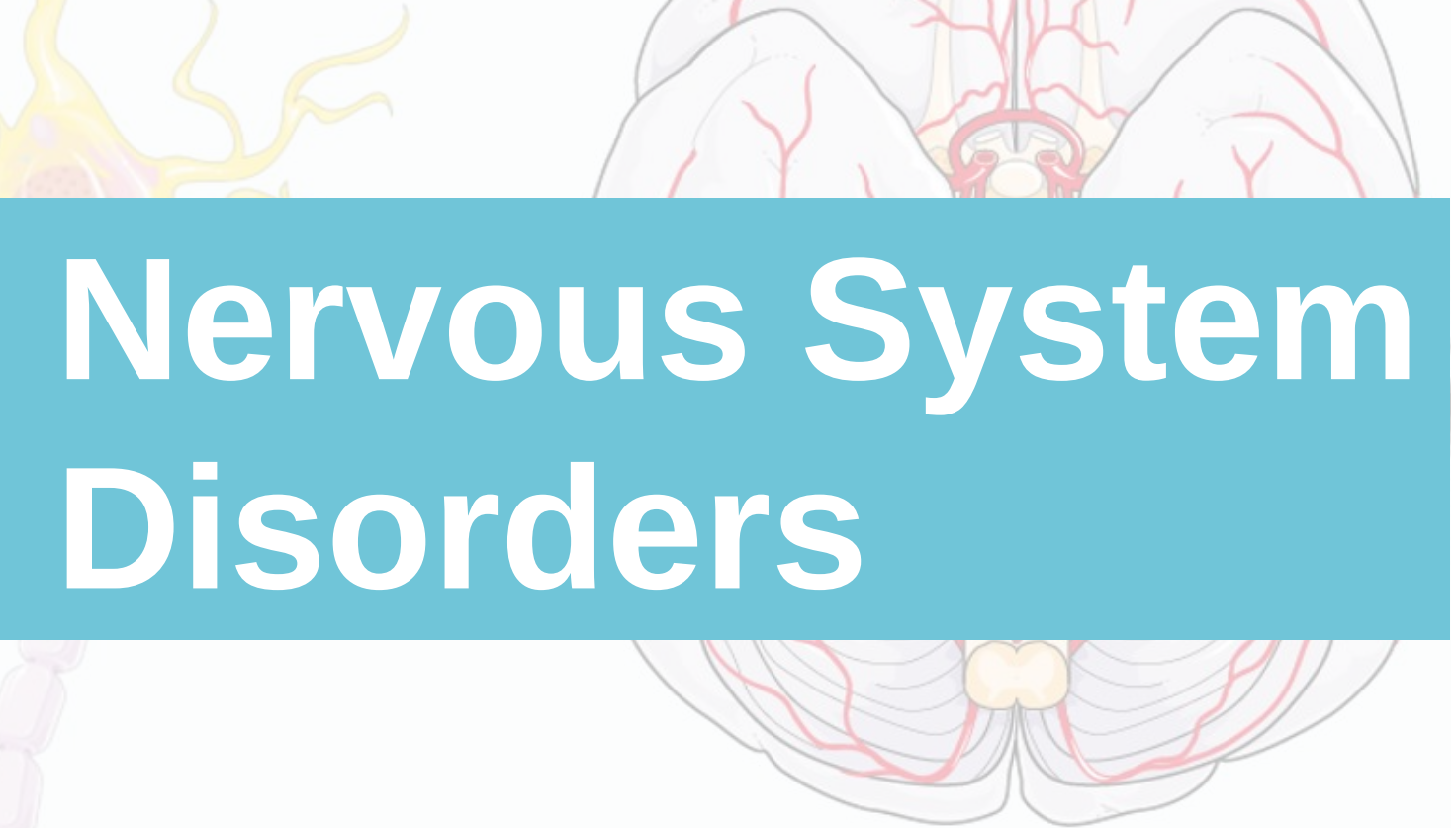
# Nervous System Disorders



# Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

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- Absence Seizures
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- Parkinson’s Disease
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- Spinal Cord Injury
- Spinal Shock
- Tonic-Clonic Seizures
- Trigeminal Neuralgia

# 1 Absence Seizures

- Usually a disease of children
- The child appears to be daydreaming and has no recall of the event.
- Abnormal EEG.



**Pathophysiology**

- Generalized seizures that may be due to alternating gamma-aminobutyric acid type B (GABAB) receptor–mediated inhibition alternating with glutamate-mediated excitation.
- Calcium channel abnormalities are also implicated in the genesis of absence seizures.

# 2 Alzheimer's Disease

- Short-term memory loss
- Forgetfulness
- Confusion
- Inability to recognize loved ones or self



**Pathophysiology**

- Neuropathologic findings in SDAT include amyloid plaques, neurofibrillary tangles, and synaptic and neuronal cell death.
- Degeneration occurs first in the hippocampus, (short-term memory), then damage spreads to the temporal area. Frontal damage causes personality changes and incontinence.
- Acetylcholine levels in the cerebral cortex become deficient.
- Ventricles of the brain become larger as the brain tissue is destroyed. The brain shrinks in size.

