

PATHO PHYSIOLOGY BIBLE

OVER 70 CONCEPT MAPS

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NEURO: CNS

Alzheimer's disease

PLAN OF CARE: Safety/ LOC/ stress free

Path physiology

The classic neuropathology findings in AD include amyloid plaques, neurofibrillary tangles, and synaptic and neuronal cell death. Granulovacuolar degeneration in the hippocampus and amyloid deposition in blood vessels might also be seen on tissue examination, but they are not required for the diagnosis

Signs & Symptoms

- Early
 - Subtle changes such as forgetfulness
 - recent memory loss
 - poor concentration
- Late
 - Severe memory loss
 - Inability to hold a conversation
 - Inability to think abstractly or formulate concepts
 - Poor hygiene and grooming
 - Inappropriate dress
 - Inability to perform instrumental activities of daily living
- Behavioral changes
 - Depression
 - Anxiety
 - Wandering
 - Impulsive behavior
 - Catastrophic reactions
 - Imitation
 - Emotional lability
 - Withdrawal

Nursing Dx	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired thought processes related to decline in cognitive function • Risk for injury related to decline in cognitive function • Anxiety related to 	<ul style="list-style-type: none"> • Provide initial and ongoing assessments • Administer prescribed medications. • Maximize effective communication 	<ul style="list-style-type: none"> • Impairment of visual perception increases the risk of falling. Identify potential risks in the environment and heighten awareness so that caregivers 	<ul style="list-style-type: none"> • Creating living conditions that are as stress-free as possible will help keep the patient calm and help strengthen his cognitive abilities,

<p>confused thought processes</p> <ul style="list-style-type: none"> • Imbalanced nutrition: less than body requirements related to cognitive decline • Activity intolerance related to imbalance in activity/rest pattern • Deficient self-care related to cognitive decline • Impaired social interaction • Deficient knowledge of family/caregiver related to care for patient as cognitive function declines • Ineffective family processes related to decline in patient's cognitive function 	<ul style="list-style-type: none"> • Maximize environmental safety • Promote optimal functioning • Optimize nutrition and fluid balance • Optimize elimination • Reducing anxiety and agitation • Promoting independence in self-care activities • Providing for socialization and intimacy needs • Promoting balanced activity and rest • Provide discharge planning 	<p>more aware of the danger.</p> <ul style="list-style-type: none"> • An impaired cognitive and perceptual disorder are beginning to experience the trauma as a result of the inability to take responsibility for basic security capabilities, or evaluating a particular situation. • Maintain security by avoiding a confrontation that could improve the behavior / increase the risk for injury. • Provide the basis for the evaluation / comparison that will come, and influencing the choice of intervention. • Noise, crowds, the crowds are usually the excessive sensory neurons and can increase interference. • Cause concern, especially in people with perceptual disorders. • The name is a form of self-identity and lead to recognition of reality and the individual. • Increasing the possibility of understanding. 	<p>but that can be a tall order.</p>
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Brain Tumors

PLAN OF CARE: Decrease ICP, pain, n/v, photophobia, monitor RR & o2

Path physiology

Brain tumors may be classified into several groups: those arising from the coverings of the brain (e.g., Dural meningioma), those developing in or on the cranial nerves (e.g., acoustic neuroma), those originating with in brain tissue and metastatic lesions originating elsewhere in the body. Tumors of the pituitary and pineal glands and of cerebral blood vessels are also types of brain tumors. Relevant clinical considerations include the location and the histology character of the tumor. Tumors may be benign or malignant.

A benign tumor CAN BE SERIOUS!! If occurs in a vital area and can grow large enough to have effects as serious as those of a malignant tumor.

Signs & Symptoms

- Severe headache in the morning, increased when coughing, bending
- Convulsions
- Signs of increased intra-cranial pressure: blurred vision, nausea, vomiting, decreased auditory function, changes in vital signs, aphasia.
- Changes in personality
- Impaired memory
- Natural disturbance of taste

- Classic triad:
 - Headache
 - Papilledema (intra-ocular pressure)
 - Vomiting

Nursing DX	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Acute pain (headache), related to tumor and increase in intracranial pressure • Disturbed body image, related to upcoming hair loss and cranial incision 	<ul style="list-style-type: none"> • Clear the airway • Monitor vital signs • Monitor the breathing pattern, breath sounds • Monitor blood gases • Blood gas analysis • Collaboration Oxygenation • Monitor the pain scale • Give a comfortable position • Perform Massage • Observation of non-verbal signs of pain • Assess, emotional state • Note the influence of pain • Cold compresses on the head • Use of therapeutic touch technique • Observation of 	Perform pain assessment each time pain occurs. Note and investigate changes from prev. report.	<ul style="list-style-type: none"> • reduced pain • Impaired gas exchange can be resolved

	nausea, vomiting • DRUGS: analgesic, relaxant, prednisone, anti-emetics		
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Cerebrovascular Accident (CVA)

PLAN OF CARE: neuro checks, pain Manage, decrease ICP, monitor RR, Effective communication & LOC

Pathophysiology

- In a stroke, the sudden interruption of blood supply to areas of the brain results in cerebral necrosis and impaired cerebral metabolism, which permanently damages brain tissues and produces focal neurologic deficit of varying severity.
- A **cerebral aneurysm is prone to rupture**, which causes blood to leak into the subarachnoid space (and sometimes into brain tissue, where it forms a clot), resulting in increased intracranial pressure (ICP) and brain tissue damage
- In a TIA, there is a temporary decrease in blood flow to a specific region of the brain, but there is **no necrosis of brain tissue**. The symptoms (lasting seconds to hours) produce transient neurologic deficits that **completely clear within 12 to 24 hours**.

Signs & Symptoms

- Stroke
 - Hemiplegia and sensory deficit
 - Aphasia (impairment may be in speaking, listening, writing, or comprehending, most cases are mixed expressive and receptive).
 - Hemiparesis – **weakening** of one side
 - Unilateral neglect of paralyzed side
 - Bladder impairment
 - Possibly respiratory impairment
 - Impaired mental activity and psychological deficits
 - STROKE: FAST – Face, affect, smile,
- Transient Ischemic Attack
 - Temporary loss of consciousness or dizziness
 - Paresthesias
 - Garbled speech
- Cerebral aneurysm
 - Blurred vision and headache
 - Signs and symptoms of ICP
 - Nuchal rigidity and pain on neck movement
 - Photophobia

- Irritability and restlessness
- Slight temperature elevation

Nursing DX	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired physical mobility related to hemiparesis, loss of balance and coordination, spasticity and brain injury • Pain related to hemiplegia and disuse • Deficient self-care (hygiene, toileting, transfers, feeding) related to stroke sequelae • Disturbed sensory perception • Impaired swallowing • Incontinence related to flaccid bladder, detrusor instability, confusion, difficulty in communicating • Impaired thought processes related to brain damage, confusion, inability to follow instructions • Impaired verbal communication related to brain damage, confusion, inability to follow instructions • Risk for impaired skin integrity related to hemiparesis or hemiplegia, decreased mobility • Sexual dysfunction related to neurologic deficit or fear of failure • Ineffective family processes related to catastrophic illness and care giving 	<ul style="list-style-type: none"> • Provide alternative methods of communication, like pictures or visual cues, gestures or demonstration. • Anticipate and provide for patient's needs. • Talk directly to patient. Speaking slowly and directly. Use yes or no question to begin with. • Speak in normal tones and avoid talking too fast. Give patient ample time to respond. • Encourage family members and visitors to BE PATIENT persist efforts to communicate with the patient. 	<ul style="list-style-type: none"> • Provide communication need or desires based on individual situation or underlying deficit. • Helpful in decreasing frustration when dependent on others and unable to communicate desires. • It reduces confusion or anxiety and having to process and respond to large amount of information at one time. • Patient is not necessary hearing impaired and raising voice may irritate or anger the patient. • It is important for family members to continue talking to the patient to reduce patient's isolation, promote establishment of effective communication and maintain sense of connectedness or bonding with the family. 	<ul style="list-style-type: none"> • speech therapy to relearn talking and swallowing; • occupational therapy to regain as much function dexterity in the arms and hands as possible; • physical therapy to improve strength and walking; and • Family education to orient them in caring for their loved one at home and the challenges they will face.

burdens <ul style="list-style-type: none"> • Impaired cerebral perfusion due to bleeding from the aneurysm • Sensory-perceptual alteration due to the restrictions of subarachnoid precautions • Anxiety due to illness or restrictions of aneurysm precautions 			
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Epilepsy

Pathophysiology

Mechanisms of tumor-related epileptogenesis remain poorly understood. In tumor-associated epilepsy, nontumoral surrounding tissue may cause seizures.³⁹ Abnormal growth kinetics of tumors can affect surrounding neurons morphologically and biochemically, altering neuronal structure and affecting the release of neurotransmitters and neuromodulators such as gamma-aminobutyric acid (GABA) and somatostatin. These changes may cause seizures through hyperexcitability or reduced inhibition.

The hippocampus may become involved—either directly, through tumor extension, or indirectly, through increased excitatory input caused by a tumor—and may contribute to seizure amplification and propagation.

Tumors can disrupt normal electrical functional patterns, causing increased local coherence, or similarity of electrical activity seen electrographically within a cortical region, which is a similar pattern observed in epileptic foci. These changes, induced by a tumor in the surrounding tissue, contribute to the formation of the epileptogenic zone.

Cortical connections contribute to generation and maintenance of seizures. Aggressive white-matter neoplasms are less likely to cause seizures because they do not directly irritate cortex, and tumor growth may disrupt the spread of epileptic activity.

Signs & Symptoms

Generalized Seizures

Generalized seizures are caused by abnormal electrical impulses in the brain and typically occur with no warning. There are six types of generalized seizures.

Tonic-clonic (grand-mal) Seizure — This seizure causes you to lose consciousness and often collapse. Your body becomes stiff during what's called the "tonic" phase. During the "clonic" phase, muscle contractions cause your body to jerk. Your jaws clamp shut and you may bite your tongue. Your bladder may contract and cause you to urinate. After one to two minutes, you fall into a deep sleep.

- Absence (petit mal) Seizure — During these brief episodes, **you lose awareness and stare blankly**. Usually, there are no other symptoms. They tend to begin and end suddenly and last for about five to 10 seconds, although they can last longer. These seizures may occur several times a day.
- Myoclonic Seizure — These very brief seizures cause your **body to jerk**, as if **shocked by electricity**, for a second or two. The jerks can range from a single muscle jerking to involvement of the entire body.
- Clonic Seizure — This seizure cause **rhythmic jerking motions** of the arms and legs, sometimes on both sides of your body.
- Tonic Seizure — Tonic seizures cause your **muscles to suddenly stiffen**, sometimes for as long as 20 seconds. If you're standing, you'll typically fall.
- Akinetic or Atonic Seizure — This seizure causes **your muscles to relax or lose strength**, particularly in the arms and legs. Although you usually remain conscious, it can cause you to suddenly fall and lead to injuries. These seizures also are called "drop attacks."

Focal Seizures

Focal seizures, also known as local or partial seizures, are caused by abnormal electrical activity in a specific, smaller part of the brain. The part of the brain causing the seizure is called the seizure focus. Focal seizures are divided into simple and complex seizures.

Some focal seizures evolve into generalized ones and are called secondarily generalized seizures.

- Simple Focal Seizure — During these seizures, you remain conscious although some people can't speak or move until the seizure is over. Uncontrolled movements, such as jerking or stiffening, can occur throughout your body. You also may experience emotions such as fear or rage or even joy; or odd sensations, such as ringing sounds or strange smells. In addition, you may experience peculiar memories such as a feeling of "deja-vu." Typically, these seizures last less than one minute.
- Complex Focal Seizure — During these seizures, you are not fully conscious and may appear to be in a dreamlike state. Typically, they start with a blank stare. You may involuntarily chew, walk, fidget, or perform other repetitive movements or simple actions, but actions are typically unorganized or confused. These seizures typically last **between 30 seconds and a minute**.
- Secondarily Generalized Seizure — These seizures begin as a focal seizure and develop into generalized ones as the electrical abnormality spreads throughout the brain. When the seizure begins, you may be fully conscious but then lose consciousness and experience convulsions as it develops.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Risk for injury related to seizure activity • Fear related to the possibility of seizures • Ineffective individual coping related to stresses imposed by epilepsy • Deficient knowledge related to epilepsy 	<ul style="list-style-type: none"> • Administer anticonvulsant therapy as prescribed. • Protect the patient from injury during seizures. • Monitor the patient continuously during seizures. • If the patient is taking antiseizure 	<ul style="list-style-type: none"> • Seizure disorders are chronic health conditions experienced by many people with developmental disabilities. • The primary goal of care is to minimize the impact of seizure disorders on the lives of 	<ul style="list-style-type: none"> • Lack of sleep, flashing lights and prolonged television viewing may increase brain activity that may cause potential seizure activity. • Enables the patient to protect self from injury. • Minimizes injury

and its control	<p>medications, constantly monitor for toxic signs and symptoms such as slurred speech, ataxia, lethargy, and dizziness.</p> <ul style="list-style-type: none"> • Monitor the patient's compliance with anticonvulsant drug therapy. • Teach the patient to take exact dose of medication at the times prescribed. • Encourage the patient to eat balanced, regular meals. • Advise the patient to be alert for odors that may trigger an attack. • Limit or avoid alcohol intake. • Encourage to have enough sleep to prevent attacks • Avoid restraining the patient during a seizure. • Loosen any tight clothing, and place something flat and soft, such as pillow, jacket, or hand, under his head. • Avoid any forcing anything into the patient's mouth if his teeth are clenched. • Avoid using tongue blade or spoon during attacks which could lacerate the mouth and lips of displace teeth, precipitating respiratory distress. • Protect the patient's tongue, if his mouth is open, by placing a soft object between his teeth. 	<ul style="list-style-type: none"> • individuals with developmental disabilities. • The cooperation of all team members, including the individual, is required to establish • optimal levels of seizure control. • The primary care prescriber or medical consultant is the only team member who can • medically diagnose a seizure, classify the seizure type, and order treatment. • Seizures are classified according to the International Classification System of Epileptic • Seizures, permitting selection of an appropriate anticonvulsant and optimal seizure • management by the primary care prescriber. • The proper diagnosis and classification of seizure disorders may be difficult to determine • because of communication deficits, confusing clinical presentation, and absent or • insufficient history. • The primary care prescriber must rely on the description of seizures by observers to make • a reliable diagnosis. • Accurate descriptions of seizure activity and a system for recording and reporting the 	<p>should seizure occur while patient is in bed.</p> <ul style="list-style-type: none"> • Use of helmet may provide added protection for individuals during aura or seizure activity. • Patient may feel restless to ambulate or even defecate during aural phase, that inadvertently removing self from safe environment and easy observation. • Help maintain airway and reduces risk of oral trauma but should not be forced or inserted when teeth are clenched because dental or soft tissue may damage. • Gentle guiding of extremities reduces risk of physical injury when patient lacks voluntary muscle control. • Patient may be confused, disoriented after seizure and need help to regain control and alleviate anxiety in postictal phase. • Specific drug therapy depends on seizure type, with some patients Requiring polytherapy or frequent medications adjustment.
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	<ul style="list-style-type: none"> • Turn the patient's head to the side to provide an open airway. • Reassure patient after the seizure subsides by telling him that he's all right, orienting him to time and place, and informing that he's had a seizure. 	<ul style="list-style-type: none"> • activity is essential to seizure management. • Because seizures frequently occur during the absence of professional staff, all staff • involved with individuals who may have seizures must be trained in observing and • recording seizure activity, and managing and protecting the individual during and after a • seizure 	
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Head Injury

Pathophysiology

There are many different types of brain injury, depending upon the severity of the force upon the head, as well as which portion of the brain is affected. To simplify, brain injuries can be classified as traumatic or acquired, with additional types under each heading. All brain injuries are described as either mild, moderate, or severe.

Traumatic Brain Injury

Traumatic brain injury is a result of an external force to the brain that results in a change to cognitive, physical, or emotional functioning. The impairments can be temporary or permanent. Types of traumatic brain injury include:

- Diffuse axonal injury. Shaking or strong rotation of the head causes brain structures to tear. Nerve tissue is disturbed throughout the brain.
- Concussion. Caused by a physical force to the head that causes blood vessels to stretch and cranial nerves to be damaged.
- Contusion. A result of a direct impact to the head, which causes bleeding on the brain.
- Coup-contrecoup injury. The force to the brain is large enough to cause contusion at the side of impact, as well as the site opposite impact.
- Penetration injury. The impact causes a foreign object to penetrate the skull.

Acquired Brain Injury

An acquired brain injury is an injury to the brain that is not hereditary, congenital, degenerative, or the result of birth trauma. Acquired brain injury generally affects cells throughout the entire brain. Types of acquired brain injury include:

- Anoxic brain injury. This occurs when the brain doesn't receive oxygen.
- Hypoxic brain injury. This occurs when the brain receives some, but not enough, oxygen.

Signs & Symptoms

- Symptoms depend on the severity and distribution of brain injury.
- A common manifestation is loss of consciousness, ranging from a few minutes to 1 hour or longer.
- Cerebrospinal otorrhea (i.e. CSF draining from the ear), and cerebrospinal rhinorrhea (CSF draining from nose) may be present. This is determined by a positive glucose reading on a dextrose stick or halo sign. (i.e. blood surrounded by a yellowish stain).
- Ecchymosis may be seen over the mastoid (Battle's sign)
- CT scan may reveal the area that is contused or injured
- Radiographs may reveal skull fractures
- Persistent, localized pain usually suggests fracture
- Fractures of the cranial vault may or may not produce swelling in that region
- Bloody spinal fluid suggests brain laceration or contusion.
- Brain injury may have various signs, including altered level of consciousness, pupillar abnormalities, altered or absent gag reflex or corneal reflex, neurologic deficits, change in vital signs (e.g. respiration pattern, hypertension, bradycardia), hyperthermia or hypothermia, and sensory, vision or hearing impairment.
- Signs of a postconcussion syndrome may include headache, dizziness, anxiety, irritability, and lethargy.
- In acute or subacute subdural hematoma, changes in level of consciousness, papillary signs, hemiparesis, coma, hypertension, bradycardia, and slowing respiratory rate are signs of expanding mass.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Ineffective tissue perfusion (cerebral) • Risk for Injury • Decreased intracranial adaptive capacity. 	<p>Independent</p> <ul style="list-style-type: none"> • Assess contributing factors to pain (noise, wrong positioning, environment) • review medication regimen • ask client to rate pain on 0-10 scale (rated as 9 out of 10)4.provide comfort measures such as repositioning the client in a comfortable position and providing a hot or coldcompress5.provide calm and quiet environment(adjust lights, temperature and eliminate offensive odors which may contribute to headache) instructed in relaxation techniques 	<ul style="list-style-type: none"> • To determine underlying cause of pain and treat accordingly.2.certain drugs may cause fatigue and drowsiness. • To assist in evaluating impact of pain on client's life. • To allow nonpharmacological pain relief and promote good circulation to the brain and decrease vasoconstriction • To decrease environmental factors which contribute to migraine and promote rest. • To distract attention from pain and decrease tension • To conserve energy of the patient and 	<ul style="list-style-type: none"> • Goal met. Patient verbalized "I feel better. It's just a little sore from althea swelling. But it intolerable pain." rated pain as 4 out of 10. • Goal met. Patient was able to relax by utilizing bed rest and deep breathing. • Goal met. Patient was able to sleep for 6 hours straight and felt rested afterwards. • Goal met. Client was able to use deep breathing and reported pain relief afterwards. Goal met. Client was able to perform ADLs with

	<p>(deep breathing, imagery)</p> <ul style="list-style-type: none"> • encourage adequate rest periods • assist in self-care activities as tolerated • provide peaceful \and adequate resting environment (dim lights, adjust temperature, wrinkle-free bed, quiet surroundings) <p>COLLABORATIVE:</p> <ul style="list-style-type: none"> • administer medications as ordered by physician (analgesics, etc) • encourage watchers to assist patient during divisional activities (minimize noise, allow client to verbalize feelings and promote rest and sleep) 	<p>prevent fatigue</p> <ul style="list-style-type: none"> • To promote client independence as much as possible and acquire sense of function to enhance quality sleep and promote rest which harnesses energy for future use. • medications will provide synergistic effect with non pharmacologic interventions for pain relief and promote better circulation by aiding in vasodilatation for better blood flow to the brain and altering prostaglandin synthesis to decrease pain • the significant others know the client more and will be able to aid in diverting client's attention from pain to determine underlying cause of pain and treat accordingly. 2. certain drugs may cause fatigue and drowsiness. 	<p>minimal assistance from watchers (feeding, self-care, etc)</p>
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Multiple Sclerosis

Pathophysiology

- Demyelination of nerve fibers within long conducting pathway of spinal cord and brain.
- Impaired transmission of nerve impulses.
- Degenerative changes myelin sheath are scattered irregularly throughout the central nervous system. Nerve axon also deteriorates. The areas involved are not consistent when it comes to deterioration thereby showing the signs and symptoms appear whenever the nerve conduction is interrupted.
- There are periods of remission also, however there are cases that symptoms are exacerbated especially when nerve impulse travel through the patchy nerve fibers.

Signs & Symptoms

- Spastic weakness – the most common sign

- Charcots Triad: A combination of symptoms that includes nystagmus, intention tremor (motor weakness in coordination), scanning speech which is elicited by slowing enunciation with tendency to hesitate at beginning of a word.
- Hyper in emotions as well as euphoria
- Visual disturbances
- Nausea and vomiting
- Urinary retention or urinary incontinence
- Dysphagia – difficulty in swallowing
- Ataxia – a problem in coordination

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired bed and physical mobility related to weakness, muscle paresis, spasticity • Risk for injury related to sensory and visual impairment • Impaired urinary and bowel elimination (urgency, frequency, incontinence, constipation) related to nervous system dysfunction • Impaired verbal communication and risk for aspiration related to cranial nerve involvement • Disturbed thought process (loss of memory, dementia, euphoria) related to cerebral dysfunction • Ineffective individual coping related to uncertainty of course of MS • Impaired home maintenance management related to physical, psychological, and social limits imposed by MS • Potential for sexual dysfunction related to lesions or 	<ul style="list-style-type: none"> • Promoting Physical mobility • Preventing Injury • Enhancing Bladder and Bowel control • Enhancing communication and managing swallowing difficulties • Improving sensory and cognitive function • Improving Home management • Promote sexual functioning 	<ul style="list-style-type: none"> • Symptomatically, allow the patient to work on his or her own in order to let him or her to know that the situation is still under control. • Comply with the medications such as cortisone or corticotrophin. These medications help in decreasing edema and inflammation at areas of demyelination. • Coordinate with a physical therapist in order to facilitate daily living. This prevents complications of immobility. • Provide proper skin care as the patient is prone in decubitus ulcers as the demyelination progresses. • Allow the patient to get in touch with the world, his family and friends to emotionally support as he keeps his mind intact in battling this degenerative condition. • Provide a safe environment for the 	<ul style="list-style-type: none"> • Maintain normal daily activities as best you can. • Stay connected with friends and family. • Continue to pursue hobbies that you enjoy and are able to do. • Get enough rest. • Exercise • Be careful with heat.

psychological reaction		patient always. Use prescribed equipment for transport, transferring the patient as well as in mobilization.	
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Meningitis

Pathophysiology

Meningitis is an inflammation of the leptomeninges and underlying subarachnoid cerebrospinal fluid (CSF). Meningitis is the inflammation of the protective membranes covering the central nervous, known collectively as the meninges.

Meningitis can be caused from a direct spread of a severe infection such as an ear infection or sinus infection. In some cases, meningitis is noted after head trauma or an injury to the head or brain. There are several causes of meningitis. These include Bacterial infection, Viral infection, Fungal infection, A reaction to medications, A reaction to medical treatments, Lupus, Some forms of cancer, A trauma to the head or back. Anyone can catch meningitis. This is especially true if your immune system is weak.

Sometimes, however, they spread to the meninges from an infection in another part of the body. The meninges are composed of three layers of membranes enclosing the brain and spinal cord. Pia mater is the innermost layer. It is akin to a tissue paper that closely adheres to the brain and spinal cord, dipping into the various folds and crevices. Arachnoid mater is the middle layer. It is a filmy membrane that is joined to the pia mater by fine threads resembling a cobweb.

Signs & Symptoms

- Symptoms: Loss of appetite, difficulty swallowing.
- Signs: anorexia, vomiting, poor skin turgor and dry mucous membranes.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Acute pain related to infection process toxin in the circulation • Impaired Physical Mobility related to neuromuscular damage. 	<ul style="list-style-type: none"> • Place the ice bag on his head, cool clothing above the eyes, provide a comfortable head position a little bit high, range of motion exercises and active or passive massage neck muscles. • Support to find a comfortable position (head rather high-). • Give range of 	<ul style="list-style-type: none"> • .Monitor changes in orientation, kemampuan speak, the natural feelings, sensory and thought processes. • Assess awareness of sensory: touch, heat, cold. • Observations of behavioral response. • Eliminate excessive noise. 	<ul style="list-style-type: none"> • patients respiration will be reestablished and its rate return to normal range • pain level experienced will be decreased or alleviated

	<p>motion exercises active / passive.</p> <ul style="list-style-type: none"> • Use a warm moisturizer, neck or hip. • Assess the degree of immobilization of the patient. • Assistive range of motion exercises. • Give skin care, massage with moisturizer. • Check the area experiencing tenderness, given air mattresses or water body alignment are functionally notice. • Provide training programs and the use of mobilization. 	<ul style="list-style-type: none"> • Validate the patient's perception and give feedback. • Give the opportunity to communicate and move. • Collaboration physiotherapists, occupational therapy, speech and cognitive 	
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Parkinson's Disease

Pathophysiology

- Parkinson's disease is a slowly progressive degenerative neurological disorder caused by the loss of nerve cell function in the basal ganglia. The basal ganglia includes several structures (substantia nigra, striatum, globus pallidus, subthalamic nucleus and the red nucleus). Loss of nerve cells in the substantia nigra causes a reduction of dopamine production. Dopamine is the neurotransmitter essential for such functions as control of posture, supporting the body in an upright position and voluntary motions.

Signs & Symptoms

- Tremor (rhythmic, purposeless, fine trembling, quivering movement), resting or passive tremor
- Muscle rigidity (stiffness seen with resistance to passive muscle stretching), cogwheel rigidity
- Akinesia (loss of movement) and bradykinesia (slowness of voluntary movement and speech)
- Mask-like expression
- Dysphagia (difficulty of swallowing)
- Monotonous speech
- Postural disturbances (stooped posture, shuffling gait, broad-based turns)
- Generalized muscle fatigue
- Cognitive changes (impaired memory, depression)
- Drooling
- Constipation

- Orthostatic hypotension
- Urinary dysfunction

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Assess cranial nerves, cerebral function (coordination) and motor function. • Observation of gait and while doing the activity. • Review the history of symptoms and their effects on body functions. • Assess the clarity and speed of speech. • Review the signs of depression. 	<ul style="list-style-type: none"> • Monitor drug treatment to note adverse reactions and allow for dosage adjustments. Monitor for liver function changes and anemia during drug therapy. • Monitor the patient's nutritional intake and check weight regularly. • Monitor the patient's ability to perform activities of daily living. • To improve mobility, encourage the patient to participate in daily exercise, such as walking, riding stationary bike, swimming, or gardening. • Advise the patient to perform stretching and postural exercises as outlined by a physical therapist. • Teach the patient walking techniques to offset parkinsonian shuffling gait and tendency to lean forward. • Encourage the patient to take warm baths and massage muscles to help relax muscles. • Instruct the patient to rest often to avoid fatigue and frustration. • To improve the patient's nutritional status, teach the 	<ul style="list-style-type: none"> • Provide client and family teaching • Promote measures to enhance body image • Prepare the client for stereotaxic surgery to reduce tremors and rigidity if indicated. • Administer prescribed medications, which may include ant Parkinson medication, anticholinergics, antihistamines, amantadine hydrochloride, antiviral agent, and monoamine oxidase-inhibitors. • Promote measures to maintain an adequate airway. • Promote methods to ease difficulty with swallowing if indicated. Encourage semi-solid diet. • Maximize functional abilities. • Improve mobility and prevent complications of immobility. • Encourage daily exercise, stretching exercises and special walking techniques to offset the shuffling gait. • Instruct the client in ways to prevent constipation (e.g. increase fluids, 	<ul style="list-style-type: none"> • To increase mobility • To optimize the nutritional status • To maximize the ability to communicate.

	<p>patient to think through the sequence of swallowing.</p> <ul style="list-style-type: none"> • Urge the patient to make a conscious effort to control accumulation of saliva (drooling) by holding head upright and swallowing periodically. Be alert for aspiration hazard. • Have the patient use secure, stabilized dishes and eating utensils. • Suggest the patient eat smaller meals and additional snacks. • To prevent constipation, encourage patient to consume foods containing moderate fiber content (whole grains, fruits, and vegetables), and to increase his or her water intake. • Obtain a raised toilet seat to help the patient sit and stand. • Teach the patient facial exercises and breathing methods to obtain appropriate pronunciation, volume, and intonation. • Teach the patient about the medication regimen and adverse reaction.. 	<p>maintain high-fiber diet, follow regular bowel routine.</p> <ul style="list-style-type: none"> • Promote self-care • o Maximize effective communication 	
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Seizures

Pathophysiology

Epilepsy is not a singular disease, but is heterogeneous in terms of clinical expression, underlying etiologies, and pathophysiology. As such, specific mechanisms and pathways underlying specific seizure types may vary. Epileptic seizures are broadly classified according to their site of origin and pattern of spread.

