Pathophysiology of the Nervous System
Course Description:
This course is designed to assist practitioners to use the pathophysiology of the nervous system to understand neurological symptoms they encounter in a clinical setting.

Course Objectives:
At the end of this course students will:
❖ Recognize neurological symptoms presented by a patient.
❖ Understand what the pathological pathways that may lead to the symptom presentation.
❖ Relate the pathology to the disease processes that are occurring.
❖ Use the understanding of pathology to assess the progress of your treatment plan.
Why study pathophysiology?

Understand the expected progression of the disease in its untreated state.
Assess dispassionately one’s ability to manage the case and what additional professional assistance is needed.
Ascertain what criteria are appropriate for determining whether healing progress is being induced by therapeutic interventions.
Assess carefully whether interventions are achieving desired results.
What are we treating; the patient who has the disease or the disease that has the patient?

The issues are in the tissues.

Why this patient?
Why is that tissue/organ the focus of pathosis?
Why did pathosis take that form of expression?
Why did pathosis manifest at time of onset?
How the Nervous System May Fail

- Traumatic injury
- Failure of blood flow
- Inflammation
  - Infection
  - Autoimmune disease
- Genetic abnormalities
- Masses
- Neurodegenerative diseases
Pathologies of the Central Nervous System

Life threatening neurological problems: stroke, subarachnoid hemorrhage, subdural hematoma, space-occupying lesion, meningitis, encephalitis, cerebral abscess, skull fracture, and vertebral fracture.

headaches, vertigo, epilepsy, traumatic brain injury, vertebral disc disease, spinal stenosis, dementia, Parkinson’s disease, cranial synostosis, Tourette’s syndrome.
Head Injuries

❖ Evidence of increasing intracranial pressure
   Decreasing level of consciousness. Falling Glasgow Coma Score
   Cushing’s response: Bradycardia, Hypertension, and diminished respiratory rate
   Dilated pupils associated with decorticate and decerebrate posturing

❖ Complications
   Subdural, Epidural and, Subarachnoid hematoma
   Cerebral contusion: focal injuries characterized as coup or contrecoup.
   Intracerebral hemorrhage: Massive lesion with bleeding into the brain
   Diffuse axonal injury: microscopic injuries scattered throughout the brain,
   without a mass lesion, in a patient in deep coma
A hematoma is an accumulation of blood or blood clot in one area. It is potentially life threatening depending on the area affected. They may occur in both the CNS or the peripheral NS.

Brain: subdural, subarachnoid

In the limbs: Compartment syndrome

Small hematomas generally spontaneously resolve
Subarachnoid and Subdural

- Symptoms usually noted 48 to 72 hours after the injury
- Type and severity of symptoms depends on the location of the injury and the extent of the bleed.
- This is an emergency and emergency services should be contacted.
Cerebral Vascular Accident

Signs and symptoms depend upon the location of the lesion.

Stroke or CVA is caused by a secessions of blood flow any part of the brain. The blood flow may be interrupted by an arterial occlusion or a spasm of a vessel. It may also occur if there is a rupture of a vessel as in the case of a ruptured aneurism.

In the case of interrupted flow the condition is called a trans ischemic attack (TIA).
Seizures are caused by paroxysmal discharges from groups of neurons, which arise as a result of excessive excitation or loss of inhibition. The key unit of neurotransmission is the synapse, and the fundamental components of synapses are ion channels.
Adults

- **Generalized seizures**
  - Classic tonic – clonic (grand mal)
  - Non-convulsive generalized seizures (petit mal) or absence seizures

- **Partial Seizures**
  - Simple: brief sensory or motor manifestation without loss of consciousness (Jacksonian)
  - Complex: Patients manifest mental and psychological symptoms including affect changes, confusion, and hallucinations

- **Status epilepticus**
  - Seizure greater than 1 hour or serial seizures that produce an enduring epileptic condition for greater than 1 hour
  - Life threatening emergency with mortality rate of 10 – 12%
Children

- **Neonates**
  - Subtle abnormal motor activity
  - Facial movements
  - Eye deviations
  - Eye lid fluttering
  - Lip smacking / sucking
  - Respiratory alterations
  - Apnea
  - Seizure activity
    - Focal or generalized tonic seizures
    - Focal or multifocal clonic seizures
    - Myoclonic movements