# 3 Amyotrophic Lateral Sclerosis

- Fasciculations and atrophy of muscle groups with progressive weakness
- A degenerative neuromuscular disease.



## Pathophysiology

- Also known as Lou Gehrig's disease, ALS is a progressive neurodegenerative disease.
- The upper and lower motor neurons degenerate and form scar tissue, disrupting nerve transmission and leading to muscle atrophy.
- Results in swallowing and breathing difficulty.
- A genetic link is suspected as the cause. Onset is usually between ages 40 and 70 years and more prevalent in men than in women; survival varies from 3–10 years or more.

# 4 Atonic Seizures

- Sudden loss of muscle tone causing a “drop attack.”



**Pathophysiology**

- Classified as a generalized seizure, with juvenile onset lasting into adulthood. The cerebral cortex, subcortical, and spinal areas are believed to be involved in the development of generalized seizures. The electrical discharge from the cortex produces stimulation of the muscles through the motor centers.
- Evidence indicates that an abnormality exists among neurotransmitters (e.g., gammaaminobutyric acid [GABA]) or in calcium, potassium, or sodium channel activity.

# 5 Autonomic Dysreflexia

- In spinal cord injuries above T6
  - Sudden onset of headache
  - Nasal stuffiness
  - High BP
  - Flushed skin above the level of injury

**Pathophysiology**

- SCI above T6 receive peripheral sensory impulses from below the injury via the spinothalamic tract that stimulates a large sympathetic release of norepinephrine, dopamine β-hydroxylase, and dopamine. These neurotransmitters cause vasoconstriction (↑ BP) and skin pallor below the area of injury.
- The brainstem reacts to the ↑ BP by parasympathetic nervous innervation above the level of injury.
- Relieving the noxious stimuli stops the sensory signal and therefore the sympathetic response.

