

A COMPLETE GUIDE FOR NURSING MEDSURG CLASS

Medical Surgical Nursing Bundle



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| P: Pathophysiology | Dx: Diagnosis |
|----------------------|----------------------|
| R: Risk Factors | N: NursingManagement |
| S/Sx: Signs+Symptoms | C: Complications |

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STROKE

P: Neurological Deficit caused by a lack of blood flow to the brain
 S/Sx: One side weakness, Facial Drooping, Confusion, Slurred Speech
 Dx: CT scan or MRI confirms and identifies type of stroke which determines treatment
 R: Atherosclerosis, hypertension, diabetes, stress obesity, oral contraceptives, anticoagulation therapy
 N: - Airway is priority - Monitor VS, LOC, pupils + reflexes - Position client on side to prevent aspiration

Types of Strokes

Ischemic: An obstruction of a blood vessel - 87% of stroke cases Hemorrhagic: A weakened blood vessel ruptures (Risk Factor = Hypertension) TIA (transient ischemic attack): Temporary clot that resolves on its own (A warning sign for stroke)

MULTIPLE SCLEROSIS

P: A central nervous system disease characterized by demyelination of axons

S/Sx: Fatigue, weakness, ataxia, vertigo, tremors, Blurred vision, emotional changes, \$\sensation, Bladder + bowel disturbances, +Babinski reflex

Dx: Requires extensive neurological testing over many years of a slow onset of disease

N: Provide energy preservation measures, encourage independence while providing safety, Monitor for urinary + bowel dysfunction

C: Falls, psychological problems, decreased mobility

TRAUMATIC HEAD INJURY

P: A trauma or blunt force hits the skull causing damage to the brain

S/Sx: Confusion, agitation, visible head injury, sleepiness blown pupils

N: - Assess for neurological changes or change in the level of consciousness, monitor V/S, assess pt. for headache, nausea/vomiting, check for CSF drainage, Assess pupil size

C: Cerebral bleed, hematoma, \uparrow intracranial pressure, infection, seizure, \uparrow CO₂ levels, permanent damage



MENINGITIS

P: Inflammation of the arachnoid + pia mater of the CNS. Usually caused by bacteria or a virus. CSF is tested for the pathogen and used to determine the treatment

 $S/Sx: \downarrow LOC, Red macular rash, pain with neck flexion$

N: Monitor for seizures. Assess cranial nerves

SEIZURES

P: Abnormal, sudden electrical activity in the brain S/SX: Uncontrollable involuntary muscle movements (convulsions) or Gaze off into the distance with no response to stimuli. May lose consciousness for seconds or minutes. Usually, the person will not recall the events leading up to it but they may remember experiencing an aura beforehand. N: Assess seizure history. Note Time + duration. Prevent injury, but do not restrain. Monitor behavior before + after episode. <u>Turn Pt. on side at end</u> of seizure to <u>drain secretions</u> C: Status epilepticus - epileptic spasms without any rest periods which can result in brain damage R: Genetic Inheritance, Brain trauma, tumors, toxicity, metabolic disorders or infection

PARKINSON'S DISEASE

P: Decreased dopamine levels in the brain cause neurological + musculoskeletal Sx

S/Sx: Blank facial expression, Slow, monotonous, slurred speech, Rigidity and tremor of extremities and head, Forward tilt to posture, Reduced arm swinging, Short, shuffling gait

N: Monitor swallowing activity + neuro activity. Assist w/ambulation. Promote PT + OT to preserve function, Increase fluid intake to 2L/day

C: Falls, Self-care deficits, depression, constipation and poor posture

| Frontal lobeGive simple instructions, re-orientate as neededTemporal LobeSpeak clearly due to impaired hearingOccipital LobeAssist with ADL due to | Frontal lobeGive simple instructions, re-orientate as neededTemporal LobeSpeak clearly due to impaired hearingOccipital LobeAssist with ADL due to visual disturbances | Frontal lobeGive simple instructions, re-orientate as neededTemporal LobeSpeak clearly due to impaired hearingOccipital LobeAssist with ADL due to visual disturbancesBrain StemMonitor vital signs | Injured Brain Area | Nursing Intervention |
|--|---|--|--------------------|--|
| Temporal Lobe Speak clearly due to impaired hearing Occipital Lobe Assist with ADL due to | Temporal Lobe Speak clearly due to impaired hearing Occipital Lobe Assist with ADL due to visual disturbances | Temporal Lobe Speak clearly due to impaired hearing Occipital Lobe Assist with ADL due to visual disturbances Brain Stem Monitor vital signs | Frontal lobe | Give simple instructions, |
| Temporal Lobe Speak clearly due to impaired hearing Occipital Lobe Assist with ADL due to | Temporal Lobe Speak clearly due to impaired hearing Occipital Lobe Assist with ADL due to visual disturbances | Temporal LobeSpeak clearly due to impaired hearingOccipital LobeAssist with ADL due to visual disturbancesBrain StemMonitor vital signs | | re-orientate as needed |
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| Brain Stem Monitor vital signs | | | Parietal Lobe | Provide simple, one-step |
| Brain Stem Monitor vital signs Parietal Lobe Provide simple, one-step | Parietal Lobe Provide simple, one-step | Parietal Lobe Provide simple, one-step | | instructions |
| Brain StemMonitor vital signsParietal LobeProvide simple, one-step instructions | Parietal Lobe Provide simple, one-step instructions | Parietal Lobe Provide simple, one-step instructions | Cerebellum | Assist with walking |

Skull Fracture

CAUSES OF SKULL FRACTURES:

- Motor Vehicle Collision
- Falls
- Fire arms related injuries
- Assaults
- Sport related Injuries
- -Recreational Accidents
- War related injuries

Death Can Occur at 3 points in time after injury:

- 1- Immediately After
- 2-Within 2 hours after
- 3- Three Weeks after injury

Scalp Lacerations: Highly Vascular/High Risk of Blood Loss

TYPES OF SKULL FRACTURES

Simple (linear) fracture: is a break in the continuity of the bone.

Comminuted skull fracture: a splintered or multiple fracture line.

Depressed skull fractures: occur when the bones of the skull are forcefully displaced downward.

Basal skull fracture: A fracture of the base of the skull.

It allows CSF to leak from the nose and ears

Signs of Basilar Skull: Battle's Sign and Raccoon Eyes

TESTING FOR CSF ≥CEREBROSPINAL FLUID€

Dextrostix or Test-Tape Strips

Used to detect <u>glucose</u> found in CSF, however it is inaccurate if blood is in the sample as there is glucose in the blood

Halo's Sign

Allow drainage to leak onto a white gauze pad. Within a few minutes, blood should gather in the center and CSF will create a yellow ring around the blood





Glasgow Coma Scale

| | Spontaneous | 4 |
|-----|-------------------------|---|
| ш | To Voice | 3 |
| EY | To Pain | 2 |
| | NONE | 1 |
| | Oriented | 5 |
| ٩L | Confused | 4 |
| RB/ | Inappropiate Words | 3 |
| VE | Incomprehensible sounds | 2 |
| | NONE | 1 |
| | Obey command | 6 |
| | Localized Pain | 5 |
| Log | Withdraws | 4 |
| MO | Flexion | 3 |
| | Extension | 2 |
| | NONE | 1 |

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Deep Coma: 3 Comatose: ≤8 Normal: 15

Spinal Cord Injury

PRIMARY

Initial mechanical disruption of axons as a result of stretch or laceration

Types of Injuries:

- Cord Compression (r/+ bone displacement)
- Interruption of blood flow
- Traction from pulling
- Penetrating trauma (gun shot, stabbing)

SECONDARY

Ongoing, progressive injury that occurs after primary injury

Complete cord damage r/t auto destruction which occurs during weeks after initial injury

- Usually starts within 24hrs of injury

- Cannot be diagnosed sooner than 72 hrs. after injury

MECHANISM OF INJURY

Flexion Hyperflexion Flexion-Rotation Extension-rotation Compression

Most unstable because the ligaments structures that stabilizes the spine are torn

aka - Quadriplegia Tetraplegia: All 4 extremities are paralyzed Paraplegia: Loss of function of lower extremities

CLASSIFICATION

LEVEL OF INJURY

Skeletal Level

- Cervical
- Thoracic
- Lumbar

Neurological Level: The lowest segment of spinal cord with normal sensory and motor function on both sides of the body

DEGREE OF INJURY

Complete Cord Involvement: -Total loss of function below the level of injury

Incomplete Cord Involvement: -Mixed loss of voluntary and sensory functions, leaves some tracts intact











rotation

neutral

flexion

extension

lateral bending

6



RESPIRATORY

CA or \uparrow : Loss of system function mechanical ventilation needed \downarrow CA: diaphragmatic breathing if phrenic nerve if functioning (Hypoventilation)

CARDIOVASCULAR

TG or ↑: Decrease CNS – Peripheral vasodilation – Bradycardia, Hypotension and Hypovolemia – Decreased Cardiac Output

INTEGUMENTARY

Skin breakdown/pressure ulcers

Poikilothermia r/t inability to shiver or sweat below the point of injury

VTE common in 1st 3 months. DVT (difficult to diagnose due to decrease pain sensation)

T5 or \uparrow : Paralytic ileus, gastric distention (may need gastric suctioning), intraabdominal bleeding (may be hard to detect due to decreased pain sensation)

G

URINARY

Retention (If shock occurs)

AMERICAN SPINAL INJURY ASSOCIATION "ASIA" IMPAIRMENT SCALE

A- Complete: No motor or sensory function is preserved in the sacral segment S4-5

B- Incomplete: Sensory but not motor function is preserved below the neurologic level and includes the sacral segment

C- Incomplete: Motor function is preserved below the neurologic level, and more than half of key muscles below the neurologic level have muscle grade of less than 3

D-Incomplete: Motor function is preserved below the neurologic level, and at least half of the key muscles below the neurologic level have a muscle grade of >3.

E- NORMAL: Motor and sensory function are normal.

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INCOMPLETE SPINAL CORD SYNDROME Anterior Cord Syndrome Central Cord Syndrome Damage to the middle of spinal cord Damage to anterior spinal artery. Results in compromised blood flow to anterior spinal Typical occurs in cervical area Motor Weakness and sensory loss in both upper and cord lower extremities Typically caused by hyperflexion injury. Motor paralysis and loss of pain and temp sensation More common in upper extremities Posterior Cord Syndrome Lateral Cord Syndrome Damage to posterior spinal cord Brown-Sequard Syndrome RARE! Damage to half the spinal cord Loss of proprioception Typically results from penetrating trauma Ipsilateral (same side) loss of motor function and position and vibratory sense, vasomotor paralysis. Contralateral (opposite side) loss of pain and temp sensation below level of injury Cauda Equina Syndrome Conus Medullaris Syndrome Damage to conus (lowest part of the spinal cord) Damage to cauda equina (lumbar and sacral nerve Flaccid paralysis of lower limbs and areflexic bladder roots) and bowel Flaccid paralysis of lower limbs and areflexic bladder and bowel

IMMOBILIZATION

GOAL IS TO MAINTAIN NEUTRAL POSITION AND TO PREVENT LATERAL ROTATION

SKELETAL TRACTION

Used to realign or reduce injury when skin traction is not possible Ropes pulls and weights are used. Traction needs to be maintained at all times. <u>Weights must hang freely</u> and the knots in the rope are tied securely





| Metabolic Causes Electrolyte Imbalances Acidosis Hypoglycemia ETOH/Barbiturate withdrawals Dehydration / Water toxicity | Intracranial Brain tumor Head Injury Aneurism Brain Infection Extracranial Heart, lungs, kidneys disease. SLE Diabetes Septicemia | Seizures resulting from metabolic disturbances are <u>NOT</u> <u>considered epilepsy</u> if seizure stops after underlying condition is resolved. |
|--|--|---|
| Seizure in Children Birth trauma Infection Congenital Defects Fever | Seizure in Adults Head Injury ETOH Infection Stimulants Med Side Effect | Seizure in Elderly Brain Tumor Stroke |

EPILEPSY

RISK FACTORS CAUSES - Socially Disadvantages 30% Idiopathic Generalized Epilepsy - Increased risk in older adults - Parent with epilepsy - Males> females

- African American
- Hx of Alzheimer or CVA

Changes in the function of <u>astrocytes</u> my play a role in recurring seizures

Astrocytes release glutamate which triggers synchronized firing of neurons.

PHASES OF SEIZURE

Prodromal: Symptoms preceding seizure: nervousness, lightheaded, etc

Aural: Sensory Warning

Ictal: Actual seizure

Postictal: Altered state of consciousness - Can last 5-30 min after seizure





GENERALIZED SEIZURE



- Synchronized epileptic discharge in both sides of the brain
- No Warning
- Cyanosis, excessive salivation, and tongue/cheek biting may occur



- Common in Children, typically resolve by adulthood
- Precipitated by flashing lights or hyperventilation

CHARACTERISTICS

- Daydreaming" STARE
- Peculiar behavior during seizure i.e.: Blinking, twitching
- Sometimes loss of postural tone
- Confusion after seizure

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TYPICAL ABSENT SEIZURE

- Tonic episode \rightarrow loss of muscle tone
- Patient usually regains consciousness by the time they fall
- No postictal phase





aka "partial" "partial local"

- Caused by focal irritation
- Symptoms depend on location of irritation
- Unilateral



Experiences unusually feeling, sudden unexplained emotions, may sense things that are not there. No loss of consciousness



Strange Behavior. Lip smacking. Repetitive Movement. Patient doesn't remember events that occurred during seizure

PSYCHOGENIC

"Pseudoseizure". Resembles epileptic seizure <u>Psychiatric</u>: patient typically suffer from emotional abuse, physical neglect or PTSD

STATUS EPILEPTICUS



- A continuous seizure or recurrent seizures without regaining consciousness
- Seizure activity can continue even with patients that are sedated and show no physical signs.

