Exam 3 Study Guide

Advanced Nursing Skills

* EKG
* Cardiac arrhythmias
* ARDS
* Shock
* Chest Trauma
* Acute Respiratory Failure
* Pulmonary Embolism
* Chest Tubes
* Bioterrorism

Neuro

* Myasthenia Gravis
* Multiple Sclerosis
* Guillain-Barre Syndrome
* Amyotrophic Lateral Sclerosis
* Spinal Cord Injury

**PE – Pulmonary Embolism**

**ICU PowerPoint**

Risks: sepsis, severe trauma, aspiration, mass transfusion, cigarette smoking, drug, or alcohol overdose

S/s: dyspnea, sudden pleuritic chest pain, tachypnea, anxiety, fever, cough, diaphoresis, syncope, hemoptysis

Labs: d-dimer, ABG’s, BNP, Troponin, ECG, chest x-ray, CT scan of chest, pulmonary angiography

Treatment: Heparin, Warfarin, Alteplase, tPa, embolectomy, inferior vena cava filter, mechanical ventilation if patient is in respiratory distress, elevate HOB, IV fluids to decrease viscosity of blood, vasopressin to improve blood pressure, bleeding precautions.

-PE prevents adequate perfusion of blood to the alveoli. High ventilation with low perfusion.

-tachypnea causes respiratory alkalosis, you eventually end up with metabolic acidosis from hypoxia

**Book pg. 850**

-PE refers to the blocking of the pulmonary artery by a thrombus

\*Thrombus: blocks blood flow typically in the lower half of body

\*Embolus: thrombus that breaks off and moves to another part of the body

-when you have a PE there is increased dead space. So, you get ventilation to the area but decreased amounts of blood. Gas exchange is impaired or absent.

-the clot causes vasoconstriction leading to increased pulmonary vascular resistance.

-vasoconstriction 🡪 increased pulmonary artery pressure 🡪 increased right ventricular work 🡪 right ventricular failure 🡪 decreased cardiac output 🡪 decreased systemic blood pressure 🡪 shock

Assessment

-chest x ray will be normal but may show infiltrates, atelectasis, elevation of diaphragm, pleural effusion

-ECG: ST-T wave abnormalities

-ABG: may show hypoxemia and hypocapnia from tachypnea

-MDCTA: Multidetector Computed Tomography Angiography: standard for diagnosing PE. Pulmonary angiography can also be used.

Treating hemodynamically unstable PE

-stabilize cardiopulmonary system

-dissolve embolus (tPa or alteplase)

-before administering you need INR, PTT, hematocrit and platelet count

-embolectomy can be performed if thrombolytics are contraindicated

-PT with recurrent PE despite anticoagulation will have a Greenfield filter inserted into the inferior vena

cava.

-prevent new emboli from forming.

Treating hemodynamically stable PE

-anticoagulation for up to 10 days. May have anticoagulation for 6 months after to prevent reoccurrence. May be extended indefinitely for high risk PT.

**ATI Ch. 24**

Health Promotion

-promote smoking cessation

-promote appropriate weight

-healthy diet and physical activity

-prevent DVT: leg exercises, compression socks, avoid sitting for long periods

Risk Factors

-immobility

-smoking

-obesity

-surgery: especially orthopedic surgery of the lower extremities and pelvis

-central venous catheters

-heart failure

-trauma

S/s – anxiety, sudden onset of chest pressure, pain, dyspnea, cough, hemoptysis, tachycardia, hypotension, diaphoresis, S3 and S4, low grade fever, distended neck veins, syncope, cyanosis

Diagnosis

**MDCTA**: standard criterion for detecting PE

**V/Q scan**: ventilation-perfusion scan which shows circulation of air and blood in the lungs and can detect a PE.

**Pulmonary angiography**: gold standard when MDCTA not available. Very invasive and costly.

**Chest x ray**: can provide data that supports PE such as an elevated diaphragm.

Nursing Care

-administer O2

-high fowlers

-assess respiratory and cardiac status

-monitor LOC

MEDS

-Anticoagulants such as low molecular weight heparin (PTT), enoxaparin, and warfarin (PT) are used to prevent clots and keep clots from getting bigger.