- **Older infants and Children**
  - Present with the same categories of seizures as adults, including Status

- **Febrile Seizures**
  - Often the first sign of a febrile illness
  - Generally tonic / clonic in nature
  - Can be Limpness
  - Staring with stiffness
  - Jerking without prior stiffness
Space occupying lesions

Malignant

- Brain stem – Astrocytoma
- Cerebellum – Astrocytoma, medulloblastoma
- Cerebrum – Astrocytoma, oligodendroglioma
- Cranial nerves – Glioma of the optic nerve, Schwannoma of 5<sup>th</sup> and 8<sup>th</sup> cranial nerve
- Meninges – meningioma, sarcoma, glioma
Vertigo

An abnormal sensation of rotary movement associated with difficulty in balance, gait, and navigation of the environment. Sensation may be subjective; the patient feels as if he is moving in relation to his environment, or it may be objective: he feels as if the environment is moving in relation to him.
Causes of Vertigo

❖ Lesions or disturbances of the inner ear

❖ Problems with the 8th cranial nerve

❖ Lesions of the vestibular nuclei or their pathways in the brain stem or cerebellum
Meniere's Disease The exact pathophysiology of Ménière disease is controversial. The underlying mechanism is believed to be distortion of the membranous labyrinth resulting from over accumulation of endolymph. Some authors have questioned whether endolymphatic hydrops is actually a marker of disease rather than a cause.

Tinnitus
Vertigo
Nausea and vomiting
Deafness
Problems in the ear

Labyrinthitis

- Post viral
- Purulent
Vestibular neuronitis

An inflammation of the vestibular division of the 8th cranial nerve thought to be viral in origin. Though the exact etiology remains largely unknown, vestibular neuronitis appears to be a sudden disruption of afferent neuronal input from 1 of the 2 vestibular apparatuses. This imbalance in vestibular neurologic input to the central nervous system (CNS) causes symptoms of vertigo. It presents with vertigo, nausea and vomiting. There is persistent nystagmus toward the affected side. It is considered to be self limiting and may occur once or multiple times over a 12 to 18 month course.
Benign paroxysmal positional Vertigo is caused by calcium carbonate particles called otoliths that are inappropriately displaced into the semicircular canals of the vestibular labyrinth of the inner ear. ... The otoliths may become displaced from the utricle by aging, head trauma, or labyrinthine disease. Vertigo, nausea, vomiting and nystagmus may occur.
Craniosynostosis is a defect involving the fusion of one or more bones in the skull before it has finished growing which affects the head size and shape and can affect the growth of the brain. The defect is often associated with other conditions. Symptoms are determined by which skull bones are prematurely fused.
The list of signs and symptoms mentioned in various sources for Craniosynostosis includes the 7 symptoms listed below:

- Abnormal head shape
- Increased intracranial pressure
- Developmental delay
- Mental retardation
- Seizures
- Blindness
- Dysmorphic facial features
Tourette syndrome

A neurological disorder in which you display unusual movements or sounds over which you may have little or no control (tics). For instance, you may repeatedly blink your eyes, shrug your shoulders or jerk your head. TS is thought to result from a complex interaction between social and environmental factors and multiple genetic abnormalities.
Tics are the hallmark sign of Tourette syndrome. Symptoms range from very mild to severe and debilitating. The first sign of Tourette is often a facial tic, such as eye blinking. But the spectrum of tics that people experience is amazingly diverse, and there's no typical case.

The tics involve movement (motor tics) and sound (vocal tics). They are classified in two ways:

- Simple tics, which are sudden, brief and repetitive and may involve a limited number of muscle groups
- Complex tics, which are distinct, coordinated patterns of movements involving several muscle groups
Some of the more common tics seen in Tourette syndrome:

Motor tics
- Simple tics: Eye blinking, Touching the nose, Head jerking, Touching other people, Shoulder shrugging, Smelling objects, Eye darting, Obscene gestures, Finger flexing, Flapping the arms, Sticking the tongue out, Hopping

Complex tics
- Hiccupping, Using different voice intonations, Yelling, Repeating one's own words or phrases, Throat clearing, Repeating others' words or phrases, Barking, Obscene language
The pathophysiology of Parkinson's disease is death of dopaminergic neurons as a result of changes in biological activity in the brain with respect to Parkinson's disease. There are several proposed mechanisms for neuronal death in PD; however, not all of them are well understood. The disease if characterized by a loss of neurons in an area of the brain called the substantia nigra pars compacta, and the presence of globs of a protein called alpha-synuclein found in neurons, called Lewy bodies.
Healthy Patient

Parkinson’s Patient
Parkinson's disease develops gradually, often starting with a barely noticeable tremor in just one hand. But while tremor may be the most well-known sign of Parkinson's disease, the disorder also commonly causes a slowing or freezing of movement.