MEDICATION MANAGEMENT

ONSET of Seizure: Diazepam, Lorazepam MAINTENANCE: Dilantin, Phenobarbital



Intracranial Pressure "ICP

factors that affect ICP

- Arterial Pressure
- Venous Pressure
- Intra-abdominal/Intra-thoracic pressure
- Posture
- Temperature
- Blood Gases

MAINTAINING A NORMAL ICP

- 1- Changes in CSF
 - Altering the CSF absorption/production
 - Displace the CSF into the spinal subarachnoid space
- 2- Changes in Intracranial Blood Volume
 - Collapse of cerebral veins and dural sinuses
 - Regional Cerebral vasoconstriction or dilation
 - Changes in venous flow
- 3- Changes in brain tissue volume
 - Distention of dura
 - Compression of brain tissue

INCREASE ICP STAGES

Stage 1: Total compensation related to autoregulation

Stage 2: Decreased compensation, Risk of Increase ICP

Stage 3: Failing compensation, clinical manifestation of

ICP appear (Cushing Triad)

Stage 4: Herniation and death likely to occur



Normal ICP: 5-15 mmHg

Elevated ICP: >20 mmHg, sustained

Monro-Kellie doctrine If one component increases, another decreases to maintain normal ICP

CEREBRAL BLOOD FLOW

The amount of blood in mm passing through 100g of brain tissue in 1 min

AUTOREGULATION: The atumatic

adjustment in the diameter of the cerebral blood vessels by the brain to maintain a constant blood flow during changes in arterial BP.

Only effective if the MAT is between 70-150

MAP – Mean Arterial Pressure

Average Pressure exerted against vessel walls by blood

MAP = <u>Systolyc + 2xDiastolic</u>

3



Treat underlying condition. Adequate Oxygenation Intubation, Mechanical Ventilation, Surgery – AS NEEDED

<u>Drug Therapy:</u> MANNITOL (Osmitrol) ***

- Increase CBF. Plasma Expansion, Reduces blood viscosity, Vascular Osmotic diuretic effect Hypertonic Saline

- Move water into blood

Corticosteroids:

- Vasogenic Edema

PRN Med: Antiseizure, Antipyretics, Sedatives, Analgesics, Barbiturates



Sudden interruption of blood flow to part of the brain, killing brain cells and destroying or impairing body functions controlled by that part of the brain.

During a stroke, brain tissue fails to receive adequate oxygen, leading to tissue damage and necrosis

Temporary Loss of neurologic function due to ischemia

Carotid: Slurred speech, aphasia, 1-side weakness

S/Sx last less than 24hrs, longer than 1hr

Brain uses 20% of body's total oxygen, it has no oxygen reserve. Anoxia: >2-4 min - Cell Damage 10 mins – Irreversible Damage Glucose is the main source of energy



TREATMENT

Medical: Antiplatelets, Anticoagulants, Vasodilators

Surgical: Carotid Endarterectomy, Angioplasty, Stents, Extracranial/Intracranial Bypass

ISCHEMIC

Depend on vessel involved:

Vertebral: vertigo, diplopia, ataxia

80% OF ALL STROKES

Inadequate blood flow due to occlusion of an artery

Thrombotic:

Occurs in large arteries. Occurs from injury to a blood vessel wall, formation of a blood cloth Gradual Onset. Typically occurs at night. Commonly precedes by TIA



Embolic:
 Clot can be made up of:
 Blood, fat, bacteria or air.
 Caused when embolus lodges/occludes
 cerebral arteries.
 Sudden onset

HEMORRHAGIC

Sudden onset of symptoms. Progression over minutes to hours because of ongoing bleeding

- Most commonly caused by Hypertension
- Typically occurs during activity

Symptoms: Severe, sudden headache. N/V, Nuchal rigidity, Rapid deterioration of function, HTN



STROKE

Clinical Manifestations by affected side

LEFT HEMISPHERE DAMAGE

Paralysis, weakness on RIGHT side Right visual field deficit Aphasia

- Expressive
- -Receptive
- Global

Altered intellectual ability Slow, cautious behavior Increased level of frustration Depression



RIGHT HEMISPHERE DAMAGE

Paralysis, weakness on LEFT side Left visual field deficit Spatial-Perceptual deficit Increased Distractibility Impulsive behavior/poor judgment Lack of awareness of deficits Abilities overestimated

DIAGNOSTIC TEST

NON-contrast CT/MRI - to determine ischemic or hemorrhagic

Lumbar Puncture, Cerebral Angiography or Angioplasty, Digital Subtraction Angiography, Transcranial Doppler Ultrasound

PT/INR, PTT

TREATMENT

HEMORRHAGIC

Management of HTN Surgery (based on cause) - Evaluate hematoma

- Clip aneurism
- -Resection

Prevent ICP

Seizure prophylaxis if needed

ISCHEMIC

- Thrombolytic Therapy
- (Tissue Plasminogen Activator)
- MUST be given within 3.5-4 hrs of onset
- MUST rule out hemorrhage via CT
- Criteria:
 - BP<185/110
- PT <15, INR < 1.7
- Not on coumadin >18 years old

Cranial Nerves

I: OLFACTORY

| Smell | SENSORY |
|---------------------------|------------|
| Pt should be able to ID | aromas. |
| -Assess Patency occluding | g one |
| nostril at the time. | - |
| -Close eyes occlude one n | ostril and |

-<u>Close eyes</u>, occlude one nostril and smell.

II: OPTIC

Visual Acuity 🏴

Ask Pt to read Snellen's Chart about 20 ft away. Close one eye at the time.

If Pt with Glasses, leave them on. Remove only reading glasses

III: OCULOMOTOR

Pupillary Constriction (PERRLA) Assess ocular movements and pupil reaction. <u>PERRLA</u>: Pupils Equal, Round, React to Light, and Accommodation.

IV: TROCHLEAR

Vertical Eye Movement Ask Pt to move eyeballs up and down (following object)

V: TRIGEMINAL

S: Face Sensation Light touch, wipe forehead, cheeks and chin with cotton (eyes closed) M: Mastication Muscles Palpate Temporal and masseter muscles as Pt clenches the teeth

VI: ABDUCENS

Horizontal Eye Movement Ask Pt to move eyeballs laterally (following object)

мото

VII: FACIAL

| S: Taste anterior $2/3$ | MD |
|--------------------------------|-------|
| Ask Pt to ID various taste | S |
| placed on the tip and side of | i the |
| mouth | |
| M: Facial Expression | |
| Ask Pt to do facial expression | SNS, |
| smile, frown, raise eyebrows | |

VIII: ACOUSTIC

Hearing + Equilibrium SENSORY Hear loud and soft-spoken words. Whispered Voice Test. Tuning Fork: Hearing by air and bone conduction. WEBER - RINNE

IX: GLOSSOPHARYNGEAL

S: Taste posterior 1/3 M: Pharynx Gag Reflex

Depress tongue, Pt says "Ahhhh" uvula and soft palate should rise to midline

MIX

X: VAGUS

S: Sensation pharynx, viscera, carotid body M: Pharynx and Larynx Ask Pt to swallow, Assess Speech for hoarseness.

XI: SPINAL ACCESSORY

Movement Trapezius and Sternomastoid Muscles Ask Pt to rotate the head against resistance applied to side of chin. Ask Pt to shrug the shoulders

against resistance.

XII: HYPOGLOSSAL

Movement of Tongue

Inspect the tongue. Tongue in midline as Pt protrudes the tongue. Ask Pt to say: "light, tight, dynamite"

мото

Cranial Nerves



SOME SAY MARRY MONEY BUT MY BROTHER SAYS BIG BRAINS MATTER MORE I II III IV V VI VII VIII IX X XI XII

S: SENSORY

M: MOTOR

B: BOTH ≥MIX€

LUNGS

Tracheal Sound: Harsh, hollow Bronchial Sound: High pitched, loud, hollow Bronchovesicular: Low Pitched, hollow, Anterior and Posterior Vesicular: Low pitched, blowing Anterior and Posterior



ADVENTITIOUS BREATH SOUNDS

| NAME | LOCATION | CAUSE | SOUND |
|----------------|---|--|---|
| CRACKLES | R+L lung bases | Sudden reinflation of alveoli or fluid in small airways | Crinkle of crackle Fine and short Coarse of Medium Can be cleared with cough |
| Ronchi | Trachea Bronchi | Fluid or Secretions in large airways | Loud and low pitched Heard on expiration Fluid through a straw |
| WHEEZING | Can be heard over all lung fields. Usually heard louder posteriorly | Narrowed or obstructed Bronchi | High pitched Prolonged Heard on expiration |
| PLEURAL RUB | Lateral Lung Fields | Inflamed Pleura | Rubbing or grating sound heard on inspiration |
| STRIDOR | Upper lungs | Disrupted air flow of larynx or Trachea Croup, foreign body in airways, infection | High pitched, wheezing Mostly heard on inspiration |

ASK ABOUT

- ✓ Tabaco Use or Smoking
- production
- ✓ Chest Pain
- ✓ Environmental Exposures
- ✓ Chronic hoarseness
- ✓ Persistent cough or sputum ✓ Uncharacteristic Shortness of Breath
 - ✓ Family history of TB
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Respiratory Disorders

PNEUMONIA

P: Infection of pulmonary tissue

- S/Sx: Chills, fever, tachypnea, rhonchi, wheezes, labored breathing
- N: Encourage coughing and deep breathing. Provide O_2 therapy or CPT

 \mathcal{R} : Previous respiratory tract infection, smokers, patients who recently had surgery, elderly, those with a weakened immune system

CHRONIC OBSTRUCTIVE PULMONARY DISEASE > COPD <

P: A progressive disorder of the airway that restricts airflow and alveolar expansion. Exacerbated by infection. Caused by emphysema or bronchitis

S/Sx: Cough, excess mucus, wheezes, crackles, barrel chest, use of accessory muscles to breathe + prolonged expiration

N: Administer oxygen therapy as ordered. Monitor pulse oximetry. Monitor sputum color/

characteristics. Place in Fowlers position to aid in breathing. Suction Pt. air way PRN.

E: Avoid extremely hot, cold or spicy foods. Avoid exposure to those with infection. Eat a nutritional diet. Stop smoking. Use pursed-lip breathing when in distress.

ASTHMA

P: Chronic inflammatory disease of airway. Smooth muscles constrict in bronchi

S/Sx: Recurrent episodes of wheezing, breathlessness, chest tightness and cough that is usually brought on by exercise or triggers

N: Monitor vital signs + pulse oximetry frequently. Administer oxygen as prescribed. Stay with the patient to reduce anxiety. Administer bronchodilators + corticosteroids as ordered

R: Asthma Triggers: Animal dander, mold, exhaust, pollen, stress, hormonal changes, GERD, chemicals, plastics, shrimp, potatoes

PLEURAL EFFUSION

P: Collection of fluid in pleural space

S/Sx: Progressive dyspnea, dry cough, sharp pain on inspiration. Decreased breath sounds

N: Identify the underlying cause. Prepare for possible thoracentesis. Encourage Cough/deep breathing

ACUTE RESPIRATORY DISTRESS SYNDROME

P: Respiratory failure caused by an underlying cause like a lung trauma or inflammation. Interstitial edema causes airway compression

S/Sx: Abnormal ABG values, tachypnea, hypoxemia, pulmonary infiltrates

N: Prepare for intubation or mechanical ventilation

Pneumonia

PATHOPHYSIOLOGY

An infection of the lung parenchyma. Usually your epiglottis, cough reflex, mucous membranes and bronchoconstriction can protect the lungs from becoming infected, but they can become overwhelmed and allow bacteria and viruses to grow.

EARLY SYMPTOMS

- Purulent sputum
- Diminished lung sounds
- Fatique
- Cough
- Sore throat

DISEASE PROCESS

LATE SYMPTOMS

- Chest pain Hemoptysis
- Tachycardia Respiratory distress
- Sepsis
- Dyspnea
- Activity

COMMON CAUSES

- Abdominal/thoracic surgery
- IV drug use
- Air pollution
- Immunosuppressive
- disease/meds
- Age of 65+
- Intestinal/gastric feeding via Diabetes
- NG tube
- Altered consciousness
- Malnutrition

- Bed rest/immobility
- Tracheal intubation
- Smoking
- Chronic disease
- Upper respiratory infection
- Exposure to farm animal
- Lung cancer
- CKD
- Recent antibiotics



MEDICAL INTERVENTIONS

Labs ABG, CBC, WBC Blood cultures Sputum culture

Radiology Chest X-Ray Chest CT

Pharmacology Antibiotics (macrolides) Corticosteroids Glucocorticoids

Other Treatment

- 02 therapy
- IV therapy
- chest physiotherapy
- suctioning
- early mobilization

Pneumonia

Nursing Management

ASSESSMENT

Lung sounds, VS, SaO₂ 90, Health Hx, Medications, Recent Surgeries, Smoking, Mobility Level, Fatigue LABs ABGs, Sputum Culture, WBCs

NUTRITIONAL CONSIDERATIONS

- Fruits + Vegetables build Immune System
- Protein Rich Foods help Repair Tissue

- Drink Plenty of Water and fluid to Maintain Fluid - Electrolyte Balance - Avoid Throat Irritating Foods Like

Milk That Can Cause Excess Secretions

- PREVENTIONS
- Wash Hands Frequently
- Eat A Balanced Diet
- Get Adequate Rest
- Exercise Regularly
- Cough + Sneeze into Elbow
- Stop Smoking
- Avoid Others who Are ill



INTERVENTIONS

- Teach good handwashing
- Change position frequently
- Promote expectoration
- Limit visitors to prevent spread of infection
- Encourage adequate rest
- Educate pt. to report chest pain, fever, changes in sputum or altered sensorium
- Provide comfort for pain
- Administer antipyretics as ordered
- Continuously monitor pulse oximetry
- Suction secretion as needed
- Encourage early

ambulation/mobilization to speed up recovery

Care Plan

Dx: Impaired gas exchange r/+ fluid and mucous accumulation

Goal: Improve ventilation and oxygenation of tissues Interventions:

- 1- Assess respiratory rate, depth and effort frequently
- 2- Administer oxygen therapy (Will help maintain PaO2 levels)
- 3- Assess skin color, mucous membranes + nails for cyanosis (Cyanosis can be a sign of hypoxemia)
- 4- Monitor Arterial blood gasses (ABGs) + pulse oximetry (helps alert healthcare team to changes in condition)