-Thrombolytics such as alteplase dissolve clots

Complications

-decreased cardiac output: hypotension, tachycardia, cyanosis, JVD, syncope

-hemorrhage: from anticoagulant therapy

**Anthrax**

**Incubation period:** the time from when the infection occurred to the onset of S/s or the 1st positive test.

-infective only in the spore form.

-standard precautions

-PT is not contagious. Cannot be spread from person to person.

-odorless and invisible

-incubation period 1-6 days

-methods of infection

1. skin contact: most common. Causes edema and pruritis.

2. GI: n/v, fever, abdominal pain, bloody diarrhea, ascites

3. Inhalation: mimics flu, cough, and headache

**Book pg. 2232**

-causes hemorrhage, edema and necrosis

Treatment

-penicillin, ciprofloxacin, levofloxacin, and doxycycline.

-treatment must begin within 24 hours of infection and continue for 60 days.

**Smallpox**

-12-day incubation period

-extremely contagious. Spread through direct contact with clothing or linens. Also spread through droplets only after the fever has decreased and the rash phase has begun.

-1972: last child vaccinated

S/s: high fever, malaise, headache, backache, maculopapular rash appears starting on the face, mouth, pharynx, and forearms.

Treatment: antibiotics, isolation

**Book pg. 2234**

-incubation period 7 – 17 days.

-aerosolization would cause widespread dissemination.

-patient remains contagious until rash crusts and falls off.

-30% fatality rate

-airborne and contact precautions

**Mass Casualty**

Black tag: dead

Green tag: walking wounded. Transported first

Red: serious injury but treatable

Yellow: injury but can wait on treatment

**Acute Respiratory Failure (ARF)**

-when one or both gas-exchange functions of the lungs are compromised.

Hypoxemic respiratory failure: V/Q mismatch

S/s: tachycardia, tachypnea, confusion, anxiety, cyanosis, coma

Hypercapnic respiratory failure: hypoventilation

S/s: headache, confusion, decreased LOC, somnolence

Risks: pulmonary edema, pneumonia, PE, asthma, narcotic overdose, myasthenia gravis

Diagnosis: ABGs, venous O2 sat, Hgb and Hct, chest x ray, sputum cultures

Treatment: supplemental O2, positive pressure ventilation, PEEP, BIPAP, CPAP

**Book pg. 556**

**Mechanical Ventilation**

**Acute Respiratory Distress Syndrome**

ARF with:

-Hypoxemia even with 100% O2 is given

-decreased pulmonary compliance

-dyspnea

-non cardiac associated pulmonary edema

-dense pulmonary infiltrates on x ray

Risk Factors: sepsis, severe trauma, aspiration, cigarette smoking, cardiopulmonary bypass, PE, drug/alcohol overdose

S/s: tachypnea and tachycardia, severe dyspnea

Treatment: mechanical ventilation, high flow nasal cannula, ECMO – extracorporeal membrane oxygenation (uses a pump to circulate blood through an artificial lung outside of the body), prone position

MEDS: antibiotics, neuromuscular blockade for PT on vent, hydration, enteral nutrition

Complications: barotrauma, renal failure, multisystem organ dysfunction syndrome, vent associated pneumonia

**Ventilators: PEEP meaning and reason, high alarm, low alarm, nursing interventions for the alarms, sedation used**

-peep: keeps alveoli open longer

-high alarm: kink in the system

-low alarm: leak in the system

**CPR proper hand placement and why**

-above xyphoid process

-1.5 inches deep

-hard surface

**Pneumothorax causes, s/s, nursing interventions, treatment, causes of tension pneumothorax and s/s**

A picture containing table

Description automatically generatedCause: partial to complete collapse of the lung due to accumulation of air in the pleural space

Cause of tension pneumothorax: sucking chest wound, prolonged clamping of the tubing, kinks, or obstruction.

S/s of tension pneumothorax: tracheal deviation. Absent breathe sounds on one side, distended neck veins, respiratory distress, asymmetry of the chest, cyanosis.

**EKG: identify basic arrhythmias and treatments for all**

EKG Analysis: rhythm, rate, p-waves, pr interval, QRS complex

Step 1. Is the speed of the rhythm between 60-100?

Step 2. Is it regular?

Step 3. Is the complex narrow? Narrow is normal, wide is abnormal

Step 4. Is it preceded by a P-wave?