- Focal or partial seizures arise from a localized region of the brain and have clinical manifestations that reflect that area of brain. Focal discharges can remain localized or they can spread to nearby cortical areas, to subcortical structures and/or transmit through commissural pathways to involve the whole cortex. The latter sequence describes the secondary generalization of focal seizures. As an example, a seizure arising from the left motor cortex may cause jerking movements of the right upper extremity. If epileptiform discharges spread to adjacent areas and then the entire brain, a secondary generalized tonic-clonic seizure ensues.
- Primary generalized seizures begin with abnormal electrical discharges in both hemispheres simultaneously. Generalized seizures involve reciprocal connections between the thalamus and neocortex. The manifestations of such widespread epileptiform activity can range from brief impairment of consciousness (as in an absence seizure) to generalized motor activity accompanied by loss of consciousness (generalized tonic-clonic seizure).

Signs & Symptoms

- Sensory/Thought:
 - Black out
 - Confusion
 - Deafness/Sounds
 - Electric Shock Feeling
 - Loss of consciousness
 - Smell
 - Spacing out
 - Out of body experience
 - Visual loss or blurring
- Emotional:
 - Fear/Panic
 - Pleasant feeling
- Physical:
 - Chewing movements
 - Convulsion
 - Difficulty talking
 - Drooling
 - Eyelid fluttering
 - Eyes rolling up
 - Falling down
 - Foot stomping
 - Hand waving
 - Inability to move
 - Incontinence
 - Lip smacking

- Making sounds
- Shaking
- Staring
- Stiffening
- Swallowing
- Sweating
- Teeth clenching/grinding
- Tongue biting
- Tremors
- Twitching movements
- Breathing difficulty
- Heart racing
- Bruising
- Difficulty talking
- Injuries
- Sleeping
- Exhaustion
- Headache
- Nausea
- Pain
- Thirst
- Weakness
- Urge to urinate/defecate

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired physical mobility related to hemiparesis, loss of balance and coordination, spasticity and brain injury • Pain related to hemiplegia and disuse • Deficient self-care (hygiene, toileting, transfers, feeding) related to stroke sequelae • Disturbed sensory perception • Impaired swallowing • Incontinence related to flaccid bladder, detrusor 	<ul style="list-style-type: none"> • Explore with patient the various stimuli that may precipitate seizure activity. • Discuss seizure warning signs (if appropriate) and usual seizure pattern. Teach SO to recognize warning signs and how to care for patient during and after seizure. • Keep padded side rails up with bed in lowest position, or place bed up against wall and pad floor if rails not available/appropriate. • Encourage patient not to smoke except while supervised. • Evaluate need 	<ul style="list-style-type: none"> • Alcohol, various drugs, and other stimuli (e.g., loss of sleep, flashing lights, prolonged television viewing) may increase brain activity, thereby increasing the potential for seizure activity. • Enables patient to protect self from injury and recognize changes that require notification of physician/further intervention. Knowing what to do when seizure occurs can prevent injury/complications and decreases SO's feelings of helplessness. • Minimizes injury should seizures 	<ul style="list-style-type: none"> • Seizures activity controlled. • Complications/injury prevented. • Capable/competent self-image displayed. • Disease process/prognosis, therapeutic regimen, and limitations understood. • Plan in place to meet needs after discharge.

<p>instability, confusion, difficulty in communicating</p> <ul style="list-style-type: none"> • Impaired thought processes related to brain damage, confusion, inability to follow instructions • Impaired verbal communication related to brain damage, confusion, inability to follow instructions • Risk for impaired skin integrity related to hemiparesis or hemiplegia, decreased mobility • Sexual dysfunction related to neurologic deficit or fear of failure • Ineffective family processes related to catastrophic illness and care-giving burdens • Impaired cerebral perfusion due to bleeding from the aneurysm • Sensory-perceptual alteration due to the restrictions of subarachnoid precautions • Anxiety due to illness or restrictions of 	<p>for/provide protective headgear</p> <ul style="list-style-type: none"> • Use tympanic thermometer when necessary to take temperature. 	<p>(frequent/generalized) occur while patient is in bed. Note: Most individuals seize in place and if in the middle of the bed, individual is unlikely to fall out of bed.</p> <ul style="list-style-type: none"> • May cause burns if cigarette is accidentally dropped during aura/seizure activity. • Use of helmet may provide added protection for individuals who suffer recurrent/severe seizures. • Reduces risk of patient biting and breaking glass thermometer or suffering injury if sudden seizure activity should occur. 	
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aneurysm precautions			
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Spinal Cord Injury

Pathophysiology

- Spinal cord injuries causes myelopathy or damage to white matter or myelinated fiber tracts that carry signals to and from the brain. It also damages gray matter in the central part of the spine, causing segmental losses of interneurons and motorneurons. Spinal cord injury can occur from many causes, including:
 - Trauma such as automobile crashes, falls, gunshots, diving accidents, war injuries, etc.
 - Tumor such as right, ependymomas, astrocytomas, and metastatic cancer.
 - Ischemia resulting from occlusion of spinal blood vessels, including dissecting aortic aneurysms, emboli, arteriosclerosis.
 - Developmental disorders, such as spina bifida, meningomyelcoele, and other.
 - Neurodegenerative diseases, such as Friedreich's ataxia, spinocerebellar ataxia, etc.
 - Demyelinative diseases, such as Multiple Sclerosis.
 - Transverse myelitis, resulting from spinal cord stroke, inflammation, or other causes.
 - Vascular malformations, such as arteriovenous malformation (AVM), dural arteriovenous fistula (AVF), spinal hemangioma, cavernous angioma and aneurysm.

Signs & Symptoms

- Impaired physical mobility
- Disturbed sensory perception
- Acute pain
- Anticipatory grieving
- Low self-esteem
- Constipation or bowel incontinence
- Impaired urinary elimination

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired physical mobility related to neuromuscular impairment. 	Independent: <ul style="list-style-type: none"> • <input type="checkbox"/> Continually asses motor function (as spinal shock or edema resolves) by requesting patient to perform certain actions. • <input type="checkbox"/> Provide means to summon help. • <input type="checkbox"/> Assist in range of motion exercises on all extremities 	<ul style="list-style-type: none"> • Evaluates status of individual situation (motor-sensory impairment may be mixed and/ or not clear) for a specific level of injury, affecting type and choice of intervention. • Enables patient to have sense of control, and 	<ul style="list-style-type: none"> • Able to demonstrate techniques or behaviors that enable resumption of activity.

	<p>and joints, using slow, smooth movements.</p> <ul style="list-style-type: none"> • <input type="checkbox"/> Plan activities to provide uninterrupted rest periods. Encourage involvement within individual tolerance or ability. • <input type="checkbox"/> Reposition periodically even when sitting in chair. Teach patient how to use weight • <input type="checkbox"/> shifting techniques. Inspect the skin daily. Observe for pressure areas, and provide meticulous skin care. <p>Collaborative:</p> <ul style="list-style-type: none"> • <input type="checkbox"/> Consult with physical or occupational therapist. • <input type="checkbox"/> Administer muscle relaxants or antispasticity as prescribed 	<p>reduces fear of being left alone.</p> <ul style="list-style-type: none"> • Enhances circulation, restores or maintains muscle tone and joint mobility, and prevent disuse contractures and muscle atrophy. • Prevents fatigue, allowing opportunity for maximal efforts or participations by patient. • Reduces pressure areas, promotes peripheral circulation. • Altered circulation, loss of sensation, and paralysis potentiate pressure sore formation. • Helpful in planning and implementing individualized exercise program and identifying or developing assistive devices to maintain function, enhance mobility and independence. • May be useful in limiting or reducing pain associated with spasticity 	
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NEURO: PNS

Guillain-Barre Syndrome

Pathophysiology

Guillain-Barré syndrome is the result of a cell-mediated and humoral immune attack on peripheral nerve myelin proteins that causes inflammatory demyelination. With the autoimmune

attack, there is an influx of macrophages and other immune-mediated agents that attack myelin, cause inflammation and leave the axon unable to support nerve conduction.

Signs & Symptoms

- Autonomic changes
 - Tachycardia, bradycardia, hypertension, or **orthostatic** hypotension
 - Increased sweating
 - Increased salivation
 - Constipation
- Dyskinesia (inability to executive involuntary movements)
- Weakness usually begins in the legs and progress upward (ascending paralysis)
- Hyporeflexia (decreased DTRs)
- Paresthesia (numbness), clumsiness
- Blindness
- Inability to swallow (dysphagia) or clear secretions
- Alternate hypotension/hypertension; feared complication: arrhythmias

Nursing Assessment	Nursing Intervention & Rationale	Goal
<ul style="list-style-type: none"> • Ineffective breathing pattern and impaired gas exchange related to rapidly progressive weakness and impending respiratory failure • Impaired bed and physical mobility related to paralysis • Imbalanced nutrition, less than body requirements, related to inability to swallow • Impaired verbal communication related to cranial nerve dysfunction • Fear and anxiety related to loss of control and paralysis 	<ul style="list-style-type: none"> • Monitor respiratory status through vital capacity measurements, rate and depth of respirations, and breath sounds. • Monitor level of muscle weakness as it ascends toward respiratory muscles. Watch for breathlessness while talking which is a sign of respiratory fatigue. • Monitor the patient for signs of impending respiratory failure. • Monitor gag reflex and swallowing ability. • Position patient with the head of bed elevated to provide for maximum chest excursion. • Avoid giving opioids and sedatives that may depress respirations. • Position patient correctly and provide range-of-motion exercises. • Provide good body alignment, range-of-motion exercises, and change of position to prevent complications such as contractures, pressure sores, and dependent edema. • Ensure adequate nutrition without the risk of aspiration. • Encourage physical and occupational therapy exercises to help the patient regain strength during rehabilitation phase. • Provide assistive devices as needed (cane or wheelchair) to maximize independence and activity. 	<ul style="list-style-type: none"> • Maintain airway patency • Demonstrate progressive weight gain. • Enable to express self.

	<ul style="list-style-type: none"> • If verbal communication is possible, discuss the patient's fears and concerns. • Provide choices in care to give the patient a sense of control. • Teach patient about breathing exercises or use of an incentive spirometer to reestablish normal breathing patterns. • Instruct patient to wear good supportive and protective shoes while out of bed to prevent injuries due to weakness and paresthesia. • Instruct patient to check feet routinely for injuries because trauma may go unnoticed due to sensory changes. • Urge the patient to maintain normal weight because additional weight will further stress monitor function. • Encourage scheduled rest periods to avoid fatigue. 	
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Myasthenia Gravis

Pathophysiology

- In myasthenia gravis, antibodies directed at the acetylcholine receptor sites impair transmission of impulses across the myoneural junction. Therefore, fewer receptors are available for stimulation, resulting in voluntary muscle weakness that escalates with continued activity.
- Eighty percent of people with myasthenia gravis have either thymic hyperplasia or a thymic tumor, and the thymus gland is believed to be the site of antibody production

Signs & Symptoms

- Ptosis - check palpebral fissure for drooping of upper eyelids
- Double vision
- Mask like facial expression
- Weakened laryngeal muscles leads to dysphagia (difficulty of swallowing, without food); odynophagia ang with food
- Hoarseness of voice
- Respiratory muscle weakness leads to respiratory arrest
- Extreme muscle weakness especially during activity or exertion in AM

Nursing Assessment	Nursing Intervention & Rationale	Goal
<ul style="list-style-type: none"> • Weakness and fatigue • Difficulty chewing • Dysphagia • Ptosis 	<ul style="list-style-type: none"> • Listen to the patient's concerns and answer the questions honestly. • Administer medications on time and at evenly spaced intervals, as ordered, to prevent relapses. 	<ul style="list-style-type: none"> • Will verbalize decreasing fatigue when performing ADLs. • Will state the correct method of medication

<ul style="list-style-type: none"> • Diplopia • Weak, hoarse voice • Difficulty breathing • Diminished breath sounds • Respiratory paralysis and failure 	<ul style="list-style-type: none"> • Plan exercise, meals, patient care, and activities to make the most of energy peaks. • When swallowing is difficult, give semi-solid foods instead of liquids to lessen the risk of choking. • After severe exacerbations, try to increase social activity as soon as possible. • Establish accurate neurologic and respiratory baseline. • Stay alert for signs of impending myasthenic crisis such as increased muscle weakness and difficulty talking or chewing. • Help the patient plan daily activities to coincide with energy peaks. • Stress the need for frequent rest periods. • If surgery is scheduled, provide perioperative teaching. 	
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Gastro Intestinal (Upper)

Esophageal Disorders

The esophagus is a tube that connects the back of the mouth to the stomach. Abnormalities of the esophagus generally fall into one of four categories: structural abnormalities, motility disorders, inflammatory disorders, and malignancies.

Pathophysiology

The esophagus is the tube that carries food, liquids and saliva from your mouth to the stomach. You may not be aware of your esophagus until you swallow something too large, too hot or too cold. You may also become aware of it when something is wrong.

The most common problem with the esophagus is gastroesophageal reflux disease (GERD). It happens when a band of muscle at the end of your esophagus does not close properly. This allows stomach contents to leak back, or reflux into, into the esophagus and irritate it. Over time, GERD can cause damage to the esophagus. Other problems include heartburn and cancer.

Treatment depends on the problem. Some get better with over-the-counter medicines or changes in diet. Others may need prescription medicines or surgery.

Signs & Symptoms

- Abdominal pain

- Abdominal swelling, distension or bloating
- Bad breath
- Belching
- Burning feeling in the chest or stomach
- Change in bowel habits
- Constipation
- Diarrhea
- Flatulence

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Heartburn • Regurgitation • Pain • Dysphasia • Belching • Worsening symptoms after eating or when in recumbent position 	<ul style="list-style-type: none"> • Avoid very cold or very hot and irritating them personally. • Eat slowly and chew properly. • Perform a comprehensive assessment of pain to include location, characteristics, onset, duration, frequency, quality, intensity or severity, and precipitating factors of pain • Teach the use of nonpharmacologic techniques (e.g., relaxation, guided imagery, music therapy, distraction, and massage) before, after, and if possible during painful activities; before pain occurs or increases; and along with other pain 	<ul style="list-style-type: none"> • Pain is a subjective experience and must be described by the client in order to plan effective treatment. • The use of noninvasive pain relief measures can increase the release of endorphins and enhance the therapeutic effects of pain relief medications • Ensures that the nurse has the right drug, right route, right dosage, right client, right frequency 	<ul style="list-style-type: none"> • Able to find the relaxing position.

	relief measures. <ul style="list-style-type: none"> • Check the medical order for drug, dose, and frequency of anal-gesic prescribed 		
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Gastritis

Pathophysiology

In gastritis, the Gastritis mucous membrane becomes edematous and hyperemic (congested with fluid and blood) and undergoes superficial erosion. It secretes a scanty amount of gastric juice, containing very little acid but much mucus. Superficial ulceration may occur and can lead to hemorrhage.

Signs & Symptoms

- Indigestion (dyspepsia)
- Heartburn
- Abdominal pain
- Hiccups
- Loss of appetite
- Nausea
- Vomiting, possibly of blood or material that looks like coffee grounds
- Dark stools

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Anxiety related to treatment • Imbalance nutrition 	<ul style="list-style-type: none"> • Reducing Anxiety • Promoting optimal nutrition 	<ul style="list-style-type: none"> • Able to calm the patient about the pain and treatment modalities. Able to explain the procedures and treatments according to the patients level of understanding • Able to provide physical and emotional 	<ul style="list-style-type: none"> • Reduce anxiety, avoidance of irritating foods, adequate intake of nutrients, maintenance of fluid balance, increased awareness of dietary management and relief pain.

<ul style="list-style-type: none"> • Risk of imbalance fluid • Deficient knowledge about dietary management • Acute pain 	<ul style="list-style-type: none"> • Promoting fluid balance. • Relieving Pain. 	<p>support and helps the patients manage the symptoms,, which may include nausea, vomiting, heartburn and fatigue. No food intake by mouth.</p> <ul style="list-style-type: none"> • Able to monitor early signs of dehydrations. • Help relieve pain instructing the patients to avoid foods and beverages that may be irritating to the gastric mucosa. 	
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Gastroesophageal Reflux Disease (GERD)

Pathophysiology

- Gastroesophageal reflux disease (GERD) includes all consequences of reflux of acid or other irritants from the stomach into the esophagus. The main cause of gastroesophageal reflux is incompetence of the antireflux barriers at the esophagogastric junction.
- Gastric pepsin duodenal contents exacerbate the action of acid and deleterious effect on the production of esophagitis.
- The antireflux barriers include two "sphincter" mechanisms: the lower esophageal sphincter (LES), and the crural diaphragm that functions as an external sphincter.
- Gastroesophageal reflux occurs when LES pressure is lower than the intragastric pressure such as in LES hypotension, increased frequency of transient lower esophageal sphincter relaxation (TLESR), when the intragastric pressure increases.

- The severity of GERD increases progressively with reflux that is mainly in the postprandial period to that in the upright posture, to that in the supine or that is bipositional reflux. Nighttime reflux leads to severe GERD.
- Hiatal hernia results from multiple mechanisms and is associated with a decreased LES pressure, decreased acid clearance, increased reflux, and more severe esophagitis.
- Mucosal defense mechanisms may be overcome by prolonged exposure of the esophageal mucosa to a pH <4 that may lead to severe and complicated esophagitis.
- Esophageal mucosal inflammation may affect nerves and muscle that alter LES function and esophageal body motility. A vicious cycle of inflammation and impaired motility may cause progressive disease.
- Patients with GERD may develop endoscopically visible erosive esophagitis or endoscopically negative nonerosive or negative endoscopy reflux disease (NERD). In NERD, factors such as visceral hypersensitivity or more proximal reflux of acid or nonacid material may be important. Acid and inflammatory mediators may gain access to sensory pathways and produce symptoms either by a direct action on the nerves or by producing abnormal muscle contraction.

Signs & Symptoms

- Difficulty in swallowing
- Chest pain due to heart burn
- Nausea in the morning
- Some ear, nose and throat problems
- Lung and breathing problems such as coughing, wheezing, pneumonia, permanent widening and damage to air passages in lungs called bronchiectasis and chronic asthma.
- Trouble swallowing (dysphagia)
- Blood in the stool
- Hoarseness (laryngitis)
- Frequent belching
- Sleep apnea leading to restlessness, morning headaches and after drowsiness
- Anemic (iron deficiency in blood) caused due to blood loss from ulcers in esophagus.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Imbalanced nutrition 	<ul style="list-style-type: none"> • Encourage adequate nutrition intake 	<ul style="list-style-type: none"> • Encourage to eat slowly and to chew all food thoroughly so that it can pass easily into the stomach. 	<ul style="list-style-type: none"> • Achieves an adequate nutritional intake.
<ul style="list-style-type: none"> • Risk for aspiration related to difficulty 	<ul style="list-style-type: none"> • Decreasing risk of aspiration 	<ul style="list-style-type: none"> • Kept in semi-fowler's position to decrease the 	<ul style="list-style-type: none"> • Doesn't aspirate or develop pneumonia

<p>swallowing or to tube feeding</p> <ul style="list-style-type: none"> • Acute pain related to difficulty swallowing • Deficient knowledge about the esophageal disorder. 	<ul style="list-style-type: none"> • Relieving pain • Providing patient education 	<p>risk of aspiration. The patient can be instructed in the use of oral suction to decrease the risk of aspiration further.</p> <ul style="list-style-type: none"> • Small frequent feedings are recommended, because large quantities of food overload the stomach and promote gastric reflux. • Able to provide physical and emotional support and helps the patients manage the symptoms,, which may include nausea, vomiting, heartburn and fatigue. 	<ul style="list-style-type: none"> • Free of pain • Increases knowledge level of esophageal condition, treatments and prognosis..
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Hiatal Hernia

Pathophysiology

The esophagus passes through the diaphragmatic hiatus in the crural part of the diaphragm to reach the stomach. The diaphragmatic hiatus itself is approximately 2 cm in length and chiefly consists of musculotendinous slips of the right and left diaphragmatic crura arising from either side of the spine and passing around the esophagus before inserting into the central tendon of the diaphragm. The size of the hiatus is not fixed, but narrows whenever intra-abdominal pressure rises, such as when lifting weights or coughing.[1]

The lower esophageal sphincter (LES) is an area of smooth muscle approximately 2.5-4.5 cm in length. The upper part of the sphincter normally lies within the diaphragmatic hiatus, while the lower section normally is intra-abdominal. At this level, the visceral peritoneum and the phrenoesophageal ligament cover the esophagus. The phrenoesophageal ligament is a fibrous layer of connective tissue arising from the crura, and it maintains the LES within the abdominal cavity. The A-ring is an indentation sometimes seen on barium studies, and it marks the upper part of the LES. Just below this is a slightly dilated part of the esophagus, forming the vestibule. A second ring, the B-ring, may be seen just distal to the vestibule, and it approximates the Z-line or squamocolumnar junction. The presence of a B-ring confirms the diagnosis of a hiatal hernia. Occasionally, the B-ring also is called the Schatzki ring.

Any sudden increase in intra-abdominal pressure also acts on the portion of the LES below the diaphragm to increase the sphincter pressure. An acute angle, the angle of His, is formed between the cardia of the stomach and the distal esophagus and functions as a flap at the gastroesophageal junction and helps prevent reflux of gastric contents into the esophagus

The gastroesophageal junction acts as a barrier to prevent reflux of contents from the stomach into the esophagus by a combination of mechanisms forming the antireflux barrier. The components of this barrier include the diaphragmatic crura, the LES baseline pressure and intra-abdominal segment, and the angle of His. The presence of a hiatal hernia compromises this reflux barrier not only in terms of reduced LES pressure but also reduced esophageal acid clearance. Patients with hiatal hernias also have longer transient LES relaxation episodes particularly at night time. These factors increase the esophageal mucosa acid contact time predisposing to esophagitis and related complications.

Signs & Symptoms

- Acidic taste in the mouth
- Belching
- Difficulty swallowing
- Epigastria pain or burning, which can run from the stomach area up to the mouth
- Heartburn
- Indigestion
- Nausea and vomiting

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Discomfort or pain in the esophagus • Nausea and vomiting • Unexplained coughing 	<ul style="list-style-type: none"> • Relieving pain • Encourage adequate nutrition intake 	<ul style="list-style-type: none"> • Small frequent feedings are recommended , because large quantities of food overload the stomach and promote gastric reflux. • Encourage to eat slowly and to chew all food thoroughly so that it can pass easily into the 	<ul style="list-style-type: none"> • Free of pain • Reduce, avoidance of irritating foods, adequate intake of nutrients, maintenance of fluid balance, increased awareness of dietary management and relief pain.

	<ul style="list-style-type: none"> Promoting fluid balance 	<ul style="list-style-type: none"> stomach. Able to monitor early signs of dehydrations. 	
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Peptic Ulcer Disease

Pathophysiology

Peptic Ulcer is a lesion in the mucosa of the lower esophagus, stomach, pylorus, or duodenum. Also known as ulcer pepticum, PUD or peptic ulcer disease, is an ulcer (defined as mucosal erosions equal to or greater than 0.5 cm) of an area of the gastrointestinal tract that is usually acidic and thus extremely painful. Causative factors include mucosal infection by the bacterium *Helicobacter pylori* (mechanism unclear) or use of non-steroidal anti-inflammatory drugs (NSAIDs), especially aspirin. Genetic factors such as cigarette smoking, stress, and lower socio-economic status may also play a role. Complications include GI hemorrhage, perforation, and gastric outlet obstruction.

Signs & Symptoms

- Vomiting blood
- Vomiting food eaten hours or days before
- Difficulty swallowing
- Nausea
- Black or tar-like stool (indication that there is blood in the stool)
- Sudden, severe pain in the abdominal area
- Pain that radiates to the back
- Pain that doesn't go away when you take medication
- Unintended weight loss
- Unusual weakness, usually because of anemia

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> Acute pain r/t Chemical burn of gastric mucosa 	Independent <ul style="list-style-type: none"> Note reports of pain, including location, duration, intensity (0–10 scale) Review factors that aggravate or alleviate pain. Identify and limit foods 	<ul style="list-style-type: none"> Pain is not always present, but if present should be compared with patient's previous pain symptoms. This comparison may assist in diagnosis of etiology of 	<ul style="list-style-type: none"> Demonstrated relaxed body posture and be able to sleep/rest appropriately.

	<p>that create discomfort such as spicy or carbonated drink.</p> <ul style="list-style-type: none"> • Encourage small, frequent meals • Encourage patient to assume position of comfort. <p>COLLABORATIVE</p> <ul style="list-style-type: none"> • Provide and implement prescribed dietary modifications. • Administer medications as indicated analgesics, e.g., morphine sulfate anticholinergics, e.g., belladonna, atropine 	<p>bleeding and development of complications</p> <ul style="list-style-type: none"> • Helpful in establishing diagnosis and treatment needs. • Food has an acid neutralizing effect and dilutes the gastric contents. • Small meals prevent distension and the release of gastrin • Reduces abdominal tension and promotes sense of control. • Patient may receive nothing by mouth (NPO) initially. When oral intake is allowed, food choices depend on the diagnosis • May be narcotic of choice to relieve acute/severe pain and reduce peristaltic activity. Note: Meperidine (Demerol) has been associated with increased 	
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		<p>incidence of nausea/vomiting</p> <ul style="list-style-type: none"> • Decreases gastric acidity by absorption or by chemical neutralization . Evaluate choice of antacid in regard to total health picture, e.g., sodium restriction • May be given at bedtime to decrease gastric motility, suppress acid production, delay gastric emptying, and alleviate nocturnal pain associated with gastric ulcer. 	
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Gastro Intestinal (Lower)

Appendicitis

Pathophysiology

- Appendicitis is usually caused by blockage of the lumen of the appendix. Obstruction causes the mucus produced by mucous appendix suffered dam. The longer the mucus is more and more, but the elastic wall of the appendix has limitations that lead to increased intra-luminal pressure. These pressures will impede the flow of lymph resulting in mucosal edema and ulceration. At that time there was marked focal acute appendicitis with epigastric pain.
- When mucus secretion continues, the pressure will continue to increase. This will cause venous obstruction, increased edema and bacteria will penetrate the wall so that the inflammation of the peritoneum arising widespread and can cause pain in the lower right abdomen is called acute suppurative appendicitis.

- If the flow is disrupted arterial wall infarction will occur followed by gangrene appendix. This stage is called appendicitis gangrenosa. If the appendix wall fragile, there will be a perforation, called perforated appendicitis.
- When the process is slow, the omentum and the adjacent bowel will move toward the appendix to appear appendicularis infiltrates.
- In children because it shortens the omentum and appendix is longer, thinner walls. The situation is coupled with the immune system that is still less easy to occur perforation, whereas in the elderly prone to occur because there is blood vessel disorders..

Signs & Symptoms

- Aching pain that begins around your navel and often shifts to your lower right abdomen
- Pain that becomes sharper over several hours
- Tenderness that occurs when you apply pressure to your lower right abdomen
- Sharp pain in your lower right abdomen that occurs when the area is pressed on and then the pressure is quickly released (rebound tenderness)
- Pain that worsens if you cough, walk or make other jarring movements
- Nausea
- Vomiting
- Loss of appetite
- Low-grade fever
- Constipation
- Inability to pass gas
- Diarrhea
- Abdominal swelling

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Acute pain related to inflammation of tissues. 	Independent: <ul style="list-style-type: none"> • <input type="checkbox"/> Investigate pain reports, noting location, duration, intensity (0-10 scale), and characteristics (dull, sharp, constant). • <input type="checkbox"/> Maintain semi fowler's position. • <input type="checkbox"/> Move patient slowly and deliberately. • <input type="checkbox"/> Provide comfort measure like back rubs, deep breathing. Instruct in relaxation or • Visualization exercises. Provide 	<ul style="list-style-type: none"> • <input type="checkbox"/> Changes in location or intensity are not uncommon but may reflect developing complications. • <input type="checkbox"/> Reduces abdominal distention, thereby Reduces tension. • <input type="checkbox"/> Reduces muscle tension or guarding, which may help minimize pain of movement. • <input type="checkbox"/> Promotes relaxation and may enhance 	<ul style="list-style-type: none"> • After nursing interventions the patient will demonstrate the use of relaxation skills, other methods to promote comfort.

	divisional activities. <ul style="list-style-type: none"> □ Provide frequent oral care. Remove noxious environmental stimuli. Collaborative: <ul style="list-style-type: none"> □ Administer analgesics as prescribed. 	patient's coping abilities by refocusing attention. <ul style="list-style-type: none"> □ Reduces nausea and vomiting, which can increase intra-abdominal pressure or pain. □ Reduce metabolic rate and aids in pain relief and Promotes healing. 	
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Small Bowel Obstruction (SBO)

Pathophysiology

Intestinal contents, fluid and gas accumulative above the intestinal obstruction. The abdominal distention and retention of fluid reduce the absorption of fluids and stimulate more gastric secretion. With increasing distention, pressure within the intestinal lumen increases, causing a decrease in venous and arteriolar capillary pressure. This causes edema, congestion, necrosis and eventual rupture or perforation of the intestinal wall, with resultant peritonitis.