Dx: Activity intolerance r/+ SOB + general fatigue + weakness

Goal: Regain baseline activity levels without complications

Interventions:

- 1- Evaluate response to activity (Allows you to anticipate the interventions needed)
- 2- Assist with ambulation + self-care (prevents exhaustion and decrease the likelihood of falls)
- 3- Turn + reposition every 2 hrs. (Prevents complication like pressure ulcers and fluid accumulation)
- 4- Group care together (Minimizes exhaustion + conserves oxygen)
- 5- Ensure pt. is receiving adequate rest (It is important to conserve rest to promote healing + save energy)

Dx: Risk for infection r/t inadequate immune defense

Goal: Recover from infection without complications Interventions:

- 1- Educate patient about importance of clearing secretions (sputum accumulation can cause secondary infection)
- 2- Provide mouth care frequently (keeps bacteria from growing + spreading to lungs)
- 3- Ensure pt. is practicing good hand hygiene (helps prevent the spread of infection + save energy)



PATHOPHYSIOLOGY

- COPD is characterized by airflow obstruction that is caused by chronic bronchitis or emphysema
- The obstruction is caused by inflammation which changes the structural function of the lung that makes it harder to expire CO2.
- The air becomes trapped causing the chest to hyper expand and become barrel shaped. This prevents more air from being expired.
- Because of decreased expiration the pt. will become hypercaphic ($\uparrow CO_2$) and hypoxic ($\downarrow O_2$)
- The excess pressure can damage alveoli further causing a snowball effect of decreased function

POSSIBLE COMPLICATIONS

Pulmonary Insufficiency - Impaired gas exchange r/t backflow from the Pulmonary Artery to Right ventricle Acute Exacerbation - Worsening or Symptoms. Tx: Assess ABGs, maintain fowler's position, suction airway if necessary

Pulmonary hypertension - Excess Pressure in Lungs. Tx: Diuretics, vasodilators, anticoagulants + Calcium Channel Blockers

Cor Pulmonale - Right Ventricle Hypertrophy. Tx: Treated with diuretics + management of underlying cause

RISK FACTORS

SMOKING - The major risk factor for developing COPD - hyperplasia, ↑mucus, ↓cilia OCCUPATIONAL - Chemicals + Dusts (Dusts, vapors, irritants, fumes can increase the risk of COPD) AIR POLLUTION - Urban air pollution coal + biomass fuels used for heating INFECTION - Recurring infection in childhood are linked to reduced function GENETICS OR AAT DEFICIENCY - Linked to poor lung function AGING - Loss of recoil, stiffening of chest wall + impaired gas exchange ASTHMA - Can be secondary to COPD or contribute to progression of it

SIGNS & SYMPTOMS

Early Stages

- Symptoms develop slowly
- Chronic intermittent cough
- Dyspnea that increase in severity
- Inability to take a deep breath
- Prolonged expiration and Jlung sounds Late Stages
- Dyspnea at rest
- Relies on accessory muscles to breathe
- Wheezing, chest tightness
- Fatigue, weight loss, anorexia

 \downarrow FEV₁ = \uparrow Obstruction

FEV1 / FVC < 70% = COPD

 $FEV_1 = Forced Expiratory Volume / 1 Sec$

- History and physical Exam

- Spirometry required
- Chest X-Ray
- A1 antitrypsin levels (AAT)
- Blood gasses in severe stage
- 6 min walk test

SPIROMETRY MEASURES FEV

DIAGNOSIS

| | Classification | Severity | FEV1 |
|-----|----------------|-------------|--------|
| | Stage 1 | Mild | >80% |
| | Stage 2 | Moderate | 50-80% |
| Sec | Stage 3 | Severe | 30-50% |
| | Stage 4 | Very Severe | <30% |
| | | | |





TREATMENT

Minimally invasive

- Smoking cessation
- Airway clearance techniques
- Hydration (if indicated)
- Long term O2 (if indicated)
 Exercise Plan (walking + upper
- body)

| - Bronchodilators |
|--------------------|
| (↓Dyspnea, ↑FEV1) |
| - Anticholinergics |
| (↓Exacerbations) |
| - Corticosteroids |
| |

Pharmacology

Surgical - Lung volume reduction - Bullectomy - Lung transplant

Pulmonary rehab

- Exercise training (ambulation + upper limb exercises)
- Smoking cessation
- Nutrition counseling
- Education (Importance of sleep
- and good nutrition)

NURSING MANAGEMENT

Assessment

Subjective Data

- Hx of exposure to
- pollutants/irritants?
- Hx of recent infection or hospital stay?
- Do they use O_2 therapy?
- Medications they're on?
- 1) bronchodilators
- 2) corticosteroids
- 3) Anticholinergics
- 4) OTC
- Smoker? Pack years/ quit date
- Weight Loss or Anorexia?
- Exercise / Activity Level?
- Anxiety / Depression? Sleep Pattern?

Objective Data

General -Restlessness, Fatigue, Sitting upright Integument Cuansis Door turgor clubbing

Cyanosis, poor turgor, clubbing, bruising, edema, thin skin

Respiratory

- <u>Rapid + shallow breathing</u>, prolonged exp.,

- ↓Breath sounds, accessory muscle breathing

- ↓Diaphragm movement, resp. acidosis

Cardiovascular

- Tachycardia, <u>Jugular vein distention</u>,

<u>edema in feet</u>, dysrhythmias

Planning

- Goals - Prevent disease progression
- Maintain ability to care for self
- Relieve symptoms avoid complications

Diagnosis

- Ineffective breathing pattern
- Impaired gas exchange
- Ineffective airway clearance

Implementation

Interventions

- Counsel smoking cessation
- Breathing retraining: <u>Pursed-lip</u> (PLB) To prolong expiration. Easier to learn + should be 1^{st} choice in acute situation
- Diaphragmatic breathing: use of abdomen instead of accessory muscles to prevent Fatigue and slow Respiratory rate
- Airway clearance (ACTs): loosen mucus/secretions then cleared by huff coughing
- <u>Chest Physiotherapy</u> (CPT): Percussion / vibration loosens mucus
- Postural drainage: Repositioning to drain secretions from specific areas
- Nutritional therapy: Increase Kcals and protein

Education

- Encourage Pt. to avoid or control exposure to pollutants
- Caution Pt. to avoid others who are sick and practice good hand hygiene
- Explain importance of reporting changes in conditions to HCP
- -Remind Pt. to follow O_2 therapy as ordered to prevent oxygen toxicity
- Suggest nutritional meals options

Evaluation

- Assess need to change flow rate
- Evaluate compliance to meds.
- Monitor for signs of complications
- Determine O2 therapy effectiveness
- NursingStoreRN



PATHOPHYSIOLOGY

- Chronic lung disease that causes narrowing and inflammation of bronchi and bronchioles Asthma Attack:

1- Sooth muscle constricts = Chest Tightness dyspnea

2- Mucosa lining + goblet cells = more inflamed + excessive mucus production

goblet cells: collect bacteria to prevent going in the airways

S/SX

Early S/Sx

- 1- Shortness of breath
- 2- Easy fatigue
- 3- Cough at night, trouble sleeping
- 4- Sneezing, tired, scratchy throat
- 5- Wheezing
- 6- ↓Peak flow best

Active S/Sx

- 1- Chest Tight
- 2-Wheezing
- 3- Cough
- 4- Dyspnea
- 5- ↑HR
- 6-Tachy
- 6-02Sat<90%

VERY BAD!

- 1-Rescue inhaler doesn't work
- 2- Can't speak
- 3- Chest retractions
- 4- Cyanosis lips/Skin
- 5- Sweaty

TRIGGERS

- Smoke, pollen, pollution, perfume, dander, dust, pest, mold, cool and dry air, GERD, respiratory infection, exercise, hormonal shift, beta blockers/NSAIDS, Aspirin, sulfites

INTERVENTIONS

- v/s
- Keep Pt. calm
- High Fowlers
- Oxygen / Bronchodilators
- Assess: lungs, cyanosis, ease of speak

BRONCHODILATORS

1- ALBUTEROL - Short Acting, fast relieve -NOT for daily Tx-2- SALMETEROL - Long Acting -NOT for acute attack-3- IPRATROPIUM - Short acting

ANTI-INFLAMMATORIES

1- CORTICOSTEROIDS - "-sone" "solone" -NOT for acute attack-2- MONTELUKAST - Oral - Relaxes smooth muscle, ↓mucus. for CONTROL and MAINTENANCE -NOT for acute attack-





Oxygen Therapy

| DEVICE | NAME | O ₂ RATE | ADVANTAGE | DISADVANT |
|--------|-------------------------|---|--|--|
| 12M | Nasal Cannula | 1-6 L/min FiO ₂ 24-44% | Lightweight Inexpensive Pt. can talk and eat | Easily dislodged, skin breakdown Mucosal drying |
| | Simple Face Mask | 6-10 L/min FiO ₂ 40-60% | Simple to use, inexpensive. Can have humidification | Poor fitting, must remove to eat |
| | Partial- Rebreathing | 6-12 L/min FiO ₂ 50-7590 | Moderate O_2 Concentration | Warm, poorly fitting, remove to eat |
| Valves | Non- Rebreathing | 10-15 L/min FiO2 80-95 | HIGH FLOW O_2 Concentration | Poorly fitting, remove to eat |
| | Venturi | 4-10 L/min FiO ₂ 24-60% | MOST PRECISE & ACCURATE | Remove to eat |

Chest Tube

Uses:

- Removing Air, Fluid or Blood
- Preventing drained air and fluid from returning to the pleural space
- Restoring Negative Pressure with the pleural space to re-expand the lung



Placement:

Mid-anterior axillary line at the 4^{th} or 5^{th} intercostal space on affected side

Complications:

- Bleeding
- Infection
- Air leak / Crepitus
- Clogged tubing DO NOT MILK / STRIP TUBING
- Tube disconnects from drainage system Place chest tube in sterile water until new system is set up

Heimlich Valve:

One-way used with a chest tube to prevent air from entering the pleural space



Assessments (q2h):

- Pulmonary Status
- Dressing Status
- Assess for crepitus
- Check tubing
- Keep CDU (Chest Drainage Unit) below patient's Chest Level
- Monitor Water Levels
- Assess for bubbling in water chamber
- Assess Drainage

HEIMLICH CHEST DRAINAGE VALVE







Tension Pneumothorax

Life-threatening condition that develops when air is trapped in the pleural cavity under positive pressure, displacing mediastinal structures. The air that enters the chest cavity with each inspiration is trapped



Open Pneumothorax

Endocrine Disorders

ADRENAL HORMONES

Addison's [Cortisol]ACTH

S/Sx: Decreased vascular tone, hypotension, bronze skin tone, weight loss and weakness Tx: lifelong replacement of glucocorticoids or mineral corticoids

Cushing's Cortisol ACTH

S/Sx: Moon face, weight gain, hypertension, fragile skin Tx: Glucocorticoid treatment, adrenalectomy with synthetic glucocorticoid replacement therapy for life.

ANTIDIURETIC HORMONE

Diabetes Insipidus JADH

S/Sx: Excretion of large amounts of dilute urine. Polydipsia, headache, low specific gravity, dehydration Tx: Vasopressin therapy, avoiding foods/beverages that are diuretics

GROWTH HORMONE

Acromegaly 1GH

S/Sx: Gigantism, long arms and extremities, oily skin, deep voice. Tx: suppress GH with a GH inhibition medication

Pituitary Dwarfism JGH

S/Sx: Short height, reduced cardiac output, moderate obesity Tx: If caught early, can be cured with GH Supplementation

THYROID HORMONE

Hyperthyroidism $\uparrow T_3 + T_4$

S/Sx: Tumors, nervousness, tachycardia, weight loss, cramps, diarrhea Tx: Anti-thyroid medications that inhibit the creation of thyroid hormone

Hypothyroidism JT3+T4

S/Sx: Drowsiness, fatigue, excessive hunger, weight gain Tx: Thyroid hormone replacement therapy based on T3-T4 levels

DIABETES MELLITUS

Type I - Inability to make Insulin

S/Sx: Polyuria, polydipsia, polyphagia, weight loss, blurred vision Tx: Consistency in food intake, close monitoring + correction of blood glucose level.

Type II - Inability to absorb Insulin

S/SX: Polyphagia, polydipsia, poor wound healing, weight gain TX: Exercise, diet changes + weight loss are preferred treatment, but if these are unsuccessful medications like metformin and Insulin are used







Diabetes Type 1 - 11

Concepts:

Pancreas: Beta cells produce and secrete Insulin

Glucose [Sugar]: Fuels cells in the body. Will only enter the cells with the help of insulin

Insulin: Secreted by Beta cells to attach glucose so that It can be used to regulate blood sugar

Liver: Stores Excess glucose as glycogen for a later time when your body needs it

Glucagon: Helps increase blood glucose levels. When released, it causes the liver to release glycogen (glucose)

WHAT'S NORMAL:

A patient with HIGH Sugar: Pancreas releases Insulin to attach to glucose to enter the cell

A Patient with LOW Sugar: Pancreas releases glucagon to tell the liver to release glycogen-glucose

Treatment:

1- Nutrition

2- Insulin

DIABETES TYPE 1

Diagnosed in Children and Young adult. Insulin-Dependent

- 1- Immune System attacks beta cells responsible for Insulin production.
- 2- NO Insulin in the bloodstream Increase glucose
- 3- Muscle unable to use glucose
- 4- Glycogen and Protein breakdown cause ketoacidosis

DIABETES TYPE 2

Three Causes:

- 1- Pancreas doesn't produce enough Insulin
- 2-Body doesn't use Insulin appropriately
- 3- Liver inappropriately produces glucose

Most common type usually occurs over 35%/0. 80%-90% patients are obese

GESTATIONAL DIABETES

Develops during pregnancy. Detected at 24-28 weeks of gestation Usually, glucose levels normalize at 6 weeks post-partum.

