Little Rock Critical Care Program

CCRN/PCCN Review

November 6th

Day 2		
0800-1000	Endocrine Adrenal insufficiency Diabetes insipidus & Mellitus DKA Hyperglycemia Hyperosmolar hyperglycemic state SIADH Thyroid disorders	Patti
1000-1015	*Break*	
1015-1230	Hematology/Immunology Anemia Autoimmune disorders Coagulopathies Myelosuppression (thrombocytopenia, neutropenia Oncologic complications (tumor lysis syndrome, neutropenia) Transfusion reactions (TRALI, TACO) Infectious diseases	Patti
1230-1315	*Lunch*	
1315-1530	Neurological Acute cord injury Brain death Encephalopathy Stroke Hydrocephalus Neurogenic shock Neurologic infection Neurological storming Neuromuscular disorders Neurosurgery Neurovascular abnormalities Seizure disorders Space-occupying lesions Spinal surgeries Traumatic brain injury (epidural, subdural, concussion, non-accidental trauma)	Kelly
1530-1600	Integumentary Cellulitis IV infiltration Necrotizing fasciitis Pressure injury Skin failure (perfusion injuries) Wounds	Lynnette
1600-1630	Practice questions	Lynnette

CCRN/PCCN Review - Endocrine System

Patti Esmail, MSN/ED RN CCRN, CHI St Vincent RN Clinical Educator

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Adrenal Insufficiency

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Adrenal Insufficiency

- Deficit of glucocorticoid and /or mineralocorticoid production
  - electrolyte and fluid abnormality
  - potentially life threatening cardiovascular collapse
- Due to
  - issues with the adrenal gland
  - infection/sepsis
  - issue with pituitary gland
  - drug induced
  - long term glucocorticoid use or abrupt cessation of corticosteroids

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Adrenal Insufficiency

- Signs and Symptoms
  - neurologic signs
  - CV symptoms
  - GI and joint
  - fever
- Diagnostic studies
  - low sodium, high potassium, high calcium, hypoglycemia
  - low AM cortisol or low cortisol in stress states
  - ACTH is 2X greater than upper limit of normal
  - test renin and aldosterone to determine mineralocorticoid deficit

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Adrenal Insufficiency

- Blood Pressure and tissue perfusion maintained
- Cortisol levels are restored

Treatment

- Fluid and electrolytes administration
- Hormone replacement
  - glucocorticoid
  - mineralocorticoid if aldosterone deficiency
- treat hypoglycemia

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Diabetes Insipidus

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Diabetes Insipidus

- Either the hypothalamus or posterior pituitary is affected and there is interference with ADH synthesis and transport or release
  - deficiency causes
    - inability to conserve water
    - excretion of large amounts of dilute urine

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Diabetes Insipidus

- Central Diabetes Insipidus: ADH deficiency
  - Idiopathic >50%; Autoimmune; familial
  - Head trauma or surgical injury
  - Pituitary tumor
  - Infection
  - Inflammatory/Autoimmune disorders
  - Lung cancer, leukemia, lymphoma
  - Increased intracranial pressure, brain death

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Diabetes Insipidus

- Nephrogenic Diabetes: ADH resistance
  - Renal
  - Metabolic
  - Familial
  - Drugs
  - Obstructive renal disease

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Diabetes Insipidus

- Polydipsia: high fluid intake creates excess free water excretion
  - Primary polydipsia
    - diagnosis of exclusion
  - Psychogenic polydipsia
    - Psychiatric disorders
      - schizophrenia
        - sensation of dry mouth

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Diabetes Insipidus

- Signs and Symptoms
  - persistent polydipsia
  - polyuria 40-50 mL/kg/24h
  - decreased skin turgor, dry mucous membranes
  - tachycardia; hypotension if dehydrated
  - Symptoms
    - CDI - often abrupt presenting in weeks to months of onset
    - NDI - more insidious onset for months or even years

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Diabetes Insipidus

- Noninvasive studies
  - elevated plasma osmolality >300 mOsm/kg
  - hypernatremia > 145 mEq/:
  - decreased urine osmolality <300 mOsm/kg
  - low specific gravity 1.001-1.005
  - low urine sodium
  - 2-step water deprivation test
    - water deprivation for 8 hour
    - DDAVP administered intramuscular or subcutaneous

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Diabetes Insipidus

DI may spontaneously resolve or require lifetime medication

- ADH deficiency is corrected
- fluid and electrolyte balance restored
- hypoperfusion restored
- Pharmacology
  - ADH - CDI
    - vasopressin, DDAVP, lypressin
  - Thiazide diuretics - NDI
    - Amiloride - adjunctive treatment with thiazide diuretics
  - Spironolactone, ACE inhibitors, ARB's

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Diabetes Mellitus

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Diabetes Mellitus

- Type 1, 1.5 and 2
- Type 1
  - autoimmune destruction of beta cells
    - insufficient insulin secretion
    - postprandial hyperglycemia
    - inability of the body to suppress hepatic glucose production during meal absorption
    - decreased peripheral glucose uptake and utilization
  - results in glucose toxicity
    - glucose transporters are severely reduced or inactivated

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Diabetes Mellitus

- Type 1.5
  - maturity-onset diabetes of the young
  - latent autoimmune diabetes of adults
    - autosomal dominant form of DM
      - mutation of hepatocyte nuclear factor or glucokinase genes
      - glucose abnormal sensing by the beta cells and impaired insulin secretion
  - symptom vary depending on severity of disease