# 6 Bell's Palsy

- Unilateral drooping of the face
- Inability to blink the eye

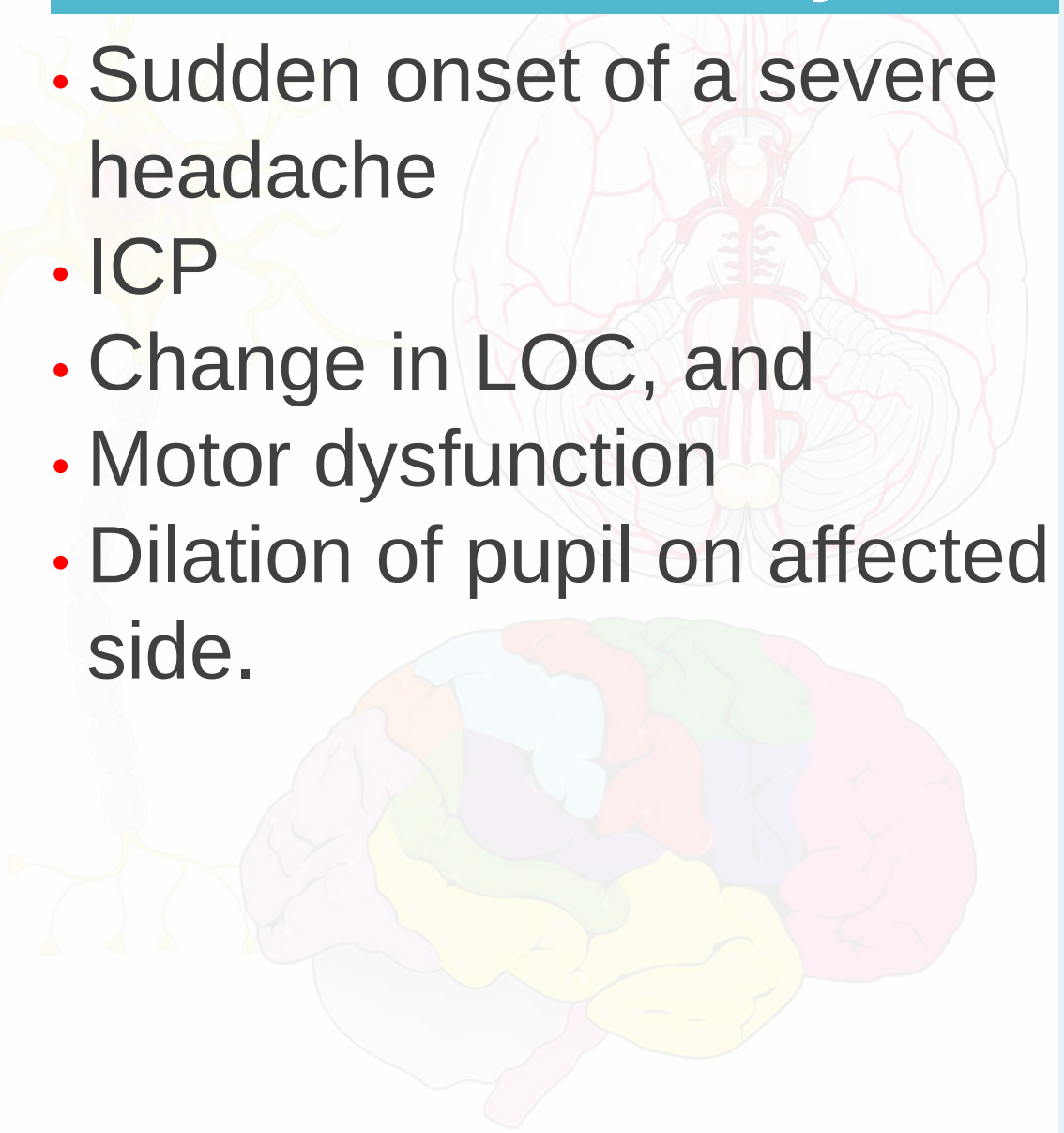


**Pathophysiology**

- Inflammation thought to be caused by autoimmune, viral, bacterial, or traumatic processes of the seventh cranial nerve (facial nerve), causing interruption of nerve transmission.
- Motor control is lost usually on one side of the face, although it can occur bilaterally (1%).
- More likely to occur in pregnancy, immune dysfunction (e.g., human immunodeficiency virus), or diabetes.

# 7 Cerebral Aneurysm

- Sudden onset of a severe headache
- ICP
- Change in LOC, and
- Motor dysfunction
- Dilation of pupil on affected side.



**Pathophysiology**

- The endothelial lining of vessel walls become damaged, lose elasticity, and become vulnerable to rupture. Contributing conditions include HTN, atherosclerosis, natural presence of arterial bifurcation, and congenital high-pressure areas such as AVMs.
- Ruptured aneurysms have abnormally high levels of inflammatory cell infiltration.

# 8 Cerebrovascular Accident

- Inability to form words
- Drooping of the face
- Inability to see out of one eye.



**Pathophysiology**

- Result of a thrombotic block to blood flow or bleeding into the brain that drastically diminishes blood flow to the neurons, causing the cerebrovascular accident (CVA).
- Injured cells fill up with free zinc ions that are believed to hasten their demise.
- Production of glutamate increases the metabolic needs of the already depleted neurons.
- Inflammation causes cerebral edema.

# 9 Complex Partial Seizures (Psychomotor)

- Staring, running away, picking at clothing, or standing still withnlip smacking or other socially awkward behavior.
- May lose consciousness.



## Pathophysiology

- Complex partial seizures arise from one hemisphere of the brain, usually in the temporal lobe.
- May spread and become a generalized seizure.
- Neurotransmitter abnormalities, especially in gamma-aminobutyric acid (GABA), as well as calcium, potassium, or sodium channel abnormalities, may produce the abnormal electrical charge.
- Loss of consciousness occurs and may last 2–15 minutes.

# 10 Concussion

- A blow to the head resulting in changes in LOC.



## Pathophysiology

- Also known as traumatic brain injury, concussion is caused by a nonpenetrating, or closed, head injury. Mechanisms of injury include acceleration injury, deceleration injury, or a combination of both. Rotational injuries cause traumatic shearing of the brain tissue. Most injuries are related to automobile accidents, but playing contact sports and falls are also frequent causes.
- After injury, the compromised cells require increased glucose in order to remain alive. However, with the onset of cerebral edema, the capillary bed may become displaced from the cells by fluid. With decreased blood flow, neuronal loss can continue to occur.

# 11 Encephalitis

- Inflammation of the brain leading to ICP
- most frequently caused by viruses (e.g., West Nile virus), parasites,
- toxins, bacteria, vaccines, or fungi.

**Pathophysiology**

- Neurons are damaged and inflamed, leading to cerebral edema and increased ICP.
- Causative agents are viruses, ticks, mosquitoes, parasites, toxins, bacteria, vaccines, or fungi. Those with a compromised immune system, the very young, and the very old are especially at risk.
- Herpes simplex virus may be the most common non-insect-borne cause of the disease.

# 12 Epidural and Subdural Hematoma

- Head injuries that may be arterial or venous in nature, causing ICP and change in LOC.
- May cause rapid ICP or be insidious, chronic, or become fatal.

## Pathophysiology

- Traumatic brain injury that is nonpenetrating and caused by rotational injury, acceleration injury, deceleration injury, or both.
- Epidural hematomas are arterial, so symptoms are more severe due to rapid accumulation of blood above the dural layer.
- Subdural hematomas are venous, so symptoms may be more insidious.
- These hematomas may occur together.