RISKs

Increased risk for C-Section, Perinatal death and neonatal complications. Increased Risk for developing type 2 DM in 5-10 years





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Diabetes Complications

DIABETIC KETOAC

DKA usually develops rapidly, over 24h period.

Signs/Symptoms

- Blood Glucose >250
- Ketones + Acidosis = DH<7.35
- Dehydration
- Kussmaul Respirations rapid, deep breathing, fruity breath
- Abdominal Pain
- Polyuria, Polydipsia, weakness, fatigue and



TREATMENT

- 1- Treat Dehydration 0.9% Normal Saline
- 2- Lower Blood Sugar
- 3- Hourly BG Checks + Heart Monitor

>250: IV Regular Insulin only -Add K+ during IV Insulin <200 or if Ketones resolve SubQ Insulin + IV D5W



Blood Glucose >600 (Severe 600-2400mg/dL) Dehydration PH >7.30



- 1- Treat Dehydration 0.9% NS
- 2- Lower Blood Sugar
- 3- Hourly BG Checks
- 4- Assess Rehydration: Stable BP, Pink skin, warm temp, Urine Output >30mL/hr

IV Regular Insulin, then titrate with SubQ Insulin + IV D5W

Hydration Insulin

Electrolyte

Replacement

Insulin Mixing Cheat Sheet

| Insulin | Starts to Work In (hours) | Peak Action (hours) | Duration of Action (hours) | Maximum Duration (hours) | When to Take |
|-----------------------|------------------------------|------------------------|-------------------------------|--------------------------------|---|
| | | Rapid Acting | | | |
| Lispro (Humalog) | 15 to 30 minutes | 1 to 2 hours | 3 to 6 hours | 4 to 6 hours | |
| Aspart (Novolog) | 15 to 30 minutes | 1 to 2 Hours | 3 to 6 hours | 4 to 6 hours | 0 to 15 minutes before meal |
| Glulisine (Apidra) | 15 to 30 minutes | 1 to 2 hours | 3 to 6 hours | 4 to 6 hours | |
| | | Short Acting | | | |
| Regular | 30 minutes to 1 hour | 2 to 4 hours | 3 to 6 hours | 6 to 8 hours | 30 minutes before meal |
| | Ir | termediate Actir | ng | | |
| NPH | 2 to 4 hours | 8 to 10 hours | 10 to 18 hours | 14 to 20 hours | Does not need to be given with meal |
| | | Long Acting | | | |
| Glargine (Lantus) | 1 to 2 hours | None | 19 to 20 hours | 24 hours | Does not need |
| Detemir (Levemir) | 1 to 2 hours | None | 19 to 20 hours | 20 hours | meal |







Withdraw enough air equal to the total amount of insulin.
 Inject the air into the NPH without touching the insulin.
 Inject remaining air into the regular insulin then withdraw the regular dosage.
 Withdraw the NPH dosage.



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Mouth – Amylase breaks down starch Esophagus - Peristalsis brings foods to Stomach Stomach - HCL breaks up food + activates enzymes. Pepsin converts proteins Small Intestine - Duodenum contains bile, pancreatic ducts Large Intestine - H2O absorption + waste elimination. Vit K synthesis Pancreas - Maltase – Maltose > monosaccharides Lactase – Lactose > galactose/glucose Gallbladder – Stores, Concentrates Bile Liver - Kupffer cells remove bacteria in the portal venous blood

GASTROESOPHAGEAL REFLUX DISEASE GERP

P: Backflow of gastric and duodenal contents up into the esophagus caused by a dysfunctional lower sphincter
 S/Sx: Frequent heartburn and epigastric pain, nausea, dyspepsia, dysphagia, regurgitation
 N: Teach pt. to avoid irritants like peppermint, chocolate, coffee, fatty foods, alcohol, smoking. Avoid eating 2
 hrs before bedtime. Avoid anticholinergics, NSAIDs. Keep HOB elevated after eating

PEPTIC ULCER DISEASE

P: An ulceration that erodes the lining of the stomach or S.I. Caused by irritation, H. pylori, NSAIDs S/SX: Sharp pain in left/mid epigastric area after meals 30-60 mins=gastric 90-180mins= duodenal RX: Proton pump inhibitors + H2 blockers

Tx: Surgical: resection, vagotomy. Total gastrectomy, pyloroplasty

CHOLECYSTITIS

P: Inflammation of the gallbladder can be caused by slow bile emptying, contracted gallbladder or bacterial invasion S/Sx: Murphy's sign > can't take deep breath when fingers are placed on the hepatic margin due to pain Belching, flatulence, RUQ pain

N: Maintain NPO status during exacerbations. Educate pt. to eat small low-fat meals.

INFLAMMATORY BOWEL DISEASE SIBS - UC - CROHN'S

P: Inflammatory diseases of the bowel

S/Sx: Diarrhea, abdominal cramps for > 6 weeks

N: Educate about a low FODMAP diet, help decrease triggers and stress, avoid use of NSAIDS to 1 GI bleeding

APPENDICITIS

P: Acute inflammation of the appendix + surrounding tissue

- S/SX: Sharp, constant, abdominal pain that moves to the RLQ
- N: Administer pain meds, prep for imaging or surgery

PANCREATITIS

P: Acute inflammation of pancreas

S/Sx: Nausea, vomiting, diarrhea, diffuse abdominal pain and cramping

N: Pain control, nausea medication administration, limit oral intake



A peptic ulcer is an excavation (hollowed-out area) that forms in the mucosa of the stomach, in the duodenum or in the esophagus.

CAUSES:

Everything that reduces the protective mucosa layer:

- 1- H. Pylori Bacteria that attacks the mucosa
- 2- NSAIDS Inhibit prostaglandins \downarrow Bicarbonate, = \downarrow Defense \uparrow Acid

Peptic Ulcer Disease

- 3- Smoking, ETOH, Genetics, STRESS
- when damaged: histamine release parietal cells stimulated to release more HCL acid

GASTRIC ULCER:

-Food makes it worst. Pain occurs IMMEDIATELY after eating.

- -Pyrosis vomiting, constipation or diarrhea, and bleeding.
- -If bleeding ulcer, hematemesis or melena (black, tarry stools)

DUODENAL ULCER:

-Food makes it better. Pain occurs 2-3 hours after meals.

- Pt. awake with pain during the night.
- Stool- Dark, Tarry

TREATMENT:

Medication:

- PPI Proton Pump Inhibitors (-prazole)
- Antibiotics If confirmed H. Pylori
- Bismuth (Pepto-Bismol)
- $-H_2$ Blockers (-tidine)
- Antacids (Mag. Hydroxide, Calcium Carbonate, Sucralfate, Carafate)

FOODS TO AVOD



Hot Peppers





Coffee



Carbonated

beverages

Refined sugar and most sweeteners









Fried foods





Tea

Chocolate



Peppermint



Omega-6 fats

(nuts, seeds, etc.)



Dairy products



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Gastroesophageal Reflux Disease



Backflow of gastric or duodenal contents into the esophagus, due to a weak/damaged lower esophageal sphincter (LES)

DIAGNOSTICS:

Endoscopy – Will assess narrowing or ulcers formed pH Monitoring – Measures the amount of acid in the esophagus

SIGNS/SYMPTOMS:

Most Common - Pyrosis (heartburn)

- Epigastric Pain
- -Regurgitation
- Dry cough worse at night/ hoarseness
- Nausea
- Difficulty swallowing
- symptoms may mimic those of a heart attack [Brunner & Suddarth's Med Surg 14e page 1283]

TREATMENT:

Lifestyle changes: Small meals

- last meal 30min before bedtime
- Sit up 1hr after meals
- Weight loss, smoking cessation

Avoid: fatty, ETOH, coffee, peppermint, acid foods (citrus, tomatoes)

Medication

- Antacids – Interferes with many drugs. Give alone, wait 1-2 hrs before taking another meds

- Histamine Receptors Blockers lowers Histamine Lowers Inflammation
- PPIS Protect lining of the stomach
- Bethanechol Protect lining of stomach

Fundoplication Surgery - Reinforces the LES by wrapping a portion of the stomach around

the esophagus



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Crohn's Disease

Inflammation or ulceration (or both) of the bowel.

Characterized by periods of remission and exacerbation.

May affect anywhere in the GI. Most common in ileum and the ascending colon.

Scattered patches - Not continuous with cobblestone appearance

SIGNS/SYMPTOMS:

- 1-Right Lower abdominal pain
- Mouth or GI ulcers
- Diarrhea (sometimes with blood, pus, mucus)
- Loss of appetite / weight
- Fissures with anal bleeding
- Abdominal bloating

COMPLICATIONS:

- 1- Abscesses: Form in the intestinal wall
- 2- Fistula: Worsening of abscess may lead to a hollow hole
- 3-Malnourishment: If affecting the Small Intestine
- 4- Fissures: If affecting anal area loss of integrity
- 5- Strictures: Narrowing, Intestinal Blockage

TREATMENT:

1- Diet Education – AVOID high fiver, nuts vegies, fruits, dairy, spicy, high fat, gas causing food Encourage- LOW fiber, HIGH protein, HIGH fluids

- 2- Medication
- 1st Line- Mild case: sulfasalazine.

Steroids: \downarrow Inflammation, NOT long term, \uparrow Infection risk

- 2^{nd} line-Immunosuppressors: \uparrow risk of infection, cancer, \downarrow Inflammation
- 3- Teach Ostomy care if surgery occurs
- 4- Smoking Cessation
- 5- In severe cases, TPN for malnourishment Monitor weight
- 6- Monitor bowel movement, frequency and characteristics/ Bowel sounds



Ulcerative Colitis

Chronic ulcerative and inflammatory disease in the INNERMOST lining of the Colon and Rectum ONLY. (There is NO abscesses, fistulas or fissures -usually) Continuous - Not Scattered

SIGNS/SYMPTOMS

- Cells of intestinal lining die from ulcers that pus and bleed.
- Intestine can't absorb water as usual Watery diarrhea that Includes Pus and Blood
- Urge to defecate frequently

- Periods of remission and exacerbation. Ulcer sites heal, but lining stays damaged, may form polyps

SEVERE

Lead-pipe Sign – large intestine starts to
lose its form. Will appear smooth (no Haustra)
Repeated Ulceration – Rupture of bowels –
peritonitis

- Toxic Megacolon - Large intestine dilates due to inflammation - Unable to function properly

TREATMENT

 Surgery – Proctocolectomy ileoanal anastomosis
 Diet Education – AVOID high fiver, nuts vegies, fruits, dairy, spicy, high fat, gas causing food
 Encourage- LOW fiber, HIGH protein, HIGH fluids
 Medication:

1st Line- Mild case: sulfasalazine.

Steroids: \downarrow Inflammation, NOT long term, \uparrow Infection risk

2nd line- Immunosuppressors: *risk* of infection, cancer,

↓Inflammation

Also, Abx during flares up Antidiarrheals








Causes: HF, MI, Cardiomyopathy

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Cardiac Disorders

| | | | P: Pathophysiology R: Risk Factors S/Sx: Signs+Symptoms | Dx: Diagnosis N: NursingManagement C: Complications |
|---|---|---------------------------------|---|---|
| | CORONARY / | ARTERY DISI | EASE | |
| Patho: Narrowing plague buildup/ at DX: ECG, Catheter N: Educate about C: 1 Perfusion, HT | or obstruction of a coronary arte herosclerosis rization, blood lipids ↓ Kcal/fat,↑ fiber diet & exercise N, angina, MI | ry due to | | Faty Deposits |
| | AORTIC | ANEURISM | | |
| Patho: Stretching vessel weakness S/Sx: Thoracic - ne Abdominal - Ruptured - | of the medial wall of an artery c eck, shoulder, ↓back pain, ↑HR, dys · pulsating mass in abdomen, Abd, severe Abd/back pain, shock, ↓B1 | aused by Spnea 'back pain | | |

DX: Ultrasound, CT Scan, arteriography

N: Monitor Vitals, check peripheral pulses, assess for abdominal tenderness, ask pt. if abdominal or back pain is present



CARDIOGENIC SHOCK

Patho: Reduced cardiac output and tissue perfusion. Usually caused by a corona artery blockage

S/Sx: Hypotension, pallor, tachy, disorientation, chest pain, cool, clammy skin

 $\ensuremath{\mathsf{N}}\xspace$: Administer O2, morphine sulfate as ordered. Prep for intubation, Monitor blood gas levels



Map

Coronary Artery with Thrombus



VENTRICULAR FIBRILLATION

Patho: Ventricles depolarize in a completely disorganized way **S/Sx:** Cardiac output ceases no pulse, BP, Respirations and Pt. is unconscious

N: Activate Emergency response, Administer CPR, defibrillate and administer O2 as ordered

PREMATURE VENTRICULAR CONTRACTION

Patho: Ventricles contract prematurely due to impulse initiation by purkinje fibers instead of the SA node N: Assess O2 saturation. Monitor anticoagulant and electrolytes as ordered Bigeminy - PVC every other heartbeat Trigeminy - PVC every 3rd heartbeat

Quadrigeminy - PVC every 4th heartbeat

ATRIAL FIBRILLATION

Patho: Multiple depolarizations from the atrium occur in a

disorganized way causing the atria to quiver

DX: ECG - no P wave seen

 \mathbf{N} : Administer O2 and anticoagulants as ordered. Educate pt. about therapy.

C: Thrombus formation, stroke



MYOCARDIAL INFARCTION

Patho: Cardiac tissue no longer has Oxygen Supply which can lead to necrosis. Blockage of 1 or more arteries of the heart. S/Sx: Chest pain, SOB, nausea, low back pain, diaphoresis, pallor, fear + anxiety Dx: Troponin levels, CK, CK-MB, Myoglobin, ECG N: Administer O2, Establish IV access, Obtain 12-lead EKG, Administer thrombolytic therapy, assess pulses, Monitor for Blood Pressure Changes Morphine - Pain and relaxes the heart Oxygen - $\uparrow O_2$ in the heart Nitroglycerin - vasodilates Aspirin - blood thinner







Cardiac Valve Stenosis



Aortic Valve Regurgitation

Blood leaks backward from aorta unto Left Ventricle

Leads to Left Ventricle enlargement due to volume overload from inadequate / incomplete emptying during systole



Symptoms

Varies depending on cause/severity Increased CD (early compensation) Paradoxical Nocturnal Dyspnea Pulmonary Edema Right Side Heart Failure Shock – Acute A.R.