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Diabetes Mellitus

- Type 2
  - dysfunction of insulin production
  - dysfunction of peripheral receptors
  - insulin secretion
    - low
    - normal
    - high
  - postprandial hyperglycemia
    - receptors resist, allows insulin to bind to the site
    - glucose is unable to enter the cell for utilization and storage

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Diabetes KetoAcidosis

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Diabetic KetoAcidosis

- Serious metabolic complication
  - decreased insulin level with gluconeogenesis & increased insulin resistance; exaggerated hepatic glucose production
  - Ketosis and metabolic acidosis
  - Fluid and electrolyte imbalance
  - Osmotic diuresis
  - Altered mental status

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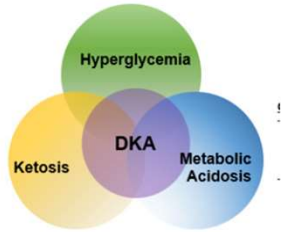
Diabetic KetoAcidosis

Risk factors



- diagnosed diabetes mellitus
  - too little insulin
  - infection, trauma, MI, CVA, pancreatitis
  - poor compliance
  - medications
- undiagnosed diabetes mellitus
- euglycemic DKA
  - SGLT2 inhibitor use; pregnancy; low caloric intake; atypical antipsychotics

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Diabetic KetoAcidosis

- blurred vision, fatigue, weakness
- n/v, abdominal cramping
- polyuria, polydipsia, weight loss
- fruity odor to breath
- tachycardia, hypotension
- tachypnea, Kussmaul's respirations
- seizures

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Diabetic KetoAcidosis

- Diagnostic findings
  - plasma glucose >250 mg/dL
    - euglycemia - <250 mg/dL in 10% of DKA cases
  - Metabolic acidosis
    - arterial pH <7.3
    - serum HCO_3 < 18 mEq/dL
    - positive serum and urine ketones
    - Anion gap > 10 mEq/l (Na - {Cl +HCO3})
      - calculations must be adjusted for hyperglycemia
    - Hyponatremia, hypocalcemia, hyperkalemia, hyperphosphatemia

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Diabetic KetoAcidosis

Goals of care

- Restoration of balance of acid-base electrolytes and fluid
- Blood glucose levels are normalized
- Elimination of ketosis
- Neurologic and pulmonary status ar normal
- Identification of the underlying cause

Treatment

- Pharmacology
  - Fluid resuscitation
  - Regular insulin infusion
  - Electrolyte replacement
- Treatments
  - monitor glucose
  - monitor electrolytes
  - I & O
- Psychosocial

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Hyperglycemia

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Hyperglycemia

glucose production outweighs glucose utilization with serum glucose > 140 mg/dL

- Type 1 diabetes
- Type 1.5 diabetes
- Type 2 diabetes
- Stress induced
- Admin of hyperglycemic-provoking agents
- Holding hypoglycemic medication

Hyperglycemia in nondiabetics is a predictor of mortality

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Hyperglycemia - Signs & Symptoms

- polyuria, polydipsia
- serum glucose > 140 mg/dL in hospitalized patient
  - occurs frequently in critically ill patient
  - implementation of appropriate interventions to control and maintain glucose levels
    - while avoiding wide swings in glucose levels

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Hyperglycemia

Goal: keep serum glucose 140-180 mg/dL

- May need
  - Insulin if glucose persists >180 mg/dL
    - continuous infusion of regular insulin
    - Subcutaneous insulin
  - appropriate glucose monitoring
  - nutrition
    - regular meals - glucose testing AC & HS
    - enteral or parenteral - glucose testing every 4 to 6 hours

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Hyperglycemia

- Diabetic or not Hyperglycemia is associated with:
  - poor clinical outcomes
  - increased mortality and morbidity
    - demonstrated associations with
      - immune functions
      - inflammatory process
      - vascular alterations
      - neuronal damage

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Hyperglycemic Hyperosmolar State

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Hyperglycemic Hyperosmolar State

- Life-threatening hyperglycemic emergency
  - hyperosmolality
  - severe dehydration
  - alterations in neurologic status
  - with or without mild ketosis
- Relative insulin deficiency
  - sufficient insulin inhibits lipolysis or ketogenesis in liver
  - insufficient to control hyperglycemia

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Hyperglycemic Hyperosmolar State

Risk factors

- inadequate insulin secretion and/ or action
- inadequate dose or poor compliance
- advanced age and severe dehydration
- infection, sepsis
- stroke, MI
- lack of access to fluids or inability to recognize or express need for fluid
- Medications

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Hyperglycemic Hyperosmolar State

Signs and Symptoms

- lethargy > progressive mental status decline > coma
- polydipsia, polyuria
- flushed skin, dry mucous membranes
- tachycardia, hypotension
- shallow, rapid respirations

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Hyperglycemic Hyperosmolar State

- Diagnostic findings
  - severely elevated glucose - >600 mg/dL
  - no or minimal ketosis
  - plasma hyperosmolality > 320 mOsm/kg
  - sodium and potassium levels vary
    - vary with state of hydration
    - often severely depleted, result of osmotic diuresis

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Hyperglycemic Hyperosmolar State

Goals of Care

- restore fluid & electrolyte balance
- normalize blood glucose
- peripheral tissue perfusion restored
- underlying precipitating factor identified