Step 5. Do all the complexes look the same?

**Normal Rhythms**

Yes to all 5 Q’s means normal sinus rhythm – Normal Sinus Rhythm

\*No treatment if BP is normal

Yes to all except rate. Rate is above 100 – Sinus Tachycardia

\*Treat the underlying condition which is typically fever, anxiety, pain, dehydration, or hypoxemia

Yes to all except rate. Rate is below 60 – Sinus Bradycardia

\*Treat with atropine or epinephrine. May need pacemaker.

**Abnormal Rhythms**

Yes to all 5 Q’s except QRS s not preceded by a P wave. It’s preceded by an F wave – Atrial Flutter

\*Treat with digoxin, verapamil, or diltiazem to slow ventricular rate and amiodarone, sotalol, or flecainide to get back to sinus rhythm.

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All normal except rate is irregular and no p wave, f wave – Atrial Fibrillation

\*Treatment: digoxin, verapamil, or diltiazem to keep rate from getting too fast. Cardioversion, amiodarone, sotalol, or flecainide to get back to sinus rhythm

Yes, to all but rate is extremely fast (140-300) – Supraventricular tachycardia. So fast you can’t see a P wave

\*Treat with vagal maneuvers, adenosine, beta blockers, verapamil, or cardioversion.

Fast rate, no p waves, wide complex – Ventricular Tachycardia

\*Pulse: lidocaine, amiodarone, procainamide, and sotalol, if BP unstable cardioversion, lidocaine

\*Pulseless: call code, defibrillation, CPR, epinephrine.

LAP

Lidocaine

Amiodarone

Procainamide

Loss of all coordinated activity, saw toothed wave – Ventricular fibrillation

\*Treat with CPR and defibrillation, epi, lidocaine, amiodarone, procainamide, magnesium

No electrical or mechanical activity – straight line - Asystole

\*Treat with CPR, epi, atropine, pacemaker

Depolarization SA Node (P wave) 🡪 AV Node (P-R interval, delays impulse so atria and ventricle don’t fire at the same time) 🡪 Purkinje Fibers (QRS Complex) 🡪 Repolarization T wave

Small box = 0.04 seconds

Large box = 0.2 seconds

30 large boxes in a 6 second strip

-count the number of complexes in a six second strip and multiply by 10 for the HR/minute

Normal PR interval 0.12 – 0.20 seconds (3-5 small boxes)

-PR interval determines if impulse from SA node is going to the ventricle through the normal pathways.

Normal QRS duration 0.04 – 0.08

-QRS duration determines how long impulse travels through the ventricles

-Wide = slowed conduction

**NEURO**

**Medications**

Baclofen – antispasmodics: used in MS

Cholinergics

Atropine: used in bradycardia

alpha interferon

plasma pheresis – treats MG

IG g

**Book Page 2103**

**GB: prognosis, pathophysiology, treatments, s/s, nursing interventions, diagnostic test**

-idiopathic polyneuritis

-autoimmune attack on peripheral nerve myelin

-ASCENDING WEAKNESS with dyskinesia (inability to execute voluntary movements), hyporeflexia, and paresthesia.

-70% recover fully

-30% have some disability

-5-10% of cases die

Patho

-cell-mediated attack on peripheral nerve myelin proteins that cause inflammatory demyelination.

S/s

-muscle weakness

-hyporeflexia may lead to tetraplegia

-paresthesia of hands and feet.

-may include tachycardia, bradycardia, HTN, or orthostatic hypotension.

Diagnostic Test

-Hx of viral illness in previous weeks

-elevated protein in CSF

Treatment

-ICU

-may need mechanical ventilation

-preventing complications of immobility: anticoagulant, sequential compression boots

-IVIG and plasmapheresis

-IV fluids to manage hypotension

Nursing Intervention

-maintain respiratory function

-enhancing physical mobility

-provide adequate nutrition

-improving communication

-decreasing anxiety

-reducing fatigue

-monitor and manage potential complications

**Book PG. 2094**

**MS: pathophysiology, s/s, risk factors, teaching to prevent relapse, nursing interventions, teaching based on Symptoms, diagnostic test**

-immune mediated

-progressive demyelinating disease of the CNS. AKA destruction of myelin which is the fat and protein surrounding certain nerve fibers in brain and spinal cord.

