Concepts of Neurologic Dysfunction

Chapter 14
Alterations in Arousal

- Coma is produced by either:
  - Bilateral hemisphere damage or suppression
  - Brain stem lesions or metabolic derangement that damages or suppresses the reticular activating system
Clinical manifestations

- Pattern of breathing
  - **Posthyperventilation apnea (PHVA)**: irregular breathing seen when consciousness decreases.
  - **Cheyne-Stokes respirations (CSR)**: abnormal pattern of breathing characterized by progressively deeper and sometimes faster breathing, followed by a gradual decrease that results in a temporary stop in breathing.
  - **Agonal breathing (gasps)**: deep slow breaths not true breaths, respiratory center failing, seen in dying victims.

- **Vomiting** may indicate damage to medulla.

- **Pupillary changes**: ischemia and hypoxia cause dilated pupils.

- **Motor responses**: loss of movement with spasticity indicates upper motor neuron damage while flaccid paralysis indicates spinal cord and lower motor neuron damage.
Brain Death (Brain Stem Death)

- Body can no longer maintain internal homeostasis

- Brain death criteria
  - Completion of all appropriate and therapeutic procedures
  - Unresponsive coma (absence of motor and reflex responses)
  - No spontaneous respirations (apnea)
Brain Death (Brain Stem Death)

- Brain death criteria
  - No ocular responses
  - Isoelectric EEG
  - Persistence for 30 minutes to 1 hour and 6 hours after onset if not on drugs or hypothermic
  - Confirming test of absent cerebral circulation
Cerebral Death

- Cerebral death (irreversible coma) is death of the cerebral hemispheres exclusive of the brain stem and cerebellum
- No behavioral or environmental responses
- The brain can continue to maintain internal homeostasis
Cerebral Death

● Survivors of cerebral death
  - Remain in coma
  - Emerge into a vegetative state ("wakeful unconscious state") open eyes may give yes/no responses, smile
  - Progress into a minimal conscious state (MCS)
    • Locked-in syndrome thought and level of arousal are intact but cannot communicate
Seizure Disorder

- Sudden explosive and disorderly discharge of cerebral neurons characterized by sudden transient alteration in brain function

- Seizure vs. Epilepsy
  - Seizure is not a disease but a symptom of underlying CNS dysfunction
  - Epilepsy is a disease characterized by recurrent seizures that are not provoked by other illnesses or circumstances
  - Genetic predisposition?
Seizure Disorder

- Provoked seizures
  - Fever
  - Electrolyte imbalances (hypocalcemia, alkalosis)
  - Hypoglycemia
  - CNS infection or damage

- Unprovoked seizures: cause unknown
Seizure Disorder

- Absence (petit mal): disturbances in consciousness
- Atonic: loss of muscle tone
- Myoclonic: muscles contract
- Tonic-clonic (grand mal): muscle contraction and loss of consciousness
- Generalized convulsive status epilepticus: seizures continue without recovery between them
Cognitive Disorders

- **Declarative memory** memories which can be consciously recalled such as facts and events
- **Nondeclarative memory** procedural memory
- **Image processing**
  - Retrograde amnesia forget past memories
  - Anterograde amnesia cannot form new memories
Data Processing Deficits

- **Agnosia** loss of ability to recognize objects, persons, sounds, shapes, or smells while the specific sense is not defective nor is there any significant memory loss
  - Tactile, visual, auditory, etc.
- **Aphasia** difficulty in producing or comprehending spoken or written language
- **Dysphasia**
  - Expressive dysphasia know what to say or write but motor cannot do it
  - Receptive dysphasia motor works but the language info coming in does not make sense
Acute Confusional States (ACS)

- Acquired mental disorder characterized by deficits in attention and coherence of thoughts and action
  - Secondary to drug intoxication, metabolic disorder, or nervous system disease
Dementia

- Progressive failure of cerebral functions that is not caused by an impaired level of consciousness

- Caused by Alzheimer’s, Parkinson’s, Huntington’s, infections (HIV, mad cow disease), compression, atherosclerosis
Alzheimer Disease (AD)

