Review Eye Anatomy

- Physiology of Vision
  - Light rays enter the eye and are bent (refracted) as they pass through eye structures to the retina.
  - Eye adjusts (accommodates) to seeing objects at various distances by flattening or thickening the lens.
  - Light rays are absorbed by photoreceptors, changed to electrical activity and transmitted via the optic nerve to the brain for processing.

- Aging Eyes
  - Decreased flexibility of the lens – decreases the ability of the eye to focus (accommodate) for near work. PRESBYOPIA
  - Decreased color perception
  - Smaller pupil size allows less light in causing impaired night vision.
  - A 60 year old needs about twice as much light to see as they did when they were 20 years old.
  - Glare
  - Decreased field of vision
  - Decrease in lens transparency – cataracts
  - Aqueous humor production decreases
  - Decreased tears
  - Duller appearance and may feel tight, scratchy or dry

Macular Degeneration

- Neovascular (wet or exudative)
  - Proliferation of new fragile blood vessels that leak in macular area and damage the macula. Scarring occurs and vision declines.

- Nonneovascular (nonexudative or dry)
  - Waste materials deposit and cause atrophy of retina

Symptoms include
  - Perceived dark spots, missing areas, distorted wavy lines
  - Visual blurring and distortion
  - Central vision loss
  - Decreased ability to distinguish colors
  - Loss of Central Vision

- Treatment
  - Nonneovascular – no treatment
  - Neovascular – laser therapy to stop leakage from vessels
    - Vision is not improved – additional loss of central vision is often spared

- Nursing Intervention
  - Promote regular eye examinations
  - Promote Rules for Eye Safety (Pg 1892 in Phipps)
  - Promote adequate nutrition for eye health. Vitamin A and B are especially needed to maintain eye health.
  - Referral to community agencies for those who have declining vision.
  - Magnifying glasses, high intensity reading lights

Glaucoma

- Caused by progressive optic nerve atrophy causing loss of vision.
- Obstruction in outflow channels for aqueous humor results in back up of fluid and a rise in IOP. (Normal is 10-21 mm Hg)
- Atrophy is caused by elevated intraocular pressure (IOP).
• Risk factors
  o Age
  o Race – African American
  o Myopia
  o Family history
  o Race – Asian race – angle closure glaucoma

• Types of Glaucoma
  o Open-angle glaucoma
  o Angle closure glaucoma
  o Primary when etiology is unknown
  o Secondary when it results from another eye disorder
    ▪ Refer to types on pg 1893 of Phipps

• Open Angle Glaucoma
  o IOP greater than 24 mm Hg
  o Slow loss of vision
  o Peripheral vision loss first, (tunnel vision) then central, then blindness
    ▪ Note Phipps pg 1894
  o Difficulty adjusting to darkness
  o Failure to detect color changes
  o Normal Vision
  o Peripheral Vision Loss

• Angle Closure Glaucoma
  o Acute severe ocular pain
  o Pupil enlarged and fixed
  o Colored halos around lights
  o May have N/V
  o Dramatically increased IOP; may exceed 50 mm Hg
  o Permanent blindness if marked increase in IOP for 24 – 48 hours.

• Common Medications
  o Decrease aqueous humor production
    ▪ Beta-Adrenergic Antagonists
    ▪ Carbonic Anhydrase Inhibitors
    ▪ Adrenergic Agents
  o Increases outflow of aqueous humor
    ▪ Miotics
    ▪ Cholinesterase Inhibitors
    ▪ Adrenergic Agents
    ▪ Prostaglandin Agonist

• Medications are administered topically or systemically
  o Davis Drug Guide – Appendix pg 1146-1157
  o Be able to look up a ophthalmic medication and determine how it helps decrease IOP.

• Surgical Management
  o Indicated if conservative management fails to control IOP.