Treatment

- Pharmacology
  - fluid resuscitation with appropriate solutions
  - regular insulin infusion
  - electrolyte replacement
- Treatments
  - monitor glucose
  - monitor electrolytes
  - I & O

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Hyperthyroidism

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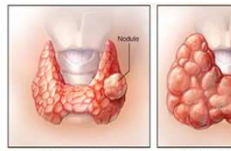
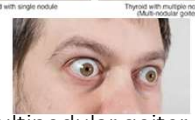
Hyperthyroidism - Thyroid Crisis

- Life threatening emergency
  - increased action of T_3 and T_4
- Systemic effects
  - sympathetic nervous system stimulation
  - increased metabolic activity
  - intolerance to heat
- Common causes
  - Graves' disease - autoimmune toxic goiter & multinodular goiter
  - inappropriate secretion of TSH
    - pituitary neoplasm secreting TSH-like substance
    - nonneoplastic pituitary secretion of TSH





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Hyperthyroidism

- Can be primary or secondary
  - Primary - TSH levels are elevated
  - Secondary - TSH levels are decreased
- Hyperthyroidism - relatively common
  - thyroid storm is rare - 10%
    - carries a 20-30% mortality rate even with prompt treatment
    - more common in women (10%) than men (2%)
    - peak age of occurrence - 20-49 years
    - can last for considerable period of time due to long half-lives of T_3 (22 hrs) and T_4 (approx 7 days)

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Hyperthyroidism

- Diagnosis primarily based on presentation
  - Measuring thyroid levels are useful
    - TSH level is the gold standard to differentiate between primary and secondary
      - decreased in primary disease
      - increased in secondary disease
- Treatment - early rapid recognition
  - decreases mortality
  - use of first-line medications

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Hyperthyroidism

- Medications
  - PTU or Tapazole
  - Iodides
  - Propranolol
  - Dexamethasone or Hydrocortisone
- Supplemental oxygen
- Nutritional Support
- if exopthalmos - eye dropes and eye sheilds
- May require surgery

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Hypoglycemia

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Hypoglycemia

- by definition below or equal to 70 mg/dL
  - can result in severe injury or death
    - ADA Workgroup on Hypoglycemia defines severe hypoglycemia as
      - event requiring assistance of another person to actively administer CHO, glucagons or other corrective actions
    - Blood glucose below 70 may seem high but....

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Hypoglycemia

- in diabetics
  - normal physiologic responses of counterregulation are impaired
    - insulin levels do not decrease
    - glucagon levels do not increase
    - epinephrine levels are increased
      - lower glycemic threshold for epinephrine secretion
- Signs and symptoms fall into two categories
  - adrenergic - increased production of epinephrine
  - neuroglycopenic - inadequate glucose supply to CNS

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Hypoglycemia

- Treatment in acute care needs to be consistent
  - Standardized protocols
  - Oral CHOs should be the first choice in an alert patient able to eat and drink
  - Rule of 15
    - 15 gms of CHO will raise the blood glucose 30-45 mg/dL
    - Adm 15 gms of CHO, recheck the blood glucose in 15 min
    - continue this cycle every 15 min until the blood glucose level is > 100 mg/dL

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Hypothyroidism

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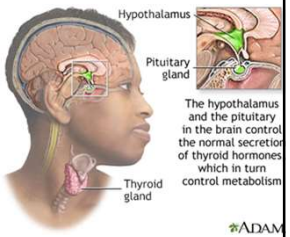
Hypothyroidism - Myxedema Coma

- Life threatening emergency
  - most common thyroid dysfunction
- System effects
  - sympathetic nervous system inhibition
  - decreased metabolic activity
  - intolerance to cold
- Common Causes
  - older females
  - undiagnosed or subclinical hypothyroidism
  - cold, infection, stress of illness or medications



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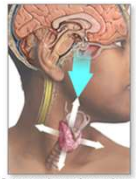
Hypothyroidism

- Can be Primary or Secondary
  - Primary- 95% are thyroid gland dysfunction
    - Hashimoto's thyroiditis
    - response to antithyroid treatment
  - Secondary
    - dysfunction in hypothalamus or pituitary
    - failure to produce TSH
  - Iatrogenic
- Hypothyroidism
  - Myxedema Coma uncommon; most severe form



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Hypothyroidism

- Diagnosis
  - Symptoms
  - Non-invasive studies
- Care delivery
  - Pharmacology
  - Treatments

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Hypothyroidism

- Medications
  - Thyroid hormone
  - glucocorticoids
  - IV fluids and electrolyte administration as appropriate
  - Hypertonic 3% saline if severe symptomatic hyponatremia
- Treatment
  - Rewarm
  - Support airway and ventilation
  - ECG monitoring
  - Prevent infection

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Syndrome of Inappropriate Anti-Diuretic Hormone

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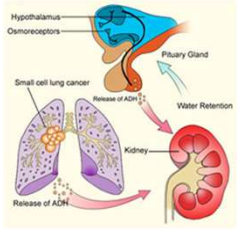
Syndrome of Inappropriate Antidiuretic Hormone

- Definition
  - hypotonicity, hyponatremia, excessive electrolyte excretion
  - abnormally high levels of ADH
- System effects
  - excessive water retention
  - low serum sodium, low serum osmolality
  - high urine sodium, high urine osmolality
- Common causes
  - abnormally high or continuous secretion of ADH or ADH like substance



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Syndrome of Inappropriate Antidiuretic Hormone