# 13 Guillain-Barré Syndrome

- Paralysis of the legs, ascending to the upper body.
- May affect the ability to breathe on one's own.



## Pathophysiology

- GB syndrome (inflammatory polyneuritis), is an inflammatory disorder characterized by a distinct progression of paralysis. GB often follows a viral infection. Occurs in those older than age 45 years and with higher frequency in Caucasians than in African Americans.
- Peripheral nerves are infiltrated by immune cells that lead to inflammation and demyelination of the axon. Paralysis begins in the legs and ascends. If the disease reaches the lungs, respiratory support is required. The plateau stage is the most severe, but it signals the end of the progression; remyelination occurs and the symptoms regress.
- A descending form of the disease exists.
- Miller-Fisher syndrome causes ataxia and extra ocular paralysis but no respiratory or sensory loss.

# 14 Huntington's Disease

- Onset of jerking movements of the upper extremities, face, and neck progressing to the rest of the body accompanied by progressive psychotic behavior.
- Genetic testing reveals mutation of the IT15 gene resulting in huntingtin protein.

**Pathophysiology**

- A disorder that causes a mutation in the IT15 gene that results in transcription of an abnormal protein called huntingtin protein. Abnormalities in DNA trigger cellular death. Cells affected are those that control motor and cognitive function.
- An autosomal dominant disorder; each offspring of an affected parent has a 50% chance of inheriting the disorder.
- Onset may be in childhood or in midlife.

# 15 Malignant Hyperthermia

- Temperature increase and muscle rigidity following exposure to anesthetics.
- Creatinine and BUN levels increase.
- Urine is brown in color.



## Pathophysiology

- Autosomal dominant disorder in which exposure to a certain anesthetic agent causes temperature increase and muscle rigidity.
- Affects skeletal muscle tissue primarily. Free ionized calcium concentration can increase to damaging levels and cause multiple contractions of skeletal muscle. The temperature may rise to over 105°F from repeated contractions.
- The hypermetabolic state causes lactate formation, resulting in acidosis.

# 16 Meningitis

- Nuchal rigidity and pain as the meninges are stretched by moving the legs or flexing the neck to the chin.
- Turbid CSF with low glucose and high protein.



## Pathophysiology

- The meninges include the dura matter, arachnoid layer, and pia matter; and surround the brain and spinal cord.
- Causative agents include bacteria, viruses, mycobacteria, fungi, amebas, cancer, and noninfectious sources. Entrance via the respiratory system is most common.
- Infections that occur close to the CNS, basilar fracture, CNS surgery or presence of an indwelling shunt, and blood-borne illnesses cause meningitis.
- TNF-α and IL-1 are major mediators of inflammation that increase permeability and transit of the causative agent through the blood–brain barrier.
- Inflammation causes ↑ ICP in meningitis.

# 17 Multiple Sclerosis

- Exacerbating and remitting periods of degenerating motor function.
- The MRI shows demyelination of the white matter of the brain.