• Glaucoma – Nursing Intervention
  o Promote regular screening of older population
  o Assess ability to purchase medications and refer prn
  o Assess ability to administer eye drops

Cataracts
  • Clouding or opacity of lens that leads to gradual painless blurring and eventual loss of vision.
  • Risk factors
- Ultraviolet radiation a suspected cause
- Higher incidence in warm sunny climates
- Vitamin deficiency of Vitamin A, C, E a suspected cause
- Eye injury
- Secondary to other systemic diseases
- Age and Sex (65yo and women)
- Signs and symptoms of cataracts
  - Painless blurring and loss of vision
  - Peripheral vision affected first
  - Glare
  - Halos
  - Loss of ability to “see” hues
  - Cloudy white opacity on pupil
- Surgical management is effective 90-95% of the time.
  - A same day surgery under local anesthesia
  - Lens is implanted during surgery

**Ear Anatomy and Physiology**
- Review
- Sound Transmission
  - Air conduction
    - Transmits sound from middle to inner ear
  - Bone conduction
    - Transmits sound from skull to inner ear
  - Sound energy is transformed to neural energy for transmission to the brain. Via the 8th cranial nerve.
  - Ear sends impulse to brain to assist in maintaining balance / equilibrium.

**Hearing Loss**
- Conductive hearing loss- a mechanical problem in outer or middle ear interfering with conduction of sound waves.
- Sensorineural hearing loss- a nerve problem interfering with conduction of sound waves.
- Conductive Hearing Loss
  - Caused by anything that blocks the external ear
    - Wax
    - Infection
    - Foreign body
    - Tumors
    - Scar tissue
- Sensorineural Hearing Loss
  - Disease or trauma to inner ear, nerves, nerve pathways
    - Diabetes, arteriosclerosis, infectious
- Noise induced hearing loss (greater than 90 decibels for prolonged time)
- Age related presbycusis
  - Tinnitus
- Ototoxic Drugs
  - Note Table 50-1 on pg 1913 of Phipps
  - Aminoglycosides
  - Vancomycin
  - Loop Diuretics – rapid parenteral administration
  - Erythromycin
  - Salicylates
• NSAIDS
  • ***Tinnitus is a common preliminary symptom***

• Hearing Aids
  • Know how to care for a hearing aid
  • Pg 1923 in Phipps

• Know how to communicate with the hearing impaired
  • Pg 1923 in Phipps

Endocrine System
  • The endocrine system is a cellular communicating system involving hormones.
  • A hormone is a molecule secreted from one organ that travels in the blood and has an effect on a distant organ.

• Thyroid Gland
  • Two lobes
  • Two cell types
    ▪ Follicular – produce T3 and T4
    ▪ Parafollicular - synthesize and secrete calcitonin

• Endocrine Feedback Loop
  • Hypothalamus produces thyroid releasing hormone
  • \( \rightarrow \) Pituitary to produce thyroid stimulating hormone (TSH)
  • \( \rightarrow \) The Thyroid gland to produce
    ▪ Thyroxine (T4)
    ▪ Triiodothyromine (T3)
  • Iodine is necessary for the synthesis of these hormones

• Clinical Manifestations of Hypothyroidism
  • Decreased metabolic rate, heat production and oxygen consumption.
  • Cold intolerance, decreased body temperature
  • Cool dry skin
  • Decreased appetite, weight gain
  • Myxedema facies (Phipps pg 900)
  • Fatigue
  • Anemia

• Thyroid Function Testing
  • TSH assay –
    ▪ increased levels indicate hypothyroidism
    ▪ decreased levels indicate hyperthyroidism
    ▪ used to monitor thyroid hormone replacement therapy
  • Free T4 – (free means not bound to protein)
  • Free T3
  • Thyroid Stimulating Hormone
    ▪ The earliest marker of hypothyroidism is an elevated TSH
    ▪ Normal Adult 0.4-4.2 micro Units / ml
    ▪ Probable hypothyroidism – greater than 7.0 micro units/ml
    ▪ Desired level when receiving thyroxine therapy – 0.5-3.5 micro units/ml
  • Thyroxine - T4
    ▪ Hormone produced in thyroid gland from iodine and thyroglobulin.
    ▪ Production occurs in response to the effects of TSH on the thyroid gland.
    ▪ When released, 99.6% of T4 is bound to protein. Bound hormone acts as reservoir.
    ▪ The remaining 0.4% is “free” throxine and is biologically active.
• Medication
  o Daily oral dose of Sodium Levothyroxine (L-thyroxine)
  o 1.6-1.8(micrograms/kg) of body weight
  o A pt weighing 70Kg would be prescribed 112 –126 mcg per day
  o Commonly a pt is started on 50mcg/day and increased q 2-3 weeks. Maintenance dose is 75-125 mcg/day