Causes

Congenital Heart Valve Disease Age-Related heart changes Endocarditis Rheumatic Fever Trauma

TREATMENT

- Balloon Valvuloplasty
- Commissurotomy

- Annuloplasty
- Valve Replacement

Mitral Valve Regurgitation

Backward of Blood from the Left Ventricle to Left Atrium due to an incompetent valve



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Symptoms

Weakness

Fatigue

Paradoxical Nocturnal Dyspnea Murmur

A-Fib

Causes

Mitral Valve Prolapse Rheumatic Fever Endocarditis Heart Attack Cardiomegaly Trauma

TREATMENT

- Medication to Increase CO
- Valvuloplasty

- Annuloplasty
- MV Repair / Replacement

ANGINA

Angina / Chest Pain: A narrowing of the coronary artery that supply the heart with blood and oxygen. It occurs in times of HIGH demand for Oxygen (Exercise or Emotional Stress). If it goes untreated, ischemia or myocardial infarction can occur.

Risk Factors: Smoking, diabetes, High BP, High Cholesterol, sedentary lifestyle, obesity, family history, MEN>45 | WOMEN >55

| Dx: Coronary Angiography - CT scan with dye to see occlusion EKG + Echocardiogram LFT's Lipid Profile - Cholesterol Stress test to the heart Blood test to see risk for Myocardial Infarction | S/Sx: Chest Pain constricting that radiates, pressure to the jaws, arms, back. Depending on the severity: Nausea, pallor, SOB, diaphoresis, upper GI discomfort |
|---|---|
| Blood test to see risk for Myocardial Infarction | |

TREATMENT

1-<u>Immediate relief</u> - Nitroglycerin (dilates heart arteries to 1 blood flow

| Pill | PATCH |
|--|---|
| For stable angina 1 pill q5m (up to 3 doses) Call 911 if symptoms persist 5 min after 1st tablet Heat + Light sensitive. Don't leave in the car, and keep it in dark bottle Don't take with Sidenafil. HA and flushing are normal | For Unstable Angina Rotate daily Clean, dry, shaved area Shower ok Wear gloves |

2- Surgical: PCI- Stent in Artery | CABG-reroute around artery

3- Beta Blockers; CCB; -statins, anticoagulants

| STABLE | | UNSTABLE |
|---|---|--|
| 1- Occurs with exertion or stress | 1 | 1- Occurs with exertion, stress and REST |
| 2- Short duration – less than 5 min | | 2- Longer duration - > 30min indicative of Heart |
| 3- Sx of CP relieved by rest or Nitroglycerin | | attack |
| 4-Predictable | | 3- Unrelieved by medication or rest |
| | | 4- Unpredictable |



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| Nursing interventions for dysrhythmias - Defibrillation for ventricular tachycardia - Cardioversion for all other dysrhythmias - CPR for a client who is pulseless or not b - Lidocaine IV bolus for a client who has v | or ventricular fibrillation reathing entricular dysrhythmia | Patient teaching about prevention of athero - Smoking cessation - Maintain an appropriate weight - Eat a low-fat diet | sclerosis |
|---|--|---|-----------------------------|
| Cardiovascular dys- - Murmur: sustained swishing or blowing s flow through a valve, vessel, or heart cha - S4 (atrial gallop): involves an extra hear resulting from decreased ventricular comp - Pericardial friction rub: scratchy, high-pi infection, inflammation, or infiltration and | functions ound caused by turbulent bloc mber ~t sound that occurs before s liance tched sound associated with can be a manifestation of | Holter monitor - Records and transmits electrical impuls heart and alerts the nurse to dysrhythu myocardial injury, or conduction defects - Allows the client freedom of movement cardiac activity is recorded | es of the nias, while |
| pericarditis - S3 (ventricular gallop): extra heart sound immediately following S2, and is caused by decreased vascular compliance | | Cardiac catheterization - Hematoma formation nursing interventions - Greatest risk = bleeding - Apply firm pressure to stop bleeding | |
| Cardiac measurements - Cardiac output: heart rate times stroke - Echocardiogram: non-invasive ultrasound - Telemetry: detects the ability of cardiac muscle - Cardiac catheterization: measurement o | Volume, measures the amou procedure, evaluates heart cells to generate a spontane f coronary artery blood flow | nt of blood ejected by the heart over 1 minute ralve function and structure ous and repetitive electrical impulse through t | ne heart |
| Dopamine: give to client in cardiogenic sh force of contractions Increases blood pressure by causing v Nitroglycerine: vasodilator that decrease Decreases blood pressure Nitroprusside: vasodilator that decrease relax Decreases cardiac output Decrease blood pressure Morphine: opioid analgesic and vasodilato Decreases blood pressure | Cardiac medicat ock because produces inotropi (asoconstriction of blood vess es cardiac preload and afterlo es cardiac preload and afterlo r that can decrease cardiac p | ons c effect and improves cardiac output by streng els ad ad by causing the arterial and venous smooth v preload and afterload | .thening nuscles to |
| Instruct client to allow the tablets to d Moisten mouth if dry Onset of relief should begin 1-3 minutes If client's chest pain has not eased in Nitroglycerin is inactivated by heat, ligh- Nurse should instruct the client to kee Client should take the medication at the Instruct client to lie down after taking | Sublingual Nitrogl issolve under the tongue or b after administration 5 minutes, client should take t, and moisture p the medication in its origin onset of angina, regardless the medication because hypo- | Jeerin etween cheek and gums ; another tablet and call 911 al dark glass container with the lid closed tight of food intake tension can occur quickly, leading to dizziness and | ·ly d syncope |
| Angina Precipitating Factors: 4 E's Exertion: physical activity and exercise | Heart Murmur Causes: SP Stenosis of a valve | 4SM | |

Eating Emotional distress Extreme temperatures: hot or cold weather Stenosis of a Valve Partial obstruction Aneurysms Septal defect Mitral regurgitation



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Extra: Are there any extra or abnormal complexes?



mm/mV 1 square = 0.04 sec/0.1mV

PR Interval: 0.12-0.20

Normal Sinus Rhythm

Sinus Tachycardia

Sinus Bradycardia

Paroxysmal Supraventricular Tachycardia



Atrial Flutter

2nd Degree AV Block – Type I

1st Degree AV Block



2nd Degree AV Block – Type II





Ventricular Fibrillation

Ventricular Tachycardia



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| Arrhythmias | Description | | Treatment |
|------------------------------------|---|--|---|
| Sinus Arrhythmia | Irregular atrial and ventricular rhythms. Normal P wave preceding each QRS complex. | Normal variation of normal sinus rhythm in athletes, children, and the elderly. Can be seen in digoxin toxicity and inferior wall MI. | Atropine if rate decreases below 40bpm. |
| Sinus Tachycardia | Atrial and ventricular rhythms are regular. Rate > 100 bpm. Normal P wave preceding each QRS complex. | Normal physiologic response to fever, exercise, anxiety, dehydration, or pain. May accompany shock, left-sided heart failure, cardiac tamponade, hyperthyroidism, and anemia. Atropine, epinephrine, quinidine, caffeine, nicotine, and alcohol use. | Correction of underlying cause. Beta-adrenergic blockers or calcium channel blockers for symptomatic patients. |
| Sinus Bradycardia | Regular atrial and ventricular rhythms. Rate < 60 bpm. Normal P wave preceding each QRS complex. | Normal in a well-conditioned heart (e.g., athletes). Increased intracranial pressure; increased vagal tone due to straining during defecation, vomiting, intubation, mechanical ventilation. | Follow ACLS protocol for administration of atropine for symptoms of low cardiac output, dizziness, weakness, altered LOC, or low blood pressure. Pacemaker |
| Sinoatrial Block | Atrial and ventricular rhythms are normal except for missing complexes. Normal P wave preceding each QRS complex. Pause not equal to multiple of the previous rhythm. | Infection Coronary artery disease, degenerative heart disease, acute inferior wall MI. Vagal stimulation, Valsalva's maneuver, carotid sinus massage. | Treat symptoms with atropine I.V. Temporary pacemaker or permanent pacemaker if considered for repeated episodes. |
| Wandering Atrial Pacemaker | Atrial and ventricular rhythms vary slightly. Irregular PR interval. P waves irregular with changing configurations indicating that they aren't all from SA node or single atrial focus; may appear after the QRS complex. QRS complexes are uniform in shape but irregular in rhythm. | Rheumatic carditis due to inflammation involving the SA node. Digoxin toxicity Sick sinus syndrome | No treatment if patient is asymptomatic Treatment of underlying cause if patient is symptomatic. |
| Premature Atrial Contraction (PAC) | Premature, abnormal-looking P waves that differ in configuration from normal P waves. QRS complexes after P waves except in very early or blocked PACs. P wave often buried in the preceding T wave or identified in the preceding T wave. | May prelude supraventricular tachycardia. Stimulants, hyperthyroidism, COPD, infection and other heart diseases. | Usually no treatment is needed. Treatment of underlying causes if the patient is symptomatic. Carotid sinus massage. |

| Arrhythmias Description | | Causes | Treatment |
|---|--|--|--|
| Paroxysmal Supraventricular Tachycardia | Atrial and ventricular rhythms are regular. Heart rate > 160 bpm; rarely exceeds 250 bpm. P waves regular but aberrant; difficult to differentiate from preceding T waves. P wave preceding each QRS complex. Sudden onset and termination of arrhythmia When a normal P wave is present, it's called paroxysmal atrial tachycardia; when a normal present, it's called paroxysmal junctional tachycardia. | Physical exertion, emotion, stimulants, rheumatic heart diseases. Intrinsic abnormality of AV conduction system. Digoxin toxicity. Use of caffeine, marijuana, or central nervous system stimulants. | If the patient is unstable prepare for immediate cardioversion. If the patient is stable, vagal stimulation, or Valsalva's maneuver, carotid sinus massage. Adenosine by rapid I.V. bolus injection to rapidly convert arrhythmia. If a patient has normal ejection fraction, consider calcium channel blockers, beta-adrenergic blocks or amiodarone. If a patient has an ejection fraction less than 40%, consider amiodarone. |
| Atrial Flutter | Atrial rhythm regular, rate, 250 to 400 bpm Ventricular rate variable, depending on degree of AV block Saw-tooth shape P wave configuration. QRS complexes are uniform in shape but often irregular in rate. | Heart failure, tricuspid or mitral valve disease, pulmonary embolism, cor pulmonale, inferior wall MJ, carditis. Digoxin toxicity. | If a patient is unstable with ventricular rate > 150bpm, prepare for immediate cardioversion. If the patient is stable, drug therapy may include calcium channel blockers, beta-adrenergic blocks, or antiarrhythmics. Anticoagulation therapy may be necessary. |
| Atrial Fibrillation | Atrial rhythm grossly irregular rate > 300 to 600 bpm. Ventricular rhythm grossly irregular, rate 160 to 180 bpm. PR interval indiscernible. No P waves, or P waves that appear as erratic, irregular base-line fibrillatory waves | Heart failure, COPD, thyrotoxicosis, constrictive pericarditis, ischemic heart disease, sepsis, pulmonary embolus, rheumatic heart disease, hypertension, mitral stenosis, atrial irritation, complication of coronary bypass or valve replacement surgery | If a patient is unstable with ventricular rate > 150bpm, prepare for immediate cardioversion. If stable, drug therapy may include calcium channel blockers, beta-adrenergic blockers, digoxin, procainamide, quinidine, ibutilide, or amiodarone. Anticoagulation therapy to prevent emboli. Dual chamber atrial pacing, implantable atrial pacemaker, or surgical maze procedure may also be used. |
| Junctional Rhythm | Atrial and ventricular rhythms are regular. Atrial rate 40 to 60 bpm. Ventricular rate is usually 40 to 60 bpm. P waves preceding, hidden within (absent), or after QRS complex; usually inverted if visible. PR interval (when present) < 0.12 second QRS complex configuration and duration normal, except in aberrant conduction. | Inferior wall MI, or ischemia, hypoxia, vagal stimulation, sick sinus syndrome. Acute rheumatic fever. Valve surgery Digoxin toxicity | Correction of underlying cause. Atropine for symptomatic slow rate Pacemaker insertion if patient is refractory to drugs Discontinuation of digoxin if appropriate. |
| Premature Junctional Conjunctions | Atrial and ventricular rhythms are irregular. P waves inverted; may precede be hidden within, or follow QRS complex. QRS complex configuration and duration normal. | MI or ischemia Digoxin toxicity and excessive caffeine or amphetamine use | Correction of underlying cause. Discontinuation of digoxin if appropriate. |
| First-degree AV block | Atrial and ventricular rhythms regular PR interval > 0.20 second. P wave preceding each QRS complex. QRS complex normal. | Inferior wall MI or ischemia or infarction, hypothyroidism, hypokalemia, hyperkalemia. Digoxin toxicity. Use of quinidine, procainamide, beta-adrenergic blocks, calcium | Correction of the underlying cause. Possibly atropine if PR interval exceeds 0.26 second or symptomatic bradycardia develops. Cautious use of digoxin, calcium channel blockers, and zbeta-adrenergic blockers. |