- SIADH is the most common cause of hyponatremia
  - underlying disease must be ruled out to confirm diagnosis
- Syndrome of Inappropriate Antidiuretic Hormone
  - Four types
    - Type A SIADH - 40% of cases
    - Type B SIADH - 35% of cases
    - Type C SIADH - 25% of cases
    - Type D SIADH - 10% of cases
  - Three mechanisms - ectopic production, increased ADH release, or idiopathic

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Syndrome of Inappropriate Antidiuretic Hormone

- Diagnosis
  - serum and urine lab values
  - CXR or CT of head
- Care Delivery
  - if underlying cause identified and treated
    - symptoms will resolve
  - if cause cannot be identified
    - patient will require
      - ongoing electrolyte monitoring throughout recovery and discharge

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Syndrome of Inappropriate Antidiuretic Hormone

- Medications
  - agents to reduce secretion of ADH or block effects of ADH
- Treatment
  - restore flud balance
    - fluid restriction
      - 500-1500 ml/day
  - restore electrolyte balance
    - sodium replacement
      - 8-10 mEq in the first 24 hours
      - 3% sodium chloride for severe hyponatremia (<115 mEq/L) [53]

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Questions??

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CCRN/PCCN REVIEW
Hematology

Anemia/Leukopenia/Thrombocytopenia/Coagulopathies
Oncological Complications
Blood Transfusions & Complications

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Anemia

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Anemia

- Pathophysiology
  - Reduced
    - Number of RBC's
    - Hemoglobin
    - Volume of RBC's
  - Acute blood loss
    - Trauma
    - Surgery
    - Vessel disruption

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Anemia

- Etiology and risk factors
  - Poor RBC production
  - Increased RBC destruction
  - Blood loss
    - Acute
    - Chronic

- Signs and Symptoms
  - Signs
  - Symptoms

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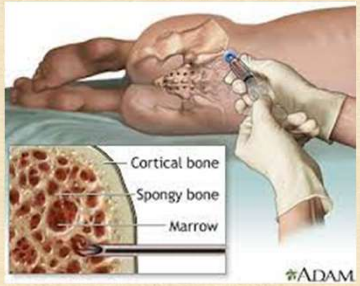
Anemia

- Diagnostic findings
  - Laboratory
    - Blood
    - Urine
    - Stool
  - Radiologic
    - GI Studies
  - Biopsy
    - Bone Marrow
  - Endoscopy
    - Detect source



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Anemia

- Management of patient care
  - Goals of Care
  - Interventions
  - Potential complications
  - Additional nursing considerations
    - If transfusion required
      - Follow guidelines of AABB

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Leukopenia

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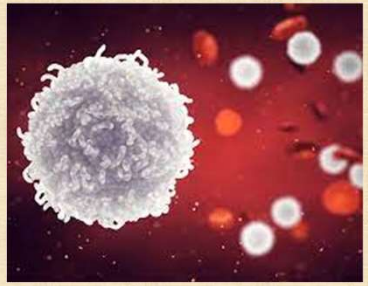
Leukopenia

- Low white blood cell count – usually a lack of neutrophils
  - Anemia
  - Myelodysplastic syndrome
  - HIV/AIDS
  - Rheumatoid arthritis
  - Tuberculosis
  - Lymphoma
- Increased risk of infection
  - Pneumonia
  - Sepsis
  - Skin
  - UTI
  - GI tract



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Leukopenia

- Etiology and risk factors
  - Malignant cells
  - History chemotherapy
  - Radiation therapy
  - Medication
    - Antivirals, antipsychotics
  - Autoimmune disorders
    - Lupus erythematosus
    - Rheumatoid arthritis
- Signs and Symptoms
  - Signs
  - Symptoms
- Diagnostic Studies
  - Laboratory
  - Radiologic
  - Bone marrow biopsy

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Leukopenia

- Goals of Care
  - No infections
  - Early detection and intervention
- Interventions
  - Focused
    - Assessment of patient
    - Prevention of infection
    - If infection, appropriate treatment
- Potential complications
  - Infection with neutropenic fever
    - Antibiotics
    - Antifungals

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Thrombocytopenia

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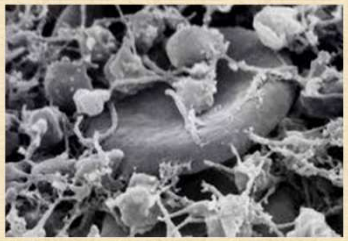
Thrombocytopenia

- Pathophysiology
  - Platelet count is diminished
  - Increased risk of bleeding
  - Low enough, spontaneous bleeding



- Etiology and Risk Factors
  - Decreased production
    - Malignant cells
    - Current or recent antineoplastic treatment
    - Radiation to bone
  - Increased destruction
    - DIC
    - Antibody mediated
      - ITP
      - HIT
    - Thrombotic Thrombocytopenia Purpura
      - TTP
    - Hemolytic uremic Syndrome
    - Sepsis
    - Mechanical injury
  - Sequestration of platelets

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Thrombocytopenia

- Signs and Symptoms
  - Petechiae
  - Purpura
  - Ecchymosis
  - Epistaxis
  - Oozing from sites
  - Bleeding
- Diagnostic Studies
  - Laboratory
    - Blood
    - Urine
    - Stool
  - Radiologic
    - US spleen
    - Scan liver-spleen
  - Biopsy
    - Bone marrow

