    – Congenital nephrotic syndrome
    – Secondary nephrotic syndrome (after another cause of glomerular damage)
• Changes in Nephrotic Syndrome
  – Glomerular membrane:
    • Normally impermeable to large proteins
    • Becomes permeable to proteins, especially albumin
    • Albumin lost in urine (hyperalbuminuria)
    • Serum albumin decreased (hypoalbuminemia)
    • Fluid shifts from plasma to interstitial spaces:
      • Hypovolemia
      • Ascites
• Edema phase
• Remission phase
• Patho Review
• Management of Nephrotic Syndrome
  – Supportive care
  – Diet:
    • Low to moderate protein
    • Sodium restrictions when large amounts of edema are present
  – Steroids:
    • 2 mg/kg divided into b.i.d. doses
    • Prednisone drug of choice (least expensive and safest)
  – Immunosuppressant therapy (cyclophosphamide [Cytoxan])
  – Diuretics
• Treatment Goals
  – Reduce excretion of urinary protein
  – Reduce fluid retention in tissues
  – Prevent infection
  – Minimize complications of therapy
    • Low salt, often fluid restricted diet
    • Diuretics
    • IV albumin
    • Steroids
• Review
• Prognosis
  • Usually good
  • Usually self limiting (80%)
• Family Issues with Nephrotic Syndrome
  – Chronic condition with relapses
  – Developmental milestones
Social isolation:
- Lack of energy
- Immunosuppression, protection
- Change in appearance due to edema—self-image

Nursing Interventions for Nephrotic Syndrome
- Aseptic technique during catheterizations
- Avoid unnecessary catheterization and early removal of indwelling catheters
  - Prevent nosocomial infections:
    - Wash hands before and after contact
    - Wear gloves for care of urinary system
- Routine and thorough perineal care for all hospitalized patients
- Avoid incontinent episodes by answering call light and offering bedpan at frequent intervals
- Ensure adequate fluid intake (patient with urinary problems may think drinking fluids will be more uncomfortable):
  - Dilutes urine, making bladder less irritable
  -Flushes out bacteria before they can colonize
  -Avoid caffeine, alcohol, citrus juices, chocolate, and highly spiced foods
    - Potential bladder irritants
- Discharge-to-home instructions
- Follow-up urine culture
  - Recurrent symptoms typically occur in 1-2 weeks after therapy
  - Encourage adequate fluids even after infection
  - Low-dose, long-term antibiotics to prevent relapses or reinfections
  - Explain rationale to enhance compliance

Wilms Tumor
- AKA nephroblastoma
- Malignant renal and intraabdominal tumor of childhood
- Three times more common in African-American children
- Peak age of diagnosis is 3 years
- More frequent in males
- Etiology – arises from malignant undifferentiated cells
- Diagnostic evaluation – evaluation of abdominal swelling or mass. Don’t palpate or can seed tumor.
- Therapeutic management
  - Surgical removal
  - Chemotherapy and/or radiation if tumor large, bilateral or mets.
- Nursing considerations – prognosis best of all childhood cancers, stages I-II 90% cure rate.
Leukemia

Symptoms of Leukemia
- ALL: lymphatic, lymphocytic, lymphoblastic, and lymphoblastoid leukemia
- AML: granulocytic, myelocytic, monocytic, myelogenous, monoblastic, and nonmyeloblastic leukemia

Pathophysiology of Leukemia
- Leukemia is an unrestricted proliferation of immature WBCs in the blood-forming tissues of the body
- Liver and spleen are the most severely affected organs
- Although leukemia is an overproduction of WBCs, often acute form causes low leukocyte count
- Cellular destruction takes place by infiltration and subsequent competition for metabolic elements

Consequences of Leukemia
- Anemia from decreased RBCs
- Infection from neutropenia
- Bleeding tendencies from decreased platelet production
- Spleen, liver, and lymph glands show marked infiltration, enlargement, and fibrosis

Diagnostic Evaluation of Leukemia
- Based on history, physical manifestations
- Peripheral blood smear:
  - Immature leukocytes
  - Frequently low blood counts
- Lumbar puncture to evaluate CNS involvement
- Bone marrow aspiration or biopsy
- CSF Results
- Bone Marrow Aspiration

Therapeutic Management of Leukemia
- Chemotherapeutic agents
- Cranial irradiation (in some cases)
- Four Phases of Therapy for Leukemia
  - Induction therapy: 4-6 weeks
  - CNS prophylactic therapy: intrathecal chemotherapy
  - Intensification (consolidation) therapy: to eradicate residual leukemic cells and prevent resistant leukemic clones
  - Maintenance therapy: to preserve remission