| Arrhythmias | Description | Causes | Treatment |
|--|--|--|---|
| Second-degree AV block Mobitz I (Wenckebach) | Atrial rhythm regular. Ventricular rhythm irregular. Atrial rate exceeds ventricular rate. PR interval progressively, but only slightly, longer with each cycle until QRS complex disappears. PR interval shorter after dropped beat. | Severe coronary artery disease, anterior wall MI, acute myocarditis. Digoxin toxicity | Atropine, epinephrine, and dopamine for symptomatic bradycardia. Temporary or permanent pacemaker for symptomatic bradycardia. Discontinuation of digoxin if appropriate. |
| Third-degree AV block (complete heart block) | Atrial rhythm regular. Ventricular rhythm regular and rate slower than atrial rate. No relation between P waves and QRS complexes. No constant PR interval. QRS interval normal (nodal pacemaker) or wide and bizarre (ventricular pacemaker). | Inferior or anterior wall MI, congenital abnormality, rheumatic fever. | Atropine, epinephrine, and dopamine for symptomatic bradycardia. Temporary or permanent pacemaker for symptomatic bradycardia. |
| Premature ventricular contraction (PVC) | Atrial rhythm regular Ventricular rhythm irregular QRS complex premature, usually followed by a complete compensatory pause QRS complexes are wide and distorted, usually >0.14 second. Premature QRS complexes occurring singly, in pairs, or in threes; alternating with normal beats; focus from one or more sites. Ominous when clustered, multifocal, with R wave on T pattern. | Heart failure; old or acute myocardial ischemia, infarction, or contusion. Myocardial irritation by ventricular catheters such as a pacemaker. Hypercapnia, hypokalemia, hypocalcemia. Drug toxicity by cardiac glycosides, aminophylline, tricyclic antidepressants, beta-adrenergic. Caffeine, tobacco, or alcohol use. Psychological stress, anxiety, pain | If warranted, procainamide, lidocaine, or amiodarone I.V. Treatment of underlying cause. Discontinuation of drug causing toxicity. Potassium chloride IV if PVC induced by hypokalemia. Magnesium sulfate IV if PVC induced by hypomagnesaemia. |
| Ventricular Tachycardia ID#: 856/107213625 Mag87 21:42:8 II II Mag87 21:42:8 II Mag87 21:42:8 II Mag87 21:42:8 | Ventricular rate 140 to 220 bpm, regular or irregular. QRS complexes wide, bizarre, and independent of P waves P waves no discernible May start and stop suddenly | Myocardial ischemia, infarction, or aneurysm Coronary artery disease Rheumatic heart disease Mitral valve prolapse, heart failure, cardiomyopathy Ventricular catheters. Hypokalemia, Hypercalcemia. Pulmonary embolism. Digoxin, procainamide, epinephrine, quinidine toxicity, anxiety. | If pulseless: initiate CPR; follow ACLS protocol for defibrillation. If with pulse: If hemodynamically stable, follow ACLS protocol for administration of amiodarone; if ineffective initiate synchronized cardioversion. |
| Ventricular Fibrillation | Ventricular rhythm and rate are rapid and chaotic. QRS complexes wide and irregular, no visible P waves | Myocardial ischemia or infarction, R-on-T phenomenon, untreated ventricular tachycardia, Hypokalemia, hyperkalemia, Hypercalcemia, alkalosis, electric shock, hypothermia. Digoxin, epinephrine, or quinidine toxicity. | If pulseless: start CPR, follow ACLS protocol for defibrillation, ET intubation, and administration of epinephrine or vasopressin, lidocaine, or amiodarone; ineffective consider magnesium sulfate. |
| Asystole | No atrial or ventricular rate or rhythm. No discernible P waves, QRS complexes, or T waves | Myocardial ischemia or infarction, aortic valve disease, heart failure, hypoxemia, hypokalemia, severe acidosis, electric shock, ventricular arrhythmias, AV block, pulmonary embolism, heart rupture, cardiac tamponade, hyperkalemia, electromechanical dissociation. Cocaine overdose. | • Start CPR. |

















AV BLOCK SECOND AND THIRD DEGREES



Integumentary Disorders



HERPES ZOSTER

P: Reactivation of the varicella zoster virus from previous Chickenpox infection

S/Sx: Red skin vesicles along areas of sensory nerves.

N: Isolate patient in contact precautions, prevent scratching - irritation or vesicles. Admin antivirals as ordered

STEVENS - JOHNSON SYNDROME

P: A skin reaction caused by an immunological response to taking certain medications S/Sx: Vesicles, erosions, flulike symptoms and redness N: Discontinue the medication that is causing the reaction. Administer antibiotics and corticosteroids as ordered

PSORIASIS

P: Chronic, non-contagious inflammation of the skin due to over-keratinization Ed: Teach client to avoid scratching and wear nonirritating clothing like cotton. Ensure pt. doesn't use any OTC meds without approval.

FROSTBITE

P: Damage to tissue/vessels as a result of prolonged exposure to cold S/Sx: White plaque around redness, blisters, bluish skin and numbness of extremities Tx: Rewarm slowly with moist heat + monitor CMS and for signs of compartment syndrome

PRESSURE INJURIES

Pressure against a vessel near the skin prevents adequate blood flow and causes skin breakdown (especially near pony areas)

- Stage 1: Non-Blanchable but intact/unbroken skin

- Stage 2: partial-thickness injury, extends up to epidermis or dermis.

- Stage 3: full thickness injury extends past dermis FAT visible.

- Stage 4: full thickness injury extends past subcutaneous/ BONE visible.

- Unstageable: unable to see thickness layers due to excess exudate.

- Wound healing is promoted by a diet that is rich in protein and Vitamin C.



Stage 3



Stage 4



Stage 2



PRIMARY LESIONS



MACULE Flat area of color change (no elevation or depression)

Example: Freckles



PAPULE Solid elevation less than 0.5 cm in diameter

Example: Allergic eczema



NODULE Solid elevation 0.5 to 1 cm in diameter. Extends deeper into dermis than papule

Example: Mole

TUMOR Solid mass—larger than 1 cm

Example: Squamous cell carcinoma



PLAQUE Flat elevated surface found on skin or mucous membrane

Example: Thrush



WHEAL Type of plaque. Result is transient edema in dermis

Example: Intradermal skin test



VESICLE Small blister—fluid within or under epidermis

Example: Herpesvirus infection



BULLA Large blister (greater than 0.5 cm)

Example: Burn

PUSTULE Vesicle filled with pus

Example: Acne

SECONDARY LESIONS



SCALES Flakes of cornified skin layer

Example: Psoriasis

Dried exudate on skin Example: Impetigo

CRUST

1-1

FISSURE Cracks in skin

Example: Athlete's foot



ULCER Area of destruction of entire epidermis

Example: Decubitus (pressure sore)



SCAR Excess collagen production after injury

Example: Surgical healing



ATROPHY Loss of some portion of the skin

Example: Paralysis







Classification of Burns:

Cold: Frostbite Thermal: liquid, steam, fire Radiation: Dun, Radiation Chemical: powder, gas (inhalation injury) Friction: Road rash Electric: usually have an entry or exit wound. Injuries may be internal



NURSING CARE

- Ensure patient Tetanus shot if >5-10 years
- Watch for temperature loss = shivering
- Pain control IV rout (best)
- Wound Care Premedicate
- debridement remove necrotic tissue
- No pillows for the ear or neck. Use rolled towel under shoulder
- Watch for Webbing



System used to estimate the percentage of body surface involved in a burn injury, and to estimate the severity of the burn

Rule of 9's - Burns



Once stablished the total body surface area 70 burned, we use the Parkland Burn Formula, for 2^{nd} and 3^{rd} Degree Burn

4mL x TBSA % x weight (Kg)

1st HALF of the Solution, over the 1st 8 Hours

2nd HALF of the Solution, over the next 16 Hours

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*TBSA90 - Total Body Surface Area Burned

Renal & Uninary Disorders

PYELONEPHRITIS

Patho: Inflammation of the renal pelvis caused by bacterial infection

S/SX: Fever, chills, tachypnea, nausea, flank pain, frequency, urgency, cloudy urine, hematuria

N: Monitor temperature, Encourage increased fluid intake and decreased protein. Administer analgesics, antipyretics. Monitor I/O + Weight

HYDRONEPHROSIS

Patho: Distension of the renal pelvis caused by an obstruction. Trapping urine proximally

S/Sx: Colicky or dull flank pain that radiates to the grain. Headache + hypertension

N: Monitor vitals, electrolytes, specific gravity, and dehydration

BENIGN PROSTATIC HYPERTROPHY

Patho: Slow enlargement of the prostate gland that can compress the urethra

S/Sx: Diminished urinary stream, Nocturia, urgency, hematuria, retention, dysuria, bladder pain

N: Increase fluid intake. (2-3L/day) Encourage patient to decrease caffeine + artificial sweetness intake. Educate about a timed voiding schedule

RENAL CALCULI

Patho: Stones that form in the urinary tract

S/Sx: Severe intermittent pain, nausea, vomiting, low-grade fewer, hematuria

N: Monitor temperature, encourage increased fluids, apply heat to flank area, diet modification

C: Scar tissue formation, infection and obstruction

POLYCYSTIC KIDNEY DISEASE

Patho: Cyst formation and hypertrophy of the kidneys causing scar tissue, infection, nephron damage

S/SX: Flank or lumbar pain that worsens with activity + improves upon lying down, Hematuria, proteinuria, recurrent UTI

N: Monitor for hematuria which could indicate a rupture. Increase sodium + water intake. Educate about possible need for surgical interventions



Patho: Slow progressive loss of kidney function resulting in uremia and hypervolemia - the inability to conserve sodium and water

Chronic Kidney Disease

S/Sx: Polyuria, decreased skin turgor, edema, diluted urine, proteinuria

N: low protein, potassium, high phosphorus diet. Educate about fluid restriction and possible dialysis treatment

R: Diabetes, Hypertension, AKI, Recurrent Infections, Renal Occlusions





Acute Kidney Injury

Sudden loss of kidney ability to regulate volume, remove waste products, release hormones or maintain body's acid-base balance.

CAUSES

- Prolonged Renal Ischemia
- Nephrotoxic Injury leading to tubular necrosis
- INTRARENAL Injury occurring from disease within the kidneys Causes:
 - Acute Tubular Nephritis
 - Nephritis
 - Nephrotoxic Injury
 - Acute Glomerular Nephritis
 - Thrombolytic Disorders
 - Malignant Hypotension
 - SLE
 - Infection

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Initiation Phase: Onset of Injury / Onset of symptoms

Oliguria Phase: Decrease urine output to 400ml/day, usually 1-7 days after injury

Diuretic Phase: Increase urine output to 1-3L/day, caused by inability to concentrate

Trisk of hyponatremia, hypokalemia, dehydration

Recovery Phase: Increase in filtration rate, BUN/Creatinine

- Decreased cardiac output POSTRENAL

PRERENAL

the kidneys.

Causes: - Vasoconstriction

- Hypotension

- Hypovolemia

Caused by a reduced blood flow to

Occurs when there is an obstruction of urinary flow causing intraluminal pressure

Causes:

- BPH
- Bladder Cancer
- Calculi
- Prostate Cancer
- Trauma

PHASES

Musculoskeletal Disorders

RHEUMATOID ARTHRITIS

- P: An autoimmune response that causes deformities
- S/Sx: Fatigue, anorexia, stiffness, weight loss Event may trigger: Childbirth, Infection, Stress Permanent deformity, Symmetrical
- D: Rheumatoid factor-Blood Test + >60 u/mL
 - \uparrow Erythrocyte sedimentation rate (ESR) -non specific CRP, ANA
 - Radiology showing joint space deterioration
- C: Nodular myositis, contractures Sjogren's syndrome, cataracts
- Tx: Surgery to restore function



Normal

Rheumatoid Arthritis

OSTEOPOROSIS

- P: Bone demineralization caused by loss of calcium and phosphorus. Bone resorption occurs faster than bone formation
- S/Sx: Loss of bone density and easily fractured bones
- N: Encourage a well balanced diet high in protein, calcium, iron, vit D + C

OSTEOARTHRITIS

- P: On inflammatory degeneration, gradual loss of articular cartilage. Asymmetrical
- R: trauma, aging, obesity, smoking, lestrogen, genetic changes
- S/Sx: Exacerbated by temperature + climate changes. Joint space narrowing. One leg shorter than other. Pain is increased after rest
- T: Regular exercise -> preventative. Avoid prolonged standing, kneeling and squatting. Apply cold for inflammation / Heat for stiffness

HERNIATED DISK

P: A vertebral disk slips out of place which can cause pain due to compression of spinal nerve
R: Numbness and tingling in back and extremities. Severe pain.

Tx: Surgery to realign vertebra, physical therapy and adjustment by a chiropractor can alleviate pain but doesn't fix the herniation



GOUT

P: Uric acid crystals build up in joints and body tissues. Can result from poor metabolism of purine

S/Sx: Swelling + inflammation of joints, low grade fever, malaise, itchiness + pain at joints

N: Low purine diet, increase fluid intake.

Ed: Instruct client to avoid alcohol and excessive use of the joint

FRACTURES

- 1- Transverse: A break that is perpendicular to the long axis
- 2- Comminuted: The bone fragments into pieces
- 3- Open / Compound: Part of the bone is through skin
- 4- Greenstick: The bone is splintered on one side

CASTS

N: Elevate for 24-48 hours to promote venous drainage. Allow plasters casts to dry for 24-72 Hours Ed: Instruct client to report skin irritation and hot spot

TRACTION

N: Ensure weights are freely hanging + off the floor. Assess skin integrity frequently with skin traction

FRACTURE COMPLICATION

Fat Embolism: Altered mental status, impaired respiratory function, decreased perfusion distal to embolus site.

Compartment Syndrome: Pressure is an extremity that can't escape, i.e., under a cast. Numbress + tingling, pain that increase with elevation, Pallor, pain W/ Movement

JOINT INJURIES

Sprains: The ligament connecting two bones becomes torn or stretched sTrains: The muscle or Tendon attached to a bone becomes injured or over stretched

AMPUTATION

Ensure residual limb sock is worn at all times, position is prone position as prescribed. Educate patient about cleaning prosthesis socket daily.