- Familial, early and late onset
- Nonhereditary (sporadic, late onset) MOST are

Theories
- Mutation for encoding amyloid precursor protein
- Alteration in apolipoprotein E
- Loss of neurotransmitter stimulation of choline acetyltransferase
Alzheimer Disease (AD)

- Neurofibrillary tangles
- Senile plaques
- Clinical manifestations
  - Forgetfulness, emotional upset, disorientation, confusion, lack of concentration, decline in abstraction, problem solving, and judgment
- Diagnosis is made by ruling out other causes of dementia
Cortical Atrophy
Increased Intracranial Pressure (IICP)

- Normal 5 to 15 mm Hg
- Caused by an increase in intracranial content
  - Tumor growth, edema, excessive CSF, or hemorrhage
Herniation

Compresssion of the opposite cerebral peduncle against the unyielding tentorium

Herniation of cingulate gyrus under falx cerebri

Herniation of temporal lobe into tentorial notch

Downward displacement of brain stem through tentorial notch
Herniation Syndromes

- **Supratentorial herniation**
  - **Uncal**
    - Uncus or hippocampal gyrus (or both) shifts from the middle fossa through the tentorial notch into the posterior fossa
  - **Central**
    - Downward shift of the diencephalon through the tentorial notch
  - **Cingulate**
    - Cingulate gyrus shifts under the falx cerebri
Herniation Syndromes

- Infratentorial herniation
  - Cerebellar tonsil shifts through foramen magnum
Cerebral Edema

- Increase in the fluid (intracellular or extracellular) within the brain

- Types
  - Vasogenic due to increased capillary permeability (injury)
  - Cytotoxic toxic factors damage brain cells
  - Ischemic cells die release digestive enzymes
  - Interstitial CSF gets into extracellular spaces of brain tissue
Hydrocephalus

- Excess fluid within the cranial vault, subarachnoid space, or both
- Caused by interference in CSF flow
  - Decreased reabsorption
  - Increased fluid production
  - Obstruction within the ventricular system
Hydrocephalus

- Noncommunicating hydrocephalus
  - Obstruction within the ventricular system, seen more in kids
- Communicating (extraventricular) hydrocephalus: due to defective reabsorption of CSF, seen more in adults
- Acute hydrocephalus due to trauma
Alterations in Motor Function

- **Muscle tone**
  - **Hypotonia**: decreased muscle tone
  - **Hypertonia**: increased muscle tone
  - **Spasticity**: hyperactive stretch reflexes
  - **Gegenhalten (paratonia)**: the involuntary resistance to passive movement of the extremities in proportion to the force
  - **Dystonia**: sustained involuntary twisting movements, may be because reciprocal inhibition messed up
  - **Rigidity**: overactive gamma motor neurons
Upper motor neuron

- Paresis (partial paralysis) and paralysis
  - Pyramidal motor syndromes
  - Upper motor neuron syndromes
    - Hemiparesis or hemiplegia
    - Paraparesis or paraplegia
    - Quadriparesis or quadriplegia
    - **Spinal shock:** loss of sensation with motor paralysis with gradual recovery of reflexes, following a spinal cord injury – most often a complete transection. Reflexes in the spinal cord inferior are depressed (hyporeflexia) or absent (areflexia) but then return hyperactive,
Alterations in Movement

- Lower motor neuron syndromes
  - Flaccid paresis or flaccid paralysis
  - Hyporeflexia or areflexia
  - Fibrillation: contraction of a single muscle fiber, not seen clinically
  - Fasciculations: muscle quivers
Lower Motor Neuron Syndromes
Lower Motor Neuron Syndromes

- Amyotrophies: any lower motor neuron syndrome in ant horn or cranial nerve motor nuclei
  - **Paralytic poliomyelitis** caused by immune response to infection
  - **Nuclear palsies**: caused by damage to several cranial nerve nuclei, called **bulbar palsy** if it involves IX, X, XII
  - **Progressive spinal muscular atrophy**: damaged anterior horns due to inherited metabolic disorders
Alterations in Movement

- Motor cortex
  1. Birth injuries (cerebral palsy of childhood)
  2. Neoplasms
  3. Trauma
  4. Inflammations