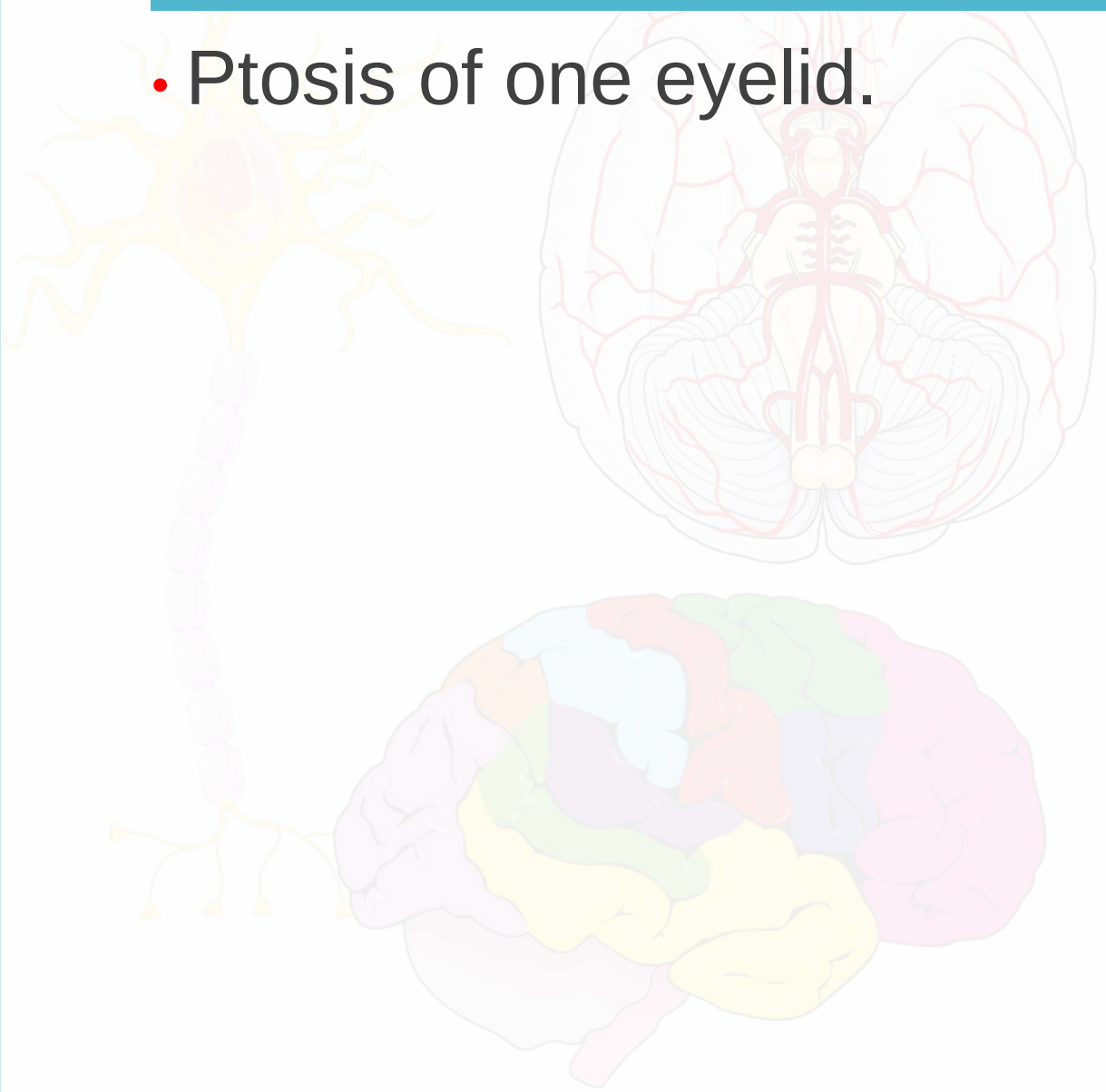


**Pathophysiology**

- Exacerbating and remitting disease characterized by demyelination of brain white matter, damage to axons, and decreased number of oligodendrocytes in the CNS affecting young adults (ages 20–40) and women more than men.
- Autoimmune inflammatory disease involving cell-mediated (T-cell) and antibody (B-cell) activity.
- Exacerbation of symptoms can be caused by extreme heat or cold, fatigue, infection, stress, or pregnancy.

# 18 Myasthenia Gravis

- Ptosis of one eyelid.