-peak onset between 20 and 50.

-affects women three times more than men.

-most frequent affected areas are optic nerve, chiasm, and tracts, the cerebrum, the brain stem and cerebellum, and the spinal cord. Once the axons degenerate there is permanent and irreversible damage.

Types

Less Severe

1. Radiologically isolated syndrome - RIS: typically, no symptoms
2. Clinical isolated syndrome – CIS: unilateral optic neuritis (inflammation of optic nerve), focal symptoms (paralysis, loss of muscle control, paresthesia, numbness, decreased sensation), partial myelopathy (injury of spinal cord due to compression)

More Severe

1. Remitting-relapsing: usually have complete recovery after each relapse, but there are deficits over time. Eventually becomes secondary progressive.
2. Secondary progressive: disease progression occurs whether or not there is a relapse
3. Primary progressive: steady increase in disabling symptoms.
4. Progressive relapsing: least common. Relapses with continuing disabling progression between exacerbations.

S/s

-usually based on location of legion.

-fatigue, depression, numbness, loss of balance, spasticity, and pain.

-blurred vision, diplopia, scotoma (patchy blindness), total blindness

-paresthesia, dysesthesias, proprioception loss (losing sense of where you are in space), spasticity (increase in muscle tone or stiffness)

Teaching

-avoid hot temperatures

-treat anemia and depression

-change meds

-OT and PT

Diagnostic tests

-MRI looking for plaques

-Cerebrospinal fluid has elevated protein levels and slight increase in WBC’s

Nursing Interventions

-PT, OT, speech therapy, and rehab

-monitor: vision, speech, fatigue, swallowing, activity intolerance, and skin integrity, LOC

**Book Page 2100**

**MG: s/s, Crisis s/s and treatment, nursing interventions, diagnostic tests**

-autoimmune disorder

-purely motor disorder. Does not affect sensation or coordination.

-varying degrees of weakness of the voluntary muscles

-most common in women 20-30 y/o and men over 50.

-antibodies attack ach receptors leaving fewer sites for ach to bind to which inhibits muscle contraction.

Cause: thymus gland believed to be cause of antibody production.

-thymic hyperplasia

-thymic tumor

S/s

Ocular: diplopia and ptosis

Generalized: weakness of face (bland facial expression) and throat muscles (dysphonia – voice impairment, dysphagia – difficulty swallowing), respiratory weakness.

-Generalized weakness affects all extremities including intercostal muscles which leads to decreased vital capacity (greatest volume of air that can be expelled from the lungs after taking the deepest possible breath) and respiratory failure. This causes Myasthenia crises.

Diagnostic Test

1. Acetylcholinesterase inhibitor test: administer edrophonium chloride IV, 30 seconds after injection s/s should resolve for about 5 minutes. Immediate improvement means the test is positive and confirms muscle weakness.

-have atropine to counteract side effects such as bradycardia, asystole, bronchoconstriction, sweating, and cramping.

2. Ice Test: indicated for patients with heart conditions and asthma. Hold ice pack over eyes for 1 minute. Ptosis should temporarily resolve if they have MG.

3. Blood test for ACH antibodies

4. Repetitive nerve stimulation – RNS – will show decrease in potentials

5. Single fiber electromyography – EMG – shows delay or failure of neuromuscular transmission.

6. MRI – detects enlarged thymus gland

Treatment

-there is no cure.

-treatment is aimed at improving function and reducing the circulating antibodies

1. Pyridostigmine bromide – anticholinesterase medication – first line of therapy.

-inhibits breakdown of ACH and increases availability at neuromuscular junction.

2. Immunosuppressive agents – given if pyridostigmine bromide doesn’t improve muscle strength.

-goal is to reduce production of antibodies – Prednisone

3. Cytotoxic drugs given if PT not responsive to corticosteroids. Azathioprine inhibits T lymphocytes and B-cell proliferation which reduces antibody levels. Takes 3 – 12 months for therapeutic effect.

4. IVIG: treats exacerbations and can also be used long term in certain patients. Symptoms improve in a few days to a week.

5. Plasmapheresis: helps get rid of antibodies.

6. Thymectomy: surgical removal of the thymus gland. Takes about 3 years for therapeutic affect since the antibodies live so long.