Reflux vomiting may be caused by abdominal distention. Vomiting results in a loss of hydrogen ions and potassium from the stomach, leading to a reduction of chlorides and potassium in the blood and to metabolic alkalosis. Dehydration and acidosis develop from loss of water and sodium. With acute fluid losses hypovolemic shock may occur.

Signs & Symptoms

- **Crampy abdominal pain that comes and goes**
- **Nausea**
- **Vomiting**
- **Diarrhea**
- **Constipation**
- **Inability to have a bowel movement or pass gas**
- **Swelling of the abdomen (distention)**

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Crampy Pain that is wavelike and colicky. • Vomiting 	<ul style="list-style-type: none"> • Promoting fluid balance. • Promoting optimal nutrition 	<ul style="list-style-type: none"> • Able to monitor early signs of dehydrations. • Able to 	<ul style="list-style-type: none"> • Reduce anxiety, avoidance of irritating foods,

	<ul style="list-style-type: none"> • Eat slowly and chew properly • Avoid very cold or very hot and irritating them personally. 	<p>provide physical and emotional support and helps the patients manage the symptoms,, which may include nausea, vomiting</p> <ul style="list-style-type: none"> • The use of noninvasive pain relief measures can increase the re- lease of endorphins and enhance the therapeutic effects of pain relief medications • Pain is a subjective experience and must be described by the client in order to plan effective treatment. 	<p>adequate intake of nutrients, maintenance of fluid balance, increased awareness of dietary management and relief pain.</p> <ul style="list-style-type: none"> • Achieves an adequate nutritional intake.
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Constipation

Pathophysiology

Constipation, costiveness, or irregularity, is a condition of the digestive system in which a person experiences hard feces that are difficult to expel.

- This usually happens because the colon absorbs too much water from the food. If the food moves through the gastro-intestinal tract too slowly, the colon may absorb too much water, resulting in feces that are dry and hard.
- Defecation may be extremely painful, and in severe cases (*fecal impaction*) lead to symptoms of bowel obstruction.

Causes of constipation:

- may be dietary
- hormonal
- anatomical a side effect of medications (e.g. some opiates)

- or an illness or disorder.

Signs & Symptoms

- Pass fewer than three stools a week
- Experience hard stools
- Strain excessively during bowel movements
- Experience a sense of rectal blockage
- Have a feeling of incomplete evacuation after having a bowel movement
- Need to use manual maneuvers to have a bowel movement, such as finger evacuation or manipulation of your lower abdomen

Nursing Assessment	Nursing Intervention	Rationale	Goal
Constipation related to decreased dietary intake.	<p>Independent:</p> <ul style="list-style-type: none"> • Determine stool color, consistency, frequency, and amount. • Auscultator bowel sounds. • Encourage fluid intake of 2500- 3000 ml/day within cardiac tolerance. • Recommend avoiding gas forming foods. • Assist in per anal skin condition frequently, noting changes or beginning breakdown. • Discuss use of stool softeners, mild stimulants, bulk-forming laxatives, or enemas as indicated. Monitor effectiveness. • Encourage to at high-fiber rich foods. <p>Collaborative:</p> <ul style="list-style-type: none"> • Consult with dietitian to provide well-balanced diet high in fiber and bulk. 	<ul style="list-style-type: none"> • Assists in identifying causative or contributing factors and appropriate interventions. • Bowel sounds are generally decreased in constipation. • Assists in improving stool consistency. • Decrease gastric distress and abdominal distension. • Prevents skin excoriation and breakdown. • Facilitates defecation when constipation is present. • To enhance easy defecation. • Fiber resists enzymatic digestion and absorbs liquids in its passage along the intestinal tract and thereby produces bulk, which acts as a stimulant to defecation. 	<ul style="list-style-type: none"> • Have regular mealtimes, no skipped meals. • Chew your food well. • Eat slowly. • Be more active. Get some daily exercise. • Use the bathroom at a regular time each day. • Choose a time when you won't have to rush. • Get 7-8 hours sleep (per 24 hours).

Hernia

Pathophysiology

A hernia occurs when part of an internal organ bulges through a weak area of muscle. Most hernias occur in the abdomen. There are several types of hernias, including

- Inguinal, the most common type, is in the groin
- Umbilical, around the belly button
- Incision, through a scar
- Hiatal, a small opening in the diaphragm that allows the upper part of the stomach to move up into the chest.
- Congenital diaphragmatic, a birth defect that needs surgery
- Hernias are common. They can affect men, women and children. A combination of muscle weakness and straining, such as with heavy lifting, might contribute. Some people are born with weak abdominal muscles and may be more likely to get a hernia.

The usual treatment for a hernia is surgery to repair the opening in the muscle wall. Untreated hernias can cause pain and health problems.

Signs & Symptoms

Symptoms of a hiatal hernia

Most people who have a hiatal hernia do not have symptoms and are unaware of the condition. When symptoms of hiatal hernia do occur, they can be related to acid reflux (regurgitation of stomach acid into the esophagus). This is because some people with hiatal hernia also have a condition called GERD (gastroesophageal reflux disease). Large hiatal hernias can be accompanied by symptoms that range in severity from mild to severe and include:

- Acidic taste in the mouth
- Belching
- Difficulty swallowing
- Epigastric pain or burning, which can run from the stomach area up to the mouth
- Heartburn
- Indigestion
- Nausea and vomiting

Symptoms of inguinal and femoral hernias

The hallmark symptom of inguinal and femoral hernias is a small bump or bulge in one or both sides of the groin or testicles (inguinal) or upper thigh (femoral). The bump may be associated with the following symptoms:

- Burning or tenderness
- Pain when lifting something heavy or when exercising
- Pressure in the groin or thigh
- Swelling or pain in the testicle area

Symptoms of an umbilical hernia

The main symptom of an umbilical hernia is a bulge around the belly button that is particularly visible when the affected infant, child or adult is upright or when he or she cries, coughs or strains. Umbilical hernias are typically painless.

Symptoms of a congenital diaphragmatic hernia

Symptoms of a congenital diaphragmatic hernia can be observed in the affected infant when still in the uterus or right after he or she is born. Prenatal signs of a hernia include:

- Excessive amount of amniotic fluid
- Ultrasound showing contents of abdominal cavity in the chest area

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Discomfort or pain in the esophagus • Nausea and vomiting • Unexplained coughing 	<ul style="list-style-type: none"> • Relieving pain • Encourage adequate nutrition intake • Promoting fluid balance 	<ul style="list-style-type: none"> • Small frequent feedings are recommended, because large quantities of food overload the stomach and promote gastric reflux. • Encourage to eat slowly and to chew all food thoroughly so that it can pass easily into the stomach. • Able to monitor early signs of dehydrations. 	<ul style="list-style-type: none"> • Free of pain • Reduce, avoidance of irritating foods, adequate intake of nutrients, maintenance of fluid balance, increased awareness of dietary management and relief pain.

Paralytic Illius

The bowel, or intestine, is the part of the digestive tract that absorbs nutrients from foods we eat. The residue of digested food passes through the bowel and is excreted during elimination, the final stage of digestion. This process can be interrupted or halted by the presence of a bowel obstruction, a blockage that prevents the passage of intestinal contents, such as feces and fluid.

Paralytic ileus is the occurrence of intestinal blockage in the absence of an actual physical obstruction. This type of blockage is caused by a malfunction in the nerves and muscles in the intestine that impairs digestive movement. Causes of ileus include electrolyte imbalances, gastroenteritis (inflammation or infection of the stomach or intestines), appendicitis, pancreatitis (inflammation of the pancreas), surgical complications, and obstruction of the mesenteric artery,

which supplies blood to the abdomen. Certain drugs and medications, such as opioids and sedatives, can cause ileus by slowing peristalsis, the contractions that propel food through the digestive tract.

Pathophysiology

A bowel obstruction occurs when there is a blockage that prevents the passage of intestinal contents. Paralytic ileus is the occurrence of an intestinal blockage in the absence of an actual obstruction. Paralytic ileus is caused by malfunction of the nerves and muscles in the intestines that impairs movement and digestion.

Causes of paralytic ileus include electrolyte imbalances, gastroenteritis (inflammation or infection of the stomach or intestines), appendicitis, pancreatitis (inflammation of the pancreas), surgical complications, and obstruction of the mesenteric artery, which supplies blood to the abdomen. Certain drugs and medications, such as opioids and sedatives, can cause ileus by slowing peristalsis, the contractions that propel food through the digestive tract.

Causes of paralytic ileus

A number of conditions are known causes of paralytic ileus. These include:

- Appendicitis
- Botulism (poisoning with botulinum, a neurotoxin)
- Certain medications, such as opiates and sedatives
- Diabetic ketoacidosis (life-threatening complication of diabetes)
- Electrolyte imbalance
- Gastroenteritis (inflammation or infection of the stomach or intestines)
- Neonatal necrotizing enterocolitis (disease that causes death of intestinal tissue in newborns)
- Obstruction of the mesenteric artery, which supplies blood to the abdomen
- Pancreatitis
- Porphyria (metabolic disorder)
- Surgical complications

Signs & Symptoms

- Abdominal swelling, distension or bloating
- Constipation
- Diarrhea
- Foul-smelling breath
- Gas
- Lack of bowel sounds
- Nausea with or without vomiting
- Stomach pain and spasms

Nursing Assessment	Nursing Intervention	Rationale	Goal
Constipation related to decreased dietary intake.	Independent: <ul style="list-style-type: none"> • Determine stool color, consistency, frequency, and amount. • Auscultator bowel sounds. 	<ul style="list-style-type: none"> • Assists in identifying causative or contributing factors and appropriate interventions. • Bowel sounds are 	<ul style="list-style-type: none"> • Have regular mealtimes, no skipped meals. • Chew your food well. • Eat slowly. • Be more active. Get

	<ul style="list-style-type: none"> • Encourage fluid intake • Recommend avoiding gas forming foods. • Assist in per anal skin condition frequently, noting changes or beginning breakdown. • Discuss use of stool softeners, mild stimulants, bulk-forming laxatives, or enemas as indicated. Monitor effectiveness. • Encourage to eat high-fiber rich foods. <p>Collaborative:</p> <ul style="list-style-type: none"> • Consult with dietitian to provide well-balanced diet high in fiber and bulk. 	<p>generally decreased in constipation.</p> <ul style="list-style-type: none"> • Assists in improving stool consistency. • Decrease gastric distress and abdominal distension. • Prevents skin excoriation and breakdown. • Facilitates defecation when constipation is present. • To enhance easy defecation. • Fiber resists enzymatic digestion and absorbs liquids in its passage along the intestinal tract and thereby produces bulk, • which acts as a stimulant to defecation. 	<p>some daily exercise.</p> <ul style="list-style-type: none"> • Use the bathroom at a regular time each day. • Choose a time when you won't have to rush.
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Ishemic Bowel

Pathophysiology

The small intestine receives blood via the coeliac artery (CA) and the superior mesenteric artery (SMA). The colon receives blood via the SMA and the inferior mesenteric artery (IMA). The rectum also receives blood via branches of the internal iliac artery. Several collateral arteries exist between the SMA and the IMA, including the marginal artery of Drummond and the arc of Riolan. The splenic flexure and the recto-sigmoid junction are 2 watershed areas where **collateralization** of blood flow may be limited. [View image](#) [View image](#)

Ischaemia occurs secondary to hypo-perfusion of an intestinal segment. When hypo-perfusion occurs, collateral blood flow may preclude or **minimize** ischaemia; however, the regions of the intestine with a solitary arterial supply, and the watershed areas, are both at increased risk of developing ischaemia. The degree of intestinal injury is dependent on the duration and severity of ischaemia. Acute or subacute mucosal sloughing and ulcerations occur as a result of ischaemia. The loss of the mucosal barrier allows for bacterial translocation and toxin or cytokine absorption. Re-perfusion injury can also occur if blood supply is re-established after a prolonged interruption. Segments of bowel which do not cause acute necrosis or perforation can heal with stenosis or stricture. These can cause ischaemic bowel disease with long-term sequelae, which is either mild and chronic or acute and resolved.

Thromboembolic events that lead to mesenteric ischaemia usually involve the SMA instead of the other mesenteric arteries (IMA and celiac artery). This is because of the anatomical position of the

SMA; the SMA is positioned vertically while the other vessels form more oblique angles from the aorta.

Signs & Symptoms

Symptoms of ischemic bowel disease may include:

- Abdominal pain:
 - Abdominal pain is usually worse after meals
 - Abdominal pain may suddenly become severe
 - Often described as cramping abdominal pain
 - Pain is usually generalized or all over the abdomen
 - Lower abdominal pain
 - Upper abdominal pain
- Abdominal tenderness
 - Right lower abdominal tenderness
 - Left lower abdominal tenderness
 - Right upper abdominal tenderness
 - Left upper abdominal tenderness
 - Upper abdominal tenderness
 - Lower abdominal tenderness
- Blood in the stool:
 - Black stool
 - Rectal bleeding
 - Red stools
 - Maroon stools
 - Constipation
 - Indigestion
 - Diarrhea
 - Nausea
 - Vomiting
 - Anorexia

Nursing Assessment	Nursing Intervention	Rationale	Goal
Constipation related to decreased dietary intake.	Independent: <ul style="list-style-type: none"> • Determine stool color, consistency, frequency, and amount. • Auscultator bowel sounds. • Encourage fluid intake of 2500- 3000 ml/day within cardiac tolerance. • Recommend avoiding gas forming foods. • Assist in per anal skin condition frequently, noting changes or beginning 	<ul style="list-style-type: none"> • Assists in identifying causative or contributing factors and appropriate interventions. • Bowel sounds are generally decreased in constipation. • Assists in improving stool consistency. • Decrease gastric distress and abdominal distension. • Prevents skin excoriation and breakdown. 	<ul style="list-style-type: none"> • Have regular mealtimes, no skipped meals. • Chew your food well. • Eat slowly. • Be more active. Get some daily exercise. • Use the bathroom at a regular time each day. • Choose a time when you won't have to rush. • Get 7-8 hours sleep (per 24 hours).

	<p>breakdown.</p> <ul style="list-style-type: none"> • Discuss use of stool softeners, mild stimulants, bulk-forming laxatives, or enemas as indicated. Monitor effectiveness. • Encourage to eat high-fiber rich foods. <p>Collaborative:</p> <ul style="list-style-type: none"> • Consult with dietitian to provide well-balanced diet high in fiber and bulk. 	<ul style="list-style-type: none"> • Facilitates defecation when constipation is present. • To enhance easy defecation. • Fiber resists enzymatic digestion and absorbs liquids in its passage along the intestinal tract and thereby produces bulk, • which acts as a stimulant to defecation. 	
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Volvulus

A volvulus is a bowel obstruction with a loop of bowel that has abnormally twisted on itself.

Pathophysiology

In simple mechanical obstruction, blockage occurs without vascular compromise. Ingested fluid and food, digestive secretions, and gas accumulate above the obstruction. The proximal bowel distends, and the distal segment collapses. The normal secretory and absorptive functions of the mucosa are depressed, and the bowel wall becomes edematous and congested. Severe intestinal distention is self-perpetuating and progressive, intensifying the peristaltic and secretory derangements and increasing the risks of dehydration and progression to strangulating obstruction.

Strangulating obstruction is obstruction with compromised blood flow; it occurs in nearly 25% of patients with small-bowel obstruction. It is usually associated with hernia, volvulus, and intussusceptions. Strangulating obstruction can progress to infarction and gangrene in as little as 6 h. Venous obstruction occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall. The ischemic bowel becomes edematous and infarcts, leading to gangrene and perforation. In large-bowel obstruction, strangulation is rare (except with volvulus).

Perforation may occur in an ischemic segment (typically small bowel) or when marked dilation occurs. The risk is high if the cecum is dilated to a diameter ≥ 13 cm. Perforation of a tumor or a diverticulum may also occur at the obstruction site.

Signs & Symptoms

The patient with volvulus complains of severe abdominal pain and may report bilious vomiting. If the patient is an infant, the parents may report increased vomiting of feedings. The history may also reveal the passage of bloody stools.

On inspection, the patient appears to be in pain. Abdominal inspection and palpation may reveal distention and a palpable mass.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Acute Pain • Abdominal Nausea • Imbalance nutrition • Impaired oral mucous membrane: Dryness • Fear and anxiety 	<ul style="list-style-type: none"> • Relieving pain • Encourage adequate nutrition intake • Promoting fluid balance 	<ul style="list-style-type: none"> • Small frequent feedings are recommended , because large quantities of food overload the stomach and promote gastric reflux. • Encourage to eat slowly and to chew all food thoroughly so that it can pass easily into the stomach. • Able to monitor early signs of dehydrations. 	<ul style="list-style-type: none"> • Free of pain • Reduce, avoidance of irritating foods, adequate intake of nutrients, maintenance of fluid balance, increased awareness of dietary management and relief pain.

Diverticulitis

Diverticulitis is a common digestive disease particularly found in the large intestine. Diverticulitis develops from diverticulosis, which involves the formation of pouches (diverticula) on the outside of the colon. Diverticulitis results if one of these diverticula becomes inflamed.

Pathophysiology

Diverticula are small mucosal herniations protruding through the intestinal layers and the smooth muscle along the natural openings created by the vasa recta or nutrient vessels in the wall of the colon. These herniations create small pouches lined solely by mucosa. Diverticula can occur anywhere in the gastrointestinal tract but are usually observed in the colon. The sigmoid colon has the highest intraluminal pressures and is most commonly affected. Diverticulosis is defined as the condition of having uninflamed diverticula. The cause of diverticulosis is not yet conclusive, but it appears to be associated with a low-fiber diet, constipation, and obesity.

Diverticulitis is defined as an inflammation of one or more diverticula. Its pathogenesis remains unclear. Fecal material or undigested food particles may collect in a diverticulum, causing obstruction. This obstruction may result in distension of the diverticula secondary to mucous secretion and overgrowth of normal colonic bacteria. Vascular compromise and subsequent microperforation or macroperforation then ensue. Alternatively, some believe that increased intraluminal pressure or inspissated food particles cause erosion of the diverticular wall, resulting

in inflammation, focal necrosis, and perforation. The disease is frequently mild when pericolic fat and mesentery wall off a small perforation. However, larger perforations and more extensive disease lead to abscess formation and, rarely, intestinal rupture or peritonitis.

Fistula formation is a complication of diverticulitis. Fistulas to adjacent organs and the skin may develop, especially in the presence of an abscess. In men, colovesicular fistulas are the most common. In women, the uterus is interposed between the colon and the bladder, and this complication is only seen following a hysterectomy. The uterus precludes fistula formation from the sigmoid colon to the urinary bladder. However, colovaginal and colocutaneous fistulas can form but are uncommon.

Recurrent attacks of diverticulitis can result in the formation of scar tissue, leading to narrowing and obstruction of the colonic lumen.

Signs & Symptoms

People with diverticulosis often have no symptoms, but they may have bloating and cramping in the lower part of the belly. Rarely, they may notice blood in their stool or on toilet paper.

Symptoms of diverticulitis are more severe and often start suddenly, but they may become worse over a few days. They include:

- Tenderness, usually in the left lower side of the abdomen
- Bloating or gas
- Fever and chills
- Nausea and vomiting
- Not feeling hungry and not eating

Nursing Assessment	Nursing Intervention & Rationale	Goal
<ul style="list-style-type: none"> • Pain related to inflamed bowel and possible peritonitis • Risk for deficient fluid volume related to inflammation • Impaired tissue integrity: Gastrointestinal related to perforated diverticulum • Deficient knowledge related to disease process and 	<ul style="list-style-type: none"> • Assess comfort status frequently, providing analgesics as needed. • Maintain intravenous infusion as prescribed. • Measure intake and output; weigh daily. • Provide mouth care every 2 to 4 hours until oral intake resumes, then every 4 hours until client assumes self-care. • Measure temperature every 4 hours. • Advance diet from clear liquids to low-residue diet when allowed. • Provide instruction and dietary consultation for high-fiber diet 	<ul style="list-style-type: none"> • Verbalize adequate pain relief. • Experience no adverse effects of prescribed bed rest. • Maintain adequate fluid balance while hospitalized, as demonstrated by balanced intake and output, stable weight, good skin turgor and mucous membrane moisture, and laboratory value within the normal range. • Heal adequately without further evidence of peritonitis. • Verbalize

dietary management		understanding of the recommended high-fiber diet and the need to increase physical activity and fluid intake to promote optimal bowel function at home.
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Resection of Intestines

Small bowel resection is surgery to remove part or all of your small bowel. It is done when part of your small bowel is blocked or diseased.

The small bowel is also called the small intestine. Most digestion (breaking down and absorbing nutrients) of the food you eat takes place in the small intestine.

Description

You will receive general anesthesia at the time of your surgery. This will make you asleep and pain-free.

If you have laparoscopic surgery:

- You will have three to five small cuts in your lower belly. The surgeon will pass a camera and medical instruments through these cuts.
- You may also have a cut of about 2 to 3 inches if your surgeon needs to put a hand inside your belly to feel the intestine or remove the diseased segment.
- Your belly will be filled with gas to expand it. This makes it easy for the surgeon to see and work.

If you have open surgery, you will probably have a cut about 6 inches long in your mid-belly.

- Your surgeon will locate the part of your small intestine that is diseased.
- Then your surgeon will put clamps on both ends of this part to close it off.
- The surgeon will remove the diseased part.

In both kinds of surgery:

- If there is enough healthy small intestine left, your surgeon will sew or staple the healthy ends of the small intestine back together. Most patients have this done.
- If you do not have enough healthy small intestine to reconnect, your surgeon will make an opening called a stoma through the skin of your belly. Your small intestine will be attached to the outer wall of your belly. Stool will go through the stoma into a drainage bag outside your body. This is called an ileostomy. The ileostomy may either be short-term or permanent.

Your surgeon may also look at lymph nodes and other organs in your belly area. Before surgery, the surgeon will talk with you about the possible need to remove other organs.

This surgery usually takes 1 to 4 hours.

Why the Procedure is Performed

Small bowel resection may be recommended for:

- A blockage in the intestine caused by scar tissue or congenital (from birth) deformities
- Bleeding, infection, or ulcers caused by inflammation of the small intestine. Three conditions that may cause inflammation are regional ileitis, regional enteritis, and Crohn's disease.
- Cancer
- Carcinoid tumor
- Injuries to the small intestine
- Meckel's diverticulum
- Noncancerous (benign) tumors
- Precancerous polyps (nodes)

Risks

Risks for any surgery are:

- Blood clots in the legs that may travel to the lungs
- Breathing problems
- Bleeding inside your belly
- Heart attack or stroke
- Infection, including in the lungs, urinary tract, and belly

Risks for this surgery include:

- Bulging tissue through the incision, called an incisional hernia
- Damage to nearby organs in the body
- Many episodes of diarrhea
- Problems with your ileostomy
- Scar tissue that forms in your belly and causes a blockage of your intestines
- Short bowel syndrome (when a large amount of the small intestine needs to be removed), which may lead to problems absorbing important nutrients and vitamins
- The ends of your intestines that are sewn together comes apart (anastomotic leak -- this may be life-threatening)
- Wound breaking open (dehiscence)
- Wound infections

Inflammatory Bowel Disease

Pathophysiology

Regional enteritis

• Is a subacute and chronic inflammation that extends through layers of the bowel walls from the intestinal mucosa. Fistula, fissures, and abscesses extend into the peritoneum, but segments of normal intestinal tissue occur between the inflammations.

Ulcerative colitis

• Is an inflammatory disease of the submucosal layer of the colon and rectum characterized by continuously occurring ulcerations and shedding of intestinal epithelium. Fat deposits and muscular hypertrophy result in a narrow, short, and thickened bowel.

Signs & Symptoms

•Regional enteritis

- Abdominal tenderness and pain, typically colicky and increased after meals
- Diarrhea, flatulence, and steatorrhea
- Fever, malaise, and anorexia
- Signs of nutritional deficits
- Perianal fistulas and abscesses
- Usually occurs in ileum and ascending colon

•Ulcerative colitis

- Severe diarrhea containing pus, blood and mucosa
- Abdominal cramping and tenderness, fever
- Anorexia and weight loss
- Usually occurs in the descending colon and rectum

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Acute Pain related to Hyperperistalsis, prolonged diarrhea, skin and tissue irritation, perirectal excoriation, fissures, fistulas. 	<ul style="list-style-type: none"> • Encourage client to report pain. • Assess reports of abdominal cramping or pain, noting location, duration and intensity. Investigate and report changes in pain characteristics. • Note nonverbal cues, such as restlessness, reluctance to move, abdominal guarding, withdrawal, and depression. Investigate discrepancies between verbal and nonverbal cues. • Review factors that aggravate or alleviate pain. • Encourage client to assume position of comfort, such as knees flexed. • Provide comfort measures and 	<ul style="list-style-type: none"> • May try to tolerate pain rather than request analgesics. • Colicky intermittent pain occurs with Crohn's disease. Predefecation pain frequently occurs in UC with urgency, which may be severe and continuous. Changes in pain characteristics may indicate spread of disease or developing complications, such as bladder fistula, perforation and toxic megacolon. • Body language or non verbal cues may be both physiological and psychological and maybe used in conjunction with verbal cues to 	<ul style="list-style-type: none"> • Bowel function stabilized. • Complications prevented/controlled • Dealing positively with condition. • Disease process/prognosis, therapeutic regimen, and potential complications are understood. • Plan in place to meet needs after discharge.

	<p>diversional activities.</p> <ul style="list-style-type: none"> • Cleanse rectal area with mild soap and water • Implement prescribed dietary modification for example, commence with liquids and increase to solid foods as tolerated. • Provide sitz bath, as appropriate. • Observe and record abdominal distention, increased temp. and decreased BP. 	<p>determine extent and severity of the problem.</p> <ul style="list-style-type: none"> • May pinpoint precipitating or aggravating factors or identify developing complications. • Reduces abdominal tension and promotes sense of control. • Promotes relaxation, refocuses attention, and may enhance coping abilities. • Protects skin from bowel acids, preventing excoriation. • Complete bowel rest can reduce pain and cramping. • Enhances cleanliness and comfort in the presence of perianal irritation and fissures. • May indicate developing intestinal obstruction from inflammation, edema, and scarring. 	
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Colorectal Cancer

Pathophysiology

Colorectal cancer is a disease in which normal cells in the lining of the colon or rectum begin to change, start to grow uncontrollably, and no longer die. These changes usually take years to develop; however, in some cases of hereditary disease, changes can occur within months to years. Both genetic and environmental factors can cause the changes. Initially, the cell growth appears as a benign (noncancerous) polyp that can, over time, become a cancerous tumor. If not treated or removed, a polyp can become a potentially life-threatening cancer. Recognizing and removing precancerous polyps before they become cancer can prevent colorectal cancer.

Signs & Symptoms

- Ascending (Right) Colon Cancer
 - Occult blood in stool
 - Anemia
 - Anorexia and weight loss
 - Abdominal pain above umbilicus
 - Palpable mass
- Distal Colon/Rectal Cancer
 - Rectal bleeding
 - Changed in bowel habits
 - Constipation or Diarrhea
 - Pencil or ribbon – shaped stool
 - Tenesmus
 - Sensation of incomplete bowel emptying

Dukes' Classification of Colorectal Cancer

- Stage A: Confined bowel mucosa, 80-90% 5-year survival rate
- Stage B: Invading muscle wall
- Stage C: Lymph node involvement
- Stage D: Metastases or locally unresectable tumor, less than 5% 5-year survival rate

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Fatigue related to altered body chemistry, side effects of pain and other medications chemotherapy 	INDEPENDENT: <ul style="list-style-type: none"> • Have patient rate fatigue, using a numeric scale, if possible, the time of day when it is most severe. • Plan care to allow rest periods. Schedule activities for periods when patient has most energy. • Assist patient with self-care needs. Keep bed in low position and assist with ambulation. • Encourage patient to do whatever possible and increase activity level as tolerated. • Perform pain assessment and provide pain management as prescribed. • Encourage nutritional intake. 	<ul style="list-style-type: none"> • Help in developing a plan for managing fatigue. • Frequent rest periods or naps are needed to restore or conserve energy. Planning will allow patient to be active during times when energy level is higher, which may restore feeling of well being and a sense of control. • Weakness may make activities of daily living and ambulation difficult, further assistance is needed. • Enhances strength and enables patient to become more active without undue fatigue. • Poorly managed cancer pain can contribute to fatigue. 	<ul style="list-style-type: none"> • patient was able to report improved sense of energy.

	COLLABORATIVE: <ul style="list-style-type: none"> Refer for physical therapy. 	<ul style="list-style-type: none"> Adequate intake of nutrients is necessary to meet energy needs and build energy reserves for activity. Programmed daily exercises and activities help patient maintain or increase strength and muscle tone which enhances sense of well being. 	
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Orthopedics (BONES)

Hip Fracture

Pathophysiology

Fracture pathophysiology includes cortical disruption, peri-osteal damage, and damage to the intra-medullary and cancellous architecture. Histomorphometric studies have shown that cortical thinning and some decrease in trabecular bone mass and connectivity can be seen especially in osteoporosis suggesting a lower quality of bone, and thus decreased mechanical strength resulting in fracture. An age-related decline in osteocyte viability has also been observed in experimental studies. An inflammatory response also occurs following fractures of the proximal femur.