Above Knee: Prevent internal and external rotation of the hip Below Knee: Discourage long period of sitting to reduce Flexion. Don't allow limb to dangle







Musculoskeletal Disorders64





Active range of motion: Can move joint without assistance Passive range of motion: Can only move w/ assistance Goniometer: Measures range of motion of a joint

Muscle Strength Scale

- O = No muscle contraction
- 1 = A barely detectable contraction
- 2 = Active muscle contraction without gravity
- 3 = Active muscle contraction against gravity
- 4 = Active muscle contraction against some resistance
- 5 = Active movement against full resistance

Abnormalities

Atrophy: Decrease Size / Strength of a muscle Ankylosis: Stiffness at a joint Kyphosis: Thoracic curvature of spine Myalgia: General Muscle Pain / Tenderness Scoliosis: Asymmetrical elevation of shoulders Paresthesia: Pins + Needles Lordosis: Excessive inward curve of spine (pregnancy)





Scoliosis

Kyphosis

Lordosis

Diagnostics

X – Ray: Remove any radiopaque obj. CT scan: Verify no shellfish allergy if contrast dye is used Bone Scan: Ensure bladder is empty

ASSESS

- Joints + muscles for crepitation or tenderness

- Muscle strength
- -Range of motion

Fall Prevention

- Eliminate scatter rugs
- Use supportive shoes that have good grip
- Use a walker or cone for support

Musculoskeletal Disorders65

SPORT RELATED INJURY

Impingement Syndrome

Soft tissue/nerves trapped under coracoacromial arch Give: NSAIDS, Rest, ROM + Strengthening

Rotator Cuff Tear -

Rest, NSAIDS + Strengthening + Surgery if Severe

Shin Sprints Periostitis in calf -> ice, stretching + supportive shoes



Tendonitis Inflammation of a tendon -> Rest, Ice, NSAIDS, brace, gradual return

Meniscus Injury Injury to fibrocartilage discs in knee -> R.I.C.E and arthroscopic surgery PRN

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DISLOCATION / SUBLUXATION

Dislocation: Complete displacement or separation of the articular surfaces of a joint Subluxation: Partial or incomplete dislocation

Nursing Care

Dislocation is an orthopedic emergency r/t the risk of vascular injury. Assist with realignment and pain management. Physical therapy and Rom exercise are imperative to achieve full recovery

1- Normal

2– Subluxation

3- Dislocation



Fractures

COMMON FRACTURE TYPES

Colles' Fracture: Fracture of the distal radius

TX: Closed reduction

Long Bone Fracture

TX: Immobilization, traction, int./ext. fixation

Hip Fraction

TX: Hip compression screw, partial replacement or total replacement

N: encourage early ambulation, assess color, temperature, cap refill, pulses, edema,

sensation motor function + pain, do not position on the affected side. Do not allow > 90° knee flexion

Ed: Teach pt. to avoid crossing legs, internally rotate hip or sit in the low chairs Stable Vertebral Fracture

TX: Immobilize spine, evaluate existence of cord damage, pain meds, kyphoplasty

FRACTURE HEALING



POSSIBLE COMPLICATIONS

Infection: A serious complication Tx: Antibiotics + surgical debridement Compartment Syndrome

Swelling causes increased pressure that can compromise nerves and blood vessels.

S/Sx: pain, pressure, paresthesia pallor, paralysis,

pulselessness. Cool skin at extremities

 $\top x$: Do not elevate or apply cold.

Fat Embolism

Fat globules from the fracture travel to the lungs, blood vessels or other organs

S/Sx: tachypnea, cyanosis, dyspnea, and low O_2 sat.

- T_{X} : Fluid resuscitation, blood transfusion, intubation
- N: encourage cough + deep breathe, provide O_2 therapy

NURSING CARE OF THE ORTHOPEDIC PATIENT



TRACTION

Pulling force to an affected extremity

- -Reduces muscle spasm
- Immobilizes
- -Reduces a fracture
- Can treat pathologic joint conditions

Skin Traction

- short term (48-72 hours)
- reduce muscle spasms
- applied directly to the skin
- 5-10 pounds

NURSING CARE

- 1. Ensuring traction weights never touch the floor
- 2. Keep patient in the correct body alignment to enhance traction
- 3. Assess for S/Sx of Compartment Syndrome
- 4. If pulleys are being used, make sure knots have enough slack
- 5. Check skin integrity around pins or skin traction site frequently
- 6. Apply ice to prevent swelling
- 7. Suggest the use of a hairdryer on cool to help relieve itching
- 8. Teach pt. importance of keeping proximal joints mobile
- 9. Ensure pt. never inserts any objects inside the cast

Skeletal Traction

- Long term (>72 hours)
- alignment of bone
- pins or wires are surgically inserted into the bone
- 5-45 pounds

Possible Complications

Atrophy: teach isotonic muscle strengthening Muscle Spasms: heat application reduces spasms

Contracture: reposition frequency + provide ROM **Pain**: determine / treat underlying cause

CASTS

A device used for long term immobilization / Allows freedom to perform most ADLS Hip spica cast: used for femur fx in children Body jacket brace: used for stable spiral spinal injury

NURSING CARE

- Never cover a plaster cast until it's dry because the heat will build up and cause a burn
- Handle with an open palm to avoid denting
- Ensure edges of cast are smooth to avoid skin irritation or breakdown
- Check color, temperature, cap. refill and pulses
- monitor for S/Sx of compartment syndrome
- S cast on a lower extremity should be elevated for the first 24hrs after application
- When a sling is used, ensure the axillary area is well padded.

IRON DEFICIENCY

- E: Inadequate diet, malabsorption, blood loss, hemolysis microcytic & hypochromic
- L: 1Hgb, 1Hct, 1MVC, 1MCH 1MCHC retic. Serum iron, TIBC
- S/Sx: Pallor, glossitis, Cheilitis, black stool
- T: Replace iron, transfusion, diet teaching, emphasize compliance
- R: Pregnancy, premenopausal women, blood loss, older adults, low socioeconomic backgrounds

COBALAMIN ≥B12€ DEFICIENCY

E: Impaired DNA synthesis, GI surgery, ETOH, Smoking, *Gastric bypass, PPI use.

- L: 1B12, macrocytic RBCs, MCV >100
- S/Sx: Neurological tingling, paresthesia, beefy tongue, weakness
- T: B12 injection or intranasally 1/week

SICKLE CELL DISEASE

- E: Genetically Autosomal Recessive
- L: Sickled RBC
- S/Sx: Occlusions, necrosis, perfusion, pain on exertion
- **T**: Avoid \uparrow Altitude + \uparrow Temp, bone marrow transplant, O₂ therapy

FOLIC ACID DEFICIENCY ANEMIA

- E: Celiac, Crohn's, alcoholism, hemodialysis, malabsorption
- L: Macrocytic (MCV > 100) 1 folate
- S/Sx: Weakness, fatique, bruising, No neuro symptoms, weight loss
- T: Replacement (green leafy veg) 1 mg/day tablet

APLASTIC ANEMIA

ΡΑΝCУΤΟΡΕΝΙΑ

E: Infection or Autoimmune

L: JPlatelets TNR + TPT/PTT

L: JRBC JWBC JPlatelets

- S/Sx: Respiratory Fatigue, Weakness
- T: Transfusion, *twBC*, Bone Marrow Transplant

THROMBOCYTOPENIA

S/Sx: Prolonged bleeding time. T: Platelet Transfusion, Bone Marrow Transplant or Corticosteroid Treatment.

N: Avoid lacerations - use electric razors, monitor Hgb, Hct and bleeding times.



latelets

Aplastic Anaemia

White Blood Cell







Red Blood Cell

MEGALOBLASTIC





Antigens on the blood Identifies the cell

Antibodies protect the cell from certain antigens.



NO antigen [AB antibodies] Donates to: A, ABReceives from: A, O

BLOOD TYPE

Donates to: AB, BReceives from: B, O



Donates ONLY to: AB Receives from: **Universal Recipient**

Donates: **Universal Donor** Receives ONLY from: O

BLOOD PRODUCTS



Contains:

RBC WBC Platelets Plasma Uses

To Increase Oxygen Carrying capacity. Restoration of Blood Volume





Contains:

Plasma

Fresh Frozen

1 Unit=250mL

Uses

Bleeding, r/t coag. factor deficiencies, DIC, Hemorrhage, Vit K Deficiency, Liver disease, Anticoagulated patients.



Packed RBC 1 Unit=250mL Replaces 500mL Loss Will ↑ HgB 190, HcT 390 Increase RBC mass Symptomatic Anemia



Platelets 1 Unit=50mL Rapid Infusion ↑ Platelets by 10,000/ Units To Prevent / Control Bleeding



Cryoprecipitate G pooled units prepared from Plasma, contains clotting factors Significant hypofibrinogenemia. Hemophilia. Excessive anticoagulation DIC Von Willebrand's



ashe

Albumin Moves water – intravascular space Infuse Slowly 5% Isotonic 25% Hypertonic

Washed RBC Rinsed w/ 1-3L of NS Hypovolemia Shock Burns Peritonitis Pancreatitis Post-Op Albumin Loss

Adverse Effects

- Pulmonary Edema
- CHF Precipitation
- -HTN
- Anaphylaxis
- Hypervolemia
- Tachycardia

Given when there is an anticipated risk of Reaction

Blood Transfusion Therapy Notes Real

Overview

Used to replace blood volume, preserve oxygen-carrying capability, or increase coagulation capabilities; autologous blood transfusions: donating your own blood before anticipated surgery; religious considerations.

Initiating a Blood Transfusion

Type and cross-matching; informed consent; infuse each unit of blood within 2 to 4 hours; begin with normal saline with Y administration set to prime tubing; do not infuse any solution containing dextrose (causes blood to lyse or be destroyed); inspect for leakage, unusual appearance (bubbles or purplish color indicate contamination); roller clamp; remain with patient for first 15 to 20 minutes; after transfusion, flush tubing with normal saline; if giving more than one unit, use fresh tubing.

Blood Transfusion Reactions

"Not feeling right," sense of impending doom, chills, fever, low back pain, pruritus (itching), hypotension, nausea and vomiting, decreased urine output, back pain, chest pain, wheezing, dyspnea (BRONCO CONSTRICTION); stop infusion immediately; infuse normal saline solution with new tubing then call provider; keep remaining blood product and send it back to pharmacy, lab, or blood bank for analysis; document all findings; reactions generally happen within first 15 minutes but some reactions occur GO to 90 minutes or days to weeks later; asses for circulatory overload.
LOW BACK PAIN DUE TO KIDNEY PAIN/ENLARGEMENT. (SYSTEM WIDE INFLAMMATION) LOW BACK PAIN = BAD
FEVER IS A SIGN OF INFLAMMATION & INFECTION

Nursing Process

Data Collection / Patient Problems

- Assess risk for fluid, electrolyte, and acid-base imbalances and presence of alterations; monitor vital signs, height, weight, neurological function, intake and output, laboratory studies, past and present medical history, medication history.

- The RN will choose the patient problem such as "compromised blood flow to tissue," "inadequate fluid volume," etc. The LPN must act accordingly.

Expected Outcomes and Planning / Goal / Outcomes

- Prioritize fluid, electrolyte, and acid-base balance.

- Baseline normal vital signs, normal skin turgor, moist mucous membranes, baseline weight, no edema, clear breath sounds; normal urine electrolytes ABG's, intake and output.

Implementation / Evaluation / Goal / Evaluative Measures

- Prevention of fluid, electrolyte, and acid-base imbalances.

- Obtain daily weight, vital signs, intake and output; auscultate lung sounds, check oral mucous membranes, check tissue turgor, monitor serum electrolyte levels.


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POLYCYSTIC OVARIAN SYNDROME = PCOS

P: Abnormalities with the metabolism of androgens and estrogen S/Sx: Hirsutism, Infertility, Diabetes, Sleep Apnea, Obesity and menstrual dysfunction Tx: Diet, Exercise, Weight loss, Oral contraceptives, anti-androgens, hypoglycemic agents

ENDOMETRIOSIS

P: The endometrium lining the uterus growth in places it should not which can cause cramping or infertility

S/Sx: Intense pelvic pain. Painful intercourse, diagnosis is confirmed by laparoscopy Tx: Monitor for S/Sx of anemia during menses, educate about the importance of annual exams, help patient relieve painful cramp with ordered meds and heat compress

PELVIC INFLAMMATORY DISEASE

P: Infection of the reportative system usually caused by STDs S/Sx: Pelvic pain, fever, discharge, cramping, painful menses

Tx: Antibiotics, education about using protection

VARICOCELE

P: An enlargement of the veins in the scrotum caused by blood pooling in veins S/Sx: A Dull, recurring pain in the scrotum, visibly large and twisted veins, a lump or swelling N: Encourage pt to wear supportive underwear or jock strap.

STERILITY

P: Inability to reproduce as a result of various causes including low sperm count, chromosomal abnormalities or inadequate hormones

 T_x : Hormone replacement, fertility drugs, surgery, artificial insemination, Psychosocial counselling to help pt. develop coping methods

ERECTILE DYSFUNCTION

P: Inability to keep an erection long enough for sexual intercourse Tx: Vasodilator or hormone therapy, Smoking Cessation

CARCINOMA

P: Any cancer originating in the epithelium

S/Sx: A growing lump with a crusty surface, slow growing flat patch of redness

R: Overexposure to sun, repetitive irritation, genetic predisposition, lighter skin, older than 60 years

SARCOMA

P: Cancer originating in the connective tissues

S/Sx: Visible lump or mass in the soft tissue

R: Lymphedema. Von Willebrand disease. Genetic predisposition

MELANOMA

P: A cancer originating in melanocytes which are located in the basal layer of epithelium S/Sx: New marks on skin, mole that changes shape or size, new pigments of the skin

LEUKEMIA

P: Cancer of blood-forming cells. Either acute or chronic S/Sx: Prevent infection by avoiding invasive procedures such as catheterizations and injections. Prevent excessive bleeding due to possible low platelet count

GENERAL NURSING INTERVENTIONS

- Treat nausea, educate about carbohydrate \downarrow for prevention
- Maintain meticulous infection control for yourself, the patient and visitors
- Provide non pharmacological and pharm pain control

TREATMENTS

Surgery: Tumor is removed or destroyed

Radiation: Localized destruction of cancer

cells. Can cause local irritation + fatigue

Chemotherapy: Kills + stops the

reproduction of neoplastic cells.

