

## Geriatrics IB

### 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
  - Age
  - Race – African American
  - Myopia
  - Family history
  - Race – Asian race – angle closure glaucoma
- Types of Glaucoma
  - Open-angle glaucoma
  - Angle closure glaucoma
  - Primary when etiology is unknown
  - Secondary when it results from another eye disorder
    - Refer to types on pg 1893 of Phipps
- Open Angle Glaucoma
  - IOP greater than 24 mm Hg
  - Slow loss of vision
  - Peripheral vision loss first, (tunnel vision) then central, then blindness
    - Note Phipps pg 1894
  - Difficulty adjusting to darkness
  - Failure to detect color changes
  - Normal Vision
  - Peripheral Vision Loss
- Angle Closure Glaucoma
  - Acute severe ocular pain
  - Pupil enlarged and fixed
  - Colored halos around lights
  - May have N/V
  - Dramatically increased IOP; may exceed 50 mm Hg
  - Permanent blindness if marked increase in IOP for 24 – 48 hours.
- Common Medications
  - Decrease aqueous humor production
    - Beta-Adrenergic Antagonists
    - Carbonic Anhydrase Inhibitors
    - Adrenergic Agents
  - Increases outflow of aqueous humor
    - Miotics
    - Cholinesterase Inhibitors
    - Adrenergic Agents
    - Prostaglandin Agonist
- Medications are administered topically or systemically
  - Davis Drug Guide – Appendix pg 1146-1157
  - Be able to look up a ophthalmic medication and determine how it helps decrease IOP.
- Surgical Management
  - Indicated if conservative management fails to control IOP.
- Glaucoma – Nursing Intervention
  - Promote regular screening of older population
  - Assess ability to purchase medications and refer prn
  - 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
  - → Pituitary to produce thyroid stimulating hormone (TSH)
  - → The Thyroid gland to produce
    - Thyroxine (T4)
    - Triiodothyronine (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
  - Daily oral dose of Sodium Levothyroxine (L-thyroxine)
  - 1.6-1.8(micrograms/kg)of body weight
  - A pt weighing 70Kg would be prescribed 112 –126 mcg per day
  - 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 diarthrodial joints
  - 80% of gerons have radiographic evidence, 5 – 10% have clinical symptoms.
  - Women – hands
  - Men – hips knees and spine
- Risk Factors
  - Weight – obesity (Body Mass Index greater than 25) causes 21% increase in knee OA
  - Family history
  - Race
  - Injury to a joint
  - OA
- Primary – no know underlying causes
- Secondary – any condition that causes damage to cartilage, chronic stress to joints or causes joint instability
- OA
  - Despite “itis” there is a small amount of low grade inflammation
  - Mechanical abnormalities in joint can cause inflammation
  - Generally considered non inflammatory to distinguish it from Rheumatoid Arthritis
- OA - Pathology
  - Erosion of articular cartilage
  - Normally smooth, white, translucent – now becomes yellow and opaque Cartilage becomes soft and gets rough, frayed and cracked. Cartilage destroyed.
  - Thickening of subchondral bone
  - Bone goes trough a remodeling process
  - Formation of osteophytes (bone spurs)
  - Bone spurs may break off and be loose
- Clinical Manifestations
  - Pain – “deep aching” in joint
  - Weather changes, increased activity
  - Swelling, joint enlargement
  - Decreased ROM
  - Muscle atrophy
  - Crepitus
  - Joint stiffness
  - Stiffness last less than 1 hour
- Diagnosis
  - Patient history
  - Physical Assessment
  - Radiographic studies
  - Narrowing of joint space
  - Osteophyte formation
  - Eburnation (sclerosis) of subchondral bone
- Focus of Therapy
  - Joint protection
  - Weight reduction
  - Use of cane or splints
  - 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 onset 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 a 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.
    - Sinemet 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 neurons.
    - 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.