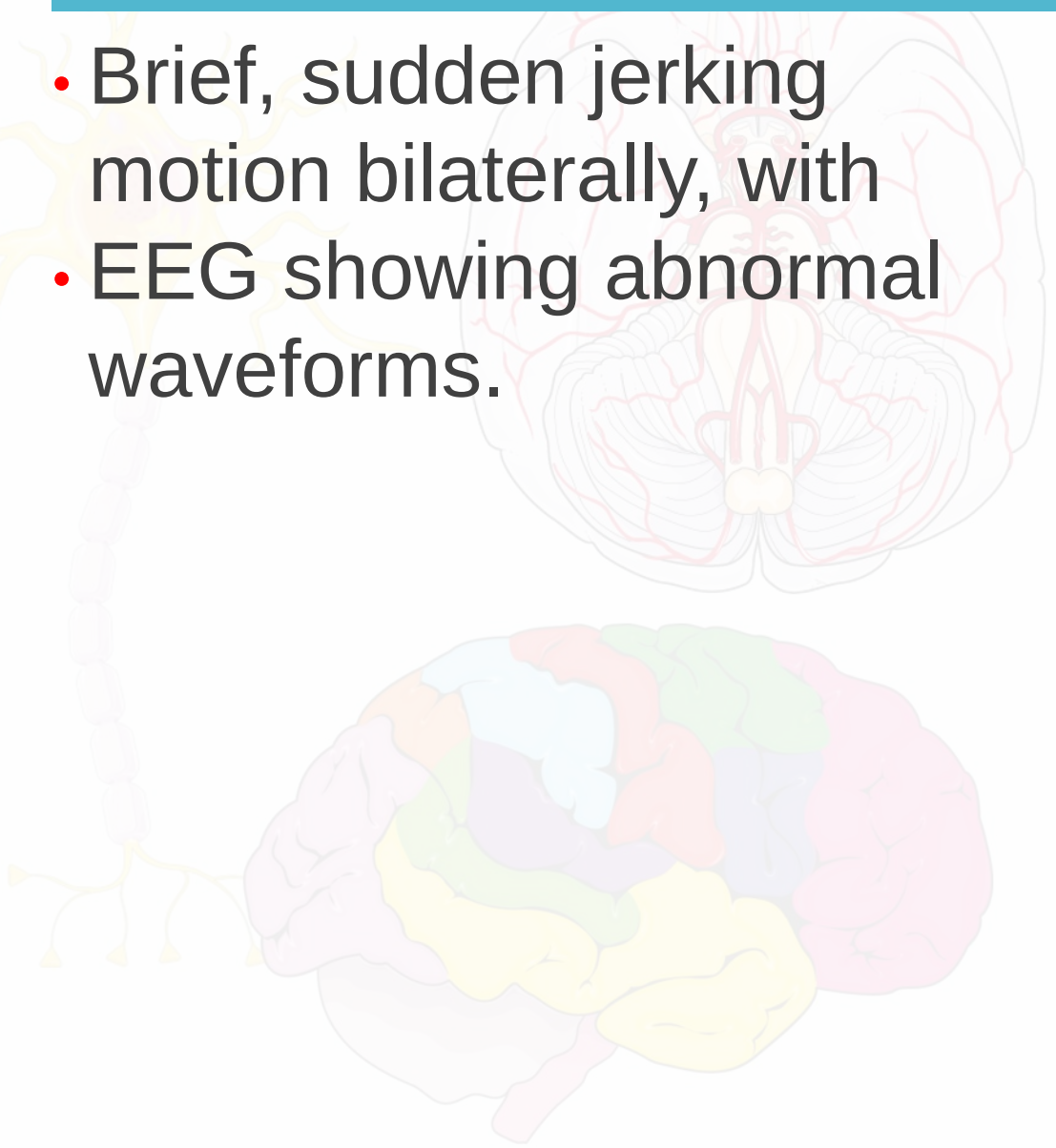


## Pathophysiology

- Meaning “grave muscle weakness,” MG is an autoimmune disease that produces antibodies that attack AChR in the NMJ of skeletal muscles.
- ACh molecules are inactivated by the enzyme AChE, which is abundantly present at the NMJ.
- The disease involves periods of exacerbation and remission.

# 19 Myoclonic Seizures

- Brief, sudden jerking motion bilaterally, with
- EEG showing abnormal waveforms.