- Internal capsule
  1. Vascular lesions (CVA, thrombosis, embolism, hemorrhage, aneurysms)
  2. Neoplasms
  3. Trauma
  4. Inflammations

- Brain stem
  1. Demyelinating diseases (multiple sclerosis)
  2. Vascular lesions
  3. Neoplasms
  4. Trauma
  5. Inflammations
  6. Degenerative diseases (Parkinson disease)

- Spinal cord
  1. Demyelinating diseases
  2. Neoplasms
  3. Trauma
  4. Inflammations

- Lower motor neuron
  - Motor end-plate
  - Muscle spindle (sensory)
  - Peripheral nerve
  - Myoneural junction
    1. Myasthenia gravis (varied locations)
  - Primary muscle
    1. Muscular dystrophies
    2. Myasthenia gravis
    3. Polymyositis

- Upper motor neuron
  - Bulbar
    1. Bulbar palsies
  - Anterior horn cell
    1. Poliomyelitis
    2. Motor system disease
    3. Polyneuritis (varied locations)
Bell’s Palsy

- Inflammation of the VII Cranial Nerve
- Rapid onset of symptoms and usually resolves spontaneously in 80% to 90% of patients
- Age Group 25 – 50 years old
- Unknown cause
  - ? Autoimmune
  - ? Viral
  - ? Ischemia
Bell’s Palsy

- Pathophysiologic Changes
  - Sagging Facial Muscles
  - Impaired ability to close eye
  - Excessive tearing

- Treatment:
  - Antiviral meds
  - Steroids
Alterations in Movement

- **Hyperkinesia**
  - Excessive movement
  - Chorea, tremor at rest, postural tremor, etc.

- **Paroxysmal dyskinesias**: involuntary movement disorders characterized by attacks of hyperkinesis with intact consciousness

- **Tardive dyskinesia**: involuntary, repetitive body movements frequently appear after long-term or high-dose use of antipsychotic drugs
Extrapyramidal Motor Syndromes

- Basal ganglia motor syndromes
- Cerebellar motor syndromes
Alterations in Movement

- Huntington disease
  - Also known as “chorea”
  - Autosomal dominant hereditary-degenerative disorder
  - Severe degeneration of the basal ganglia (caudate nucleus) and frontal cerebral atrophy
    - Depletion of gamma-aminobutyric acid (GABA)
    - Hyperkinesia manifest starting in their 30s #13 on House
Alterations in Movement

- Hypokinesia
  - Decreased amount of movement
  - Akinesia no movement
  - Bradykinesia slow movement
Parkinson Disease

- Severe degeneration of the basal ganglia (corpus striatum) involving the dopaminergic nigrostriatal pathway
  - Parkinsonian tremor
  - Parkinsonian rigidity
  - Parkinsonian bradykinesia
  - Postural disturbances
  - Autonomic and neuroendocrine symptoms
  - Cognitive-affective symptoms
Parkinson Disease
Disorders of Posture (Stance)

- **Decorticate posture**: arms flexed, or bent inward on the chest, the hands are clenched into fists, and the legs extended and feet turned inward. *Indicates that there may be damage to areas above the midbrain (cerebral hemispheres, the internal capsule, and the thalamus).*

- **Decerebrate posture**: arms and legs are extended and rotated internally. The patient is rigid, with the teeth clenched. The signs can be on just one or the other side of the body or on both sides, and it may be just in the arms and may be intermittent. *Indicates brain stem damage, It is exhibited by people with lesions or compression in the midbrain and lesions in the diencephalon.*
Disorders of Posture (Stance)
Disorders of Expression

- **Hypermimesis** overactive expression of emotions (crying, laughing,) cause? Is this what is wrong with our balling Speaker of the House

- **Hypomimesis** lack of or really low expression of emotions, possibly due to brain damage

- **Apraxias** caused by damage to specific areas of the cerebrum, characterized by loss of the ability to execute or carry out learned purposeful movements, despite having the desire and the physical ability to perform the movements. It is a **disorder of motor planning**, which may be acquired or developmental, but may not be caused by incoordination, sensory loss, or failure to comprehend simple commands
Hypermimesis