Signs & Symptoms

- Inability to move immediately after a fall
- Severe pain in your hip or groin
- Inability to put weight on your leg on the side of your injured hip
- Stiffness, bruising and swelling in and around your hip area
- Shorter leg on the side of your injured hip
- Turning outward of your leg on the side of your injured hip

Nursing Assessment	Nursing Intervention & Rationale	Goal
<ul style="list-style-type: none"> Increased risk of hypovolemia and shock related to trauma and bleeding. Increased risk of bone inflammation related to open fracture. 	<ul style="list-style-type: none"> Provide emergency care if requires (homeostasis, respiratory care, prevention of shock) Provide fracture fixation to prevent following injury of tissues Observe signs of fat embolism (especially during first hours after the fracture) Monitor fluids input and output 	<ul style="list-style-type: none"> Increase comfort, decrease pain. Prevent avoidable injury. Prevent complications of immobility. Provide optimal bone and wound healing. Then surgical intervention prescribed, prevent

<ul style="list-style-type: none"> • Increased risk of fat embolism related to fracture of the long bones. • Increased risk of severe fluid, electrolyte, and metabolic imbalances related to injury or inflammation. • Pain and immobility, related to diagnosis of fracture. • Increased risk of respiratory, cardiovascular, bowel, and skin complications related to a long period of immobility. • Anxiety related to the symptoms of disease and fear of the unknown. 	<p>continuously, insert IV catheter, urinary catheter</p> <ul style="list-style-type: none"> • Monitor client's vital signs • Monitor client's laboratory tests results for abnormal values • Administer IV therapy, analgesics, antibiotics, and other medications as prescribed • Prepare client and his family for surgical intervention if required • For client after surgical intervention provide routine postoperative care and teach about possible postoperative complications • Provide care to client with cast (observe signs of circulatory impairment – change in skin color and temperature, diminished distal pulses, pain and swelling of the extremity; protect the cast from damage) • Provide care to client in traction (check the weights are hanging freely, observe skin for irritation and site of skeletal traction insertion for signs of infection; use aseptic technique when cleaning the site of insertion) • In case of hip fracture and hip replacement maintain the adduction of the affected extremity • Provide respiratory exercises to prevent lung complications • Observe for signs of thrombophlebitis, report immediately • Provide appropriate skin care to prevent pressure sores • Encourage fluid intake and high-protein, high-vitamin, high-calcium diet • Teach the client appropriate crutch-walking techniques • Provide emotional support to client, explain all procedures to decrease anxiety and to obtain cooperation • Instruct client regarding fracture healing process, diagnostic procedures, treatment and its complications, home care, daily activities, diet, restrictions and follow-up 	<p>postoperative complications.</p> <ul style="list-style-type: none"> • Decreased anxiety with increased knowledge.
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Total Knee Replacement (TKR)

Knee replacement, or knee arthroplasty, is a surgical procedure to replace the weight-bearing surfaces of the knee joint to relieve the pain and disability of osteoarthritis. It may be performed for other knee diseases such as rheumatoid arthritis and psoriatic arthritis. In patients with severe deformity from advanced rheumatoid arthritis, trauma, or long standing osteoarthritis, the surgery may be more complicated and carry higher risk. Osteoporosis does not typically cause knee pain, deformity, or inflammation and is not a reason to perform knee replacement.

Other major causes of debilitating pain include meniscus tears, cartilage defects, and ligament tears. Debilitating pain from osteoarthritis is much more common in the elderly.

Knee replacement surgery can be performed as a partial or a total knee replacement. In general, the surgery consists of replacing the diseased or damaged joint surfaces of the knee with metal and plastic components shaped to allow continued motion of the knee.

The operation typically involves substantial postoperative pain, and includes vigorous physical rehabilitation. The recovery period may be 6 weeks or longer and may involve the use of mobility aids (e.g. walking frames, canes, crutches) to enable the patient's return to preoperative mobility.

Pathophysiology

The exact cause of the degenerative process in primary osteoarthritis is unknown. It may represent a defect in cellular (chondrocyte) repair processes. Osteoarthritic cartilage contains increased amounts of water, alterations in the type of proteoglycan, type 2 collagen abnormalities and increased levels of the cathepsins, metalloproteinases, interleukin 1 and others as a complex cascade of enzymatic process. Changes in the synovium include synoviocyte hyperplasia, an increased leukocyte population in the membrane and fluid, occasional giant cells, neovascularisation with increased vessel permeability and altered matrix and cellular cytokine formation.

Long Bone Injury

Pathophysiology

When a bone is broken, the periosteum and blood vessels in the cortex, marrow, and surrounding soft tissues are disrupted. Bleeding occurs from the damaged ends of the bone and from the neighboring soft tissue. A clot (hematoma) forms within the medullary canal, between the fractured ends of the bone, and beneath the periosteum. Bone tissue immediately adjacent to the fracture dies. This necrotic tissue along with any debris in the fracture area stimulates an intense inflammatory response characterized by vasodilation, exudation of plasma and leukocytes, and infiltration by inflammatory leukocytes and mast cells. Within 48 hours after the injury, vascular tissue invades the fracture area from surrounding soft tissue and the marrow cavity, and blood flow to the entire bone is increased. Bone-forming cells in the periosteum, endosteum, and marrow are activated to produce subperiosteal procallus along the outer surface of the shaft and over the broken ends of the bone. Osteoblasts within the procallus synthesize collagen and matrix, which becomes mineralized to form callus (woven bone). As the repair process continues, remodeling occurs, during which unnecessary callus is resorbed and trabeculae are formed along lines of stress. Except for the liver, bone is unique among all body tissues in that it will form new bone, not scar tissue, when it heals after a fracture."

Signs & Symptoms

Although bone tissue itself contains no nociceptors, bone fracture is very painful for several reasons:

- Breaking in the continuity of the periosteum, with or without similar discontinuity in endosteum, as both contain multiple nociceptors.
- Edema of nearby soft tissues caused by bleeding of torn periosteal blood vessels evokes pressure pain.
- Muscle spasms trying to hold bone fragments in place

Damage to adjacent structures such as nerves or vessels, spinal cord and nerve roots (for spine fractures), or cranial contents (for skull fractures) can cause other specific signs and symptoms.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<p>Possible Etiologies: (Related to)</p> <p>Individual</p> <ul style="list-style-type: none"> - Loss of skeletal integrity (fracture) - Movement of bone fragments - Balancing difficulties - Weakness - Lack of safety education/ precautions - History of previous trauma <p>Environment</p> <ul style="list-style-type: none"> - Slippery floors - Bathtub without hand grip - Unsteady ladder or chairs - Unlit room - Unsteady or absence of stair rails - High bed 	<ul style="list-style-type: none"> • Determine factors related to individual situation and extent of risk; evaluate the environment for appropriateness to client; and knowledge of caregiver to safety needs. • Orient the client and his caregiver to the physical setup of the facility and demonstrate the use of call bell/ light which is placed within reach of the client. • Maintain bed rest/ limb rest and provide support to joints of both below and above of the affected limb, especially during movement or turning. • Place bed board under the mattress. • Support fracture with pillows and maintain affected part in neutral position with sandbags, trochanter rolls, or footboard. • Check for resolution of edema. • Maintain the position of traction. • Make sure that all clamps are functional; lubricate pulleys and check ropes for fraying. • Avoid lifting and releasing the weights. • Assist client with proper placement of lifts under bed wheels is indicated. 	<ul style="list-style-type: none"> • This is to provide a baseline data on client's condition and could help assess the extent of risk for additional trauma. • Orientation could help the client fully maximize his full potential while within the hospital facility. • It gives stability and reduces the possibility of disturbing the alignment. • Sagging mattress may deform a wet plaster cast, crack a dry cast, or interfere with pull of traction. • It prevents unnecessary disruption of alignment and pressure deformities in the drying cast. • As swelling 	<p>Client will be able to perform correct body mechanics, reducing his risk for further injury.</p> <p>Client will be able to understand and accept skeletal integrity and will be able to recognize the need for assistance; identify and correct possible factors in the environment and demonstrate lifestyle changes in promoting bone integrity and preventing self from further injury.</p>

	<ul style="list-style-type: none"> • Instruct client about restrictions like not bending at waist or sitting with Buck traction and not turning below the waist with Russel traction. • Encourage client verbalize feelings and problems regarding fracture. • Administer medications prior to activities. • Perform and supervise client with active and passive ROM exercises. • Educate and assist in performing proper body mechanics in sitting, assisted walking as indicated. • Review X rays of client. 	<p>subsides, a readjustment of splint or application of plaster may be done to ensure alignment of bone.</p> <ul style="list-style-type: none"> • It permits pull on the long axis of the fractured part and overcomes muscle tension. • To avoid interruption of fracture approximation. • It prevents sudden pull on fracture, which could be associated with pain and muscle spasm. • It could help maintain client's proper position and function of traction by counterbalance. • It maintains the proper pull of traction. • Helps alleviate anxiety and helps client cope with situation. • It promotes muscle relaxation and encourages client to participate in rehabilitative activities. • It promotes strength and mobility of unaffected muscles and facilitates healing of 	
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		<p>surrounding trauma.</p> <ul style="list-style-type: none"> • It provides an avenue for the client to develop a sense of self reliance and would guide client appropriately within precautionary measures. • It provides visual evidence of proper alignment/ healing process of the fractured bone; the need for continued therapy. 	
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Osteoarthritis (OA)

Pathophysiology

- The most common form of arthritis.
- It causes the deterioration of the joint cartilage and formation of reactive new bone at the margins and subchondral areas of the joint.
- This chronic degeneration results from a breakdown of chondrocytes, most often in the hips and knees.
- Osteoarthritis occurs equally in both sexes after age 40.
- The earliest symptoms appear in middle age and progress with advancing age.
- Depending on the site and severity of joint involvement, disability can range from minor limitation of the fingers to near immobility in persons with hip or knee disease.
- Progression rates vary; joints may remain stable for years in the early stage of deterioration.

Etiology And Pathophysiology

- Changes in articular cartilage occur first; later, secondary soft tissue changes may occur.
- Progressive wear and tear on cartilage leads to thinning of joint surface and ulceration into bone.
- Leads to inflammation of the joint and increased blood flow and hypertrophy of subchondral bone.
- New cartilage and bone formation at joint margins results in osteophytosis, altering the size and shape of the bone.
- Generally affects adults ages 50 to 90; equal to males and females.
- Cause is unknown, but aging and obesity are contributing factors. Previous trauma cause secondary osteoarthritis.

Signs & Symptoms

- Joint pain
- Joint stiffness
- Joint tenderness
- Limited range-of-motion
- Crepitus (crackling, grinding noise with movement)
- Joint effusion (swelling)
- Local inflammation
- Bony enlargements and osteophyte formation

Nursing Assessment	Nursing Intervention & Rationale	Goal
Chronic pain related to joint deterioration.	<ul style="list-style-type: none"> • Provide rest for involved joints. Excessive use aggravates the symptoms and accelerates degeneration. • Advise the patient to avoid activities that precipitate pain. • Apply heat as directed to relieve muscle pain and stiffness. • Teach the patient correct posture and body mechanics. • Advise the patient to sleep with rolled terry cloth towel under the neck to relieve cervical pain. • Provide patient with crutches, braces, or cane when indicated to reduce-weight bearing stress on hips and knees. • Encourage patient to wear corrective shoes and metatarsal support for foot disorders. • Encourage patient to lose weight to decrease stress on weight-bearing joints. • Teach the patient range-of-motion exercises to maintain joint mobility. • Refer patient to physical and occupational therapy. 	Describes risk factors, the disease process, and rehabilitation activities necessary to manage the therapeutic regimen

Rheumatoid Arthritis (RA)

Pathophysiology

Rheumatoid arthritis (RA) is a chronic, systemic inflammatory disorder that may affect many tissues and organs, but principally attacks the joints producing an inflammatory synovitis that often progresses to destruction of the articular cartilage and ankylosis of the joints.

Rheumatoid arthritis can also produce diffuse inflammation in the lungs, pericardium, pleura, and sclera, and also nodular lesions, most common in subcutaneous tissue under the skin.

Although the cause of rheumatoid arthritis is unknown, autoimmunity plays a pivotal role in its chronicity and progression.

About 1% of the world's population is afflicted by rheumatoid arthritis, women three times more often than men. Onset is most frequent between the ages of 40 and 50, but people of any age can be affected. It can be a disabling and painful condition, which can lead to substantial loss of functioning and mobility. It is diagnosed chiefly on symptoms and signs, but also with blood tests (especially a test called rheumatoid factor) and X-rays. Diagnosis and long-term management are typically performed by a rheumatologist, an expert in the diseases of joints and connective tissues.

Signs & Symptoms

- Tender, warm, swollen joints
- Morning stiffness that may last for hours
- Firm bumps of tissue under the skin on your arms (rheumatoid nodules)
- Fatigue, fever and weight loss

Nursing Assessment	Nursing Intervention	Rationale	Goal
Acute pain r/t distension of tissues by accumulation of fluid	<p><i>Independent</i></p> <ul style="list-style-type: none"> • Investigate reports of pain, noting location and intensity(scale of 0–10). Note precipitating factors and nonverbal pain cues. • Recommend/provide firm mattress or bedboard, small pillow. Elevate linens with bed cradle as needed. • Suggest patient assume position of comfort while in bed or sitting in chair. Promote bedrest as indicated. 	<ul style="list-style-type: none"> • Helpful in determining pain management needs and effectiveness of program • Soft/sagging mattress, large pillows prevent maintenance of proper body alignment, placing stress on affected joints. Elevation of bed linens reduces pressure on inflamed/painful joints. • In severe disease/acute exacerbation, total 	Demonstrated relaxed body posture and be able to sleep/rest appropriately.

	<p><i>Collaborative</i></p> <ul style="list-style-type: none"> • Apply ice or cold packs when indicated • Assist with physical therapies, e.g., paraffin glove, whirlpool baths. <p>Administer medications as indicated</p> <ul style="list-style-type: none"> • Salicylates, e.g., aspirin (ASA) (Acuprin, Ecotrin, ZORprin); <p>Tetracyclines, e.g., minocycline (Minocin);</p>	<p>bedrest may be necessary (until objective and subjective improvements are noted) to limit pain/injury to joint.</p> <ul style="list-style-type: none"> • Cold may relieve pain and swelling during acute episodes. • Provides sustained heat to reduce pain and improve ROM of affected joints • ASA exerts an anti-inflammatory and mild analgesic effect, decreasing stiffness and increasing mobility. ASA must be taken regularly to sustain a therapeutic blood level. Research indicates that ASA has the lowest toxicity index of commonly prescribed NSAIDs. <p>CHARACTERISTICS OF ANTI-INFLAMMATORY AND IMMUNE MODIFIER EFFECTS COUPLED WITH ABILITY TO BLOCK METALLOPROTEINASES</p>	
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Gout

Pathophysiology

Gout is a disorder of purine metabolism characterized by elevated uric acid levels with deposition of urate crystals in joints and other tissues. High uric acid levels result from decreased excretion of uric acid (90% of cases) due to a wide variety of causes. The disorder may progress from an asymptomatic stage through acute gouty arthritis, to chronic tophaceous gout. Complications include erosive deforming arthritis, uric acid kidney stones, and urate nephropathy caused by hyperuricemia.

Signs & Symptoms

- **Intense joint pain.** Gout usually affects the large joint of your big toe, but it can occur in your feet, ankles, knees, hands and wrists. The pain is likely to be most severe within the first 12 to 24 hours after it begins.
- **Lingering discomfort.** After the most severe pain subsides, some joint discomfort may last from a few days to a few weeks. Later attacks are likely to last longer and affect more joints.
- **Inflammation and redness.** The affected joint or joints become swollen, tender and red.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired physical mobility related to pain 	<p><i>Independent:</i></p> <ul style="list-style-type: none"> • Evaluate or continuously monitor degree of joint inflammation or pain. • Maintain bed rest or chair rest when indicated. • Schedule activities providing frequent rest periods and uninterrupted night time sleep. • Encourage adequate fluid intake. • Assist with active or passive range of motion. • Encourage patient to maintain upright and erect posture when sitting, standing, or walking. • Encourage the patient to avoid alcohol. • Review foods that are rich in purines like sardines, anchovies, shell fish and organ meats. • Provide safety needs. <p><i>Collaborative:</i></p> <ul style="list-style-type: none"> • Administer anti-inflammatory drugs and also colchicines 	<ul style="list-style-type: none"> • Level of activity or exercise depends on progression and • resolution of inflammatory process. • Systemic rest during acute attacks and important throughout all phases of disease to reduce fatigue and improve strength. • To assist with excretion of uric acid and decrease likelihood of stone formation. • Maintains or improves joint function, muscle strength, and general stamina. • Maximizes joint function, maintains mobility. • That can precipitate acute attack. • To avoid foods that precipitate acute attacks. • Help prevent accidental injuries or falls. • To relieve pain and swelling during acute attacks. 	<ul style="list-style-type: none"> • able to maintain or increase strength and function of affected or compensatory body part.

	as prescribed.		
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Vascular Disorders

Peripheral Artery Disease (PAD)

Pathophysiology

Peripheral arterial disease (PAD) is a systemic atherosclerotic process for which the major risk factors are similar to those for atherosclerosis in the carotid, coronary, and other vascular beds. Among the traditional risk factors for PAD, those with the strongest associations are advanced age, smoking, and diabetes mellitus. More recently, a number of nontraditional risk factors for PAD have also been recognized. This article briefly reviews the pathophysiology of PAD and the evidence supporting established and emerging risk factors for its development.

Signs & Symptoms

- Painful cramping in your hip, thigh or calf muscles after activity, such as walking or climbing stairs (intermittent claudication)
- Leg numbness or weakness
- Coldness in your lower leg or foot, especially when compared with the other leg
- Sores on your toes, feet or legs that won't heal
- A change in the color of your legs
- Hair loss or slower hair growth on your feet and legs
- Slower growth of your toenails
- Shiny skin on your legs
- No pulse or a weak pulse in your legs or feet
- Erectile dysfunction in men

Peripheral Vein Disease (PVD)

Pathophysiology

PVD, also known as arteriosclerosis obliterans, is primarily the result of atherosclerosis. The atheroma consists of a core of cholesterol joined to proteins with a fibrous intravascular covering. The atherosclerotic process may gradually progress to complete occlusion of medium and large arteries. The disease typically is segmental, with significant variation from patient to patient.

Vascular disease may manifest acutely when thrombi, emboli, or acute trauma compromises perfusion. Thromboses are often of an atheromatous nature and occur in the lower extremities more frequently than in the upper extremities. Multiple factors predispose patients for thrombosis. These factors include sepsis, hypotension, low cardiac output, aneurysms, aortic dissection, bypass grafts, and underlying atherosclerotic narrowing of the arterial lumen.

Emboli, the most common cause of sudden ischemia, usually are of cardiac origin (80%); they also can originate from proximal atheroma, tumor, or foreign objects. Emboli tend to lodge at artery bifurcations or in areas where vessels abruptly narrow. The femoral artery bifurcation is the most

common site (43%), followed by the iliac arteries (18%), the aorta (15%), and the popliteal arteries (15%).

The site of occlusion, presence of collateral circulation, and nature of the occlusion (thrombus or embolus) determine the severity of the acute manifestation. Emboli tend to carry higher morbidity because the extremity has not had time to develop collateral circulation. Whether caused by embolus or thrombus, occlusion results in both proximal and distal thrombus formation due to flow stagnation.

Signs & Symptoms

The most common symptom of peripheral vascular disease in the legs is pain in one or both calves, thighs, or hips.

- The pain usually occurs while you are walking or climbing stairs and stops when you rest. This is because the muscles' demand for blood increases during walking and other exercise. The narrowed or blocked arteries cannot supply more blood, so the muscles are deprived of oxygen and other nutrients.
- This pain is called intermittent (comes and goes) claudication.
- It is usually a dull, cramping pain. It may also feel like a heaviness, tightness, or tiredness in the muscles of the legs.
- Cramps in the legs have several causes, but cramps that start with exercise and stop with rest most likely are due to intermittent. When the blood vessels in the legs are completely blocked, leg at night is very typical, and the individual almost always hangs his or her feet down to ease the pain. Hanging the legs down allows for blood to passively flow into the distal part of the legs.

Other symptoms of peripheral vascular disease include the following:

- Buttock pain
- Numbness, tingling, or weakness in the legs
- Burning or aching pain in the feet or toes while resting
- A sore on a leg or a foot that will not heal
- One or both legs or feet feel cold or change color (pale, bluish, dark reddish)
- Loss of hair on the legs
- Impotence

Aneurysms

Pathophysiology

Studies were performed to evaluate the contributions of elastin and collagen to the formation of arterial aneurysms. Dog carotid arteries and human external and internal iliac arteries were

excised, mounted horizontally in a tissue bath, and were pressurized. Vessel diameter and longitudinal force were measured. The vessels were treated with elastase or collagenase. Those treated with elastase dilated, but never ruptured. Those treated with collagenase dilated still more and, in every case, ruptured. Circumferential stability resulted from recruitment of previously non-loaded collagen fibers, and from a change in geometry from a cylinder to a sphere. The laminated thrombus lining the lumen has little intrinsic strength and therefore does not confer strength to the aneurysmal wall. Treatment with elastase also reduces the retractive force exerted by the vessel in the longitudinal direction. Therefore loss of elastin permits the vessel to elongate and to become tortuous. In aged human arteries collagen also contributes a small portion of the retractive force. Progressive enlargement of aneurysms results from continued failure of wall connective tissues reflecting a) genetically defective collagen and or b) activity of the immune system.

Signs & Symptoms

Signs and symptoms of an aneurysm depend on the type and location. The signs and symptoms also depend on whether the aneurysm has ruptured or is interfering with other muscles, organs and structures in the body. The signs and symptoms are not known until an aneurysm ruptures or grows sufficiently to press against nearby organs or tissues or may block the flow of blood.

I. Aortic Aneurysms:

1) Thoracic Aortic Aneurysm: Symptoms of thoracic aortic aneurysm are as follows:

- Pain in jaw, neck, upper back or chest.
- Cough, hoarseness or experiencing trouble in breathing.
- Pain in left shoulder or between shoulder blades.

2) Abdominal Aortic Aneurysms (AAAs): Symptoms of AAAs include:

- Deep penetrating pain the back or side of abdomen.
- Steady gnawing pain in the abdomen lasting for hours or days.
- Coldness, numbness or tingling of feet.
- In case of a rupture of the AAA, symptoms include sudden severe pain in lower abdomen and back; nausea and vomiting; sweaty skin, light headedness and rapid heart rate when standing up.

II. Cerebral Aneurysm: Signs and symptoms of cerebral aneurysm are:

- Drooping of eyelids.
- Double vision or blurred vision.
- Pain above or behind the eye.
- A dilated pupil.
- Numbness or weakness on one side of the face.
- A cerebral aneurysm rupture leads to sudden severe headache, nausea and vomiting, stiff neck and loss of consciousness.

III. Peripheral Aneurysm: Signs and symptoms of peripheral aneurysm are as follows:

- Pulsating lump felt in the neck, arm or leg
- Pain in the leg or arm or cramping with exercise
- Painful sores on toes or fingers

- Gangrene (i.e., death of tissue) due to severe blockage of blood in the limbs

An aneurysm in the popliteal artery can compress the nerves and cause pain, weakness and numbness in knee and leg (1) & (4).

Respiratory

Bronchial Asthma

Pathophysiology

Bronchial asthma is a chronic inflammatory disease of the airways, associated with recurrent, reversible airway obstruction with intermittent episodes of wheezing and dyspnea. Bronchial hypersensitivity is caused by various stimuli, which innervate the vagus nerve and beta adrenergic receptor cells of the airways, leading to bronchial smooth muscle constriction, hypersecretion of mucus, and mucosal edema.

Signs & Symptoms

- a feeling of tightness in the chest;
- difficulty in breathing or shortness of breath;
- wheezing; and
- coughing (particularly at night).

Nursing Assessment	Nursing Intervention	Rationale	Goal
Ineffective airway clearance related to increased production of secretions.	Independent: · Auscultate breath sounds. Note adventitious breath sounds like wheezes, crackles and rhonchi. · Elevate head of the bed, have patient lean on overbed table or sit on edge of the bed. · Keep environmental pollution to a minimum like dust, smoke and feather pillows, according to individual situation. · Encourage or assist with abdominal or pursed lip breathing exercises. · Assist with measures to improve effectiveness of cough effort.	· Some degree of bronchospasm is present with obstructions in airway and may or may not be manifested in adventitious breath sounds. · Elevation of the bed facilitates respiratory function by use of gravity. · Precipitators of allergic type of respiratory reactions that can trigger or exacerbate onset of acute episode. · Provides patient with some means to cope with or control dyspnea and reduce air tapping. · Coughing is most	the patient will be able to demonstrate behaviors to improve airway clearance.

	<ul style="list-style-type: none"> · Increased fluid intake to 3000 ml/ day. Provide warm or tepid liquids. <p>Collaborative:</p> <ul style="list-style-type: none"> · Administer bronchodilators as prescribed. 	<p>effective in an upright position after chest percussion.</p> <ul style="list-style-type: none"> · Hydration helps decrease the viscosity of secretions, facilitating expectoration. Using warm liquids may decrease bronchospasm. · To reduce the viscosity of secretions. 	
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Bronchitis

Pathophysiology

Bronchitis is an inflammation of the air passages within the lungs. It occurs when the trachea (windpipe) and the large and small bronchi (airways) within the lungs become inflamed because of infection or other causes.

Signs & Symptoms

Nursing Assessment	Nursing Intervention	Rationale	Goal
Ineffective airway clearance related to excessive, thickened mucous secretions.	<p>Independent:</p> <ul style="list-style-type: none"> □ □ Assess respiratory rate, depth. Note use of accessory muscles, pursed lip breathing, inability to speak. □ □ Elevate head of the bed, assist patient assume position to ease work of breathing. Encourage deep slow or pursed lip breathing as individually tolerated or indicated. □ □ Routinely monitor skin and mucous membrane color. · Encourage expectoration of sputum; suction when indicated. · Evaluate level of activity tolerance. Provide calm and quiet environment. · Evaluate sleep 	<ul style="list-style-type: none"> · Useful in evaluating the degree or respiratory distress and chronicity of the disease process. · Oxygen delivery may be improved by upright position and breathing exercises to decrease airway collapse, dyspnea and work of breathing. · Cyanosis may be peripheral in nail beds or central in lips or earlobes. Dusky and central cyanosis indicate advanced hypoxemia. · Thick, tenacious, copious secretions are major source of ineffective airways. · Deep suctioning 	Improved ventilation and adequate oxygenation of tissues and Arterial blood gases (ABGs) within normal range and free from symptoms of respiratory distress.

	<p>patterns, note report of difficulties and whether patient feels well rested.</p> <ul style="list-style-type: none"> · Monitor vital signs and cardiac rhythm. <p>Collaborative:</p> <ul style="list-style-type: none"> · Administer supplemental oxygen as indicated by ABG results and patients tolerance. 	<p>may be required when cough is ineffective for expectoration of secretions.</p> <ul style="list-style-type: none"> · During severe or acute respiratory distress, patient may be totally unable to perform basic self care activities because of hypoxemia and dyspnea. · Multiple external stimuli and presence of dyspnea may prevent relaxation and inhibit sleep. · Tachycardia, dysrhythmias, and changes in blood pressure can reflect effect of systemic hypoxemia on cardiac function. · May correct or prevent worsening of hypoxia 	
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Chronic Obstructive Pulmonary Disease (COPD)

Pathophysiology

COPD disrupts airway dynamics, resulting in obstruction of **airflow** into or out of the lungs.

Chronic Bronchitis.

Hypertrophy and hypersecretion in goblet cells and bronchial mucus glands leading to increased sputum secretions, bronchial congestion, narrowing of bronchioles, and small bronchi.

Emphysema

Increased size of air spaces (i.e. "dead space") with loss of elastic recoil of lung due to hyperinflation of distal airways causing airway obstruction. Destruction of alveolar walls and diffuse airway narrowing causes resistance to airflow because of loss of supporting structure and bronchospasm further impede airflow.

Signs & Symptoms

Chronic Bronchitis

- History of productive cough that lasts 3 months per year for 2 consecutive years
- Persistent cough, known as smoker's cough usually in cold weather

- Persistent sputum production
- Recurrent acute respiratory infection
- Dusky color leading to cyanosis
- Clubbing of fingers

Emphysema

- History of chronic bronchitis
- Slow onset of symptoms (typically over several years) which can lead to right-side heart failure (i.e. cor pulmonale)
- Progressive dyspnea, initially only on exertion and later also at rest
- Progressive cough and increased sputum production, especially bouts of infection, use of accessory muscles
- Anorexia with weight loss and profound weakness
- Dyspnea with insidious onset progressing to severe dyspnea with slight exertion (major symptom)
- Chronic cough, wheezing, dyspnea, fatigue, and tachypnea
- On inspection, "barrel chest" due to air trapping, muscle wasting, and pursed-lip breathing
- On auscultation, diminished breath sounds with crackles, wheezes, rhonchi, and prolonged expiration.
- Hyperresonance with percussion and a decrease in fremitus
- Anorexia, weight loss, weakness, and inactivity
- Hypoxemia and hypercapnia, morning headaches in advanced stages
- Inflammatory reactions and infections from pooled secretions

Nursing Assessment	Nursing Intervention	Rationale	Goal
Ineffective airway Clearance related to Increased production of secretions.	Independent: · Assist patient to assume position of comfort, e.g., elevate head of bed, encourage patient to lean on overbed table or sit on the edge of the bed. · Keep environmental pollution to a minimum, e.g., dust, smoke and feather pillows, according to individual situation · Encourage or assist with pursed lip breathing exercises. · Observe characteristics of cough like persistent or hacking or moist. Assist with measures to improve effectiveness of cough effort.	· Elevation of the head of the bed facilitates respiratory function by use of gravity. · Precipitators of allergic type or respiratory reactions that can trigger or exacerbate onset of acute episode. · Provides patient with some means to cope or control dyspnea and reduce air trapping. · Coughing is most effective in an upright position or head down position after chest percussion. · A variety of medications may be used to decrease mucus and to improve respiration. · Humidity helps	able to demonstrate behaviors to improve airway clearance. e.g. cough effectively and expectorate secretions.