Friends and family may notice that your face shows little or no expression and your arms don't swing when you walk. Speech often becomes soft and mumbling. Parkinson's symptoms tend to worsen as the disease progresses.

While there is no cure for Parkinson's disease, many different types of medicines can treat its symptoms. In some cases, your doctor may suggest surgery.
The symptoms of Parkinson's disease vary from person to person. Early signs may be subtle and can go unnoticed for months or years. Symptoms typically begin on one side of the body and usually remain worse on that side. Parkinson's signs and symptoms may include:

- **Tremor.** The characteristic shaking associated with Parkinson's disease often begins in a hand. A back-and-forth rubbing of your thumb and forefinger, known as pill-rolling, is common. However, many people with Parkinson's disease do not experience substantial tremor.

- **Slowed motion (bradykinesia).** Over time, Parkinson's disease may reduce your ability to initiate voluntary movement. This may make even the simplest tasks difficult and time-consuming. When you walk, your steps may become short and shuffling. Or your feet may freeze to the floor, making it hard to take the first step.

- **Rigid muscles.** Muscle stiffness often occurs in your limbs and neck. Sometimes the stiffness can be so severe that it limits the range of your movements and causes pain.
Impaired posture and balance. Your posture may become stooped as a result of Parkinson's disease. Imbalance also is common, although this is usually mild until the later stages of the disease.

Loss of automatic movements. Blinking, smiling and swinging your arms when you walk are all unconscious acts that are a normal part of being human. In Parkinson's disease, these acts tend to be diminished and even lost. Some people may develop a fixed staring expression and unblinking eyes. Others may no longer gesture or seem animated when they speak.

Speech changes. Many people with Parkinson's disease have problems with speech. You may speak more softly, rapidly or in a monotone, sometimes slurring or repeating words, or hesitating before speaking.

Dementia. In the later stages of Parkinson's disease, some people develop problems with memory and mental clarity. Alzheimer's drugs appear to alleviate some of these symptoms to a mild degree.
Spinal Cord Injuries
Spinal cord injuries can be caused by trauma to the spinal column (stretching, bruising, applying pressure, severing, laceration, etc.). The vertebral bones or intervertebral disks can shatter, causing the spinal cord to be punctured by a sharp fragment of bone. Usually, victims of spinal cord injuries will suffer loss of feeling in certain parts of their body. In milder cases, a victim might only suffer loss of hand or foot function. More severe injuries may result in paraplegia, tetraplegia, or full body paralysis (called Quadriplegia) below the site of injury to the spinal cord.
Damage to upper motor neuron axons in the spinal cord results in a characteristic pattern of ipsilateral deficits. These include hyperreflexia, hypertonia and muscle weakness. Lower motor neuronal damage results in its own characteristic pattern of deficits. Rather than an entire side of deficits, there is a pattern relating to the myotome affected by the damage. Additionally, lower motor neurons are characterized by muscle weakness, hypotonia, hyporeflexia and muscle atrophy.
Spinal shock and neurogenic shock can occur from a spinal injury. Spinal shock is usually temporary, lasting only for 24-48 hours, and is a temporary absence of sensory and motor functions. Neurogenic shock lasts for weeks and can lead to a loss of muscle tone due to disuse of the muscles below the injured site.
The pathophysiology of spinal stenosis is related to cord dysfunction elicited by a combination of mechanical compression and degenerative instability. With aging, the intervertebral disk may degenerate and collapse, leading to spur formation. This results in spinal cord or nerve root impingement. The patients symptoms reflect the area of the spinal column affected.
Lumbar Spinal Stenosis, effects the Legs, calves, and buttocks. Patients with lumbar spinal stenosis may feel pain, weakness, or numbness in the legs, calves or buttocks.