-Skin, hair, nail, GI cells also impacted

WARNING SIGNS

- Change in bowel/bladder
- Any sore that doesn't heal
- Unusual bleeding/discharge
- Thickening or lumps
- Indigestion
- Obvious skin changes

- Nagging cough/hoarseness <u>NursingStoreRNbyANA</u>

WNG CANCER





SMALL CELL LUNG CANCER >NSCLC

- Usually begin in the bronchi
- Spread more quickly than NSCLC
- Early metastasis to Lymph
- Poorest Prognosis
- Survival Rate of 12-18 months
- Staging not useful due to
- aggressive nature

NON-SMALL CELL LUNG CANCER SNSCLCS

Adenocarcinoma

- Associated with scarring (chronic fibrosis)
- -Resection attemptable
- Most common in non-smoker

Squamous Cell

- Slow growing
- -Resectable
- Often causes Bronchial
- Obstructions

Large Cell

- Associated with Tobacco abuse
- Highly metastatic
- High reoccurrence
- Surgery not attempted

Occult-stage:

Cancer cells are found in sputum, but no tumor can be found in the lung by imaging tests or bronchoscopy, or the tumor is too small to be checked.

Stage D

Cancer at this stage is also known as carcinoma in situ. The cancer is tiny in size and has not spread into deeper lung tissues or outside the lungs.

Stage I

Cancer may be present in the underlying lung tissues, but the lymph nodes remain unaffected.

Stage II

The cancer may have spread to nearby lymph nodes or into the chest wall.

Stage III

The cancer is continuing to spread from the lungs to the lymph nodes or to nearby structures and organs, such as the heart, trachea and esophagus.

Stage IV

The most advanced form of the disease. In stage IV, the cancer has metastasized, or spread, beyond the lungs into other areas of the body.







Wedge Resection

Removal of "wedge" of lung tissue

Segmentectomy - Segmental Resection

A portion of the lung is removed. Larger than a wedge, while leaving a portion of the lobe.

Lobectomy

Removal of a Single Lobe

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Pneumonectomy

Removal of the entire lung.

Post Op Consideration – Place pt. on operative side to facilitate expansion of remaining lung.

Lab Values

| Electrolytes: | | Arterial Blood Gases (ABGs) |
|---|---|--|
| Sodium (Na ⁺): 135-145 mEq/L | | PH: 7.35-7.45 |
| Chloride (Cl ⁻): 98-106 mEg/L | | PaCO2: 35-45 mmHg |
| Calcium (Ca^{2+}) ; 9-10.5 mg/dL | | PaO2: 80-100 mmHg |
| Potassium (K+): 3.5-5.0 mEg/L | | HCO3: 22-26 mEq/L |
| Phosphate (PO_4) : 3-4.5 mg/dL | | SaO2: 95-100%; <95% Indicates Hypoxemia |
| Magnesium (Mg ²⁺): 1.5-2.5 mEg/L | | |
| | | WBC Differential Count: |
| | | Neutrophils: 55-70% |
| Anticoagulant Therapy Coagulation Times Therapeutic INR: 2-3 sec | | Lymphocytes (T & B Cells): 20-40% |
| | | Monocytes: 2-8% |
| | | Eosinophils: 1-4% |
| (<u>NOT MAL KANGE: U.D-1.1</u>) | | Basophils: 0.5-1.5% |
| F1: 11-12.5560 | | Liver Function Tests |
| 101015. 190,000 | - 400,000 mm | Albumin: 3,5-5,0 a/dL |
| Liver Enzymes | BMIRanges | Ammonia: 10-80 ma/dL |
| ALT: 4-36 u/L AST: 0-35 u/L ALP: 30-120 u/L | Underweight: <18.5 Healthy: 18.5-24.9 Overweight: 25-29.9 Obese: >30 | Total bilirubin: 0.3-1.0 ma/dL |
| | | Indirect/unconjugated bilirubin: 0.2-0.8 ma/dL |
| | | Total protein: 6-8 a/dL: |
| | | Prealbumin: 19-38 ma/dL |
| Bland Glusses La | (alc | Tutake & Output (TRO) |
| DIDOU GIUCOSE LEVEIS | | Eluid intake: 2,000,3,000 ml (day |
| Church (TASTING): 70-110 Mg/AL | | Taily using autouts 1200 1 EDD mal (day |
| (11405412702 NEMOGIODIN (HDH10): 4-600 | | Laurely unive output: 1,200-1,300 ML/May |
| Thyroid | | for > 2 concecutive loover = CONCERNIL |
| T3: 70-205 na/dL | | Polyuria (concictently laida uring valume) |
| T4: 4-12 mca/dL | | Polyaria (consistenting high arme volume): |
| Thyroid Stimulating Hormone (TSH): 2-10 mU/L | | >2,000-2,500 ML/aay |
| | | Therapeutic Medication Monitoring |
| | | Digoxin level: 0.8-2.0 ng/mL |
| Urinalysis Specific gravity: 1.005-1.030 Bratein D. D. G. maldu | | Lithium level: 0.4-1.4 mEq/L |
| | | Phenobarbital: 10-40 mcg/mL |
| Glucose: 50-300 malday | | Theophylline: 10-20 mcg/mL |
| рн: 4.6-8 | | Dilantin: 10-20 mcg/mL |
| | | Carbamazepine level: 4-10 mcg/mL |
| | | Valproic Acid level: 50-100 mcg/mL |
| | | II |







Bed Positions

Fowler's: A bed position where the head and trunk are raised, typically between 40-90°. This position is often used for patients who have cardiac issues, trouble breathing, or a nasogastric tube in place. Lateral: This position involves the patient lying on either her right or left side. Right lateral means the patient's right side is touching the bed, while left lateral means the patient's left side is touching the bed. A pillow is often placed in between the leas for patient comfort. Lithotomy: This position involves the patient lying flat on her back with legs elevated to hip level or above, often supported by stirrups. It is commonly used For gynecological procedures and childbirth. Prone: A position where the patient lies on his stomach with his back up. The head is typically turned to one side. This position allows for drainage of the mouth after oral or neck surgery. It also allows for full flexion of knee and hip Reverse Trendelenburg: The patient is supine with the head of the bed elevated and the foot of the bed down. This position may be used in surgery to help promote perfusion in obese patients. It can also be helpful in treating venous air embolism and preventing pulmonary aspiration. Sim's Position: A prone/lateral position in which the patient lies on his side with his upper leg flexed and drawn in towards the chest, and the upper arm flexed at the elbow. Sim's position is useful for administering enemas, perineal examinations, and for comfort in pregnancy. Supine: A position where the patient is flat on his back. Supine is considered the most natural "at rest" position, and is often used in surgery for abdominal, Trendelenburg: his position involves a supine patient and sharply lowering the head of the bed and raising the foot, creating an "upside down" effect. In the past, this position was frequently used to treat hypotension, although this has fallen out of favor in recent years due to studies showing it to be ineffective

Orthopheic or tripod position places the patient in a sitting position or on the side of the bed with an overbed table in front to lean on and several pillows on the table to rest on. Widely used for patient with COPDS. This position helps to open up lungs.

and potentially dangerous. It is helpful during gynecological and abdominal hernia

joints.

facial, and extremity procedures.

surgeries, and in the placement of central lines.

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STEPS TO ABG ANALYSIS:

- 1- Look at the pH (7.35 7.45)
 If the pH is HIGH, this is ALKALOSIS
 If the pH is LOW, this is ACIDOSIS
 Look at the PaCD2 (35 45) PaCD2 Respiratory
 If PaCO2 is HIGH, this is ACIDOSIS
 If PaCO2 is LOW, this is ALKALOSIS
 Look at the HCD3 (22 26) HCO3 Metabolic
 If HCO3 is HIGH, this is ALKALOSIS
- If HCO3 is LOW, this is ACIDOSIS

Interpret

Step 1: Analyze the pH. It will tell you ACIDOSIS or ALKALOSIS

Step 2: Analyze the Paco2 and the HCO3

- Is PaCO2 below 35? It is Alkalotic. Above 45 it is Acidic

- Is HCO3 below 22? It is Acidic. Above 26 it is Alkalotic

Step 3: Match the PacO2 or the HCO3 with the PH

For example, if the pH is acidotic, and the PaCO2, then the Acid-Base disturbance is being caused by the respiratory

system. Therefore, we call it Respiratory Acidosis

Step 4: Does the PaCO2 or the HCO3 go the opposite direction of the PH?

If so, there is compensation by the systems. For example, if the pH is acidotic, and the PaCO2 is acidotic, and the HCO3 is

alkalotic.

If they don't go the opposite direction, It is UNCOMPENSATED

Step 5: Is the pH in normal range? Fully Compensated / Partially Compensated / Uncompensated

If there is Compensation, and the pH is in normal range (7.35-7.45), then it is Fully Compensated

If there is Compensation, and the pH is out of range, then it is Partially Compensated Step 6: Are the pO2 and the O2 saturation normal?

If they are below normal, there is evidence of Hypoxemia

| PH: 7.35 - 7.45 | | | |
|---------------------------|--|--|--|
| PACO ₂ 35 - 45 | | | |
| HCO ₃ 22 - 26 | | | |
| PAO ₂ 80 - 100 | | | |
| 0 ₂ SAT >95% | | | |

1- Practice Question

A 72 yr. old with pneumonia. pH - 7.31 (Acidic)

PaCO2 – 60 (Acidic)

HCO3 - 34 (Alkalotic)

PO2 - 50 (LOW)

| РН: | 7.35 - 7.45 |
|-------------------|-------------|
| PACO ₂ | 35 - 45 |
| HCO₃ | 22 - 26 |
| PAO ₂ | 80 - 100 |
| 02SAT | >95% |

#1 - pH is below 35, so It is Acidosis #2 – Who is doing the same as the pH (Acidic)? PaCO2 It is Respiratory #3 - Does the HCO3 go in opposite direction as the pH? YES - Alkalotic So, there is Compensation #4 - Is the pH in normal range? NO So, it is Partially Compensated #5 – Is the pO2 in normal range? NO The patient has Hypoxemia The full Diagnosis is: Partially Compensated Respiratory Acidosis with Hypoxemia 2- Practice Question A 20 years old, acute renal failure PH - 7.18 (Acidic) Paco2 - 44 (Normal) HC03 - 16 (Acidotic) #1 - pH is below 35, so It is Acidosis PO2 - 92 (Normal) #2 – Who is doing the same as the pH (Acidic)? HCO3 It is Metabolic #3 - Does the PaCO2 go in opposite direction as the pH? NO So, there is NO Compensation #4 - Is the pH in normal range? NO

So, it is Uncompensated

#5 – Is the pO2 in normal range? YES

The patient doesn't have Hypoxemia

The full Diagnosis is:

Uncompensated Metabolic Acidosis.

PRACTICE

- 1. pH: 7.11 CO2: 51 HCO3: 27
- 2. pH: 7.39 CO2: 54 HCO3: 38
- 3. pH: 7.14 CO2: 51 HCO3: 28
- 4. pH: 7.39 CO2: 53 HCO3: 27
- 5. pH: 7.45 CO2: 40 HCO3: 22
- 6. pH: 7.50 CO2: 44 HCO3: 31
- 7. pH: 7.35 CO2: 20 HCO3: 17
- 8. pH: 7.12 CO2: 44 HCO3: 14
- 9. pH: 7.28 CO2: 54 HCO3: 26
- 10. pH: 7.30 CO2: 35 HCO3: 17
- 11. pH: 7.19, CO2: 39, HCO3: 18
- 12. pH: 7.7, CO2: 52, HCO3: 35
- 13. pH: 7.42, CO2: 54, HCO3: 28
- 14. pH: 7.84, CO2: 49, HCO3: 30
- 15. pH: 7.75, CO2: 43, HCO3: 37
- 16. pH: 7.87, CO2: 26, HCO3: 24
- 17. pH: 7.37, CO2: 20, HCO3: 15
- 18. pH: 7.14, CO2: 31, HCO3: 20
- 19. pH: 7.58, CO2: 50, HCO3: 36
- 20. pH: 7.43, CO2: 32, HCO3: 12

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ANSWER

1. pH: 7.11, CO2: 51, HCO3: 27 - Partially Compensated Respiratory Acidosis 2. pH: 7.39, CO2: 54, HCO3: 38 - Fully Compensated Respiratory Acidosis 3. pH: 7.14, CO2: 51, HCO3: 28 - Partially Compensated Respiratory Acidosis 4. pH: 7.39, CO2: 53, HCO3: 27 - Fully Compensated Respiratory Acidosis 5. pH: 7.45, CO2: 40, HCO3: 22 - Normal 6. pH: 7.5, CO2: 44, HCO3: 31 - Uncompensated Metabolic Alkalosis 7. pH: 7.35, CO2: 20, HCO3: 17 - Fully Compensated Metabolic Acidosis 8. pH: 7.12, CO2: 44, HCO3: 14 - Uncompensated Metabolic Acidosis 9. pH: 7.28, CO2: 54, HCO3: 26 - Uncompensated Respiratory Acidosis 10. pH: 7.3, CO2: 35, HCO3: 17 - Uncompensated Metabolic Acidosis 11. pH: 7.19, CO2: 39, HCO3: 18 - Uncompensated Metabolic Acidosis 12. pH: 7.7, CO2: 52, HCO3: 35 - Partially Compensated Metabolic Alkalosis 13. pH: 7.42, CO2: 54, HCO3: 28 - Fully Compensated Metabolic Alkalosis 14. pH: 7.84, CO2: 49, HCO3: 30 - Partially Compensated Metabolic Alkalosis 15. pH: 7.75, CO2: 43, HCO3: 37 - Uncompensated Metabolic Alkalosis 16. pH: 7.87, CO2: 26, HCO3: 24 - Uncompensated Respiratory Alkalosis 17. pH: 7.37, CO2: 20, HCO3: 15 - Fully Compensated Metabolic Acidosis 18. pH: 7.14, CO2: 31, HCO3: 20 - Partially Compensated Metabolic Acidosis 19. pH: 7.58, CO2: 50, HCO3: 36 - Partially Compensated Metabolic Alkalosis 20. pH: 7.43, CO2: 32, HCO3: 12 - Fully Compensated Respiratory Alkalosis