	Dependent: · Administer medication as prescribed by the physician. · Provide supplemental humidification like nebulizer.	reduce viscosity of secretions, facilitating expectoration, and may reduce or prevent formation of thick mucus plugs in bronchioles.	
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Emphysema

Pathophysiology

The pathophysiology of emphysema is best explained on the basis of decreased pulmonary elastic recoil. At any pleural pressure, the lung volume is higher than normal. Additionally, the altered relation between pleural and alveolar pressure facilitates expiratory dynamic compression of airways. Such compression limits airflow during forced expiration and, in severe instances, during tidal expiration. Another factor contributing to airflow limitation is disease of the airways, both large and small. In general, patients with relatively pure emphysema maintain blood gases in or near the normal range until very late in their course. PaO₂ is maintained because of the preserved matching of ventilation and perfusion as alveolar walls are destroyed. PaCO₂ is maintained because the ventilatory response to CO₂ is not usually impaired. It is not clear why patients who are categorized clinically as "chronic bronchitics" are more likely to respond to an increased flow-resistive work of breathing by hypoventilating. Physical findings in emphysema are not specific. Radiologic changes are insensitive and are of less value than physiologic measurements.

Signs & Symptoms

- **Shortness of Breath**
- **Rapid Breathing**
- **Chronic Cough (With or Without Sputum)**
- **Wheezing**
- **Reduced Exercise Tolerance**
- **Loss of Appetite Leading to Weight Loss**
- **Barrel Chest**

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Patients can maintain adequate gas exchange 	<ul style="list-style-type: none"> • Assess for signs and symptoms of hypoxia and hypercapnia • Monitor and record blood gas examination, examine the trend in the 	<ul style="list-style-type: none"> • Respiratory distress and changes in vital signs may occur as a results of physiological stress and pain or may indicate 	<ul style="list-style-type: none"> • Beep net pulmonary • The color of normal skin • Blood gases within normal limits for the estimated

	<p>increase or decrease in PaO₂ PaCO₂</p> <ul style="list-style-type: none"> • Help with the provision of mechanical ventilation according to indications, assess the need for CPAP or PEEP. • Auscultation chest to listen to breath sounds every hour • Review the daily chest X-ray examination, or deviations noticed improvement • Monitor cardiac rhythm • Provide appropriate parenteral fluid orders • Provide customized medicines: bronchodilators, antibiotics, steroids. • Evaluation of AKS in conjunction with a decrease in oxygen demand. 	<p>development of shock due to hypoxia.</p> <ul style="list-style-type: none"> • To facilitate maximal lung expansion/improve ventilation and reduce venous return to the right side of the heart. • Breath sounds may be diminished or absent in a lobe lung segment or entire lung field. Atelectatic area will have no breath sound, and partially collapsed areas have decreased sounds. Regularly scheduled evaluation also helps determine areas of good air exchange and provides a baseline to evaluate resolution of pneumothorax 	age
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Hemothorax

Pathophysiology

A hemothorax is managed by removing the source of bleeding and by draining the blood already in the thoracic cavity. Blood in the cavity can be removed by inserting a drain (chest tube) in a procedure called a tube thoracostomy. Usually the lung will expand and the bleeding will stop after a chest tube is inserted. The blood in the chest can thicken as the clotting cascade is

activated when the blood leaves the blood vessels and is activated by the pleural surface, injured lung or chest wall, or contact with the chest tube. As the blood thickens, it can clot in the pleural space (leading to a retained hemothorax) or within the chest tube, leading to chest tube clogging or occlusion. Chest tube clogging or occlusion can lead to worse outcomes as it prevents adequate drainage of the pleural space, contributing to the problem of retained hemothorax. In this case, patients can be hypoxic, short of breath, or in some cases, the retained hemothorax can become infected (empyema). Therefore adequately functioning chest tubes are essential in the setting of a hemothorax treated with a chest tube. To attempt to minimize the potential for clogging, the surgeons will often place more than one tube, or large diameter tubes. Maintaining an adequately functioning chest tube is an active process, usually for the nurses, that often requires tapping the tubes, milking the tubes, or stripping the tubes to minimize potential for clogging in the tube in the setting of a hemothorax. When these efforts fail a new chest tube must be placed, or the patient must be taken to the operating room by a surgeon to open the chest and remove the blood clot, and re insert adequately functioning chest tubes.

Thrombolytic agents have been used to break up clot in tubes or when the clot becomes organized in the pleural space, however this is risky as it can lead to increased bleeding and the need for reoperation. Therefore, ideally, the tubes maintain their function so that the blood cannot clot in the chest or the tube.

In some cases bleeding continues and surgery is necessary to stop the source of bleeding. For example, if the cause is rupture of the aorta in high energy trauma, the intervention by a thoracic surgeon is mandatory.

Signs & Symptoms

- Tachypnea
- Dyspnea
- Cyanosis
- Decreased or absent breath sounds on affected side
- Tracheal deviation to unaffected side
- Dull resonance on percussion
- Unequal chest rise
- Tachycardia
- Hypotension
- Pale, cool, clammy skin
- Possibly subcutaneous emphysema
- Narrowing pulse pressure

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Ineffective breathing pattern related to decreased lung expansion 	Independent: <ul style="list-style-type: none"> • Identify etiology or precipitating factors. • Monitor vital signs. • Assess lung sounds, respiratory rate and effort and the 	<ul style="list-style-type: none"> • Understanding the cause is necessary for choice of therapeutic measures. • Monitoring the vital signs is necessary to evaluate the degree of 	<ul style="list-style-type: none"> • Establish a normal and effective breathing pattern within client's normal range

	<p>use of accessory muscles.</p> <ul style="list-style-type: none"> Evaluate respiratory function, noting rapid or shallow respirations, dyspnea, reports of "air hunger," and changes in vital signs. Observe skin and mucous membranes for signs of cyanosis. Encourage adequate rest and limit activities within client's level of tolerance. Promote a calm and restful environment. <p>Dependent:</p> <ul style="list-style-type: none"> Administer supplemental oxygen as ordered by the physician. Administer medications as prescribed by the physician 	<p>compromise.</p> <ul style="list-style-type: none"> Respiratory rate less than 12 or more than 24 or use of accessory muscles indicate distress. Diminished lung sounds indicate possible poor air movement and impaired gas exchange. Respiratory distress and changes in vital signs occur as a result of physiologic stress and pain, or may indicate development of shock due to hypoxia or hemorrhage Cyanosis indicates poor oxygenation. Oral mucous membrane cyanosis indicates serious hypoxia. Helps limit oxygen needs and consumption. Supplemental oxygen decreases hypoxia. To treat underlying conditions 	
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Pneumonia

Pathophysiology

Pneumonia is an acute inflammatory disorder of lung parenchyma that results in edema of lung tissues and movement of fluid into the alveoli. These impair gas exchange resulting in hypoxemia. Pneumonia can be classified in several ways. Based on microbiologic etiology, it may be viral, bacterial, fungal, protozoal, myobacterial, mycoplasmal, or rickettsial in origin. Based on location,

pneumonia may be classified as bronchopneumonia, lobular pneumonia, or lobar pneumonia. Bronchopneumonia involves distal airways and alveoli; lobular pneumonia, part of the lobe; and lobar pneumonia, the whole lobe.

Pneumonia occurs in both sexes and at all ages, but older adults run a greater risk of developing it because their weakened chest musculature reduces their ability to clear secretions. Bacterial pneumonia is the most common type of pneumonia found in older adults; viral pneumonia is the second most common type. Aspiration pneumonia occurs in older adults due to impaired swallowing ability and diminished gag reflex. These changes can occur after a stroke or any prolonged illness.

Signs & Symptoms

- Sudden chills, rapidly rising fever (38.5°C to 40.5°C), and profuse perspiration.
- Pleuritic chest pain aggravated by respiration and coughing
- Severely ill patient has marked tachypnea (25 to 45 breaths/min) and dyspnea; orthopnea when not propped up.
- Pulse rapid and bounding, may increase beats/min per degree of temperature elevation
- Dullness with consolidation on percussion of chest
- Bronchial breath sounds auscultated over consolidated lung fields
- Shaking chills (with bacterial pneumonia)
- Dyspnea, respiratory grunting, and nasal flaring
- Severe pneumonia: flushed cheeks, cyanotic lips and nail beds
- Sputum purulent, rusty, blood-tinged, viscous, or green depending on etiologic agent.
- Anxiety and confusion
- In elderly clients, the only signs may be mental status change and dehydration.
- Chest radiograph shows density changes, primarily in the lower lung fields.
- Sputum culture and sensitivity are positive for a specific causative organism.
- White blood cell (WBC) count is elevated in pneumonia of bacterial origin, WBC count is depressed in pneumonia of mycoplasmal or viral origin.

Nursing Assessment	Nursing Intervention	Rationale	Goal
Impaired Gas Exchange related to: <ul style="list-style-type: none"> • oxygen-carrying blood disorders, • impaired oxygen delivery characterized by: <ul style="list-style-type: none"> • Dyspnea, cyanosis • Tachycardia • Nervous / mental changes • Hypoxia 	<ul style="list-style-type: none"> • Assess the frequency / depth and ease of breathing • Observe the color of skin, mucous membranes and nails. Note the presence of peripheral cyanosis (nail) or central cyanosis. • Assess mental status. • Elevate the head and thrust frequently change position, breathe deeply and cough 	<ul style="list-style-type: none"> • the manifestation of respiratory distress depends on the indication of the degree of lung involvement and general health status. • nails showed cyanosis vasoconstriction 	<ul style="list-style-type: none"> • Establish a normal and effective breathing pattern within client's normal range

	<p>effectively.</p> <ul style="list-style-type: none"> • Collaboration 	<p>body's response to fever / chills, but cyanosis on the ears, mucous membranes and skin around the mouth indicate systemic hypoxemia.</p> <ul style="list-style-type: none"> • nervous irritability, confusion and somnolence may indicate cerebral hypoxia or decreased oxygen. • This action increases the maximum inspiration, increased spending secretions to improve ventilation ineffective. • to maintain PaO₂ above 60 mmHg. Oxygenation provided with a method that provides precise delivery. 	
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Pneumothorax

Pathophysiology

Pneumothorax refers to gas within the pleural space. Normally, the alveolar pressure is greater than the intrapleural pressure, while the intrapleural pressure is less than atmospheric pressure. Therefore, if a communication develops between an alveolus and the pleural space or between the atmosphere and the pleural space, gases will follow the pressure gradient and flow into the pleural space. This flow will continue until the pressure gradient no longer exists or the abnormal communication has been sealed. Since the thoracic cavity is normally below its resting volume, and the lung is above its resting volume, the thoracic cavity enlarges and the lung becomes smaller when a pneumothorax develops.

A tension pneumothorax is a medical emergency and occurs when the intrapleural pressure exceeds atmospheric pressure, especially during expiration, and results from a ball valve mechanism that promotes inspiratory accumulation of pleural gases. The build-up of pressure within the pleural space eventually results in hypoxaemia and respiratory failure from compression of the lung.

The pathophysiology of catamenial pneumothoraces is not known. It has been suggested that air gains access to the peritoneal cavity during menstruation and then secondarily the pleural space through diaphragmatic defects. Alternatively, it has been hypothesized that ectopic intrathoracic endometriosis results in visceral pleural erosions, thus causing a pneumothorax.

Signs & Symptoms

Signs and symptoms of a pneumothorax usually include:

- **Chest pain.** Sudden, sharp chest pain on the same side as the affected lung — this pain doesn't occur in the center of your chest under the breast bone. And it doesn't worsen when you breathe in and out.
- **Shortness of breath.** This may be mild or severe, depending on how much of your lung is collapsed and whether you have underlying lung disease.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Breathing pattern ineffective may related to • Decreased lung expansion • Musculoskeletal impairment • pain/anxiety • inflammatory process 	<ul style="list-style-type: none"> • Identify etiology precipitating factors • Evaluate respiratory functions, noting rapid/shallow respirations, dyspnea 	<ul style="list-style-type: none"> • Understanding the cause of lung collapse is necessary for proper chest placement and choice other therapeutic measures. • Respiratory distress and changes in vital signs may occur as a results of physiological 	Patient maintains optimal gas exchange as evidenced by:-a. <ul style="list-style-type: none"> • Normal arterial blood gases (ABGs) • Pulse oximetry results within normal range. • Usual mental status • Normal respiration rate

	<ul style="list-style-type: none"> • Monitor for synchronous respiratory pattern when using mechanical ventilator • Auscultate breath sounds • Assess fremitus 	<p>stress and pain or may indicate development of shock due to hypoxia.</p> <ul style="list-style-type: none"> • Difficulty breathing "with" ventilator or increasing airway pressure suggests worsening of condition/development of complications • Breath sounds may be diminished or absent in a lobe lung segment or entire lung field. Atelectatic area will have no breath sound, and partially collapsed areas have decreased sounds. Regularly scheduled evaluation also helps determine areas of good air exchange and provides a baseline to evaluate resolution of pneumothorax • Voice and tactile fremitus is reduced in fluid filled/consolidated tissue 	
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Pulmonary Embolism

Pathophysiology

A thrombus that has separated from its site of origin travels through the circulation to the inferior vena cava. The right ventricle pumps this thrombus to the pulmonary arteries where the thrombus finally lodges. PE may occur singly or multiply. They can be microscopic in size or be big enough to occlude the major branches of the pulmonary artery.

The embolus obstructs flow in the pulmonary arteries and thus causes an increase in resistance to blood flow in the pulmonary vessels. Severe pulmonary hypertension, RV strain, and cardiac heart failure occur when more than 50-60% decrease in perfusion. In addition, intrapulmonary reflexes stimulate the **release** of humoral substances that lead to vasoconstriction throughout the lungs and thus increases pulmonary vascular resistance.

10% of PE will progress to pulmonary infarction. The lung depends on 3 sources of oxygen (airways, bronchial circulation, pulmonary circulation) and therefore the chance that all 3 sources will be compromised simultaneously are not great.

Recurrent PE may gradually obstruct the pulmonary vasculature and ultimately lead to chronic obstructive pulmonary hypertension and cor pulmonale.

The most important pathophysiological consequence of PE is V/Q mismatch in which there is "dead space" ventilation in some parts of the lung and overperfusion in others. "Dead space" ventilation refers to ventilation of lung segments that have obstructed vascular supply and thus no perfusion. On the other hand, overperfusion and decreased vascular resistance in other parts of the lung leads to right-to-left intrapulmonary shunting with insufficient oxygenation of a large portion of perfused blood.

Signs & Symptoms

Symptoms of pulmonary embolism may be vague, or they may resemble symptoms associated with other diseases. Symptoms can include:

- Cough
 - Begins suddenly
 - May produce bloody sputum (significant amounts of visible blood or lightly blood streaked sputum)
- Sudden onset of shortness of breath at rest or with exertion
- Splinting of ribs with breathing (bending over or holding the chest)
- Chest pain
 - Under the breastbone or on one side
 - Especially sharp or stabbing; also may be burning, aching or dull, heavy sensation
 - May be worsened by breathing deeply, coughing, eating, bending, or stooping
- Rapid breathing
- Rapid heart rate (tachycardia)

Additional symptoms that may be associated with this disease:

- Wheezing
- Clammy skin
- Bluish skin discoloration
- Nasal flaring
- Pelvis pain
- Leg pain in one or both legs
- Swelling in the legs (lower extremities)
- Lump associated with a vein near the surface of the body (superficial vein), may be painful
- Low blood pressure
- Weak or absent pulse

- Lightheadedness or fainting
- Dizziness
- Sweating
- Anxiety

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Impaired gas exchange related to decrease pulmonary perfusion associated with obstruction of pulmonary arterial blood flow by the embolus. 	<ul style="list-style-type: none"> • Frequently assess respiratory status including rate, depth, effort, lung sound and SPO2 • Assess the mental status of the client • Monitor ABGs and note changes • Position the patient in high fowler's position • Administered oxygen as ordered by doctor • maintain bed rest • Administer medication as prescribed by doctor. 	<ul style="list-style-type: none"> • Impaired ventilation affects gas exchange and worsens hypoxemia (Tachypnea, dyspnea). SPO2 can be used as a non-invasive method to monitor oxygen saturation. • Restlessness is an early sign of hypoxia. Hypoxemia often causes confusion and agitation. • ABGs used to assess gas exchange of client • To facilitate maximal lung expansion/improve ventilation and reduce venous return to the right side of the heart. • To improve oxygenation. • Bed rest reduces metabolic demands for oxygen • Anticoagulant therapy is 	<p>Patient maintains optimal gas exchange as evidenced by:-a.</p> <ul style="list-style-type: none"> • Normal arterial blood gases (ABGs) • Pulse oximetry results within normal range. • Usual mental status • Normal respiration rate

		preventive by inhibiting further dot formation.	
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Respiratory Failure

Pathophysiology

Respiratory failure can arise from an abnormality in any of the components of the respiratory system, including the airways, alveoli, central nervous system (CNS), peripheral nervous system, respiratory muscles, and chest wall. Patients who have hypoperfusion secondary to cardiogenic, hypovolemic, or septic shock often present with respiratory failure.

Ventilatory capacity is the maximal spontaneous ventilation that can be maintained without development of respiratory muscle fatigue. Ventilatory demand is the spontaneous minute ventilation that results in a stable $P_a \text{CO}_2$.

Normally, ventilatory capacity greatly exceeds ventilatory demand. Respiratory failure may result from either a reduction in ventilatory capacity or an increase in ventilatory demand (or both). Ventilatory capacity can be decreased by a disease process involving any of the functional components of the respiratory system and its controller. Ventilatory demand is augmented by an increase in minute ventilation and/or an increase in the work of breathing.

Signs & Symptoms

Respiratory failure is accompanied by a number of symptoms including:

- Bluish coloration of the lips or fingernails
- Confusion or loss of consciousness
- [Fainting](#) or change in level of consciousness or lethargy
- [Fatigue](#)
- Irregular heart rate (arrhythmia)
- Rapid breathing (tachypnea) or [shortness of breath](#)

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Irregular Heart rate • Rapid Breathing • Fatigue 	<ul style="list-style-type: none"> • Monitor respiratory status, including vital signs, breath sounds, and skin color. • Place the patient in semi-fowlers position and place the diaphragm in proper position to contract. • assist in self-care 	<ul style="list-style-type: none"> • Respiratory status assessment helps gauge the patient's severity and whether it's progressing. • To increase chest expansion and to alleviate dyspnea. • To distract attention from pain and decrease tension 	<ul style="list-style-type: none"> • Goal met. Patient was able to relax by utilizing bed rest and deep breathing.

	activities as tolerated <ul style="list-style-type: none"> • provide peaceful \and adequate resting environment (dim lights, adjust temperature, wrinkle-free bed, quiet surroundings) 	<ul style="list-style-type: none"> • To conserve energy of the patient and prevent fatigue • To promote client independence as much as possible and acquire sense of function9.to enhance quality sleep and promote rest which harnesses energy for future use. 	
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Tuberculosis (TB)

Pathophysiology

The risk of TB is a higher in older people who have close contact with a newly diagnosed TB patient, those who have TB before, gastrectomy patients, and those affected with diabetes mellitus. The aging process weakens the immune system, further increasing the likelihood of tubercular infection in older adults.

Transmission occurs when droplet nuclei are produced form an infected person's coughs or sneezes. If inhaled, tubercle bacillus settles in the alveolus and infection occurs, with alveolocapillary dilation and endothelial swelling. The incubation time for TB is 4 to 8 weeks. TB is usually asymptomatic in primary infection

Signs & Symptoms

Nursing Assessment	Nursing Intervention	Rationale	Goal
Ineffective breathing pattern related to acute infection and decreased lung capacity	<ul style="list-style-type: none"> • Monitor respiratory status, including vital signs, breath sounds, and skin color. • Administer oxygen therapy as ordered. • Monitor ABG levels and oxygen saturation as ordered. 	<ul style="list-style-type: none"> • Respiratory status assessment helps gauge the patient's severity and whether it's progressing. • To provide relief from symptoms of hypoxemia and hypoxia. • ABG levels and continuous pulse oximetry measures the blood's oxygen content and are good indicators of the lung's ability 	<ul style="list-style-type: none"> • Breathing returned to normal rate and pattern • Minimal or no signs of infection.

	<ul style="list-style-type: none"> Place the patient in semi-fowlers position and place the diaphragm in proper position to contract. Collect sputum samples as ordered. 	<ul style="list-style-type: none"> to oxygenate the blood. To increase chest expansion and to alleviate dyspnea. To monitor the progress of the disease and treatment. 	
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Upper Respiratory Infection (URI)

Pathophysiology

Signs & Symptoms

- Itchy, watery eyes
- nasal discharge
- nasal congestion
- sneezing
- sore throat
- cough
- head ache
- fever
- malaise
- fatigue, weakness
- muscle pain

Nursing Assessment	Nursing Intervention	Rationale	Goal
ineffective Airway Clearance related to thick tenacious secretions and airway obstructions manifested by shallow respiration, tachypnea and fever	<ol style="list-style-type: none"> 1. Monitor VS every 2 hrs. 2. Encourage patient to position in high-Fowler's or semi-Fowler's position. 3. Turn patient every 2 hrs and prn. 4. Teach client to maintain adequate hydration by drinking at least 8-10 glasses of fluid/day (if not contraindicated). 5. Teach and supervise effective coughing techniques. 6. Perform Chest Physical therapy 	<ol style="list-style-type: none"> 1. To assess baseline data. 2.promotes maximal lung function. 3.repositioning promotes drainage of pulmonary secretions and enhances ventilation to decrease potential of atelectasis. 4.to help thin secretions. 5.to conserve energy and to reduce airway collapse. 6.CPT techniques utilizes forces of gravity and motion to 	had been able to cough effectively and clear own secretions. maintained patency of airway and had clear breath sounds

	<p>7. Instruct on splinting abdomen with pillow during coughing efforts.</p> <p>8. Monitor airway for patency and provide artificial airways as warranted.</p> <p>9. Administer bronchodilators as ordered.</p> <p>10. Instruct client/family to notify nurse if the client is experiencing shortness of breath or air hunger.</p> <p>11. Instruct client/family regarding medications, effects, side effects and symptoms of adverse effects to report to nurse or physician.</p>	<p>facilitate secretion removal</p> <p>7. Promotes increased expiratory pressure.</p> <p>8.requires if patient cannot maintain airway patency.</p> <p>9. To improve ventilation and maximizes air exchange.</p> <p>10. May indicate bronchial tubes are blocked with mucus, leading to hypoxia and hypoxemia.</p> <p>11. Promotes prompt identification of potential adverse reaction to facilitate timely intervention.</p>	
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CARDIAC (HEART)

Angina

Pathophysiology

Angina is a temporary chest pain that results from inadequate oxygen flow to the myocardium. It's usually described as burning, squeezing, or a tight feeling in the substernal or precordial chest. This pain may radiate to the left arm, neck, jaw, or shoulder blade. Typically, the patient clenches his fist over his chest or rubs his left arm when describing the pain, which may also be accompanied by nausea, vomiting, fainting, sweating, and cool extremities.

STABLE ANGINA: discomfort that often occurs with activity or stress. Angina is a type of chest discomfort caused by poor blood flow through the blood vessels (coronary vessels) of the heart muscle (myocardium) Your heart muscle is working all the time, so it needs a constant supply of oxygen. This oxygen is provided by the coronary arteries, which carry blood.

When the heart muscle has to work harder, it needs more oxygen. Symptoms of angina occur when the coronary arteries are narrowed or blocked by hardening of the arteries atherosclerosis or by a blood clot.

UNSTABLE ANGINA: condition in which your heart doesn't get enough blood flow and oxygen. It may lead to a heart attack.

Angina is a type of chest discomfort caused by poor blood flow through the blood vessels (coronary vessels) of the heart muscle (myocardium).

Coronary artery disease due to atherosclerosis is by far the most common cause of unstable angina. Atherosclerosis is the buildup of fatty material called plaque along the walls of the arteries. This causes arteries to become narrowed and less flexible. The narrowing interrupts blood flow to the heart, causing chest pain.

People with unstable angina are at increased risk of having a heart attack.

When assessing for anginal pain, older adults commonly have an increased tolerance for pain, and may be less likely to complain. Instead, they may compensate by slowing their activity levels. Older adults may not experience chest pain at all, but may report dyspnea, faintness, or extreme fatigue.

The person's health history may suggest a pattern to the type and onset of pain. If the pain is predictable and relieved by rest or nitrates, it's called **stable angina**. If it increases in frequency and duration and is more easily induced, it's referred to as unstable angina or unpredictable angina. Unstable angina may occur at rest and generally indicates extensive or worsening disease that may progress to an MI. Variant or Prinzmetal's angina is caused by coronary artery spasm, and commonly occurs at rest without initial increased oxygen demand.

Signs & Symptoms

- Chest pain, heavy sensation (retrosternal area)
- Tightness, heavy, choking or strangling sensation
- Weakness
- Numbness in the arms, wrists, and hands
- Shortness of breath
- Pallor, diaphoresis, dizziness or lightheadedness
- Nausea and vomiting
- Anxiety

Nursing Assessment	Nursing Intervention	Rationale	Goal
Acute pain related to decreased myocardial blood flow	Assess for vital signs and symptoms of pain such as facial grimacing, rubbing of neck or jaw, reluctance to move, increased blood pressure, and tachycardia.	To differentiate angina pain from pain related to other causes. To monitor the effectiveness of medications given for pain relief.	the patient will be free from pain, maintains stable vital signs, and relaxed body posture.

	<p>Note onset, duration, location, and pattern of pain.</p> <p>12-lead EKG immediately during acute chest pain.</p> <p>Use a pain rating scale to assess the patient's perception of the pain's severity.</p> <p>Administer sublingual nitroglycerin as ordered. (NOTE CONTRAINDICATED FOR PT ON VASODIALATORS LIKE VIAGRA)</p> <p>Instruct the patient to notify a nurse immediately when experiencing pain. Have the patient stop current activity, and place him on bed rest in a semi- to high Fowler's position.</p> <p>Administer oxygen as ordered.</p>	<p>To decrease myocardial oxygen demands through vasodilatation, preload and after load reduction and decreased cardiac work load.</p> <p>To minimize ischemia produced by increased myocardial work load.</p> <p>To provide optimal oxygenation to the myocardium.</p> <p>To document ischemic changes. To decrease anxiety and promote comfort.</p>	
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Arrhythmias

Pathophysiology

Regardless of the specific arrhythmia, the pathogenesis of the arrhythmias falls into one of three basic mechanisms: enhanced or suppressed automaticity, triggered activity, or re-entry. Automaticity is a natural property of all myocytes. Ischemia, scarring, electrolyte disturbances, medications, advancing age, and other factors may suppress or enhance automaticity in various areas. Suppression of automaticity of the sinoatrial (SA) node can result in sinus node dysfunction and in sick sinus syndrome (SSS), which is still the most common indication for permanent pacemaker implantation. In contrast to suppressed automaticity, enhanced automaticity can result in multiple arrhythmias, both atrial and ventricular. Triggered activity occurs when early afterdepolarizations and delayed afterdepolarizations initiate spontaneous multiple

depolarizations, precipitating ventricular arrhythmias. Examples include torsades de pointes and ventricular arrhythmias caused by digitalis toxicity. Probably the most common mechanism of arrhythmogenesis results from re-entry. Requisites for re-entry include bidirectional conduction and unidirectional block. Micro level re-entry occurs with VT from conduction around the scar of myocardial infarction (MI), and macro level re-entry occurs via conduction through (Wolff-Parkinson-White [WPW] syndrome) concealed accessory pathways.

Signs & Symptoms

- Palpitations (a feeling of skipped heart beats, fluttering or "flip-flops," or feeling that your heart is "running away").
- Pounding in your chest.
- Dizziness or feeling light-headed.
- Fainting.
- Shortness of breath.
- Chest discomfort.
- Weakness or fatigue (feeling very tired).

Nursing Assessment	Nursing Intervention	Rationale	Goal
Decrease in cardiac output associated with cardiac arrhythmias	<p>monitor cardiovascular status by using a heart monitor.</p> <p>Assess and record apical pulse, peripheral pulses, blood pressure, capillary filling time, fluid intake and output, and skin characteristics (such as striped skin, skin color, edema, temperature, and diaphoresis).</p> <p>Please provide cardiovascular treatment, as directed.</p> <p>Help your child save energy through the grouping of nursing care.</p>	<p>Indications of heart monitoring and recording of various irregularities heart normal heart rate and rhythm of children.</p> <p>Assessments provide data from the basic measurement change, possibly indicated arrhythmias.</p> <p>Cardiovascular treatment could be given to help decide electrical disturbances associated with arrhythmias.</p> <p>Clustering allows care to be a long rest period.</p>	<p>will express their understanding of the disease abank, the reason for hospitalization, and nursing home care instructions and demonstrate procedures for home care.</p>

Acute Coronary Syndrome (ACS)

Pathophysiology

Acute coronary syndrome is a term used for any condition brought on by sudden, reduced blood flow to the heart. Acute coronary syndrome can describe chest pain you feel during a heart attack, or chest pain you feel while you're at rest or doing light physical activity (unstable angina). Acute coronary syndrome is often diagnosed in an emergency room or hospital.