In the lumbar spine, symptoms often increase when walking short distances and decrease when the patient sits, bends forward or lies down.
Cervical Spinal Stenosis effects the Shoulders, arms, and legs. Cervical spinal stenosis may cause symptoms (pain, weakness, or numbness) similar to those experienced by patients with lumbar spinal stenosis.

Hands can exhibit clumsiness and gait and balance disturbances can also occur.
Most thoracic spinal stenosis is due to degenerative changes, arthritis, bone spurs, disc degeneration and other changes due to aging. Patients may experience symptoms across the ribs and occasionally in one or more of the major internal organs. As the degeneration progresses, patients may experience pain in the back and legs, either aching in the legs when walking, or pain that radiates down the back or legs. Additional symptoms may include; problems with walking or loss of bowel or bladder function.
Spinal stenosis may effect sensory as well as motor pathways. In some patients, the pain starts in the legs and moves upward to the buttocks; in other patients, the pain begins higher in the body and moves downward. This is referred to as a “sensory march.”

The pain may radiate like sciatica or may be a cramping pain. In severe cases, the pain can be constant.
Degeneration of the intervertebral disc, often called "degenerative disc disease" (DDD) of the spine, is a condition that can be painful and can greatly affect the quality of one's life. While disc degeneration is a normal part of aging and for most people is not a problem, for certain individuals a degenerated disc can cause severe constant chronic pain.
Peripheral Neurological Disease

- Multiple sclerosis
- Amyotrophic lateral sclerosis
- Myasthenia gravis
- Muscular dystrophy
- Peripheral neuropathy
Multiple sclerosis is an inflammatory demyelinating disease of the CNS in which activated immune cells invade the central nervous system and cause inflammation, neurodegeneration, and tissue damage. The underlying cause is currently unknown although some scientists have suggested an autoimmune process is at work.

Vitamin D deficiency may predict onset. A new, large-scale study in Finnish women suggests that vitamin D deficiency can significantly raise the risk of multiple sclerosis, which makes it a reliable predictive marker for the disease. Studies have also found that MD is more prevalent in northern climates.
**Multiple Sclerosis**

**Pathological Changes**
- Loss of myelin sheath
- Autoimmune response (T and B cell activation)
- Gliosis
- Release of pro-inflammatory mediators - neuroinflammation
- Disrupted synaptic glutamate handling - excitotoxicity

**Synapse Engulfment**

**Current Knowledge**
- Complement tagging of synapses
- Little evidence of microglia phagocytosing pre-synapses
- Robust human data is lacking
Multiple sclerosis may be difficult to diagnose as there are no definitive diagnostic test for this condition. MS generally appear between the ages of 20 and 40. Typically a person is seen after developing two or more distinct episodes of symptoms that resolve yet are consistent with MS. The most common early symptoms of MS include:

- Tingling
- Numbness
- Loss of balance
- Weakness in one or more limbs
- Blurred or double vision
Less common symptoms of MS may include:

- Slurred speech
- Sudden onset of paralysis
- Lack of coordination
- Cognitive difficulties

Generally, all MS symptoms are worse in hot weather
Amyotropic Lateral Sclerosis is a progressive chronic neurodegenerative disorder characterized by death of pyramidal neurons in the motor cortex (upper motor neurons) and motor neurons in the brain stem and central spinal cord (lower motor neurons). Voluntary movement gradually degenerate. The loss of these motor neurons causes the muscles under their control to weaken and waste away, leading to paralysis. The cause of this disease process is still unknown.
ALS strikes in mid-life, most often in the fifth through seventh decades of life. Men are about one-and-a-half times more likely to have the disease as women. It affects about 20,000 Americans with 5,000 new cases occurring in the United States each year.