Degenerative Joint Disease – (DJD)
• Osteoarthritis (OA) – cellular, biochemical and biomechanical factors affecting diarthroidal joints
  o 80% of gerons have radiographic evidence, 5 – 10% have clinical symptoms.
  o Women – hands
  o Men – hips knees and spine
• Risk Factors
  o Weight – obesity (Body Mass Index greater than 25) causes 21% increase in knee OA
  o Family history
  o Race
  o Injury to a joint
  o OA
• Primary – no know underlying causes
• Secondary – any condition that causes damage to cartilage, chronic stress to joints or causes joint instability
  o OA
    o Despite “itis” there is a small amount of low grade inflammation
    o Mechanical abnormalities in joint can cause inflammation
    o Generally considered non inflammatory to distinguish it from Rheumatoid Arthritis
• OA - Pathology
  o Erosion of articular cartilage
  o Normally smooth, white, translucent – now becomes yellow and opaque Cartilage becomes soft and gets rough, frayed and cracked. Cartilage destroyed.
  o Thickening of subchondral bone
  o Bone goes trough a remodeling process
  o Formation of osteophytes (bone spurs)
  o Bone spurs may break off and be loose
• Clinical Manifestations
  o Pain – “deep aching” in joint
  o Weather changes, increased activity
  o Swelling, joint enlargement
  o Decreased ROM
  o Muscle atrophy
  o Crepitus
  o Joint stiffness
  o Stiffness last less than 1 hour
• Diagnosis
  o Patient history
  o Physical Assessment
  o Radiographic studies
  o Narrowing of joint space
  o Osteophyte formation
  o Eburnation (sclerosis) of subchondral bone
• Focus of Therapy
  o Joint protection
  o Weight reduction
  o Use of cane or splints
  o Physical Therapy
- Exercise is indicated
- ROM daily
- Isometric and isotonic (that do not stress the joint - exercises daily)
- Swimming and water exercises
  - Heat and cold therapy
  - Exercise reduces fatigue, though with advanced disease exercise may exacerbate symptoms. Rest relieves most joint pain but should be avoided for prolonged periods, because immobility promotes joint stiffness.
  - Pain Relief
    - Medications
      - Analgesics (mild to moderate pain)
        - Acetaminophen – doses up to 4G/day
          - Dosing at intervals or prn
          - Watch liver and renal function
        - NSAIDS (mild to moderate pain)
          - Cox-1 and Cox-2
          - Prevent prostaglandins (pain and inflammation)
        - Intra-articular corticosteroids injection
          - No more than 3 – 4 injections per year
          - Risk of infection and cutaneous atrophy
          - Corticosteroids have many side effects
      - Opioids
        - Refer to Table 48-7 in Phipps pg 1525)
    - Surgical Management
      - Arthroplasty
      - Osteotomy
      - Athrodesis – joint fusion

**Osteoporosis**
- Bone Density
  - Osteoblastic forces (bone remodeling or building) predominate through young adulthood until peak bone mass is achieved at age 35.
  - Osteoclastic forces predominate after menopause
- Osteoporosis
  - Called the “silent thief” or “silent disease”.
  - Earliest sign may be acute inset of back pain from vertebral fracture that occurred at rest or with minimal activity.
  - Loss of height. Lower rib cage may rest on iliac crests!
  - No outward manifestations may be apparent until a fracture occurs.
- Osteoporosis- Risk Factors
  - Unchangeable
    - Aging – postmenopausal
    - Caucasian or Asian female
    - Nullparity
    - Family history
    - Small frame – low body weight
    - There has been a gene identified which controls bone density
  - Changeable
    - Diet
    - Chronic calcium deficiency
    - Vitamin D deficiency
    - Chronic alcohol abuse
    - Excessive caffeine intake
- Diet high in protein and fat
- Low peak bone mass at skeletal maturity
- Smoking
- Sedentary lifestyle - Accelerated postmenopausal bone loss

- Types of Osteoporosis
  - Primary – no cause, no underlying pathologic condition
  - Secondary – results from another cause or medical condition
  - See Box 47-9 in Phipps pg 1556