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Thrombocytopenia

- Goals of care
  - No bleeding
  - Prevent injury
- Interventions
  - ID and treat cause
  - Possible transfusion
  - DDAVP
  - Immune & glucocorticoids
  - Skin and mucous membrane precautions
- Potential Complications
  - Bleeding after trauma
  - Thromboembolism

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Coagulopathies

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Medication Induced Coagulopathy

- Pathophysiology
  - Effective hemostasis relies on platelets and procoagulant factors
    - Antiplatelet agents
    - Anticoagulants
  - Therapeutic anticoagulation
    - Risk for bleeding
      - Many patients receive anticoagulation as part of their medical treatment plan
        - Atrial fibrillation
        - Prosthetic heart valves
        - Most hospitalized patients have anticoagulation/antiplatelet orders therapy ordered
  - Nurses need awareness of the many classes of medications
    - Anticoagulant effects
    - Antiplatelet effects

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Medication Induced Coagulopathy

- Etiology & Risk Factors
  - Anticoagulant or antiplatelet agents
  - Advanced age
  - Risk for falls and other trauma
  - Invasive procedure or surgery
- Signs & Symptoms
  - Prolonged bleeding
    - Incisions and puncture sites
  - Purpura, ecchymosis, hematomas, epistaxis
  - Spontaneous and/or uncontrollable hemorrhage
    - Venipuncture
    - Tubes
    - Drains
    - Lines
    - Incisions
    - Wounds
  - Bleeding from GI tract

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Medication Induced Coagulopathy

- Goals of care
  - Identify patients at risk
  - Early recognition
  - Prevention of shock, anemia, organ /tissue damage or ischemia
- Interventions
  - Monitor labs
  - Review medication records
  - Look for bleeding
  - Reverse anticoagulation
    - Protamine
    - Warfarin
- Potential complications
  - Hypovolemic shock
  - Anemia
  - Tissue and organ ischemia and damage

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Antithrombotic Medications

From Good, V.S., Kirkwood, P.L. (2018). *Advanced critical care nursing* , 2nd ed. St. Louis, Elsevier.

Medication	Mechanism of Action	Reversal
Abciximab (Reopro)	Glycoprotein IIb/IIIa receptor blocker	Platelet transfusion
Alteplase (tPA)	Thrombolytic (Fibrinolytic)	No specific reversal agent
Apixaban (Eliquis)	Direct Xa inhibitor	Andexanet alpha (ANDEXXA)
Argatroban	Direct thrombin inhibitor	No specific reversal agent
Aspirin	Irreversible inhibitor of cyclooxygenase	Platelets, consider use of desmopressin
Bivalirudin (Angiomax)	Direct thrombin inhibitor	FFP, cryoprecipitate, FVIIa
Clopidogrel (Plavix)	Inhibition of ADP-P2Y12 receptors on platelets	Platelet transfusion
Dabigatran (Pradaxa)	Direct thrombin inhibitor	Idarucizumab (Praxbind)
Eptifibatide (Integrilin)	Glycoprotein IIb/IIIa receptor blocker	Platelets
Fondaparinux (Arixtra)	Inhibition of Xa	No specific reversal agent
Heparin (unfractionated)	Xa and thrombin inhibition	Protamine
Low-molecular-weight heparin	Same as unfractionated heparin; mainly Xa effect	Protamine
Rivaroxaban (Xarelto)	Direct anti-Xa inhibitor	Andexanet alpha (ANDEXXA)
Warfarin (Coumadin)	Vitamin K antagonist	IV vitamin K, PCC, FFP

FFP , Fresh frozen plasma; *PCC* , prothrombin complex concentrate, *tPA* , tissue plasminogen.

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Disseminated Intravascular Coagulation-DIC

- Pathophysiology
- Unique coagulopathy
  - Intravascular bleeding and clotting
  - Always a secondary diagnosis
- Clots where not needed
  - Microvasculature
- Unable to clot where bleeding
- Available platelets and clotting factors are depleted
  - Consumptive coagulopathy
- Abnormal fibrinolysis
  - Fibrin degradation products
    - FDP-FSP
    - D-dimers
- Difficult diagnosis in liver failure

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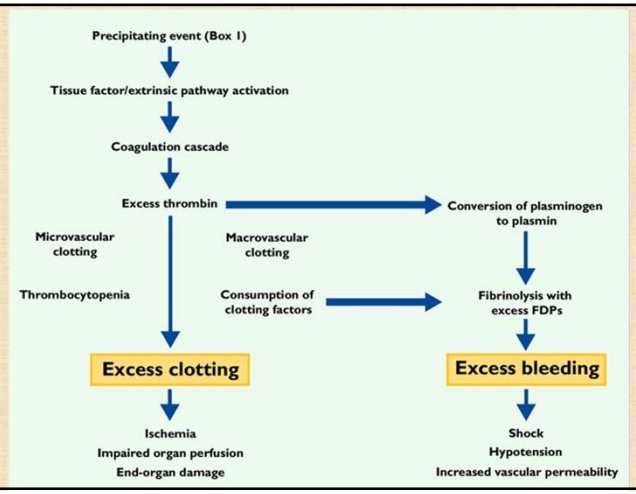

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Disseminated Intravascular Coagulation

- Etiology & Risk Factors
  - Infection
  - Trauma
  - OB complications
  - Metastatic cancer
  - Immunologic conditions
- Signs and Symptoms
  - Spontaneous bleeding
  - Petechiae & purpura
  - Ecchymosis
  - Hematomas
  - Epistaxis
  - Spontaneous or uncontrolled hemorrhage
  - Organ dysfunction