Acute coronary syndrome is treatable if diagnosed quickly. Acute coronary syndrome treatments vary, depending on your signs, symptoms and overall health condition.

Signs & Symptoms

Many acute coronary syndrome symptoms are the same as those of a heart attack. And if acute coronary syndrome isn't treated quickly, a heart attack will occur. It's important to take acute coronary syndrome symptoms very seriously. Get medical help right away if you have these signs and symptoms and think you're having a heart attack:

- Chest pain (angina) that feels like burning, pressure or tightness and lasts several minutes or longer
- Pain elsewhere in the body, such as the left upper arm or jaw (referred pain)
- Nausea
- Vomiting
- Shortness of breath (dyspnea)
- Sudden, heavy sweating (diaphoresis)
-

If you're having a heart attack, the signs and symptoms may vary depending on your sex, age and whether you have an underlying medical condition, such as diabetes. Some unusual heart attack symptoms include:

- Abdominal pain
- Pain similar to heartburn
- Clammy skin
- Lightheadedness, dizziness or fainting
- Unusual or unexplained fatigue
- Feeling restless or apprehensive

Nursing Assessment	Nursing Intervention & Rationale	Goal
Reported pain Systolic blood pressure Diastolic blood pressure Apical heart rate Urinary output	Evaluate chest pain (e.g., intensity, location, radiation, duration, and precipitating and alleviating factors) in order to accurately evaluate, treat, and prevent further ischemia. Monitor effectiveness of oxygen therapy to increase oxygenation of myocardial tissue and prevent further ischemia. Administer medications to relieve/prevent pain and ischemia to decrease anxiety and cardiac workload. Obtain 12-lead ECG during pain episode to help differentiate angina from extension of MI or pericarditis. Monitor cardiac rhythm and rate and trends in blood pressure and hemodynamic parameters (e.g., central venous pressure and	Describes risk factors, the disease process, and rehabilitation activities necessary to manage the therapeutic regimen

	<p>pulmonary artery wedge pressure) to monitor for hypotension and bradycardia, which may lead to hypoperfusion.</p> <p>Monitor vital signs frequently to determine baseline and ongoing changes.</p> <p>Monitor for cardiac dysrhythmias, including disturbances of both rhythm and conduction, to identify and treat significant dysrhythmias.</p> <p>Monitor respiratory status for symptoms of heart failure to maintain appropriate levels of oxygenation and observe for signs of pulmonary edema.</p> <p>Monitor fluid balance (e.g., intake/output, daily weight) to monitor renal perfusion and observe for fluid retention.</p> <p>Arrange exercise and rest periods to avoid fatigue and decrease the oxygen demand on myocardium</p>	
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Atrial Fibrillation (AFIB)

Pathophysiology

Atrial fibrillation occurs in three clinical circumstances:

- As a primary arrhythmia in the absence of identifiable structural heart disease;
- As a secondary arrhythmia in the absence of structural heart disease but in the presence of a systemic abnormality that predisposes the individual to the arrhythmia;
- As a secondary arrhythmia associated with cardiac disease that affects the atria (Prystowsky et al, 1996).

The most common causes of AF are listed in Box 1. Three types have been identified: acute, chronic, and lone/primary.

- Acute AF: This has an onset within 24-48 hours of the causative event and usually converts spontaneously or in response to an antiarrhythmic agent (cardioversion). It may occur in individuals who are clinically normal but who have a temporary change in their condition; for example, it may occur in people who have consumed excessive alcohol;

- Chronic AF - this may be paroxysmal, and is the most debilitating form of AF because of its abrupt onset. It may be persistent or permanent and requires intervention by cardioversion to sinus rhythm (Marriott and Conover, 1998);

- Lone or primary AF - this occurs in the absence of any other clinical evidence that would suggest a primary cardiac disorder.

Signs & Symptoms

- Atrial fibrillation may be asymptomatic, but clinical manifestations may include:
 - ** Palpitations
 - ** Dyspnea
 - ** Pulmonary edema
 - ** Signs of cerebrovascular insufficiency

Nursing Assessment	Nursing Intervention	Rationale	Goal

Cardiogenic Shock

Pathophysiology

Signs and symptoms of cardiogenic shock reflects the nature of the circulation of the pathophysiology of heart failure. Heart damage resulting in decreased cardiac output, which in turn lowers blood pressure artery to the vital organs.

Blood flow to the coronary arteries is reduced, so that the intake of oxygen to the heart decreases, which in turn increases ischemia and further decreased the heart's ability to pump, eventually there was a vicious circle.

Dysrhythmias often occur due to decreased oxygen to the heart, such as in heart failure, the use of pulmonary artery catheter to measure left ventricular pressure and cardiac output is essential to assess the severity of the problem and evaluate the management that has been done. Increased left ventricular end-diastolic pressure of sustainable (LVEDP = Left ventricle End Diastolic Pressure) indicates that the heart fails to function as an effective pump.

Signs & Symptoms

- Anxiety, restlessness, altered mental state due to decreased cerebral perfusion and subsequent hypoxia.
- Hypotension due to decrease in cardiac output.
- A rapid, weak, thready pulse due to decreased circulation combined with tachycardia.
- Cool, clammy, and mottled skin (cutis marmorata), due to vasoconstriction and subsequent hypoperfusion of the skin.
- Distended jugular veins due to increased jugular venous pressure.
- Oliguria (low urine output) due to insufficient renal perfusion if condition persists.

- Rapid and deep respirations (hyperventilation) due to sympathetic nervous system stimulation and acidosis.
- Fatigue due to hyperventilation and hypoxia.
- Absent pulse in tachyarrhythmia.
- Pulmonary edema, involving fluid back-up in the lungs due to insufficient pumping of the heart..

Nursing Assessment	Nursing Intervention	Rationale	Goal
	<p>Administer oxygen by face mask or artificial airway to ensure adequate oxygenation of tissues.</p> <p>Adjust the oxygen flow rate to higher or lower level, as blood gas measurements indicate.</p> <p>Administer an osmotic diuretic, such as mannitol, if ordered to increase renal blood flow and urine output.</p> <p>Never flex the patient's "ballooned" leg at the hip because this may displace or fracture catheter.</p> <p>To ease emotional stress, allow frequent rest periods as possible.</p> <p>Allow family members to visit and comfort the patient as much as possible.</p> <p>Monitor and record blood pressure, pulse, respiratory rate, and peripheral pulse every 1 to 5 minutes until the patient stabilizes.</p> <p>Record hemodynamic pressure readings every 15 minutes.</p> <p>Monitor ABG values, complete blood count, and electrolyte levels.</p> <p>During therapy assess skin color and temperature and note any changes. Cold and</p>		

	clammy skin may be a sign of continuing peripheral vascular constriction, indicating progressive shock.		
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Coronary Artery Bypass Graft (CABG)

Pathophysiology

Coronary Artery Bypass Graft surgery is the most common type of cardiac surgery and the most common procedure for older adults. The occluded coronary arteries are bypassed with the client's own venous or arterial blood vessel or synthetic grafts. The internal artery (IMA) is the current graft of choice because it has a 90% patency rate at 12 years after the procedure. The vessels to be bypassed should have proximal lesions occluding more than 70% of the vessel's diameter but with good distal runoff. Bypass of less occluded vessels may result in poor perfusion through the graft and early obstruction. The procedure is most effective when good ventricular function remains and the ejection fraction is more that 40% to 50%.

Signs & Symptoms

Nursing Assessment	Nursing Intervention	Rationale	Goal
Risk for decreased cardiac output may be related to altered myocardial contractility, secondary to temporary factors, such as ventricular wall surgery, recent myocardial infarction, response to certain medication and drug interactions.	<p>Independent</p> <p>Monitor and document trends in heart rate and blood pressure; especially noting hypertension.</p> <p>Observe for bleeding from incisions and chest tube (if in place).</p> <p>Observe for changes in usual mental status, orientation, ad body movement or reflexes.</p> <p>Record skin temperature and color and quantity and equality of peripheral pulses.</p> <p>Measure and document intake and output and calculate fluid imbalance.</p> <p>Schedule uninterrupted rest and sleep periods.</p> <p>Inspect for jugular vein distention.</p>	<p>Tachycardia is the most common response to discomfort, inadequate blood or fluid replacement, and the stress of surgery.</p> <p>Helps identify bleeding complications that can reduce circulating volume, organ perfusion, and cardiac function.</p> <p>May indicate decreases cerebral blood flow or oxygenation as a result of diminished cardiac output.</p> <p>Warm. Pink and strong, equal pulses are general indicators of adequate cardiac output.</p> <p>Useful in determining</p>	the patient was able to demonstrate display homodynamic stability, such as stable blood pressure and cardiac output.

	Collaborative Review serial ECGs. Administer supplemental oxygen as indicated.	fluid needs or identifying fluid excesses, which can compromise cardiac output and oxygen consumption. Prevents fatigue or exhaustion and excessive cardiovascular stress. May be indicative of acute or chronic heart failure. Most frequently done to follow the progress in normalization of electrical conduction patterns and ventricular function after surgery or to identify complications. Promotes maximal oxygenation to reduce cardiac workload and aid in resolving myocardial irritability and dysrhythmias.	
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Congestive Heart Failure (CHF)

Pathophysiology

The heart is fundamentally a blood pump. It pumps blood from the right side of the heart to the lungs to pick up oxygen. The oxygenated blood returns to the left side of the heart. The left side of the heart then pumps blood into the circulatory system of blood vessels that carry blood throughout the body.

The heart consists of four chambers.

- The two upper chambers are called atria and the two lower chambers are called ventricles.
- The right atrium and right ventricle receive blood from the body through the veins and then pump the blood to the lungs.
- The left atrium and left ventricle receive blood from the lungs and pump it out through the aorta into the arteries, which feed all organs and tissues of the body with oxygenated blood.
- Because the left ventricle has to pump blood to the entire body, it is a stronger pump than the right ventricle.

Heart failure sounds frightening because it sounds like the heart just stops working. Do not be discouraged by the term heart failure. Heart failure means the tissues of the body are temporarily not receiving as much blood and oxygen as needed. With advancements in diagnosis and therapy for heart failure, patients are feeling better and living longer.

Signs & Symptoms

Left-sided heart failure

- ** Dyspnea on exertion, paroxysmal nocturnal dyspnea, or orthopnea
- ** Moist crackles on lung auscultation
- ** Frothy, blood-tinged sputum
- ** Tachycardia with S3 heart sound
- ** Pale, cool extremities
- ** Peripheral and central cyanosis
- ** Decreased peripheral pulses and capillary refill time longer than 3 seconds
- ** Decreased urinary output (<30 ml/hour)
- ** Easy fatigability
- ** Insomnia and restlessness

Right-sided heart failure

- ** Dependent pitting edema (peripheral and sacral)
- ** Weight gain
- ** Nausea and anorexia
- ** Jugular vein distention (JVD)
- ** Liver congestion (e.g. hepatomegaly), ascites or weakness

Left and right-sided heart failure

- ** Chest radiographs reveals cardiomegaly
- ** Vascular congestion of lung fields
- ** Electrocardiogram identifies hypertrophy or myocardial damage
- ** Arterial blood gas studies reveals decreased partial pressure of arterial oxygen and increased partial pressure of CO_2
 - ** Pulse oximeter readings may be less than 95%, indicating decreased oxygen saturation.
 - ** Multilumen pulmonary artery catheter shows elevated pulmonary artery and capillary wedge pressure in left-sided heart failure and elevated central venous pressure in right-sided heart failure.

Nursing Assessment	Nursing Intervention	Rationale	Goal
Decreased cardiac output related to altered myocardial contractility /isotropic changes.	INDEPENDENT: <ul style="list-style-type: none"> · Auscultate apical pulse; assess heart rate, and rhythm. · Inspect skin for pallor, cyanosis. · Monitor urine output, noting decreasing output and dark or concentrated urine. · Note changes in sensorium. · Provide quiet environment. 	<ul style="list-style-type: none"> · Tachycardia is usually present even at rest to compensate for decreased ventricular contractility. · Pallor is an indicative of diminished peripheral perfusion secondary to inadequate cardiac output, vasoconstriction, and anemia. Cyanosis may develop in refractory heart failure. Dependent areas are	The patient will be able to display vital signs within acceptable limits, dysrhythmias controlled and no symptoms of failure.

	<p>DEPENDENT:</p> <ul style="list-style-type: none"> · Administer supplemental oxygen as indicated. · Administer diuretics as prescribed. 	<p>often blue or mottled as venous congestion increases.</p> <ul style="list-style-type: none"> · Urine output is usually decreased during the day because of fluid shifts into tissues but may be increased at night because fluid returns to circulation when patient is recumbent. · May indicate inadequate cerebral perfusion secondary to decreased cardiac output. · Psychological rest help reduce emotional stress, which can produce vasoconstriction, elevating BP and increasing heart rate or work. · Increases available oxygen for myocardial uptake to combat effects of hypoxia or ischemia. · Diuretics, in conjunction with restriction of dietary sodium and fluids, often lead to clinical improvement in patients with heart failure 	
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Coronary Artery Disease (CAD)

Pathophysiology

- "Left-sided heart failure
 - ** Dyspnea on exertion, paroxysmal nocturnal dyspnea, or orthopnea
 - ** Moist crackles on lung auscultation
 - ** Frothy, blood-tinged sputum
 - ** Tachycardia with S3 heart sound
 - ** Pale, cool extremities
 - ** Peripheral and central cyanosis
 - ** Decreased peripheral pulses and capillary refill time longer than 3 seconds

- ** Decreased urinary output (<30 ml/hour)
- ** Easy fatigability
- ** Insomnia and restlessness
- Right-sided heart failure
 - ** Dependent pitting edema (peripheral and sacral)
 - ** Weight gain
 - ** Nausea and anorexia
 - ** Jugular vein distention (JVD)
 - ** Liver congestion (e.g. hepatomegaly), ascites or weakness
- Left and right-sided heart failure
 - ** Chest radiographs reveals cardiomegaly
 - ** Vascular congestion of lung fields
 - ** Electrocardiogram identifies hypertrophy or myocardial damage
 - ** Arterial blood gas studies reveals decreased partial pressure of arterial oxygen and increased partial pressure of CO_2 ** Pulse oximeter readings may be less than 95%, indicating decreased oxygen saturation.
 - ** Multilumen pulmonary artery catheter shows elevated pulmonary artery and capillary wedge pressure in left-sided heart failure and elevated central venous pressure in right-sided heart failure."
 - Coronary artery disease is a chronic process that begins during adolescence and slowly progresses throughout life. Independent risk factors include a family history of premature coronary artery disease, cigarette smoking, diabetes mellitus, hypertension, hyperlipidemia, sedentary lifestyle, and obesity. These risk factors accelerate or modify a complex and chronic inflammatory process that ultimately manifests as fibrous atherosclerotic plaque.

The most widely accepted theory of atherosclerosis states that the process represents an attempt at healing in response to endothelial injury. The first step in the atherosclerotic process is the development of fatty streaks, which contain atherogenic lipoproteins and macrophage foam cells. These streaks form between the endothelium and internal elastic lamina. Over time, an intermediate lesion made up of an extracellular lipid core and layers of smooth muscle and connective tissue matrix eventually forms a fibrous cap. The edge of the fibrous cap (the shoulder region) plays a critical role in the development of acute coronary syndromes. The shoulder region is the site where most plaques lose their integrity, or rupture. Plaque rupture exposes the underlying thrombogenic core of lipid and necrotic material to circulating blood. This exposure results in platelet adherence, aggregation, and progressive luminal narrowing, which are associated with acute coronary syndromes.

Inflammation is emerging as a critical component of atherosclerosis genesis, activity, and potential plaque instability. Patients with established coronary artery disease who possess a confluence of risk factors known as the metabolic syndrome remain at particularly high risk for a future vascular event, such as an acute myocardial infarction or cerebrovascular accident. Biochemical markers such as elevated levels of C-reactive protein signal a higher likelihood of vascular inflammation and portend a higher risk of vascular event rates. This marker may also signal more rapidly advancing coronary artery disease and the need for aggressive preventive measures.

Signs & Symptoms

- Angina
- Nausea and vomiting
- Dizziness and syncope

- Diaphoresis and cool, clammy skin
- Apprehension or a sense of impending doom

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Acute pain related to the imbalance between myocardial oxygen supply and demand. • Ineffective tissue perfusion related to myocardial ischemia and decreased cardiac output. • Anxiety related to pain, perceived threat of death, possibly lifestyle changes, and diagnosis of CAD. • Activity intolerance related to angina, pulmonary congestion, fatigue and inadequate tissue perfusion. • Ineffective therapeutic regimen management related to lack of knowledge related to disease process, prognosis, and treatment strategies. 	<ul style="list-style-type: none"> • Provide care during an acute angina attack • Promote pain relief • Prepare the client for possible treatment • Provide client and family teaching to promote optimal management of the disease and to minimize anxiety. • Provide referrals. 	<ul style="list-style-type: none"> • Provide client teaching and discharge planning: • Reduce the probability of an episode of angina plan by balancing rest and activity. • Avoid using medications or any over-the-counter substances (diet pills, nasal decongestants) that can increase the heart rate and blood pressure without first discussing with a health care provider. • Stop smoking and other use of tobacco, and avoid second-hand smoke (because smoking increase the heart rate, blood pressure and blood carbon monoxide levels) • Eat a diet low in saturated fat, high in fiber and if indicated, lower in calories. • Achieve and maintain normal blood pressure. • Achieve and maintain normal blood glucose level. • Take medications, especially aspirin and beta-blockers as prescribed. • Carry nitroglycerin at all times; state when and how to 	<ul style="list-style-type: none"> • Reduce pain

		use it; identify its side effects	
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Hypertension (HTN)

Pathophysiology

Central Nervous System

Medulla Oblongata, relays motor and sensory impulses between other parts of the brain and the spinal cord. Reticular formation (also in pons, midbrain, and diencephalon) functions in consciousness and arousal. Vital centers regulate heartbeat, breathing (together with pons) and blood vessel diameter.

Hypothalamus, controls and integrates activities of the autonomic nervous system and pituitary gland. Regulates emotional and behavioral patterns and circadian rhythms. Controls body temperature and regulates eating and drinking behavior. Helps maintain the waking state and establishes patterns of sleep. Produces the hormones oxytocin and antidiuretic hormone.

Cardiovascular System

Baroreceptor, pressure-sensitive sensory receptors, are located in the aorta, internal carotid arteries, and other large arteries in the neck and chest. They send impulses to the cardiovascular center in the medulla oblongata to help regulate blood pressure. The two most important baroreceptor reflexes are the carotid sinus reflex and the aortic reflex.

Chemoreceptors, sensory receptors that monitor the chemical composition of blood, are located close to the baroreceptors of the carotid sinus and the arch of the aorta in small structures called carotid bodies and aortic bodies, respectively. These chemoreceptors detect changes in blood level of O₂, CO₂, and H⁺.

Renal System

Renin-Angiotensin-Aldosterone system. When blood volume falls or blood flow to the kidneys decreases, juxtaglomerular cells in the kidneys secrete renin into the bloodstream. In sequence, renin and angiotensin converting enzyme (ACE) act on their substrates to produce the active hormone angiotensin II, which raises blood pressure in two ways. First, angiotensin II is a potent vasoconstrictor; it raises blood pressure by increasing systemic vascular resistance. Second, it stimulates secretion of aldosterone, which increases reabsorption of sodium ions and water by the kidneys. The water reabsorption increases total blood volume, which increases blood pressure.

Antidiuretic hormone. ADH is produced by the hypothalamus and released from the posterior pituitary in response to dehydration or decreased blood volume. Among other actions, ADH causes vasoconstriction, which increases blood pressure.

Atrial Natriuretic Peptide. Released by cells in the atria of the heart, ANP lowers blood pressure by causing vasodilation and by promoting the loss of salt and water in the urine, which reduces blood volume.

Signs & Symptoms

- Headache,
- dizziness,

- blurred vision,
- nausea and vomiting, and
- chest pain and shortness of breath.
- Heart attack
- Heart failure
- Stroke or transient ischemic attack (TIA)
- Kidney failure
- Eye damage with progressive vision loss
- Peripheral arterial disease causing leg pain with walking (claudication)
- Outpouchings of the aorta, called aneurysms

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Risk for prone behavior related to lack of knowledge about the disease 	<p>INDEPENDENT:</p> <ul style="list-style-type: none"> • Define and state the limits of desired BP. Explain hypertension and its effect on the heart, blood vessels, kidney, and brain. • Assist the patient in identifying modifiable risk factors like diet high in sodium, saturated fats and cholesterol. • Reinforce the importance of adhering to treatment regimen and keeping follow up appointments. • Suggest frequent position changes, leg exercises when lying down. • Help patient identify sources of sodium intake. • Encourage patient to decrease or eliminate caffeine like in tea, coffee, cola and hocolates. • Stress importance of accomplishing daily rest periods. <p>COLLABORATIVE:</p> <ul style="list-style-type: none"> • Provide 	<ul style="list-style-type: none"> • Provides basis For understanding elevations of BP, and clarifies misconceptions and also understanding that high BP can exist without symptom or even when feeling well. • These risk factors have been shown to contribute to hypertension. • Lack of cooperation is common reason for failure of antihypertensive therapy. • Decreases peripheral venous pooling that may be potentiated by vasodilators and prolonged sitting or standing. • Two years on moderate low salt diet may be sufficient to control mild hypertension. • Caffeine is a cardiac stimulant and may adversely affect cardiac function. • Alternating rest and activity increases tolerance 	<ul style="list-style-type: none"> • the patient was able to verbalize understanding of the disease process and treatment regimen.

	information Regarding community resources, and support patients in making lifestyle changes.	to activity progression. <ul style="list-style-type: none"> • Community resources like health centers programs and check ups are helpful in controlling hypertension. 	
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Hyperlipidemia (high cholesterol)

Pathophysiology

Hyperlipidemia is an excess of fatty substances called lipids, largely cholesterol and triglycerides, in the blood. It is also called hyperlipoproteinemia because these fatty substances travel in the blood attached to proteins. This is the only way that these fatty substances can remain dissolved...

Signs & Symptoms

Nursing Assessment	Nursing Intervention	Rationale	Goal

Myocardial Infarction

Pathophysiology

In an MI, inadequate coronary blood flow rapidly results in myocardial ischemia in the affected area. The location and extent of the infarct determine the effects on cardiac function. Ischemia depresses cardiac function and triggers autonomic nervous system responses that exacerbate the imbalance between myocardial oxygen supply and demand. Persistent ischemia results in tissue necrosis and scar tissue formation, with permanent loss of myocardial contractility in the affected area. Cardiogenic shock may develop because of inadequate CO from decreased myocardial contractility and pumping capacity.

Signs & Symptoms

- Chest pain (typically, chest pain is persistent and crushing; located substernally with radiation to the arm, neck, jaw, or back; and unrelieved by rest or nitrates. A silent MI may produce no pain.)
- Diaphoresis and cool, clammy, pale skin
- Nausea and vomiting
- Dyspnea with or without crackles
- Palpitations or syncope
- Restlessness and anxiety or feeling of impending doom

- Tachycardia or bradycardia
- Decreased blood pressure
- Altered S3 heart sound (indicates left ventricular failure)
- Electrocardiogram: Myocardial ischemia causes the T wave to be larger and inverted; in epicardial myocardial ischemia, the ST segment is elevated; in endocardial myocardial ischemia, the ST segment is depressed.
- Serum enzyme studies reveal elevated levels of creatine phosphokinase; lactate dehydrogenase and troponin.
- The white blood cell count is elevated.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<ul style="list-style-type: none"> • Pain r/t tissue ischemia (coronary artery occlusion) 	<p>Independent:</p> <ul style="list-style-type: none"> • Obtain full description of pain from patient including location, intensity (0–10), duration, characteristics (dull/crushing), and radiation. Assist patient to quantify pain by comparing it to other experiences • Instruct patient to report pain immediately. • Provide quiet environment, calm activities, and comfort measures • Assist/instruct in relaxation techniques, e.g., deep/slow breathing, distraction behaviors, visualization, guided imagery <p>Collaborative</p> <ul style="list-style-type: none"> • Administer supplemental oxygen by means of nasal cannula or face mask, as indicated <p>Administer medications as indicated:</p> <ul style="list-style-type: none"> • Antianginals, e.g., 	<ul style="list-style-type: none"> • Pain is a subjective experience and must be described by patient • Delay in reporting pain hinders pain relief/may require increased dosage of medication to achieve relief • Decreases external stimuli, which may aggravate anxiety and cardiac strain, limit coping abilities and adjustment to current situation • Helpful in decreasing perception of/response to pain. Provides a sense of having some control over the situation, increase in positive attitude. • Increases amount of oxygen available for myocardial uptake and thereby may relieve discomfort associated with tissue ischemia • Nitrates are useful for pain control by coronary vasodilating effects, which increase coronary blood flow and myocardial perfusion. • Important second-line agents for pain 	<ul style="list-style-type: none"> • Verbalized relief/control of chest pain within appropriate time frame for administered medications.

	nitroglycerin, isosorbide dinitrate (Isordil) <ul style="list-style-type: none"> • Beta-blockers, e.g., atenolol (Tenormin), propranolol (Inderal), metoprolol (Lopressor) • Analgesics, e.g., morphine, meperidine (Demerol) 	control through effect of blocking sympathetic stimulation, thereby reducing heart rate, systolic BP, and myocardial oxygen demand <ul style="list-style-type: none"> • Although intravenous (IV) morphine is the usual drug of choice, other injectable narcotics may be used in acute 	
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Pulmonary Edema

Pathophysiology

Pulmonary edema is a condition caused by excess fluid in the lungs. This fluid collects in the numerous air sacs in the lungs, making it difficult to breathe.

In most cases, heart problems cause pulmonary edema. But fluid can accumulate for other reasons, including pneumonia, exposure to certain toxins and medications, and exercising or living at high elevations.

Pulmonary edema that develops suddenly (acute) is a medical emergency requiring immediate care. Although pulmonary edema can sometimes prove fatal, the outlook improves when you receive prompt treatment for pulmonary edema along with treatment for the underlying problem. Treatment for pulmonary edema varies depending on the cause, but generally includes supplemental oxygen and medications

Signs & Symptoms

- **Sudden (acute) pulmonary edema symptoms**
 - Extreme shortness of breath or difficulty breathing (dyspnea) that worsens when lying down
 - A feeling of suffocating or drowning
 - Wheezing or gasping for breath
 - Anxiety, restlessness or a sense of apprehension
 - A cough that produces frothy sputum that may be tinged with blood
 - Excessive sweating
 - Pale skin
 - Chest pain, if pulmonary edema is caused by heart disease
 - A rapid, irregular heartbeat (palpitations)
- **Long-term (chronic) pulmonary edema symptoms**
 - Having more shortness of breath than normal when you're physically active.

- Difficulty breathing with exertion, often when you're lying flat as opposed to sitting up.
 - Wheezing.
 - Awakening at night with a breathless feeling that may be relieved by sitting up.
 - Rapid weight gain when pulmonary edema develops as a result of congestive heart failure, a condition in which your heart pumps too little blood to meet your body's needs. The weight gain is from buildup of fluid in your body, especially in your legs.
 - Swelling in your legs and ankles.
 - Loss of appetite.
 - Fatigue.
- **High-altitude pulmonary edema symptoms**
 - Headache
 - Insomnia
 - Fluid retention
 - Cough
 - Shortness of breath

Nursing Assessment	Nursing Intervention	Goal
<ul style="list-style-type: none"> ● Impaired gas exchange related to increased pulmonary congestion secondary to increased left ventricular end diastolic pressure 	<ul style="list-style-type: none"> ● Provide supplemental oxygen via mask as indicated. ● Administer diuretic agents or nesiritide to reduce circulating volume, which will improve gas exchange. ● Monitor urine output and electrolytes. ● Administer vasodilating agents to redistribute fluid volumes, which will facilitate gas exchange. ● Morphine sulfate maybe ordered to promote preload and after load reduction and to decrease anxiety. 	<ul style="list-style-type: none"> ● Impaired gas exchange related to increased pulmonary congestion secondary to increased left ventricular end diastolic pressure

Valvular Heart Disease

Pathophysiology

Valvular heart disease is characterized by damage to or a defect in one of the four heart valves: the **mitral, aortic, tricuspid or pulmonary**.

The mitral and tricuspid valves control the flow of blood between the atria and the ventricles (the upper and lower chambers of the heart). The pulmonary valve controls the flow of blood from the heart to the lungs, and the aortic valve governs blood flow between the heart and the aorta, and thereby the blood vessels to the rest of the body. The mitral and aortic valves are the ones most frequently affected by valvular heart disease.

Normally functioning valves ensure that blood flows with proper force in the proper direction at the proper time. In valvular heart disease, the valves become too narrow and hardened (stenotic) to open fully, or are unable to close completely (incompetent).