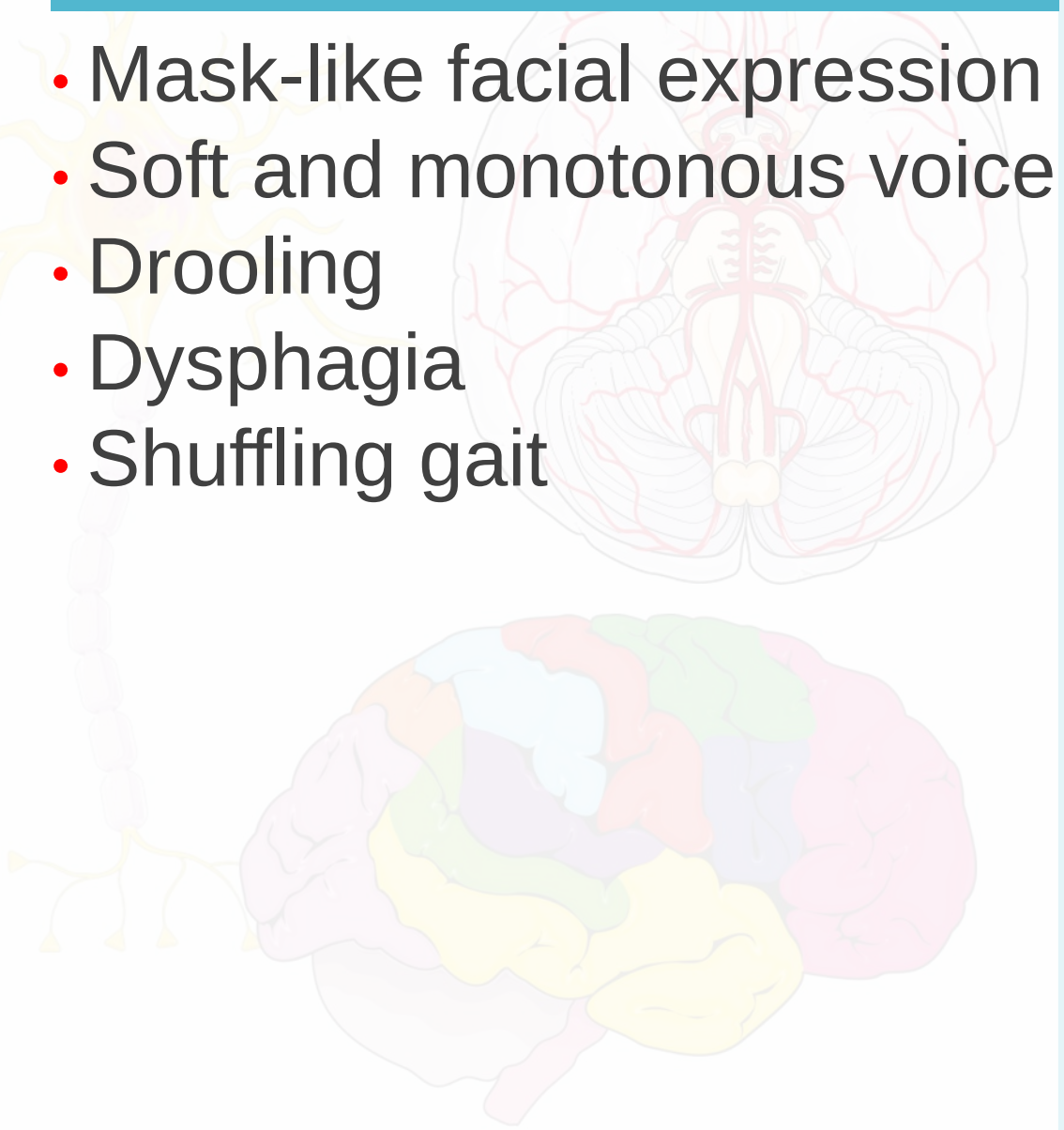


**Pathophysiology**

- A generalized seizure with juvenile onset. The cerebral cortex, subcortical, and spinal areas are believed to be involved in the development of myoclonic seizures. The electrical discharge from the cortex produces stimulation of the muscles through the motor centers.
- Acetycholine (excitatory), serotonin (inhibitory), and GABA (inhibitory) neurotransmitters are present in mismatched amounts in this disorder.
- The mechanism of myoclonic seizure disorder is related to restless leg syndrome.

# 20 Parkinson's Disease

- Mask-like facial expression
- Soft and monotonous voice
- Drooling
- Dysphagia
- Shuffling gait



**Pathophysiology**

- Under the cerebral cortex are interconnected areas of gray matter (basal ganglia), which are involved in controlling voluntary movement. Adjacent to the basal ganglia are cells of the substantia nigra that produce the neurotransmitter dopamine necessary to produce smooth and coordinated muscle movement. Death of cells in the substantia nigra leads to decreased levels of dopamine production, and impairment of EP tract.
- As dopamine levels decrease, acetylcholine levels increase.

# 21 Simple Partial Seizures (Focal)

- Lip smacking, picking at clothing, or chewing behaviors of which the client is unaware.
- Consciousness is not lost.