- Measuring Bone Mass
  - Dual-Energy X-ray Absorptiometry (DEXA)
  - Scans heel, finger, lumbar spine, non dominant proximal femur or forearm to determine bone mineral density (BMD)
  - Used to predict fracture risk
  - Z score – peak bone mass
  - T score – comparison of BMD with others the same age
    - Evaluating T Scores
      - Normal skeletal status
      - T score above –1
      - Osteopenia
        - T score between –1 to –2.5
      - Osteoporosis
        - T score at or below –2.5
  - Severe Osteoporosis
    - Below –2.5 and presence of one or more pathologic fractures

- Medications
  - Decrease bone resorption and/or increase bone formation
    - Calcium
    - Vitamin D Compounds
    - Estrogen Replacement
      - Calcitonin (IM, SC, Intranasal)
      - Biphosphonates (end in “ate”)
    - Selective Estrogen Receptor Modulators (SERM’s)

- Nursing Intervention
  - You are asked to speak to a group of perimenopausal women at a local community center. The topic in “healthy bones”. Describe essential components of your presentation.

- Your patient is considering pharmacological treatment options for severe osteoporosis. Explain the various categories of medications used.

**Parkinson’s Disease**

- Most cases are primary or idiopathic
- Secondary cases may develop from some medications, infections, brain trauma or tumors or ingestion of neurotoxins.

- Pathophysiology
  - Dopaminergic neurons in basal ganglia are destroyed. (No symptoms till 70% destroyed.)
  - Neurotransmitters in basal ganglia
  - Dopamine – produces inhibitory effects
  - Acetylcholine - produces excitatory effects
  - When the excitatory effect of acetylcholine is inadequately balanced, an individual has difficulty controlling or inhibiting voluntary movements.

- Clinical Manifestations
  - Tremor
- Rigidity
- Akinesia/Bradykinesia
- Postural instability
  - (See Clinical Manifestations Box in Phipps pg 1390)
  - (Note Secondary Manifestations in Box 43-9 in Phipps pg 1391)
- Cannot be stopped or cured – but drug therapy can control symptoms.

- Diagnosis
  - Made from patient history and symptoms
  - No definitive diagnostic test
  - Confirmed primarily from response to medication

- Medications
  - 1. Levodopa - crosses blood brain barrier and converts to dopamine.
    - Sinamet contains Levodopa and carbidopa. Carbidopa blocks conversion of levodopa in the peripheral tissues. See pt teaching guidelines in Phipps pg 1392. Most common medication given for Parkinson’s disease
  - 2. Anticholinergics – they stop the excitatory effects of the cholinergic neurions.
    - Stop tremors and decrease muscle rigidity
  - Antiviral agents
    - Potentiates the action of dopamine in the CNS
      - Dopamine agonists – stimulates dopamine receptors and increases the effect of levodopa
      - Monoamine Oxidase B Inhibitor (MAO inhibitors) – blocks metabolism of dopamine
      - COMT Inhibitors - prevents breakdown of levodopa

- Nursing Intervention
  - Describe the main components of the Nursing Care Plan for a Patient with Parkinson’s Disease in Phipps pgs 1393-1396.

Shingles – Herpes Zoster
- Persons who have had chicken pox may develop herpes zoster after being exposed to a person with a vesicular lesion of varicella zoster.
  - It is thought that a person who develops herpes zoster has only partial immunity to varicella zoster and therefore susceptible.

- Symptoms
  - Macules-vesicles are arranged in linear fashion – never crossing the midline of the body.
  - Malaise, fever, itching, pain over involved are precede the rash.
  - Rash appears on thoracic area, face, eye and scalp.
  - Vesicles develop in 1 – 2 days. Lesions clear in 2-3 weeks.
  - Pain and itching is a major problem.
    - Pain may be light burning to deep visceral type pain. It may be intermittent or constant. Can persist for 4 weeks.
    - 10% of patients get postherpetic neuralgia

- Medications
  - Anti viral therapy
    - Zovirax 800 mg five times a day X 7 days
      - Can be give IV
  - Analgesics
    - ASA with or without codeine
    - Topical agents for itching
  - Postherpetic Neuralgia
    - Pain results form nervous system damage and may last for years
  - Multidisciplinary approaches to pain management are usually needed.