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Disseminated Intravascular Coagulation

- Diagnostic Studies
  - Blood
    - Coagulation panel
      - PT
      - PTT
      - Fibrinogen
      - Platelets
      - FDP
      - D-dimers
  - Radiologic
    - Noncontributory

PT prolonged
PTT prolonged
Platelets decreased
Fibrinogen level decreased
Fibrin split products increased

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Disseminated Intravascular Coagulation

- Goals of care
  - Identification and treatment
  - Adequate tissue perfusion
  - Normal hemostasis
  - Decreased bleeding
- Interventions
  - Treat primary disorder
  - Provide critical care support
  - Prevent hypothermia
  - Transfuse as necessary
  - Vitamin K administration
  - Gently mobilize patient
- Potential complications
  - Hypovolemic shock
  - Multiple organ dysfunction syndrome
  - Monitor adverse reaction to transfusion therapy

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Blood Transfusions & Complications

25

Blood transfusions and complications

- Patient Management
  - Proper indications for transfusions
  - Correct patient monitoring pre, during and post transfusion
  - Early recognition and management of reactions is critical

26

Blood transfusions and complications

- Transfusion Reactions
  - **Febrile reaction, nonhemolytic – most common**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
Febrile	Sensitization to donor WBC's. platelets, or plasma protiens	Fever – rise 1° C, chills, flushing, nausea and vomiting	Immediately or up to 4 hours posttransfusion	✓ Stop transfusion ✓ Continue NS ✓ Notify provider and blood bank ✓ Give acetaminophen as ordered

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Blood transfusions and complications

- Transfusion Reactions
  - **Allergic reaction**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
Allergic	Recipient sensitivity (IgE antibodies) against donor plasma proteins	Hives, itching, flushing, rash, wheezing, angioedema Anaphylaxis is possible	Occurs immediately or up to 4 hours posttransfusion	✓ Stop transfusion ✓ Continue NS ✓ Notify provider and blood bank ✓ Give antihistamine as ordered ✓ If signs resolve, transfusion may be restarted with a physician order

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Blood transfusions and complications

- Transfusion Reactions
  - **Hemolytic reaction**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
Hemolytic	Transfusion of ABO- or Rh-incompatible blood	Fever, chills, hypotension, chest pain, tachypnea, tachycardia, shock or circulatory collapse, hemoglobinuria, DIC	Immediately or may occur up to 24 hours posttransfusion	✓ Stop transfusion ✓ Continue NS ✓ Call Rapid Response if warranted ✓ Notify provider and blood bank immediately; provide supportive therapy to maintain blood pressure and urine output

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Blood transfusions and complications

- Transfusion Reactions
  - **Bacterial reaction**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
Bacterial	Blood contaminated with organisms	High fever, chills, flushing, tachycardia, shock, DIC, renal failure	Within 30 minutes of the start of the transfusion	✓ Stop transfusion ✓ Notify provider and blood bank ✓ Give antibiotics, IV fluids, vasopressors

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Blood transfusions and complications

- Transfusion Reactions
  - **Transfusion Associated Circulatory Overload (TACO)**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
TACO	Transfusion is administered more rapidly than the circulation can accommodate	Dyspnea, cough, crackles, pulmonary edema, tachycardia, hypertension	Anytime during the transfusion	✓ Slow the transfusion ✓ Administer O2 ✓ Obtain stat CXR ✓ Provide frequent monitoring ✓ Administer diuretics as ordered

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Blood transfusions and complications

- Transfusion Reactions
  - **Transfusion Related Acute Lung Injury (TRALI)**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
TRALI	Reaction involving antibodies from donor and recipient WBC antigens	Severe respiratory distress, hypoxia, fever, cyanosis	Occurs immediately or up to 6 hours posttransfusion	✓ Stop transfusion ✓ Call rapid response if warranted ✓ Notify provider and blood bank ✓ Administer O2 ✓ Monitor closely, may require critical care management

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Blood transfusions and complications

- Transfusion Reactions
  - **Transfusion Related Immune Modulation (TRIM)**

Reaction	Mechanism	Signs & Symptoms	Time of Occurrence	Treatment
TRIM	Immune response to transfusion where antibodies form against antigens in the transfused blood	Decreased lymphocytes and immune function; antibodies that can make future transfusion or organ donation difficult	Initial – decreased lymphocytes Late – formation of antibodies	✓ Monitor for signs of infrection ✓ Continue to restrict transfusion therapy

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Oncological Emergencies

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Oncological Emergencies

- Pericardial Tamponade
- Tumor Lysis Syndrome
- Sepsis
- DIC
- SIADH

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Neurological Disorders

Kelly Urban, PhD, MEd, RN, CCRN-K, TCRN, CEN

UAMS Medical Center

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Topics

- Brain Death
- Anoxic Injury
- Encephalopathy
- Neurological Infections
- Seizures
- Space Occupying Lesions

2

Brain Death

- Irreversible absence of brain function
- Criteria includes:
  - Unresponsiveness
  - Lack of movement
  - Absence of cranial nerve function
  - Absence of spontaneous respirations

3

Determination of Brain Death

Apnea testing

Hyperoxygenate with 100% O_2 for at least 10 min

Obtain baseline ABG

Remove from vent (provide O_2 6L)

Monitor for spontaneous respiratory effort or more than 10% change in BP, HR, or pulse ox