ALS manifests itself in different ways, depending on which muscles weaken first. Symptoms may include tripping and falling, loss of motor control in hands and arms, difficulty speaking, swallowing and/or breathing, persistent fatigue, and twitching and cramping, sometimes quite severely.
Myasthenia gravis is an autoimmune disease of the neuromuscular junction. The normal neuromuscular junction releases acetylcholine (ACh) from the motor nerve terminal in discrete packages (quanta). The ACh quanta diffuse across the synaptic cleft and bind to receptors on the folded muscle end-plate membrane. Stimulation of the motor nerve releases many ACh quanta that depolarize the muscle end-plate region and then the muscle membrane causing muscle contraction. In acquired myasthenia gravis, the post-synaptic muscle membrane is distorted and simplified, having lost its normal folded shape. The concentration of ACh receptors on the muscle end-plate membrane is reduced, and antibodies are attached to the membrane. ACh is released normally, but its effect on the post-synaptic membrane is reduced. The post-junctional membrane is less sensitive to applied ACh, and the probability that any nerve impulse will cause a muscle action potential is reduced.
Although myasthenia gravis may affect any voluntary muscle, muscles that control eye and eyelid movement, facial expression, and swallowing are most frequently affected. The onset of the disorder may be sudden. Symptoms often are not immediately recognized as myasthenia gravis.

The degree of muscle weakness involved in myasthenia gravis varies greatly among patients, ranging from a localized form, limited to eye muscles to a severe or generalized form in which many muscles sometimes including those that control breathing are affected. Symptoms, which vary in type and severity, may include a drooping of one or both eyelids (ptosis), blurred or double vision (diplopia) due to weakness of the muscles that control eye movements, unstable or waddling gait, weakness in arms, hands, fingers, legs, and neck, a change in facial expression, difficulty in swallowing with shortness of breath, and impaired speech (dysarthria).
Muscular dystrophy is one of a group of genetic diseases characterized by progressive weakness and degeneration of the skeletal or voluntary muscles which control movement. The muscles of the heart and some other involuntary muscles may also be affected in some forms of muscular dystrophy, and a few forms involve other organs as well. Muscular dystrophy can affect people of all ages. Although some forms first become apparent in infancy or childhood, others may not appear until middle age or later.
Signs and symptoms vary according to the type of muscular dystrophy. In general, muscular dystrophy symptoms may include:

❖ Muscle weakness

❖ Apparent lack of coordination

❖ Progressive crippling, resulting in fixations (contractures) of the muscles around your joints and loss of mobility

❖ Specific signs and symptoms vary among the different forms of MD. Each type is different in the age of onset, which parts of the body the symptoms primarily affect and how rapidly the disease progresses
Dystrophinopathies

These types of muscular dystrophies are due to a genetic defect of the protein dystrophin.

Duchenne's muscular dystrophy is the most severe form of dystrophinopathy. It occurs mostly in young boys and is the most common form of MD that affects children. Signs and symptoms of Duchenne's usually appear between the ages of 2 and 3

- Frequent falls
- Large calf muscles
- Difficulty getting up from a lying or sitting position
- Weakness in lower leg muscles, resulting in difficulty running and jumping
- Waddling gait
- Mild mental retardation, in some cases

It first affects the muscles of the pelvis, upper arms and upper legs. By late childhood, most children with this form of muscular dystrophy are unable to walk. Most die by their 20s or early 30s, often from pneumonia, respiratory muscle weakness or cardiac complications.
Myotonic dystrophy

Also known as Steinert's disease, this form of muscular dystrophy produces stiffness of muscles and an inability to relax muscles at will (myotonia), as well as the muscle weakness of the other forms of muscular dystrophy. Although this form of MD can affect children, it often doesn't affect people until adulthood. It can vary greatly in its severity. Muscles may feel stiff after using them. Progression of this form of MD is slow.
Other symptoms may include: Weakening of voluntary muscles that control your arms and legs. This usually begins with the limb muscles farthest from the torso, the muscles of the feet, hands, lower legs and forearms.

Weakening of head, neck and face muscles, which may result in the face having a hollow, drooped appearance.

Weakening of muscles involved in breathing and swallowing. Weaker breathing muscles may result in less oxygen intake and fatigue. Weaker swallowing muscles increase the risk of choking.
Fainting or dizziness, which may indicate that the disease is interfering with the conduction of electrical signals that keep the heart rate normal.

Weakening of muscles of hollow internal organs such as those in the digestive tract and the uterus. Depending on which part of the digestive tract is affected, you may experience problems with swallowing as well as constipation and diarrhea. Weakness of the uterine walls may cause problems during childbirth.