Myasthenia Crisis

S/s: respiratory distress, dysphagia, dysarthria, ptosis, diplopia, and prominent muscle weakness.

-ICU

-vent assistance

-nurse assesses respiratory rate, depth, sounds, vital capacity, and negative inspiratory force.

-chest physiotherapy and postural drainage to clear secretions

-enteral tube feedings

-monitor ABG’s, serum electrolytes, I&O, daily weight

Nursing Management

-patient and family education

-medication management

-energy conservation

-strategies to help with ocular manifestations

-prevention and management of complications

-suction should be available at home to deal with aspiration

-avoid stress, infections (especially respiratory), vigorous physical activity, some meds, high temps.

**Book page 2131**

**ALS: s/s diagnostic, prognosis, teaching**

-aka Lou Gehrig Disease

Diagnosis

-based on s/s

-electromyography and muscle biopsy studies of the affected muscles indicate reduction in the number of functioning motor units.

-MRI

Prognosis

-no cure

-based on the area of CNS involvement

-death occurs b/c of infection, respiratory insufficiency, or aspiration

Teaching

-encourage patient to complete an advanced directive

S/s

-weakness in the muscles

-difficulty swallowing, talking, and eventually breathing

-regurgitate liquid through the nose

-can’t laugh, cough, or blow the nose

-aspiration risk, can’t speak or swallow

-nasally voice, unintelligible speech

-emotional lability

-cognitive impairment

**Spinal cord injury:**

**degree of paralysis**

-partial: possibility of recovery

-complete: paralyzed for life

**altered vital centers**

**Quadriplegia**

-come from cervical region injuries

-paralysis of all 4 extremities and trunk

**paraplegia**

-injuries below T1

-paralysis/paresis of the lower extremities

**nursing interventions**

**autonomic dysreflexia (pathophysiology, causes, nursing interventions, medical management, s/s)**

-exaggerated reflex response by the sympathetic nervous system

-severe HTN caused by irritating stimulus below SCI

-Risk factor injury at T6 or higher

-medical emergency

Causes

most common is bladder (distended bladder, UTI)

bowel (impaction)

skin breakdown (pressure injury, skin infection)

Patho

Sympathetic: vasoconstriction, sweat gland stimulation, increased HR, dilate pupils, bronchodilation

Parasympathetic: vasodilation, decreased HR, salivation, bronchoconstriction, constrict pupils

-disconnection between sympathetic and parasympathetic nervous system

1. Irritating stimulus

2. Exaggerated sympathetic reflex response

3. Vasoconstriction of vessels below the site of injury which elevates BP. PT is pale, cool, clammy.

4. Baroreceptors sense increased BP. Forces parasympathetic to vasodilate the above the injury. PT looks flushed

S/s

-throbbing headache

-hypertension

-flushing above

-pale, cool, clammy below

-bradycardia

-dilated pupils

-stuffy nose

-goosebumps

-sweating

-anxiety

Nursing Interventions

Prevention

1. Bladder: keep empty, assess urine output, bladder scans, prevent UTI, foley care, assessing foley tubing
2. Bowel: check for impaction, assess bowel sounds, palpate for distention, assess last BM
3. Breakdown of skin: remove binding devices, reposition Q2H. assess skin, protect from injury

Detection

1. Check BP is PT reports headache
2. Assess for S/s of AD

Action

1. Call a rapid response
2. Position PT at 90 degrees with legs lowered: decreases BP
3. Assess BP Q2-5 minutes
4. Remove binding devices or clothing
5. Investigate 3 big b’s

Medical Management

-nitro paste:

-nifedipine

**Spinal shock (s/s, meaning, resolution, management)**

**S/s**

-sensation, motor and reflex s/s

**-spinal cord’s response to the inflammation caused by the injury.**

**-MAP at least 85 to prevent further damage**

**Neurogenic shock (s/s, management)**

-complication of spinal trauma

-can occur within 24 hours of SCI

Cause

-SCI: cervical, thoracic above T6

-spinal anesthesia

S/s

-hypotension

-decreased cardiac afterload

-venous blood pooling

-decreased cardiac output

-bradycardia

-DVT

-hypothermia

-extremities warm and dry

-cool trunk

Nursing Interventions / Management

-manage ABC’s

-keep spine immobilized: cervical collar, log-rolling patient, use backboard

-maintain tissue perfusion: MAP between 85-90 mmHg

1. IV fluids: crystalloids, use with caution, watch for fluid volume overload

2. Vasopressors: causes vasoconstriction, increases BP, systemic vascular resistance, and cardiac output

3. Dopamine: vasoconstrict and increase HR

-Atropine to increase heart rate

-may need temporary pacing

-warming devices for hypothermia

-prevent DVT: ROM daily, compression stockings, anticoagulation

-foley placement

-proper positioning

-monitor for hypotension, bradycardia, dependent edema, and loss of temp regulation.