Obtain ABG (looking for ↑ CO_2)

Prior to testing, rule out all conditions which impair neuro function
- Normalize electrolytes
- Body temp above 95°F (35°C)
- Consider drug levels (neuromuscular blockade, sedation, anesthetics)

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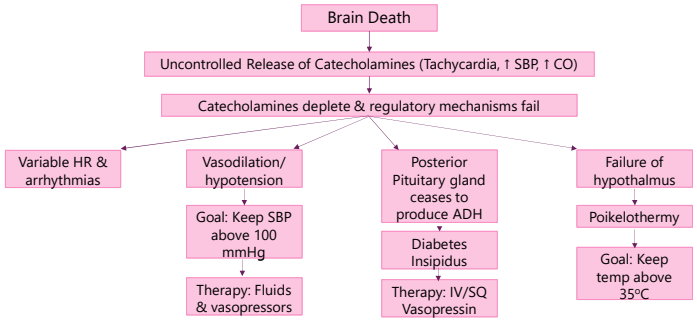
Response of Body to Brain Death



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Anoxic Injury

Brain injury is a leading cause of mortality and morbidity among cardiac arrest survivors.

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Encephalopathy

Encephalopathy is a change in how your brain functions.

It can be a temporary disturbance or it could permanently damage the brain.

There are many possible causes of encephalopathy, like an infection or an underlying condition.

Treatment depends on the cause.

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Neurological Infections

Viral infections: Encephalitis, meningitis, rabies, herpes simplex virus (HSV)

Bacterial infections: Meningitis, brain abscess, spinal cord infection

Fungal infections: Cryptococcal meningitis, histoplasmosis

Parasitic infections: Toxoplasmosis, cysticercosis

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Space Occupying Lesions (Brain Tumors)

Primary Tumors

- Meningioma
- Astrocytoma
- Glioblastoma Multiforme
- Oligodendroglioma
- Ependymoma

Metastatic Tumors

- Lung
- Breast
- Colon
- Renal
- Melanoma
- Thyroid
- Uterine

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Brain Tumors Causes

- Cause of most is unknown
- Only modifiable risk factor is exposure to ionizing radiation
- Genetic predisposition

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Brain Tumors – Signs and Symptoms

- Localized: anatomic and clinical correlation
- Generalized:
  - Decreased LOC
  - Seizures
  - Cognitive-behavioral deterioration
  - Fatigue

Associated with ICP elevation:
- Headache
- Vomiting
- Decreased LOC
- Papilledema
- Edema of optic disk

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Brain Tumors - Diagnostics

Radiologic

- CT Head (without contrast)
- MRI with and without contrast
- MRS (magnetic resonance spectroscopy)
- PET
- Cerebral angiogram
- Systemic workup

Laboratory

- Endocrine workup
  - Identify neuroendocrine involvement
- Visual
- Audiometry (hearing deficits – acoustic neuroma)

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Brain Tumors Patient Care

- Symptom Management
  - Vasogenic edema
  - Seizures
  - VTE
  - Nausea/Vomiting
  - Fatigue
- Infection Control
- Nutrition
- Fluid and electrolytes
- Bowel and Bladder elimination
- Communication, cognition, and swallow
- Skin care
- Psychosocial issues

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Brain Tumors Treatment

- Surgery
  - Biopsy
  - Craniotomy
  - Shunt
- Radiation
- Chemotherapy

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Intracranial Infections – Bacterial Meningitis

Pathophysiology

- Bacterial organisms gain access to subarachnoid space, CSF, and pia mater and arachnoid mater layers of the meninges
- Bacteria then proliferates causing meningeal inflammation (can obstruct CSF flow)
- Vasogenic edema and increased ICP results

Risk Factors

- Neisseria meningitides and Stretococcus pneumoniae most common
- Sources of infection:
  - Neurologic surgery or invasive procedures
  - Otitis media, sinusitis, mastoiditis, osteomyelitis, dental abscess, recent dental work
  - Exposure to infectious organisms
  - IV medication use

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Intracranial Infections – Bacterial Meningitis

Signs & Symptoms

- Headache (becomes progressively worse)
- General signs of infection: malaise, fever, tachycardia, chills
- Rash (meningococcal meningitis – red/purple petechiae progressing to purpura over trunk, legs, conjunctiva, and mucous membranes – does not fade when compressed)
- Neurologic (irritability, confusion; progressive decrease in LOC; seizures)
- Meningeal Irritation (headache, photophobia, nuchal rigidity, Brudzinski's sign, Kernig's sign)
- Nausea/Vomiting

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Intracranial Infections – Bacterial Meningitis Diagnostics

Laboratory

- Cultures
- Serology Tests

Radiologic

- CT scan
- Skull x-rays

Lumbar puncture

EEG

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Intracranial Infections – Bacterial Meningitis Management

- Anticipated patient trajectory
- Monitor VS/neurologic status
- Pharmacology
  - Antibiotics
  - Dexamethasone
  - IV fluids
  - Antipyretics
  - Anxiolytics
  - Anticonvulsants

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Intracranial Infections – Bacterial Meningitis Complications

- Waterhouse-Friderichsen syndrome (adrenal hemorrhage)
  - Results in adrenal insufficiency, hypotension, resp distress, and circulatory collapse
- DIC
- Brain abscess, subdural effusions, encephalitis
- Hydrocephalus
  - Purulent exudate and meningeal fibrosis in the subarachnoid space can obstruct CSF flow and reabsorption
- Increase ICP
  - Accumulation of purulent exudates, hydrocephalus, and cerebral edema
- Seizures
- Fluid/Electrolyte imbalance (SIADH)