A stenotic valve forces blood to back up in the adjacent heart chamber, while an incompetent valve allows blood to leak back into the chamber it previously exited. To compensate for poor pumping action, the heart muscle enlarges and thickens, thereby losing elasticity and efficiency. In addition, in some cases, blood pooling in the chambers of the heart has a greater tendency to clot, increasing the risk of stroke or pulmonary embolism.

The severity of valvular heart disease varies. In mild cases there may be no symptoms, while in advanced cases, valvular heart disease may lead to congestive heart failure and other complications. Treatment depends upon the extent of the disease.

Signs & Symptoms

Valve disease symptoms can occur suddenly, depending upon how quickly the disease develops. If it advances slowly, then your heart may adjust and you may not notice the onset of any symptoms easily. Additionally, the severity of the symptoms does not necessarily correlate to the severity of the valve disease. That is, you could have no symptoms at all, but have severe valve disease. Conversely, severe symptoms could arise from even a small valve leak.

Many of the symptoms are similar to those associated with congestive heart failure, such as shortness of breath and wheezing after limited physical exertion and swelling of the feet, ankles, hands or abdomen (edema). Other symptoms include:

- Palpitations, chest pain (may be mild).
- Fatigue.
- Dizziness or fainting (with aortic stenosis).
- Fever (with bacterial endocarditis).
- Rapid weight gain.

Nursing Assessment	Nursing Intervention	Rationale	Goal
<p>Cardiac Output, decreased May be related to</p> <ul style="list-style-type: none"> • Altered myocardial contractility/isotropic changes Alterations in rate, rhythm, electrical conduction Structural changes (e.g., valvular defects, ventricular aneurysm) <p>Possibly evidenced by</p> <ul style="list-style-type: none"> • Increased heart rate (tachycardia), dysrhythmias, ECG changes • Changes in BP (hypotension/hypertension) 	<ul style="list-style-type: none"> • Auscultate apical pulse; assess heart rate, rhythm (document dysrhythmias if telemetry available). • Palpate peripheral pulses. • Monitor BP • Inspect skin for pallor, Cyanosis 	<ul style="list-style-type: none"> • Tachycardia is usually present (even at rest) to compensate for decreased ventricular contractility. Premature atrial contractions (PACs), paroxysmal atrial tachycardia (PAT), PVCs, multifocal atrial tachycardia (MAT), and atrial fibrillation (AF) are 	<p>Cardiac output adequate for individual needs. Complications prevented/resolved. Optimum level of activity/functioning attained. Disease process/prognosis and therapeutic regimen understood. Plan in place to meet needs after discharge.</p>

<p>ension)</p> <ul style="list-style-type: none"> • Extra heart sounds • Decreased urine output • Diminished peripheral pulses • Cool, ashen skin; diaphoresis • Orthopnea, crackles, JVD, liver engorgement, edema • Chest pain 		<p>common dysrhythmias associated with HF, although others may also occur.</p> <ul style="list-style-type: none"> • Decreased cardiac output may be reflected in diminished radial, popliteal, dorsalis pedis, and posterior tibial pulses. Pulses may be fleeting or irregular to palpation, and pulsus alternans (strong beat alternating with weak beat) may be present. 	
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Endocrine

Diabetes Mellitus Type 1

Pathophysiology

Diabetes Mellitus (DM) is a chronic metabolic disorder caused by an absolute or relative deficiency of insulin, an anabolic hormone. Type 1 diabetes mellitus can occur at any age and is characterized by the marked and progressive inability of the pancreas to secrete insulin because of autoimmune destruction of the beta cells. It commonly occurs in children, with a fairly abrupt onset; however, newer antibody tests have allowed for the identification of more people with the new-onset adult form of type 1 diabetes mellitus called latent autoimmune diabetes of the adult (LADA). The distinguishing characteristic of a patient with type 1 diabetes is that, if his or her insulin is withdrawn, ketosis and eventually ketoacidosis develop. Therefore, these patients are dependent on exogenous insulin.

Type 1 diabetes (formerly called juvenile-onset or insulin-dependent diabetes), accounts for 5% to 10% of all people with diabetes. In type 1 diabetes, the body's immune system destroys the cells that release insulin, eventually eliminating insulin production from the body. Without insulin, cells cannot absorb sugar (glucose), which they need to produce energy. a

Signs & Symptoms

- Extreme thirst
- frequent urination
- drowsiness

- lethargy
- increased appetite
- sudden weight loss for no reason
- sudden vision changes
- sugar in urine
- ketones in urine
- heavy or labored breathing
- unconsciousness

Nursing Assessment	Nursing Intervention	Rationale	Goal
Fluid volume deficient related to osmotic diuresis from hyperglycemia	<p>Independent:</p> <p>Monitor orthostatic blood pressure changes.</p> <p>Monitor respiratory pattern like Kussmaul's respirations and acetone breath.</p> <p>Monitor temperature, skin color and moisture.</p> <p>Assess peripheral pulses, capillary refill, skin turgor, and mucous membrane.</p> <p>Monitor input and output. Note urine specific gravity</p> <p>Weigh daily.</p> <p>Maintain fluid intake at least 2500 ml / day within cardiac tolerance with oral</p>	<p>Hypovolemia may be manifested by hypotension and tachycardia.</p> <p>Lungs remove carbonic acid through respirations, producing a compensatory respiratory alkalosis for ketoacidosis.</p> <p>Fever, chills, and diaphoresis are common with infectious process; fever with flushed, dry skin may reflect dehydration.</p> <p>Indicators of level of dehydration, adequacy of circulating volume.</p> <p>Provides ongoing estimate of volume replacement needs, kidney function, and effectiveness of therapy.</p> <p>Provides the best assessment of current fluid status and adequacy of fluid replacement.</p> <p>Maintains hydration and circulating volume.</p>	the patient will be able to demonstrate adequate hydration evidenced by stable vital signs, palpable peripheral pulses, good skin turgor and capillary refill.

	intake is resumed. Promote comfortable environment. Cover patient with light sheets. Collaborative: Administer fluids as indicated.	Avoids overheating, which could promote further fluid loss. Type and amount of fluid depend on the degree of deficit and individual patient response.	
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Diabetes Mellitus Type 2

Pathophysiology

- Type 2 diabetes mellitus occurs when the pancreas produces insufficient amounts of the hormone insulin and/or the body's tissues become resistant to normal or even high levels of insulin. This causes high blood glucose (sugar) levels, which can lead to a number of complications if untreated.
- Type 2 diabetes is a chronic medical condition that requires regular monitoring and treatment. Treatment, which includes lifestyle adjustments, self-care measures, and sometimes medications, can control blood glucose levels in the near-normal range and minimize the risk of diabetes-related complications.
- Type 2 diabetes accounts for around 85% of all people with diabetes.

Signs & Symptoms

- Any symptoms of DM Type 1
- recurring or hard-to heal skin, gum or urinary tract infections
- drowsiness
- tingling of hands and feet
- itching of skin and genitals

Nursing Assessment	Nursing Intervention	Rationale	Goal
Risk for infection related to high glucose levels, decreased leukocyte function.	Independent: Observe for signs of infection and inflammation. Promote good hand washing by nurse and	Patient may be admitted with infection, which could have precipitated the ketoacidotic state, or may develop a nosocomial infection. Reduces the risk of cross contamination	the patient will be able to identify intervention to prevent or reduce risk of infection.

	<p>patient.</p> <p>Maintain aseptic technique for IV insertion procedure, administration of medications, and providing maintenance and site care. Rotate IV sites as indicated.</p> <p>Provide catheter or perinea care. Teach the female patient to clean from front to back after elimination.</p> <p>Provide conscientious skin care, gently areas. Keep the skin dry, linens dry and wrinkle free.</p> <p>Place in semi – fowler’s position.</p> <p>Encourage adequate dietary and fluid intake of 3000 ml per day.</p> <p>Collaborative: Obtain specimen for culture and sensitivities as indicated.</p>	<p>High glucose in the blood creates an excellent medium for bacterial growth.</p> <p>Minimizes the risk for infection.</p> <p>Peripheral circulation may be impaired, placing patient at increased risk for skin irritation or breakdown and infection.</p> <p>Facilitates lung expansion and reduces risk of aspiration.</p> <p>Decrease susceptibility to infection.</p> <p>Identifies organisms so that most appropriate drug therapy can be instituted.</p>	
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Hyperglycemia

Pathophysiology

Hyperglycemic hyperosmolar nonketotic syndrome portrait of insulin deficiency, and excessive hormone glucagon. Decrease insulin resistance causes glucose movement into cells, resulting in the accumulation of glucose in plasma. Increase in the hormone glucagon which causes glycogenolysis can increase plasma glucose levels. Increased glucose levels lead to hyperosmolar.

Serum hyperosmolar conditions would attract intracellular fluid into the intra vascular, which can lower the intracellular fluid volume. If the client does not feel the sensation of thirst will cause dehydration.

High levels of serum glucose are excreted in the kidneys, causing glycosuria which can lead to excessive osmotic diuresis (polyuria). The impact of polyuria would cause excessive fluid loss, and followed the loss of potassium, sodium and phosphate.

Due to lack of insulin the glucose can not be converted into glycogen to increase blood sugar levels and hyperglycemia occurs. The kidneys can not resist hyperglycemia, because the threshold for blood sugar was 180 mg% in case of hyperglycemia so that the kidneys can not filter out and absorb the amount of glucose in the blood. With respect to the nature of the sugar which absorbs all the excess water removed with the urine is called glucosuria. Simultaneously the state of glucosuria then some water is lost in the urine is called polyuria. Polyuria resulting in intra cellular dehydration, this will stimulate the thirst center so that patients will feel constantly hungry, so the patient will continue to drink the so-called polydipsi. Decreased renal perfusion resulting in increased secretion of the hormone over again and hyperglycemic hyperosmolar arise.

The lack of insulin production will cause a decrease in glucose transport into the cells so the cells are starved of food and stores carbohydrates, fats and proteins to be depleted. Because it is used to burn the body, then the client will feel hungry eat, causing many so-called polyphagia.

Failure to restore the body's homeostasis situation will lead to hyperglycemia, hyperosmolar, excessive osmotic diuresis and dehydration. Central nervous system dysfunction due to transport oxygen to the brain disorder and tends to be a comma.

Hemoconcentration increases the blood viscosity which may lead to the formation of blood clots, thromboembolism, cerebral infarction, heart.

Signs & Symptoms

- Frequency in urination
- Thirst
- Dry mouth
- Urination at night
- Drowsiness or fatigue
- Loss of weight
- Increase in appetite
- Slow healing of wounds
- Blurriness in vision
- Dry and itchy skin
- Rapid loss in weight
- Unconsciousness
- Increased confusion or drowsiness
- Breathing difficulty
- Dizziness when you stand up
- Coma

Nursing Assessment	Nursing Intervention	Rationale	Goal
Risk for Infection Risk for Disturbed Sensory Perception Powerlessness Imbalanced Nutrition	Observe for signs of infection and inflammation, e.g., fever, flushed appearance, wound	Patient may be admitted with infection, which could have precipitated the ketoacidotic state, or	Homeostasis achieved. Causative/precipitating factors

<p>Less Than Body Requirements Deficient Fluid Volume Fatigue</p>	<p>drainage, purulent sputum, cloudy urine</p> <p>Promote good handwashing by staff and patient.</p> <p>Maintain aseptic technique for IV insertion procedure, administration of medications, and providing maintenance/site care. Rotate IV sites as indicated.</p> <p>Provide catheter/perineal care. Teach the female patient to clean from front to back after elimination</p> <p>Monitor vital signs and mental status.</p> <p>Address patient by name; reorient as needed to place, person, and time. Give short explanations, speaking slowly and enunciating clearly.</p> <p>Schedule nursing time to provide for uninterrupted rest periods.</p> <p>Encourage patient/SO to express feelings about hospitalization and disease in general.</p>	<p>may develop a nosocomial infection.</p> <p>Reduces risk of cross-contamination.</p> <p>High glucose in the blood creates an excellent medium for bacterial growth</p> <p>Minimizes risk of UTI. Comatose patient may be at particular risk if urinary retention occurred before hospitalization. Note: Elderly female diabetic patients are especially prone to urinary tract/vaginal yeast infections.</p> <p>Provides a baseline from which to compare abnormal findings, e.g., fever may affect mentation.</p> <p>Decreases confusion and helps maintain contact with reality.</p> <p>Promotes restful sleep, reduces fatigue, and may improve cognition.</p> <p>Identifies concerns and facilitates problem solving.</p> <p>Recognition that</p>	<p>corrected/controlled. Complications prevented/minimized. Disease process/prognosis, self-care needs, and therapeutic regimen understood. Plan in place to meet needs after discharge.</p>
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	<p>Acknowledge normality of feelings.</p> <p>Assess how patient has handled problems in the past. Identify locus of control.</p>	<p>reactions are normal can help patient problem-solve and seek help as needed. Diabetic control is a full-time job that serves as a constant reminder of both presence of disease and threat to patient's health/life.</p> <p>Knowledge of individual's style helps determine needs for treatment goals. Patient whose locus of control is internal usually looks at ways to gain control over own treatment program. Patient who operates with an external locus of control wants to be cared for by others and may project blame for circumstances onto external factors.</p>	
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Hypoglycemia

Pathophysiology

Hypoglycemia, also called low blood glucose or low blood sugar, occurs when blood glucose drops below normal levels. Glucose, an important source of energy for the body, comes from food. Carbohydrates are the main dietary source of glucose. Rice, potatoes, bread, tortillas, cereal, milk, fruit, and sweets are all carbohydrate-rich foods.

After a meal, glucose is absorbed into the bloodstream and carried to the body's cells. Insulin, a hormone made by the pancreas, helps the cells use glucose for energy. If a person takes in more glucose than the body needs at the time, the body stores the extra glucose in the liver and muscles in a form called glycogen. The body can use glycogen for energy between meals. Extra glucose can also be changed to fat and stored in fat cells. Fat can also be used for energy.

When blood glucose begins to fall, glucagon-another hormone made by the pancreas-signals the liver to break down glycogen and release glucose into the bloodstream. Blood glucose will then rise toward a normal level. In some people with diabetes, this glucagon response to hypoglycemia is impaired and other hormones such as epinephrine, also called adrenaline, may raise the blood glucose level. But with diabetes treated with insulin or pills that increase insulin production, glucose levels can't easily return to the normal range.

Hypoglycemia can happen suddenly. It is usually mild and can be treated quickly and easily by eating or drinking a small amount of glucose-rich food. If left untreated, hypoglycemia can get worse and cause confusion, clumsiness, or fainting. Severe hypoglycemia can lead to seizures, coma, and even death.

In adults and children older than 10 years, hypoglycemia is uncommon except as a side effect of diabetes treatment. Hypoglycemia can also result, however, from other medications or diseases, hormone or enzyme deficiencies, or tumors.

Signs & Symptoms

Hypoglycemia causes symptoms such as

- hunger
- shakiness
- nervousness
- sweating
- dizziness or light-headedness
- sleepiness
- confusion
- difficulty speaking
- anxiety
- weakness

Hypoglycemia can also happen during sleep. Some signs of hypoglycemia during sleep include

- crying out or having nightmares
- finding pajamas or sheets damp from perspiration
- feeling tired, irritable, or confused after waking up

Nursing Assessment	Nursing Intervention	Rationale	Goal
sweating dizziness or light-headedness sleepiness	Ensure a patent airway. Administer liquids that contain glucose. If the patient is alert, give him juice with sugar added, followed by protein and complex carbohydrates to prevent hypoglycemia from recurring the next hour. If the patient has a decreased level of consciousness, establish a large-bore I.V.line and administer 50 ml of 50% dextrose as a bolus. If he doesn't regain consciousness in 15 minutes, repeat the	After determining which factors contributed to this incident of hypoglycemia help the patient understand how to prevent its recurrence. Teach the patient to recognize early signs and symptoms of hypoglycemia. Teach the patient how to use a glucometer at home if a chronic condition may cause hypoglycemia to recur. Emphasize the importance of having glucose tablets, hard candy, or other food	The patient will maintain airway patency and adequate circulation. The patient will display no change in neurologic status. The patient will demonstrate a blood glucose level between 60 and 150mg/dl.

	<p>bolus of dextrose. If I.V. access can't be established, administer glucose gel under the patient's tongue or give glucose-rich liquids by nasogastric tube instead of providing the IM dextrose solution.</p> <p>If none of the above interventions is possible, administer glucagon or epinephrine I.M.</p> <p>Repeat the measurement of the blood glucose level in 1 hour.</p> <p>Monitor the patient's heart rate, cardiac rhythm and blood pressure.</p> <p>Administer a normal saline bolus if hypotension occurs.</p> <p>Replace electrolytes based on laboratory test results.</p> <p>Help determine the cause of hypoglycemia by interviewing the patient and reviewing his history. Be sure to inquire about such common causes as poor food intake, medication changes, alcohol or other recreational drug use, hepatic or renal impairment that prevents gluconeogenesis, pancreatic tumor or an endocrine disorder, including impaired pituitary, thyroid, parathyroid, or adrenal glands.</p> <p>Be aware that postprandial hypoglycemia may</p>	<p>containing simple sugars readily available.</p>	
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	occur with many conditions, especially after gastric bypass surgery.		
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Diabetic Ketone Acidosis (DKA)

Pathophysiology

Diabetic ketoacidosis is a serious complication of diabetes that occurs when your body produces very high levels of blood acids called ketones.

Diabetic ketoacidosis develops when you have too little insulin in your body. Insulin normally plays a key role in helping sugar (glucose) — a major source of energy for your muscles and other tissues — enter your cells. Without enough insulin, your body begins to break down fat as an alternate fuel. In turn, this process produces toxic acids in the bloodstream called ketones, eventually leading to diabetic ketoacidosis if untreated.

Signs & Symptoms

- Deficient fluid volume (specify)
- Imbalanced nutrition less than body requirements
- Risk for infection (sepsis)
- Risk for disturbed sensory perception (specify)
- Fatigue
- Powerlessness
- Knowledge deficient (learning need) regarding condition, prognosis, treatment regimen, self-care, and discharge needs

Nursing Assessment	Nursing Intervention / Rationale	Goal
<ul style="list-style-type: none"> • sleep/rest • disturbances Weakness, • fatigue, • difficulty walking/moving • Muscle cramps, decreased muscle strength 	<ul style="list-style-type: none"> • Restore fluid/electrolyte and acid-base balance. • Correct/reverse metabolic abnormalities. • Identify/assist with management of underlying cause/disease process. • Prevent complications. • Provide information about disease process/prognosis, self-care, and treatment needs 	<ul style="list-style-type: none"> • Homeostasis achieved. • Causative/precipitating factors corrected/controlled. • Complications prevented/minimized. • Disease process/prognosis, self-care needs, and therapeutic regimen understood. • Plan in place to meet needs after discharge

Gallbladder, Liver & Appendix

Appendicitis

Pathophysiology

- Appendicitis is usually caused by blockage of the lumen of the appendix. Obstruction causes the mucus produced by mucous appendix suffered dam. The longer the mucus is more and more, but the elastic wall of the appendix has limitations that lead to increased intra-luminal pressure. These pressures will impede the flow of lymph resulting in mucosal edema and ulceration. At that time there was marked focal acute appendicitis with epigastric pain.
- When mucus secretion continues, the pressure will continue to increase. This will cause venous obstruction, increased edema and bacteria will penetrate the wall so that the inflammation of the peritoneum arising widespread and can cause pain in the lower right abdomen is called acute suppurative appendicitis.
- If the flow is disrupted arterial wall infarction will occur followed by gangrene appendix. This stage is called appendicitis gangrenosa. If the appendix wall fragile, there will be a perforation, called perforated appendicitis.
- When the process is slow, the omentum and the adjacent bowel will move toward the appendix to appear appendicularis infiltrates.
- In children because it shortens the omentum and appendix is longer, thinner walls. The situation is coupled with the immune system that is still less easy to occur perforation, whereas in the elderly prone to occur because there is blood vessel disorders..

Signs & Symptoms

- Aching pain that begins around your navel and often shifts to your lower right abdomen
- Pain that becomes sharper over several hours
- Tenderness that occurs when you apply pressure to your lower right abdomen
- Sharp pain in your lower right abdomen that occurs when the area is pressed on and then the pressure is quickly released (rebound tenderness)
- Pain that worsens if you cough, walk or make other jarring movements
- Nausea
- Vomiting
- Loss of appetite
- Low-grade fever
- Constipation
- Inability to pass gas
- Diarrhea
- Abdominal swelling

Nursing Assessment	Nursing Intervention	Rationale	Goal
Acute pain related to inflammation of tissues.	Independent: <input type="checkbox"/> Investigate pain reports, noting location, duration, intensity (0-10 scale), and characteristics (dull, sharp, constant). <input type="checkbox"/> Maintain semi fowler's position. <input type="checkbox"/> Move patient slowly	<input type="checkbox"/> Changes in location or intensity are not uncommon but may reflect developing complications. <input type="checkbox"/> Reduces abdominal distention, thereby Reduces tension. <input type="checkbox"/> Reduces muscle tension or guarding,	After nursing interventions the patient will demonstrate use of relaxation skills, other methods to promote comfort.

	<p>and deliberately.</p> <ul style="list-style-type: none"> □ Provide comfort measure like back rubs, deep breathing. Instruct in relaxation or Visualization exercises. Provide divisional activities. □ Provide frequent oral care. Remove noxious environmental stimuli. <p>Collaborative:</p> <ul style="list-style-type: none"> □ Administer analgesics as prescribed. 	<p>which may help minimize pain of movement.</p> <ul style="list-style-type: none"> □ Promotes relaxation and may enhance patient's coping abilities by refocusing attention. □ Reduces nausea and vomiting, which can increase intra-abdominal pressure or pain. □ Reduce metabolic rate and aids in pain relief and Promotes healing. 	
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Cholecystitis

Pathophysiology

- **Acute Cholecystitis Pathophysiology**

One of the most common types of cholecystitis is acute cholecystitis. This is when the onset of inflammation of the gallbladder is sudden and intense, with fast progression of the disease. More often than not, the inflammation is caused due to obstruction of the bile duct, which is known as calculous cholecystitis, as they are caused due to gallstones, or cholelithiasis. There are other causes of acute cholecystitis as well, such as ischemia, chemical poisoning, motility disorders, infections with protozoa, collagen disease, allergic reactions, etc. The obstruction results in gallbladder distension, which results in edema of the cells lining the gallbladder. This in turn results in ischemia, which spurs on inflammatory mediators, especially prostaglandins, which further aggravates the inflammation. The lining wall of the gallbladder may eventually undergo necrosis and gangrene, which is known as gangrenous cholecystitis.

The inflammation of the gallbladder wall may be bacterial in nature, or may even be sterile in some cases. In cases where it is bacterial, there is normally superinfection with gas forming organisms, which may lead to formation of gas in the wall or the lumen of the gallbladder, which leads to a condition known as emphysematous cholecystitis. However, it is normally seen that bacterial contamination is secondary to biliary obstruction, because in the early stages of gallbladder wall inflammation, the bile is seen to be sterile.

- **Acalculous Cholecystitis Pathophysiology**

The pathophysiology of acalculous cholecystitis is not very well understood. It is said that the causative factors may be many and interlinked. Functional cystic duct obstruction is normally present and is related to biliary sludge or even bile inspissation. This inspissation is caused due to dehydration, which leads to an

increase in the viscosity of bile, thus, causing bile stasis. This may be spurred on by trauma or due to systemic disease or disorder. Other reasons include burns, multisystem organ failure and parenteral nutrition. In some cases, patients that have sepsis may have direct gallbladder wall lining inflammation. This is because one needs to understand that bile is an extremely favorable growth medium for bacteria and infections in this space develop rapidly, especially when they are spurred on by a systemic infection. Acalculous cholecystitis may occur with or without localized or generalized tissue ischemia and obstruction.

At times, there may be spontaneous resolution of acute cholecystitis which may occur within five to seven days after the onset of symptoms. This is especially seen in cases of acalculous cholecystitis, due to reestablishment of cystic duct patency.

Cholecystitis symptoms are quite obvious, which greatly helps in the diagnosis. The common triad helps in diagnosing cholecystitis - jaundice, upper right quadrant pain and fever. Cholecystitis diet helps to considerably mitigate these symptoms. To properly diagnose and understand how this condition progresses, a person needs to understand cholecystitis pathophysiology. This helps to understand the prognosis and severity of this disease.

Signs & Symptoms

- Nausea or vomiting.
- Tenderness in the right abdomen.
- Fever.
- Pain that gets worse during a deep breath.
- Pain for more than 6 hours, particularly after meals.

Nursing Assessment	Nursing Intervention	Rationale	Goal
May be related to Biological injuring agents: obstruction/ductal spasm, inflammatory process, tissue ischemia/necrosis Possibly evidenced by Reports of pain, biliary colic (waves of pain) Facial mask of pain; guarding behavior Autonomic responses (changes in BP, pulse) Self-focusing; narrowed focus	Relieve pain and promote rest. Maintain fluid and electrolyte balance prevent complications provide information about disease process, prognosis and treatment needs.	Assists in differentiating cause of pain, and provides information about disease progression/resolution, development of complications, and effectiveness of interventions. Severe pain not relieved by routine measures may indicate developing complications/need for further intervention. Bed rest in low-Fowler's position reduces intra-abdominal pressure; however, patient will naturally assume least	Pain Relieved Homeostasis achieved Complications prevented and minimized Disease process, prognosis and therapeutic regimen understood Plan in place to meet need after discharge.

		<p>painful position. Reduces irritation/dryness of the skin and itching sensation. Cool surroundings aid in minimizing dermal discomfort. Promotes rest, redirects attention, may enhance coping. Helpful in alleviating anxiety and refocusing attention, which can relieve pain</p>	
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Hepatitis

Pathophysiology

- Inflammation that spreads to the liver (hepatitis) can be caused by infection by viruses and toxic reactions to drugs and chemicals. Basic functional unit of the liver called lobul and the unit is unique because it has its own blood supply.
- Along with the development of inflammation in the liver, the normal pattern in the hepatic impaired. Disruption of the normal blood supply to the cells causes hepatic necrosis and damage to liver cells. After passing his time, the liver cells become damaged eliminated from the body by the immune system response and replaced by new cells of a healthy liver. Therefore, most clients who have hepatitis recovered with normal liver function.
- Inflammation of the liver due to viral invasion would lead to an increase in body temperature and stretching the liver capsule which lead to feelings of discomfort in the upper right abdominal quadrant. This is manifested by the presence of nausea and pain in the gut.
- Onset of jaundice because the liver parenchymal cell damage. Although the number billirubin that has not undergone conjugation, into the liver remained normal, but due to liver cell damage and intra-hepatic bile ductuli, then there is the difficulty of transporting billirubin in the liver.
- There was also a difficulty in terms of conjugation. As a result, billirubin imperfect through the ductus hepaticus issued, due to retention (due to cell damage excretion) and regurgitation in the ductuli, bile has not undergone conjugation (indirect bilirubin), or already experiencing the conjugation of bilirubin (direct bilirubin). So here jaundice arising mainly due to difficulties in transport, conjugation and excretion of bilirubin.
- Feces contain little stercobilin therefore pale stools (abolis). Because water-soluble conjugated bilirubin, the bilirubin can be excreted into the urine, causing urinary bilirubin and dark colored urine. Elevated levels of bilirubin can be accompanied by an increase in the conjugated bile salts in the blood which will cause itching in jaundice.

Signs & Symptoms

The initial phase of hepatitis is called the *acute phase*. The symptoms are like a mild flu, and may include:

- Diarrhea
- Fatigue
- Loss of appetite
- Mild fever
- Muscle or joint aches
- Nausea
- Slight abdominal pain
- Vomiting
- Weight loss

The acute phase is not usually dangerous, unless it develops into the fulminant or rapidly progressing form, which can lead to death.

As the patient gets worse, these symptoms may follow:

- Circulation problems (only toxic/drug-induced hepatitis)
- Dark urine
- Dizziness (only toxic/drug-induced hepatitis)
- Drowsiness (only toxic/drug-induced hepatitis)
- Enlarged spleen (only alcoholic hepatitis)
- Headache (only toxic/drug-induced hepatitis)
- Hives
- Itchy skin
- Light colored feces, the feces may contain pus
- Yellow skin, whites of eyes, tongue (jaundice)

Nursing Assessment	Nursing Intervention	Rationale	Goal
Fluid volume, risk for deficient related to excessive losses through vomiting and diarrhea.	Independent: · Monitor intake and output, compare with periodic weight. Note enteric losses such as vomiting and diarrhea. · Assess vital signs, peripheral pulses, capillary refill, skin turgor, and mucous membranes. · Check for ascites for edema formation. Measure abdominal girth as indicated. · Use small-gauge needles for injections, applying pressure for longer than usual after venipuncture. · Have patient use cotton or sponge	· Provides information about replacement need or effects of therapy. · Indication of circulating volume or perfusion. · Useful in monitoring progression/resolution of fluid shifts. · Reduces possibility of bleeding into tissues. · Avoids trauma and bleeding of gums. · Prothrombin levels are reduced and coagulation times prolonged when vitamin K absorption is altered in GI tract and synthesis of prothrombin is	Pain Relieved Homeostasis achieved Complications prevented and minimized Disease process , prognosis and therapeutic regimen understood Plan in place to meet need after discharge.

	swabs and mouth wash instead of tooth brush. · Observe for signs of bleeding such as hematuria, ecchymosis, oozing from gums. Collaborative: · Monitor laboratory values. · Administer antidiarrheal agents. · Provide IV fluids and electrolytes. · Administer Vitamin K as indicated.	decreased in affected liver. · Reflects hydration and identifies sodium retention or protein deficits, which may lead to edema formation. · Reduces fluid or electrolyte loss from GI tract. · Provides, fluid and electrolyte acute toxic shock state. · To increase clotting factor and decrease bleeding.	
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Pancreatitis

Pathophysiology

Pancreatitis is an inflammatory disease, which varies in severity from mild to severe. Factors determining the severity of pancreatitis are not known. It is generally believed that the earliest events in the evolution of acute pancreatitis lead to premature intra-acinar cell activation of digestive zymogens and that those enzymes, once activated cause acinar cell injury. Recent studies have suggested that the ultimate severity of resulting pancreatitis may be determined by events which occur subsequent to acinar cell injury. These include inflammatory cell recruitment and activation as well as the generation and release of cytokines and other chemical mediators of inflammation. Recently, we have undertaken studies to elucidate the role of various inflammatory agents in determining the severity of pancreatitis. Results from these ongoing studies indicate that substance P acting via neurokinin-1 (NK1) receptors, chemokines interacting with CCR1 receptors and platelet activating factor play an important pro-inflammatory role in regulating the severity of pancreatitis and associated lung injury. On the other hand, complement factor 5a (C5a) acts as an anti-inflammatory agent during the development of pancreatitis.