-monitor for VTE

-nervous system issue.

-major vasodilation: SNS has lost the ability to regulate diameter of vessels

<https://www.youtube.com/watch?v=KUEunZYZgII>

**Diagnostic of Guillain Barre**

-protein in CSF

-Hx of viral illness a few weeks prior

**Prognosis of Guillain Barre**

-70% full recovery

-30% some disability

-death in 5-10% of cases

**Symptoms of Guillain Barre**

-ascending weakness

-paresthesia

-diplopia

-hyporeflexia

-dyskinesia

**Pathophysiology of Guillain-Barre**

-inflammatory demyelination

-interruption of nerve conduction and axonal loss

**Treatment options for Guillain Barre**

-respiratory therapy

-mechanical ventilation

-anticoagulant agents, sequential compression boots

-IV immunoglobin

-plasma exchange to remove antibodies

**PMH of patient with Guillain-Barre**

-virus in the previous few weeks such as campylobacter jejuni, cytomegalovirus, Epstein-barr, mycoplasma pneumoniae, h influenzae, zika

**Priority assessment during Guillain-Barre**

-changes in vital capacity and negative inspiratory force are assessed to identify impending neuromuscular respiratory failure.

**Signs of alert in patient with Guillain Barre**

-respiratory depression

**In which condition is important to assess vital capacity**

Guillain Barre

**Causes of loss of vision in multiple sclerosis**

-lesions in the optic nerve

**Pathophysiology of Multiple sclerosis**

-T cells remain in the CNS which promotes infiltration of other agents

-the immune system attacks causing inflammation which destroys myelin and oligodendroglia cells.

-conduction of impulses is slowed and the production of myelin is reduced.

**Teaching for a client with relapsing-remitting multiple sclerosis**

-avoid stress, sickness or sepsis, smoking, and sun or extreme heat

-avoid hot temps

-treat pain and anemia

-change meds

-OT and PT

-avoid physical and emotional stress

**Evaluation after administering baclofen**

-monitor for dizziness, drowsiness and muscle weakness

**Symptoms of multiple sclerosis**

-blurring of vision

-diplopia

-spasticity

-fatigue

-depression

-weakness

-numbness

-difficulty in coordination

-loss of balance

**Interferon side effects**

-flu like symptoms

-leukopenia

-headache

-depression

-skin necrosis

**Signs and symptoms of myasthenic crisis and management**

Ocular: diplopia and ptosis

Generalized: weakness of face and throat muscles, limbs, and respiratory weakness.