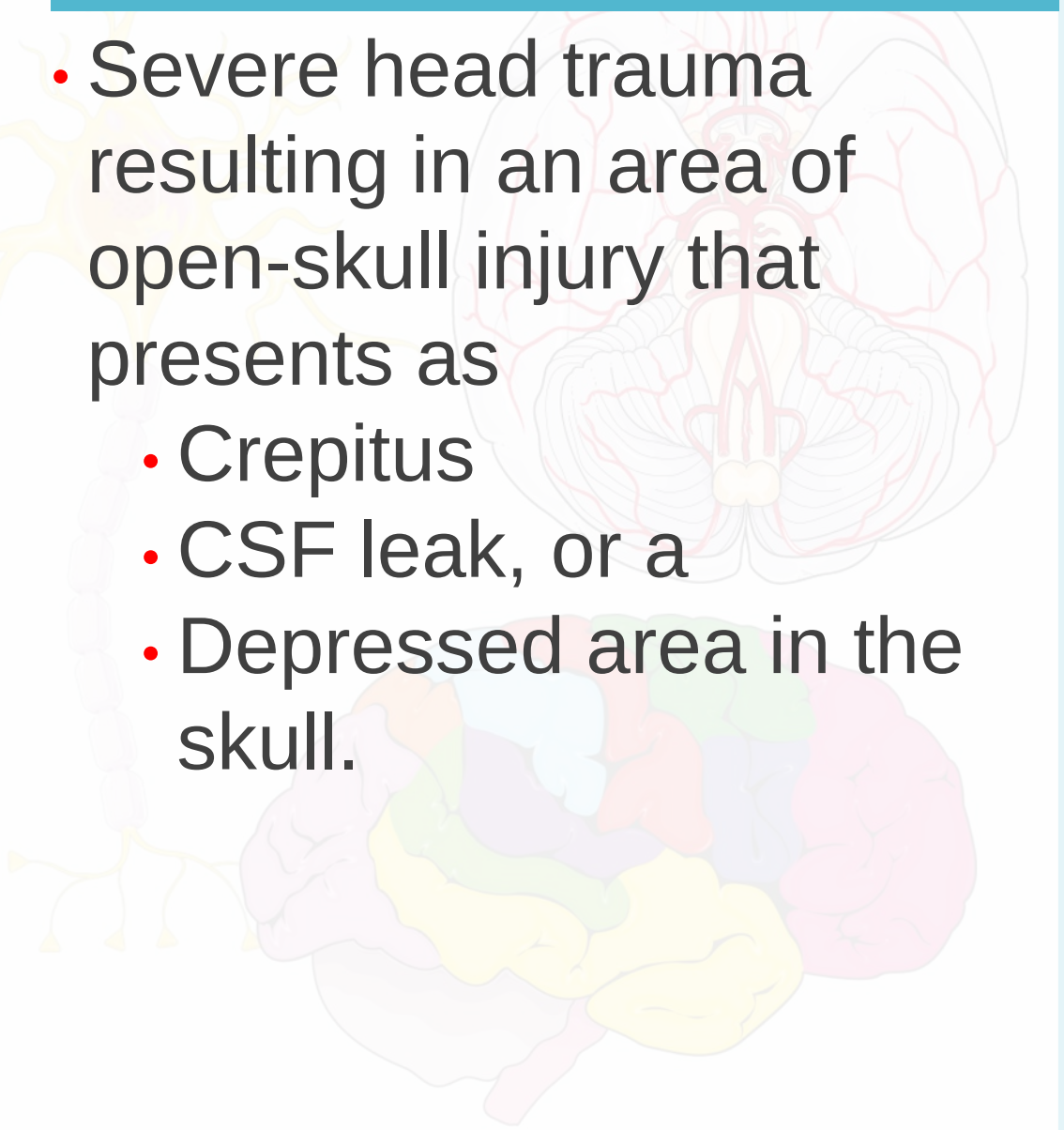


## Pathophysiology

- Simple partial seizures, or focal seizures, arise from one hemisphere of the brain, usually in the temporal lobe.
- May spread to the parietal lobe, causing transient paresthesias of the body on the opposite side of the epileptogenic focus, usually beginning in the finger, arm, and hand and then spreading to the leg and face.
- Neurotransmitter abnormalities, especially in gamma-aminobutyric acid (GABA), as well as calcium, potassium, or sodium channel abnormalities, may produce the abnormal electrical charge.
- Simple partial seizures may spread and become a generalized seizure.

# 22 Skull Fracture

- Severe head trauma resulting in an area of open-skull injury that presents as
  - Crepitus
  - CSF leak, or a
  - Depressed area in the skull.



**Pathophysiology**

- Loss of integrity of the cranial bones and/or meninges causing damage to the underlying brain tissue and creation of an avenue of infection to the CNS.
- Types include linear, comminuted, depressed, compound, and basilar.
- Acute cerebral edema and ICP occur from neuronal damage, hemorrhage, inflammation, infection, potassium leaking to the extracellular space, and lactate buildup from glycolysis.

# 23 Spinal Cord Injury

- Loss of sensation, movement, or both after trauma to the neck, thorax, lumbar, or sacral area.



## Pathophysiology

- Nerve fibers of the spinal cord are nonregenerative.
- Central cord syndrome results in weakness or paralysis that affects the upper extremities more than the lower extremities.
- Anterior cord syndrome, caused by trauma or ischemia results in weakness and decreased pain and temperature sensation below the damaged area.
- Posterior cord syndrome causes ataxia, but strength and sensation are preserved.
- Brown-Séquard syndrome results in paralysis on the affected side and sensation loss on the opposite side of injury.
- Cauda equina syndrome results in bowel and bladder dysfunction and some leg paresthesia.

# 24 Spinal Shock

- Period of time after SCI in which there is no motor or sensory transmission.
- Can last a day to several months.



**Pathophysiology**

- SCI causes a concussion-like injury to neurons known as spinal shock in which neurons below the level of the SCI are incapable of any sensory or motor transmission.
- Cytokines cause an inflammatory condition in the affected neurons.
- Phase 1 spinal shock is characterized by the absence of all reflex arcs below the SCI.
- Phase 2 spinal shock is characterized by the return of some of the reflex arcs, which signals the beginning of the end of spinal shock.
- Phase 3 and 4 are characterized by strong reflexes that occur with minor stimulation and may be followed by autonomic dysreflexia, hyperreflexia, and clonus.

# 25 Tonic-Clonic Seizures

- Presence of an aura followed by loss of consciousness with alternating cycles of stiffness and jerking movements lasting 1–2 minutes.

## Pathophysiology

- Tonic-clonic seizures are generalized seizures.
- Pathology includes an area of hyperexcitable neurons. This is the epileptogenic focus.

# 26 Trigeminal Neuralgia

- Severe knife-like facial pain unilaterally in response to movement of the musculature of the face, a touch, or cool breeze.



**Pathophysiology**

- Vascular compression or other structural disorders of the vasculature cause inflammation of the fifth cranial nerve, or TN.
- Inflammation and compression cause demyelination and remyelination of the nerve. This abnormal myelination causes abnormal sensory discharge, felt by the client as intense pain.
- Trigger zones include the lips, upper or lower gums, cheeks, forehead, and side of the nose.