Signs & Symptoms

Signs and symptoms of pancreatitis vary if it is acute or chronic in nature, depending on what the client is having.

Signs and symptoms of acute pancreatitis include:

- Abdominal pain to the upper quadrants, radiates to the clients back and worsens after meals
- Nausea and vomiting
- Tenderness on the abdomen

Signs and symptoms of chronic pancreatitis include:

- Upper abdominal pain
- Indigestion

- Sudden weight loss
- Steatorrhea (oily, foul smelling stools)

Nursing Assessment	Nursing Intervention	Rationale	Goal
<p>Acute pain related to inflammation, edema, distention of the pancreas, and peritoneal irritation</p> <p>Ineffective breathing pattern related to severe pain, pulmonary infiltrates, pleural effusion, telecasts, and elevated diaphragm</p> <p>Imbalanced nutrition, less than body requirements, related to reduced food intake and increased metabolic demands</p> <p>Impaired skin integrity related to poor nutritional status, bed rest, multiple drains, and surgical wound</p>	<p>with held oral feedings</p> <p>the patient is maintained on parenteral fluids and electrolytes</p> <p>Nasogastric suction</p> <p>frequent oral hygiene and care</p> <p>Maintain bed rest</p> <p>If experiencing severe pain, report to physician</p> <p>Provide frequent and repeated but simple explanations about the need for withholding fluids, maintenance of gastric suction, and bed rest.</p>	<p>decrease the formation of secretin to restore and maintain fluid balance</p> <p>to relieve n/v or to treat abdominal distention and paralyticileus</p> <p>to decrease discomfort from then nasogastric tube and to relieve dryness of the mouth</p> <p>to decrease the metabolic reate and reduce the secretion of pancreatic and gastric enzymes</p> <p>the client may be experiencing hemorrhage of the pancreas or the dose of the analgesic maybe inadequate.</p> <p>The patient often has clouded sensorium because of severe pain, fluid and electrolyte disturbances, and hypoxia</p>	<p>Relief of pain and discomfort</p> <p>Improved respiratory function</p> <p>Improved nutritional status</p> <p>Maintenance of skin integrity</p> <p>Prevent complication</p>

kidney (RENAL)

ARF (Acute Renal Failure)

Pathophysiology

The interaction of tubular and vascular events result in ARF. The primary cause of ATN is ischemia. Ischemia for more than two hours results in severe and irreversible damage to the kidney tubules. Significant reduction in glomular filtration rate (GFR) is a result of (1) ischemia, (2) activation of the renin-angiotensin system, and (3) tubular obstruction by cellular debris. As nephrotoxins damage the tubular cells and these cells are lost through necrosis, the tubules become more permeable. This results in filtrate absorption and a reduction in the nephrons ability to eliminate waste.

The clinical course of ARF is characterized by the following three phases:

Phase 1. Onset

ARF begins with the underlying clinical condition leading to tubular necrosis, for example hemorrhage, which reduces blood volume and renal perfusion. If adequate treatment is provided in this phase then the individual's prognosis is good.

Phase 2. Maintenance

A persistent decrease in GFR and tubular necrosis characterizes this phase. Endothelial cell necrosis and sloughing lead to tubular obstruction and increased tubular permeability. Because of this, oliguria is often present during the beginning of this phase. Efficient elimination of metabolic waste, water, electrolytes, and acids from the body cannot be performed by the kidney during this phase. Therefore, azotemia, fluid retention, electrolyte imbalance and metabolic acidosis occurs. The patient is at risk for heart failure and pulmonary edema during this phase because of the salt and water retention. Immune function is impaired and the patient may be anemic because of the suppressed erythropoietin secretion by the kidney and toxin-related shorter RBC life.

Phase 3. Recovery

Renal function of the kidney improves quickly the first five to twenty-five days of this phase. It begins with the recovery of the GFR and tubular function to such an extent that BUN and serum creatinine stabilize. Improvement in renal function may continue for up to a year as more and more nephrons regain function.

Signs & Symptoms

- Dizziness
- Dry mouth
- Low blood pressure (hypotension)
- Rapid heart rate
- Slack skin
- Thirst
- Weight loss

Nursing Assessment	Nursing Intervention	Rationale	Goal
Fluid Volume excess related to Compromised regulatory mechanism (renal failure)	1. Record accurate intake and output (I&O). Include "hidden" fluids such as IV antibiotic additives, liquid medications, ice chips, frozen treats. Measure gastrointestinal (GI) losses and estimate insensible losses, e.g., diaphoresis.	1. Low output (less than 400 mL/24 hr) may be first indicator of acute failure, especially in a high-risk patient. Accurate I&O is necessary for determining renal function and fluid replacement needs and reducing risk of fluid overload. Note: Hypervolemia occurs in the anuric phase of ARF.	Homeostasis achieved. Complications prevented/minimized. Dealing realistically with current situation. Disease process/prognosis and therapeutic regimen understood. Plan in place to meet needs after discharge.

	<p>2. Monitor urine specific gravity.</p> <p>3. Weigh daily at same time of day, on same scale, with same equipment and clothing.</p> <p>4. Assess skin, face, dependent areas for edema. Evaluate degree of edema (on scale of +1–+4).</p> <p>5. Monitor heart rate (HR), BP, and JVD/CVP.</p> <p>6. Auscultate lung and heart sounds.</p> <p>7. Assess level of consciousness; investigate changes in mentation, presence of restlessness.</p> <p>8. Plan oral fluid replacement with patient, within multiple restrictions. Intersperse desired beverages throughout 24 hr. Vary offerings, e.g., hot, cold, frozen.</p>	<p>2. Measures the kidney's ability to concentrate urine. In intrarenal failure, specific gravity is usually equal to/less than 1.010, indicating loss of ability to concentrate the urine.</p> <p>3. Daily body weight is best monitor of fluid status. A weight gain of more than 0.5 kg/day suggests fluid retention.</p> <p>4. Edema occurs primarily in dependent tissues of the body, e.g., hands, feet, lumbosacral area. Patient can gain up to 10 lb (4.5 kg) of fluid before pitting edema is detected. Periorbital edema may be a presenting sign of this fluid shift because these fragile tissues are easily distended by even minimal fluid accumulation.</p> <p>5. Tachycardia and hypertension can occur because of (1) failure of the kidneys to excrete urine, (2) excessive fluid resuscitation during efforts to treat hypovolemia/hypotension or convert oliguric phase of renal failure, and/or (3) changes in the renin-angiotensin system. Note: Invasive monitoring may be needed for assessing intravascular volume, especially in patients with poor cardiac function.</p> <p>6. Fluid overload may lead to pulmonary edema and HF evidenced by development of adventitious breath sounds, extra heart sounds. (Refer to ND: Cardiac Output, risk for decreased, following.)</p> <p>7. May reflect fluid shifts, accumulation of toxins, acidosis, electrolyte</p>	
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		imbalances, or developing hypoxia. 8. Helps avoid periods without fluids, minimizes boredom of limited choices, and reduces sense of deprivation and thirst.	
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CRF (Chronic Renal Failure)

Pathophysiology

- Regardless of the primary cause of nephron loss, some usually survive or are less severely damaged
- These nephrons then adapt and enlarge, and clearance per nephron markedly increases.
- If the initiating process is diffuse, sudden, and severe, such as in some patients with rapidly progressive glomerulonephritis (crescentic glomerulonephritis), acute or subacute renal failure may ensue with the rapid development of ESRD.
- In most patients, however, disease progression is more gradual and nephron adaptation is possible.
- Focal glomerulosclerosis develops in these glomeruli, and they eventually become non-functional.
- At the same time that focal glomerulosclerosis develops, proteinuria markedly increases and systemic hypertension worsens.
- This process of nephron adaptation has been termed the "**final common path.**"
- Adapted nephrons enhance the ability of the kidney to postpone uremia, but ultimately the adaptation process leads to the demise of these nephrons.
- Adapted nephrons have not only an enhanced GFR but also enhanced tubular functions in terms of, for example, potassium and proton secretion.

Signs & Symptoms

Chronic renal failure can be present for many years before you notice any symptoms. If your doctor suspects that you may be likely to develop renal failure, he or she will probably catch it early by conducting regular blood and urine tests. If regular monitoring isn't done, the symptoms may not be detected until the kidneys have already been damaged. Some of the symptoms - such as fatigue - may have been present for some time, but can come on so gradually that they aren't noticed or attributed to kidney failure.

Some signs of chronic renal failure are more obvious than others. These are:

- increased urination, especially at night
- decreased urination
- blood in the urine (not a common symptom of chronic renal failure)
- urine that is cloudy or tea-colored

Other symptoms aren't as obvious, but are a direct result of the kidneys' inability to eliminate waste and excess fluid from the body:

- puffy eyes, hands, and feet (called *edema*)
- high blood pressure
- fatigue
- shortness of breath
- loss of appetite
- nausea and vomiting (this is a common symptom)
- thirst
- bad taste in the mouth or bad breath
- weight loss
- generalized, persistent itchy skin
- muscle twitching or cramping
- a yellowish-brown tint to the skin

As the kidney failure gets worse and the toxins continue to build up in the body, seizures and mental confusion can result.

Nursing Assessment	Nursing Intervention	Rationale	Goal
Cardiac Output, risk for decreased related to Fluid imbalances affecting circulating volume, myocardial workload, and systemic vascular resistance (SVR), Alterations in rate, rhythm, cardiac conduction (electrolyte imbalances, hypoxia), Accumulation of toxins (urea), soft-tissue calcification (deposition of calcium phosphate).	Independent Auscultate heart and lung sounds. Evaluate presence of peripheral edema/vascular congestion and reports of dyspnea. Assess presence/degree of hypertension: monitor BP; note postural changes, e.g., sitting, lying, standing. Investigate reports of chest pain, noting location, radiation, severity (0–10 scale), and whether or not it is intensified by deep inspiration and supine position. Evaluate heart sounds (note friction rub), BP, peripheral pulses, capillary refill, vascular congestion, temperature, and sensorium/mentation. Assess activity level, response to activity. Collaborative Monitor laboratory/diagnostic studies, e.g.: Electrolytes (potassium, sodium,	S3/S4 heart sounds with muffled tones, tachycardia, irregular heart rate, tachypnea, dyspnea, crackles, wheezes, and edema/jugular distension suggest HF. Significant hypertension can occur because of disturbances in the renin-angiotensin-aldosterone system (caused by renal dysfunction). Although hypertension is common, orthostatic hypotension may occur because of intravascular fluid deficit, response to effects of antihypertensive medications, or uremic pericardial tamponade. Although hypertension and chronic HF may cause MI, approximately half of CRF patients on dialysis develop pericarditis, potentiating risk of	Fluid/electrolyte balance stabilized. Complications prevented/minimized. Disease process/prognosis and therapeutic regimen understood. Dealing realistically with situation; initiating necessary lifestyle changes. Plan in place to meet needs after discharge.

	<p>calcium, magnesium), BUN/Cr; Administer antihypertensive drugs, e.g., prazosin (Minipress), captopril (Capoten), clonidine (Catapres), hydralazine (Apresoline). Prepare for dialysis. Assist with pericardiocentesis as indicated.</p>	<p>pericardial effusion/tamponade. Presence of sudden hypotension, paradoxical pulse, narrow pulse pressure, diminished/absent peripheral pulses, marked jugular distension, pallor, and a rapid mental deterioration indicate tamponade, which is a medical emergency. Weakness can be attributed to HF and anemia. Imbalances can alter electrical conduction and cardiac function; Do Chest x-rays. Useful in identifying developing cardiac failure or soft-tissue calcification. Reduces systemic vascular resistance and/or renin release to decrease myocardial workload and aid in prevention of HF and/or MI. Reduction of uremic toxins and correction of electrolyte imbalances and fluid overload may limit/prevent cardiac manifestations, including hypertension and pericardial effusion. Accumulation of fluid within pericardial sac can compromise cardiac filling and myocardial contractility, impairing cardiac output and potentiating risk of cardiac arrest</p>	
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Nephrotic Syndrome

Pathophysiology

Proteinuria occurs because of changes to capillary endothelial cells, the glomerular basement membrane (GBM), or podocytes, which normally filter serum protein selectively by size and charge.

The mechanism of damage to these structures is unknown in primary and secondary glomerular diseases, but evidence suggests that T cells may up-regulate a circulating permeability factor or down-regulate an inhibitor of permeability factor in response to unidentified immunogens and cytokines. Other possible factors include hereditary defects in proteins that are integral to the slit diaphragms of the glomeruli, activation of complement leading to damage of the glomerular epithelial cells and loss of the negatively charged groups attached to proteins of the GBM and glomerular epithelial cells.

Signs & Symptoms

- Hypoalbuminemia (low level of albumin in the blood)
- Edema (swelling)
- Hypercholesterolemia (high level of cholesterol in the blood)

Nursing Assessment	Nursing Intervention	Rationale	Goal
Excess fluid volume related to compromised regulatory mechanism with changes in hydrostatic or oncotic vascular pressure and increased activation of the renninangiotensinaldosterone system.	INDEPENDENT: <input type="checkbox"/> Record accurate intake and output of the patient. <input type="checkbox"/> Monitor urine specific gravity. <input type="checkbox"/> Weigh daily at same time of the day, on same scale, with same equipment and clothing. <input type="checkbox"/> Assess skin, face, dependent areas of edema. Monitor heart rate and blood pressure.	Accurate Intake and output is necessary for determining renal function and fluid Replacement needs and reducing risk of fluid overload. <input type="checkbox"/> Measures the kidney's ability to concentrate urine. <input type="checkbox"/> Daily body weight is the best monitor of fluid status. A weight gain of more than 0.5 kg/day suggest fluid retention. <input type="checkbox"/> Edema occurs primarily in dependent tissues of the body. It will serve as parameter the severity of fluid excess.	After Nursing interventions, the patient was able to display stable weight, vital signs within patient's normal range, and nearly absence of edema.

	<p><input type="checkbox"/> Assess level of consciousness; Investigate changes in mentation, presence of restlessness.</p> <p>COLLABORATIVE:</p> <p><input type="checkbox"/> Monitor laboratory and diagnostic studies.</p> <p><input type="checkbox"/> Administer diuretics as prescribed.</p>	<p><input type="checkbox"/> Tachycardia and hypertension can occur because of failure of the kidneys to excrete urine.</p> <p><input type="checkbox"/> May reflect fluid shifts and electrolyte imbalances.</p> <p><input type="checkbox"/> Provide assessment of the progression and management of the dysfunction.</p> <p><input type="checkbox"/> To promote adequate urine volume that aids in prevention of further edema.</p>	
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Kidney Stone (Calculi)

Pathophysiology

- Kidney stones (renal lithiasis) are small, hard deposits that form inside your kidneys. The stones are made of mineral and acid salts. Kidney stones have many causes and can affect any part of your urinary tract — from your kidneys to your bladder. Often, stones form when the urine becomes concentrated, allowing minerals to crystallize and stick together.
- Passing kidney stones can be quite painful, but the stones usually cause no permanent damage. Depending on your situation, you may need nothing more than to take pain medication and drink lots of water to pass a kidney stone. In other instances, surgery may be needed. Your doctor may recommend preventive treatment to reduce your risk of recurrent kidney stones if you're at increased risk of developing them again.

Signs & Symptoms

- Severe pain in the side and back, below the ribs
- Pain that spreads to the lower abdomen and groin
- Pain that comes in waves and fluctuates in intensity
- Pain on urination
- Pink, red or brown urine
- Cloudy or foul-smelling urine
- Nausea and vomiting
- Persistent urge to urinate

- Urinating more often than usual
- Fever and chills if an infection is present

Nursing Assessment	Nursing Intervention	Rationale	Goal
<p>Acute pain related to inflammation, obstruction, and abrasion of urinary tract by migration of stones. Altered urinary elimination.</p>	<p>Document the pain in terms of location, duration, intensity (1-10 pain scale), and radiation. Also, observe for nonverbal cues like BP and pulse rate elevation, restlessness, crying or moaning.</p> <p>Encourage to verbalize pain noting also for the pain threshold of the client; let client explain how the pain occur or for any changes in characteristics.</p> <p>Educate and encourage client in diversional activities like focused breathing and guided imagery.</p> <p>Provide scheduled resting periods for client and also provide a peaceful environment.</p> <p>Assist client in daily ambulation and encourage increasing fluid intake of at least 3 L per day as tolerated.</p> <p>Instruct client to report for persistent or increased abdominal pain.</p>	<p>This would aid you in assessing and evaluating the effectiveness of treatment; it can also reflect the progress of calculi movement because a flank pain means the stones are still in the kidney area and upper ureter; severe pain may result to severe anxiety and restlessness.</p> <p>It will provide an avenue for timely administration of pain medication.</p> <p>It will help client in diverting pain and coping with disease condition.</p> <p>It can promote relaxation and reduces muscle tension.</p> <p>Supine position could be worse for renal colic while an increased fluid intake promotes the passing of the stone and prevents further stone formation.</p> <p>Complete obstruction of the ureter can cause the perforation of urine into the perirenal space making it a surgical</p>	<p>Pain relieved. Homeostasis achieved. Complications prevented/minimized. Disease process, prognosis, and therapeutic regimen understood. Plan in place to meet needs after discharge</p>

	<p>Administer medications like narcotics, antispasmodic and corticosteroid as prescribed by the physician.</p> <p>If indicated, a warm compress may be applied to the back.</p> <p>Insert and maintain the patency of urinary catheter.</p>	<p>emergency.</p> <p>Narcotics are given during acute periods of pain; antispasmodic is used to decrease spasm preventing colic and pain; corticosteroid is given to reduce edema, facilitating the movement of stone.</p> <p>It reduces muscle tension and spasms.</p> <p>To determine and prevent urinary retention and it can also help in lessening renal pressure and infection.</p>	
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Glomerulonephritis

Pathophysiology

The initial reaction is usually either an upper respiratory infection or skin infection due to group A beta-hemolytic streptococcus. This leads to the formation of an antigen-antibody reaction. It is followed by the release of a membrane-like material from the organism into the body's circulation. Antibodies produced to fight the invading organism also react against the glomerular tissue, thus forming immune complexes. The immune complexes become trapped in the glomerular loop and cause an inflammatory reaction in the affected glomeruli. Changes in the glomerular capillaries reduce the amount of the glomerular filtrate, thereby allowing passage of blood cells and protein into the infiltrate, and reducing the amount of sodium and water that is passed into the tubules for reabsorption. This affects the vascular tone and permeability of the kidney, resulting to tissue injury.

Signs & Symptoms

Signs and symptoms of glomerulonephritis may depend on whether you have the acute or chronic form, and the cause. Your first indication that something is wrong may come from symptoms or from the results of a routine urinalysis. Signs and symptoms may include:

- Pink or cola-colored urine from red blood cells in your urine (hematuria)
- Foamy urine due to excess protein (proteinuria)
- High blood pressure (hypertension)
- Fluid retention (edema) with swelling evident in your face, hands, feet and abdomen
- Fatigue from anemia or kidney failure

Nursing Assessment	Nursing Intervention / Rationale	Goal
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<ul style="list-style-type: none"> • Light microscopy: Enlarged glomeruli with mesangial proliferation and exudation of neutrophils • Immune of fluorescent microscopy: Granular pattern of immunoglobulin deposition • Electron microscopy: reveals electron dense humps (immunecomplex) on the epithelial side of the glomerular basement membrane 	<ul style="list-style-type: none"> • Provide best rest during the acute phase. • Perform passive range of motion exercises for the patient on bed rest. • Allow the patient to resume normal activities gradually as symptoms subside. • Consult the dietician about a diet high in calories and low in protein, sodium, potassium, and fluids. • Protect the debilitated patient against secondary infection by providing good nutrition and hygienic technique and preventing contact with infected people. • Check the patient's vital signs and electrolyte values. • Monitor intake and output and daily weight. • Report peripheral edema or the formation of ascites. • Explain to the patient taking diuretics that he may experience orthostatic hypotension and dizziness when he changes positions quickly. • Provide emotional support for the patient and his family. • If the patient is scheduled for dialysis, explain the procedure fully. 	<ul style="list-style-type: none"> • Pain relieved. • Homeostasis achieved. • Complications prevented/minimized. • Disease process, prognosis, and therapeutic regimen understood. • Plan in place to meet needs after discharge
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Transurethral Resection of Prostate (TURP)

Pathophysiology

TURP (Transurethral Resection of the Prostate) is the most common procedure used to treat BPH. It can be carried out through endoscopy. The surgical and optical instrument is introduced directly through the urethra to the prostate, which can then be viewed directly. The gland is removed in small chips with an electrical cutting loop. This procedure, which requires no incision, may be used for glands of varying size and is ideal for patients who have small glands and for those who are considered poor surgical risks. Newer technology uses bipolar electrosurgery and reduces the risk of TUR syndrome (hyponatremia, hypovolemia). TURP usually requires an overnight hospital stay. Urethral strictures are more frequent than with (non-trans-urethral) procedures, and repeated procedures may be necessary because the residual prostatic tissue grows back.

TURP rarely causes erectile dysfunction, but may trigger retrograde ejaculation because removal of the prostatic tissue at the bladder neck can cause seminal fluid to flow backward into the bladder rather than forward through the urethra during ejaculation.

Signs & Symptoms

- Urgency of urination
- Frequency of urination
- Abdominal straining
- Nocturia
- Impairment of size and force of stream
- Intermittent hesitancy
- Incomplete bladder emptying
- Terminal dribbling
- Dysuria
- Eventual renal failure from urinary obstruction

Nursing Assessment	Nursing Intervention	Rationale	Goal
Impaired Urinary Elimination urinary retention hematuria fever	<p>Monitor urinary elimination, including consistency, odor, volume, and color.</p> <p>Help the client select appropriate incontinence garment or pad for short-term management while more definitive treatment is designed.</p> <p>Instruct Patient to limit fluids for 2 to 3 hours before bedtime.</p> <p>Instruct him to drink a minimum of 1,500 mL (six 8-ounce glasses) fluids per day.</p> <p>Limit ingestion of bladder irritants (e.g., colas, coffee, tea, and chocolate).</p> <p>Instruct Patient or a family member to record urinary output.</p> <p>Catheterize for</p>	<p>These parameters help determine adequacy of urinary tract function.</p> <p>Appropriate undergarments can help diminish the embarrassing aspects of urinary incontinence.</p> <p>Decreased fluid intake several hours before bedtime will decrease the incidence of urinary retention and overflow incontinence, and promote rest.</p> <p>Increased fluids during the day will increase urinary output and discourage bacterial growth.</p> <p>Alcohol, coffee, and tea have a natural diuretic effect and are bladder irritants.</p> <p>Serves as an indicator of urinary tract and renal function and of fluid balance.</p>	<p>Able to start and stop stream</p> <p>Empties bladder completely</p> <p>Description of self-care responsibilities for ongoing care</p> <p>Description of self-monitoring techniques.</p> <p>Refrain from alcoholic beverages.</p> <p>Avoid sexual activities for a few weeks.</p> <p>Avoid driving a car for a week or more.</p> <p>Keep domestic activities to a minimum.</p> <p>Avoid weight lifting or strenuous exercise.</p> <p>Check their temperature and report any fever to the physician.</p> <p>Practice good hygiene, especially of the hands and penis.</p> <p>Drink plenty of liquids.</p>

	<p>residual urine, as appropriate.</p> <p>Implement intermittent catheterization, as appropriate</p> <p>Provide enough time for bladder emptying (10 minutes).</p> <p>Instruct the client in ways to avoid constipation or stool impaction.</p>	<p>An enlarged prostate compresses the urethra so that urine is retained. Checking for residual urine provides information about bladder emptying.</p> <p>Helps maintain tonicity of the bladder muscle by preventing over distention and providing for complete emptying.</p> <p>In addition to the effect of an enlarged prostate on the bladder, stress or anxiety can inhibit relaxation of the urinary sphincter. Sufficient time should be allowed for micturition.</p> <p>Impacted stool may place pressure on the bladder outlet, causing urinary retention.</p>	
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UTI (urinary tract infection)

Pathophysiology

A urinary tract infection (UTI) may occur in the bladder, where it is called cystitis, or in the urethra, where it is called urethritis. Upper tract infection results in pyelonephritis. Most UTIs result from ascending infections by bacteria that have entered through the urinary meatus but some may be caused by hematogenous spread. UTIs are much common in females because the shorter female urethra makes them more vulnerable to entry of organisms from surrounding structures (vagina, periurethral glands, and rectum).

Signs & Symptoms

Symptoms depend on age of person and where the UTI is located .

- Symptoms of urethritis often include:
 - Burning sensation at the start of urination
- Symptoms of cystitis often include:

- Burning sensation in the middle of urination
 - Fever
 - Lower abdominal pain
 - Funny smell, color, or appearance (cloudy, dark, blood tinged) of urine
- Symptoms of Pyelonephritis often include:
 - Pain in back, flanks, or abdomen
 - Fever
 - Nausea
 - Vomiting
- Other symptoms of UTI's:
 - Uncomfortable pressure above pubic bone
 - Fullness in rectum (in men only)
 - Small amount of urine, despite urge to urinate
 - Irritability (in children only)
 - Abnormal eating (in children only)

Nursing Assessment	Nursing Intervention	Rationale	Goal
Hydration status suprapubic tenderness – may be mild to moderate flank pain – if present refer or consult suggests upper UTI fever, rigor, chills – if present refer or consult – suggests upper UTI	<input type="checkbox"/> Assess pain, noting location, intensity (scale of 0 – 10), duration. <input type="checkbox"/> Encourage increased fluid intake. <input type="checkbox"/> Investigate report of bladder fullness. <input type="checkbox"/> Observe for changes in mental status, behavior or level of consciousness. <input type="checkbox"/> Provide comfort measure like back rub, helping patient assume position of comfort. Suggest use of relaxation technique and deep breathing exercises. <input type="checkbox"/> Encourage use of sitz baths, warm soaks to the perineum. Collaborative: <input type="checkbox"/> Administer antibacterial as prescribed	how to take medication, proper dosing, expected side effects, and follow-up increasing fluid intake to 8-10 glasses per day methods for cleaning sex toys avoiding sharing sex toys avoiding douching avoiding bubble baths returning to the clinic if fever develops or symptoms do not improve in 48-72	relieve symptoms prevent complications and ascending infection eradicate infection

		hours	
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Benign Prostate Hypertrophy (BPH)

Pathophysiology

- As males age, production of androgenic hormones decreases, causing an imbalance in androgen and estrogen levels and high levels of dihydrotestosterone, the main prostatic intracellular androgen.
- Other causes of Benign prostatic hyperplasia (BPH) include:
 - Neoplasm
 - Arteriosclerosis
 - Inflammation
 - Metabolic Imbalance
 - Nutritional disturbances.
- Complications for Benign prostatic hyperplasia (BPH)
 - Urinary stasis, urinary tract infection (UTI), or
 - Renal calculi
 - Bladder wall trabeculation
 - Detrusor muscle hypertrophy
 - Bladder diverticula and saccules
 - Urethral stenosis
 - Hydronephrosis
 - Paradoxical (overflow) incontinence
 - Acute or chronic renal failure
 - Acute postobstructive diuresis.

Signs & Symptoms

Symptoms include a slow flow of urine, the need to urinate urgently and difficulty starting the urinary stream.

Nursing Assessment	Nursing Intervention	Rationale	Goal
Acute pain. May related to mucosal irritation such as bladder distention, renal colic, urinary infection and radiation therapy	<p>Asses pain, nothing location, intensity</p> <p>Tape drainage tube to high and catheter to the abdomen, if traction not required.</p> <p>Provide comfort measure, such as backrub, helping patient assume position of comfort. Suggest use of</p>	<p>Provide information to aid in determine choice and effectiveness of interventions.</p> <p>Prevents accidental dislodging of catheter with attendant urethral trauma.</p> <p>Promotes relaxation, refocuses attention, and may enhance coping abilities</p>	The patient will able to report pain relieved or controlled, appear relaxed and be able to sleep and rest appropriately.

	relaxation and deep creating exercises and divisional activities. Encourage use of sitz baths and warm soak to perineum.	Promotes muscle relaxation	
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