-bland facial expression

-dysphonia

-dysphagia

-respiratory failure from weakness of intercostal muscles

Management: place on a vent, ABG’s, chest physiotherapy

**Physical activity recommendations for myasthenia gravis**

-conserve energy

-handicap parking pass

-schedule activities to coordinate with peak energy and strength levels

-schedule rest periods

**Diagnosis of myasthenia gravis**

-acth inhibitor test. Have atropine to counteract side effects

-MRI will show enlarged thymus gland

**How to administer myasthenia gravis treatment**

-gradually increased to a daily maximum

-4 times per day

**Surgical treatment for Myasthenia gravis**

Thymectomy: removal of thymus gland

**Mechanism of action for pyridostigmine**

-inhibits breakdown of ACH

**Medication to have during edrophonium test**

Atropine

**Priority intervention in spinal cord injury when just happens.**

-immobilization of head and neck in neutral position

-stabilization

-control of life-threatening injuries

**Priority action during autonomic dysreflexia**

-Sit the patient up to lower BP

**Teaching about preventing autonomic dysreflexia**

-prevent the three B’s: bladder, bowel, and breakdown of skin

**Pathophysiology of autonomic dysreflexia**

-breakdown between sympathetic and parasympathetic nervous system

**Signs and symptoms of autonomic dysreflexia**

-pounding headache

-hypertension

-flushing above

-cool, clammy, pale below

**Medication used to treat hypertension related to autonomic dysreflexia**

-clonodine

-hydralazine and nitroglycerin

**Causes of autonomic dysreflexia**

-bladder

-bowel

-breakdown of skin

**Priority assessment for spinal cord injury above C5**

-respiratory

**Spinal shock signs when is resolved**

s/s of spinal shock: hypotension, bradycardia

Teaching of family during the spinal shock

**Education to manage urinary retention in patients with neurological disorders**

-drink 2.5 L daily

-empty bladder frequently

-intermittent catheterization

Neurological deficit in lower SCI

**Prevention of complications of immobility in patients with SCI**

-anticoagulant therapy

Nursing intervention in a client with high thoracic SCI

**s/s of neurogenic shock – everything low**

-hypotension

-bradycardia

-decreased cardiac output

-venous pooling in the extremities

**Purpose of halo fixation device**

-traction and/or immobilization of the spinal column

**Position to place a client during respiratory distress**

-fowlers

-high fowlers

**Signs of hypoxia**

-bradycardia

-restlessness

-cyanosis

**Rational for using PEEP**

-keeps the alveoli expanded

-increases oxygenation and improves lung expansion

**Treatment for severe bradycardia**

Atropine

**Lab values for metabolic acidosis**

PH less than 7.35

HCO3 less than 22

Action taken for accidental extubation

**Lab for respiratory acidosis**

PH less than 7.35

CO2 greater than 45

**Sedation used during mechanical ventilation**

Propofol

**Risk factors for pulmonary embolism**

-immobility

-estrogen therapy

-pregnancy

-tobacco use

-obesity

-cancer

-long bone fractures

-trauma

**Pulmonary embolism medical management**

-anticoagulants

-thrombolytics

-embolectomy

-vena cava filter

**S/s of hypercapnia and hypoxia in acute respiratory failure**

-dyspnea

-cyanosis

-pallor

-hypoxemia

-tachycardia

-confusion

-restlessness

-hypercarbia

**s/s in acute respiratory distress syndrome**

-dyspnea

-pulmonary edema

-reduced lung compliance

-pulmonary infiltrates

-hypoxemia despite 100% O2

**How to ambulate a patient with a chest tube**

-keep drainage unit below the heart

-make sure tubes are functioning and the connections are secure

**Signs of leak in a chest tube chamber**

-continuous bubbling means there is a leak

**Signs of pneumothorax** – partial to complete collapse of lung due to accumulation of air in the pleural space

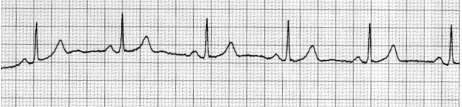
-dsypnea, distended neck veins, hemodynamic instability, pleuritic chest pain, cough, absent or reduced breath sounds on the affected side, hyperresonance on percussion of affected side, asymmetrical chest wall motion.

**Action to take after chest tube disconnect from chest**

-cover with gauze

-tell client to exhale as much as possible to remove air from chest

Normal Sinus Rhythm



SVT

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**Treatment for afib, ventricular fibrillation, flat line**

AFIB: digoxin, verapamil, diltiazem, cardioversion, amiodarone

VFIB: CPR and defibrillation, epinephrine, LAP: lidocaine, amiodarone, procainamide

Asystole: CPR, epinephrine, atropine, maybe a pacemaker

**What labs to monitor when administering corticosteroids IV**

-glucose

-electrolytes

**Intervention on a client with advanced ALS**

-mechanical ventilation

-PEG tube

**Reason that cause death on a client with ALS**

Infection, respiratory insufficiency, aspiration

**Anthrax methods of transmission**

1. skin contact: most common. Causes edema and pruritis.

2. GI: n/v, fever, abdominal pain, bloody diarrhea, ascites

3. Inhalation: mimics flu, cough, and headache

**Isolation time for smallpox**

-until scabs fall off

**Types of client with red tags, yellow tags and green tags in bioterrorism**

Red: life threatening injuries, need minimal intervention

Yellow: significant injuries but can wait hours

Green: minor injuries. Highest chance of survival