Difficulty sleeping well at night and daytime sleepiness, and inability to concentrate because of the effect of the disease on the brain.

Frontal balding in men.
Peripheral neuropathy, a result of damage to the nerves outside of the brain and spinal cord (peripheral nerves), often causes weakness, numbness and pain, usually in your hands and feet. It can also affect other areas of your body. Your peripheral nervous system sends information from your brain and spinal cord (central nervous system) to the rest of your body. The peripheral nerves also send sensory information to the central nervous system. A dermatome is an area of skin in which sensory nerves derive from a single spinal nerve root. The spinal cord has 31 segments, each with a pair (right and left) of ventral (anterior) and dorsal (posterior) nerve roots that innervate motor and sensory.
Most commonly, peripheral neuropathy may start in the longest nerves the ones that reach to your toes. Specific symptoms vary, depending on which types of nerves are affected.

- Gradual onset of numbness and tingling in your feet or hands, which may spread upward into your legs and arms
- Burning pain
- Sharp, jabbing or electric-like pain
- Extreme sensitivity to touch, even light touch
- Lack of coordination
- Muscle weakness or paralysis if motor nerves are affected
- Bowel or bladder problems if autonomic nerves are affected
Pathologies of Cranial Nerves

- Trigeminal neuralgia (Tic Douloureux)
- Bell's palsy
- Glaucoma
- Loss of smell
- Auditory neuropathy
- Glossopharyngeal nerve
<table>
<thead>
<tr>
<th>Nerve no.</th>
<th>Nerve name</th>
<th>Rhyme</th>
<th>Function</th>
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<tr>
<td>1</td>
<td>Olfactory</td>
<td>On</td>
<td>Smell (not usually tested)</td>
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<tr>
<td>2</td>
<td>Optic</td>
<td>Old</td>
<td>Visual acuity</td>
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<tr>
<td>3</td>
<td>Oculomotor</td>
<td>Olympus’</td>
<td>Opening of eyelids, eye movement (upward/medial, upward/lateral, medial, downward/lateral)</td>
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<td>4</td>
<td>Trochlear</td>
<td>Towering</td>
<td>Eye movement (downward/medial)</td>
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<td>5</td>
<td>Trigeminal</td>
<td>Top</td>
<td>Facial sensation, chewing movements</td>
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<tr>
<td>6</td>
<td>Abducens</td>
<td>A</td>
<td>Eye movement (lateral)</td>
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<tr>
<td>7</td>
<td>Facial</td>
<td>French</td>
<td>Facial muscle movement (except chewing muscles) and eyelid closing</td>
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<tr>
<td>8</td>
<td>Auditory</td>
<td>And</td>
<td>Hearing and balance</td>
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<tr>
<td></td>
<td>(vestibulocochlear)</td>
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<tr>
<td>9</td>
<td>Glossopharyngeal</td>
<td>German</td>
<td>Taste on the posterior third of the tongue (not usually tested)</td>
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<tr>
<td>10</td>
<td>Vagus</td>
<td>Viewed</td>
<td>Uvula (palate muscles) and swallowing</td>
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<td>11</td>
<td>Accessory</td>
<td>A</td>
<td>Shoulder shrug</td>
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<tr>
<td>12</td>
<td>Hypoglossal</td>
<td>Hop</td>
<td>Tongue movement</td>
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The exact pathophysiology of **Trigeminal neuralgia (tic douloureux)**, remains controversial. The etiology of trigeminal neuralgia may be central, peripheral, or both. The trigeminal nerve can cause pain, because its major function is sensory. A lack of inhibitory inputs from large myelinated nerve fibers seems to play a role. The exact mechanism is not understood. The most commonly accepted theory is compression of the trigeminal nerve, usually by a blood vessel, causing it to become irritated. This irritation causes the myelin sheath to erode over time.
Bell palsy, also termed idiopathic facial paralysis (IFP), is the most common cause of unilateral facial paralysis. It is one of the most common neurologic disorders of the cranial nerves. In the great majority of cases, Bell palsy gradually resolves over time, and its cause is unknown. Bell's palsy is thought to result from compression of the seventh cranial nerve at the geniculate ganglion.
Bell's Palsy Symptoms