Hematopoietic Stem Cell Transplantation (HSCT)
- Donors may be:
  - Relatives or nonrelatives
  - Antigen matched or mismatched
  - Peripheral stem cells may be used
  - Stem cells from umbilical cord blood
- Risks of HSCT
Significant risk of morbidity and mortality
Graft vs. host disease (GVHD)
Overwhelming infection
Severe organ damage
Cure after HSCT: up to 60%-70%

Prognosis for Leukemia
If relapse after HSCT: dismal prognosis
Identified factors for determining prognosis:
Initial WBC count
Age at time of diagnosis
Type of cell involved
Gender
Karyotype analysis

Nursing Considerations for Leukemia
Prevent child and family for procedures
Pain management
Prevent complication of myelosuppression

Increased Susceptibility to Infection
At time of diagnosis and relapse
During immunosuppressive therapy
After prolonged antibiotic therapy that predisposes to the growth of resistant organism

Infection Control
Environment
Hand hygiene
Visitor restriction
Managing Chemotherapeutic Agents
“Vesicants”—sclerosing agents even in minute amounts
Interventions for extravasation
Risk for anaphylaxis
Managing Problems of Drug Toxicity
Nausea and vomiting
Anorexia
Mucosal ulceration
Neuropathy
Hemorrhagic cystitis
Alopecia
Mood changes
Moon face

Muscular Dystrophy

Largest group of muscular diseases in children
• All have genetic origin with gradual degeneration of muscle fibers, progressive weakness, and wasting of skeletal muscles
• All have increasing disability and deformity with loss of strength
• Initial Muscle Groups Involved in MDs
• Duchenne Muscular Dystrophy (DMD)
  • Also called pseudohypertrophic muscular dystrophy
  • Most severe and most common of the MDs in childhood
  • X-linked inheritance pattern; one third are fresh mutations
  • Incidence: 1 in 3500 male births
• Characteristics of DMD
  • Onset between ages 3 and 5 years
  • Progressive muscle weakness, wasting, and contractures
  • Calf muscles hypertrophy in most patients
  • Progressive generalized weakness in adolescence
  • Death from respiratory or cardiac failure
• Diagnostic Evaluation of DMD
  • Suspected based on clinical appearance
  • Confirmation by EMG, muscle biopsy, and serum enzyme measurement
  • Serum CPK and AST levels high in first 2 years of life, before onset of weakness; levels diminish as muscle deterioration continues
• DMD: Clinical Manifestations
  • Waddling gait, frequent falls, Gower sign
  • Lordosis
  • Enlarged muscles, especially thighs and upper arms
  • Profound muscular atrophy in later stages
  • Mental deficiency common
• Therapeutic Management of DMD
  • No effective treatment has been established
  • Primary goal: maintain function in unaffected muscles as long as possible
  • Keep child as active as possible
  • ROM, bracing, performance of ADLs, surgical release of contractures prn
  • Genetic counseling for family
• DMD: Nursing Considerations
  • Help child and family cope with chronic, progressive, debilitating disease
  • Help design a program to foster independence and activity as long as possible
  • Teach child self-help skills
  • Arrange for appropriate health care assistance as child’s needs intensify (home health, skilled nursing facility, respite care for family, etc.)

Osteogenesis Imperfecta

• Osteogenesis Imperfecta (OI)
  • A group of heterogeneous inherited disorders of connective tissue
Characterized by excessive fragility and bone defects
- Defective periosteal bone formation and reduced cortical thickness of bones
- Hyperextensibility of ligaments

**Classification of OI—Type I**
- Type I-A: mild bone fragility, blue sclera, normal teeth, presenile deafness
- Type I-B: same as A except with abnormal dentition
- Type I-C: same as B but no bone fragility
- Two thirds of all cases are type I

**Classification of OI—Type II**
- Lethal; stillborn or die in early infancy
- Severe bone fragility with multiple fractures at birth
- Autosomal recessive inheritance

**Classification of OI—Type III**
- Severe bone fragility leads to severe progressive deformities
- Normal sclera, marked growth failure
- Most are autosomal recessive, but some are autosomal dominant inheritance

**Classification of OI—Type IV**
- Type IV-A: mild to moderate bone fragility, normal sclera, short stature, variable deformity, autosomal dominant
- Type IV-B: same as A except abnormal dentition (dentinogenesis imperfecta)
- Approximately 6% of OI cases are type IV-B

**Therapeutic Management of OI**
- Primarily supportive care
- Drugs of limited benefit
- May rule out OI if multiple fractures occur

**Nursing considerations**
- Caution with handling to prevent fractures
- Family education
- Occupational planning and genetic counseling