- Inability to furrow brow
- Drooping eyelid & cannot close eye
- No muscle tone in cheek
- Drooping mouth & cannot smile or pucker lips
The pathophysiology of glaucoma is caused by raised intraocular pressure. It is this raised pressure that compresses and damages the optic nerve. Once the optic nerve is damaged, it fails to carry visual information to the brain and this results in loss of vision. Although the pathogenesis of glaucoma is not fully understood, the level of intraocular pressure is related to retinal ganglion cell death.
The two main types of glaucoma are open-angle and angle-closure. These are marked by an increase of intraocular pressure (IOP), or pressure inside the eye.
In glaucoma, the passage or the drainage channel is blocked, either at its entrance or beyond. When the block is at the entrance it is called Closed Angle Glaucoma. When the blockage is not at the entrance, but beyond, somewhere inside, we call it Open Angle Glaucoma.

In open-angle glaucoma, the trabecular meshwork offers increased resistance to fluid outflow. This causes the pressure to build up inside your eye.

In closed-angle glaucoma, both the uveoscleral drain and the trabecular meshwork become blocked. Typically, this is caused by a damaged iris (colored part of the eye) blocking the outlet.
Anosmia is the complete loss of smell. Fortunately, for most people, anosmia is a temporary nuisance caused by a severely stuffy nose from a cold. Nasal congestion from a cold, allergy, sinus infection, or poor air quality is the most common cause of anosmia. The inflammation of the sinus mucus membranes puts pressure on the olfactory bulbs causing the temporary loss of smell. However, more serious conditions that affect the brain or nerves, such as brain tumors or head trauma, can cause more permanent damage.
Auditory neuropathy may have a number of causes. In some cases, the cause may involve damage to the inner hair cells—specialized sensory cells in the inner ear that transmit information about sounds through the auditory nerve to the brain. Another cause may involve direct damage to the auditory neurons that transmit sound information from the inner hair cells to the brain. There may be genetic mutations that effect the auditory system.
Glossopharyngeal neuralgia is a disorder that is associated with repeated episodes of severe pain in the tongue, throat, ear, and tonsils. These areas are all connected to the ninth cranial nerve, also called the glossopharyngeal nerve. Glossopharyngeal neuralgia is thought to be caused by irritation of the glossopharyngeal nerve, but the exact cause of the irritation is sometimes unknown.
Abnormalities of Personality and Behavior

Schizophrenia
Personality Disorder
Dementia
Abnormalities of personality and behavior generally effect the “software” of the brain and are thus harder to treat with conventional medicine. Schizophrenia is probably the most serious condition in this area. Treatment is predicated on the belief that abnormalities in neurotransmission have provided the basis for theories on the pathophysiology of schizophrenia. Most of these theories center on either an excess or a deficiency of neurotransmitters, including dopamine, serotonin, and glutamate. Sadly, these treatments do not have a consistent success rate.
There are many classification of personality disorders. Personality disorders are thought to be caused by a combination of these genetic and environmental influences. Your genes may make you vulnerable to developing a personality disorder, and a life situation may trigger the actual development. Some common problems are:

- Obsessive compulsive behavior
- Narcissism
- Bipolar disorder
- Anxiety
- Chronic depression
- Anger disorder
- Post traumatic stress disorder
Dementia is defined as a chronic or persistent disorder of the mental processes caused by brain disease or injury and marked by memory disorders, personality changes, and impaired reasoning.

Dementia affects three areas of the brain:

- Language
- Memory
- Decision making

The pathophysiology of the dementia depends entirely on the etiology and the area of the brain involved.
Alzheimer’s disease is the most common type of dementia. Beta-amyloid deposition and neurofibrillary tangles lead to loss of synapses and neurons, which results in gross atrophy of the affected areas of the brain.

The second most common type of dementia is vascular dementia. It’s caused by a lack of blood flow to the brain. Vascular dementia can happen as you age and can be related to atherosclerotic disease.

Frontotemporal dementia is a name used to describe several types of dementia, all with one thing in common: They affect the front and side parts of the brain, which are the areas that control language and behavior.

Other types of dementia include: Dementia with Lewy bodies, Parkinson's disease, Frontotemporal dementia, Creutzfeldt-Jakob disease, Wernicke-Korsakoff syndrome.
Thank you for joining us today!

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