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Intracranial Infections – Viral Encephalitis

Pathophysiology

- Inflammation of brain tissue caused by a virus; migrates through choroid plexus, cerebral capillaries, or along peripheral nerves into CNS

Etiology/Risk Factors

- Herpes simplex virus
- Enterovirus, cytomegalovirus, measles, mumps, varicella, lymphocytic choriomeningitis viruses, Epstein-Barr virus, rabies virus
- Arboviruses (tick/mosquito) (i.e. West Nile)

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Intracranial Infections – Viral Encephalitis Signs & Symptoms

- Onset/progression varies with pathogen and area of brain involved
- Common: headache, fevered, altered LOC, nuchal rigidity
- Herpes: fever, headache, N/V, altered LOC, seizures
  - Frontal and temporal lobe damage from virus may cause strange behavior, personality changes, hemiparesis, aphasia, temporal lobe seizures, hallucinations, signs of increased ICP, and eventually uncal herniation
- Arthropod-borne encephalitis: gradual onset of flu-like symptoms
  - Lymphadenopathy and erythematous rash (West Nile)
  - Changes in LOC, meningeal signs, seizures, tremors, ataxia, abnormal reflexes, muscle weakness, motor/cranial nerve deficits
  - West Nile encephalitis: severe muscle weakness or flaccid paralysis

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Intracranial Infections – Viral Encephalitis Diagnostic Studies

Lab Findings
- CSF Cultures
- Lumbar puncture
  - Increased WBCs, RBCs with cerebral hemorrhage
  - Viral cultures
  - IgM antibodies by PCR
- Serologic tests

EEG

Radiologic
- CT scan
- MRI
  - Initially normal
  - Later: abnormalities of affected areas

Brain Tissue Biopsy

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Intracranial Infections – Viral Encephalitis Patient Management

Pharmacology
- Antivirals (Herpes and varicella-zoster virus)
- IV fluids
- Antipyretics
- Anticonvulsants

Potential Complications
- Increased ICP (due to brain inflammation and cerebral edema)
- Seizures
- Fluid/electrolyte imbalance

23

Seizures

Pathophysiology
- Paroxysmal episodes of desynchronized and excessive electrical discharges from neurons that result in a sudden transient alteration in brain function

Status epilepticus: brain's excitatory and inhibitory circuits become altered, allowing prolonged or frequent recurring seizures

Seizures can increase cerebral metabolic demand and can deplete high-energy phosphates, causing failure of energy-dependent functions

Aspiration and trauma may occur during a seizure.
- Prolonged seizures can cause cerebral edema, neuronal dysfunction and injury, hyperthermia, metabolic derangements, arrhythmias, rhabdomyolysis, fractures, and death

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Seizures – Etiology & Risk Factors

- Inadequate levels of or withdrawal from an anticonvulsant therapy
- Acute withdrawal from the chronic use of sedatives or depressants
- Medication toxicity or adverse medication reaction
- Metabolic disorders (i.e. uremia, hypoglycemia, electrolyte disorders, fever)
- Neurologic pathologic conditions such as TBI, CNS infections, brain tumors, cerebral edema, stroke, cerebral anoxia, AVM, increased ICP

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Seizures – Tonic-clonic

- Generalized seizure involving the entire or large areas of both cerebral hemispheres.
- LOC followed by brief period of muscle rigidity and then rhythmic muscle jerking bilaterally
- Apnea may occur (briefly) during the tonic phase
- Hyperventilation may occur during the clonic phase (or occur as seizure ends)
- Incontinence, profuse salivation, and diaphoresis common
- Usually lasts 1-5 minutes
- Postictal: headache, amnesia (of event), confusion, myalgia, fatigue

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Seizures – Myoclonic

- Sudden, brief muscular contractions that may occur singly or repetitively
- Usually involves the extremities or face (can be generalized)

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Seizures – Partial

- Localized in an area of one hemisphere
- May be simple or complex
- Simple partial seizure – patient remains conscious
- Complex partial seizure – patient has loss of awareness and amnesia of event
- May progress and secondarily generalize to include both hemispheres with LOC
- Clinical presentation related to area of brain affected

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Seizures – Partial Clinical Presentation

- Motor events (face twitching or limb jerking)
- Automatisms (lip smacking, fidgeting, blinking) – common with complex partial seizures
- Sensory events such as numbness/tingling, visual, auditory, gustatory, or vertiginous symptoms
- Psychic events such as hallucinations and illusions
- Autonomic events such as diaphoresis, vomiting

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Status Epilepticus

- Last > 5 minutes or repetitively without full recovery between ictal episodes
- Clinical or subclinical
  - Subclinical: patient is unresponsive to all stimuli; eyes may deviate away from side of seizure focus; EEG demonstrates seizure activity

30

Seizures – Diagnostic Findings

Laboratory
- Electrolyte or metabolic abnormalities
  - Na
  - Hypomagnesemia
  - Hypoglycemia
  - Hypoxemia
- Serum enzyme levels (CPK) elevated following seizure
- Myoglobinuria common after prolonged seizure
- Other tests (toxicology screen) may reveal disorders that precipitated the seizure

Radiologic
- Determine precipitating or complicating cause

EEG
- Identifies seizure activity
- Localizes the foci

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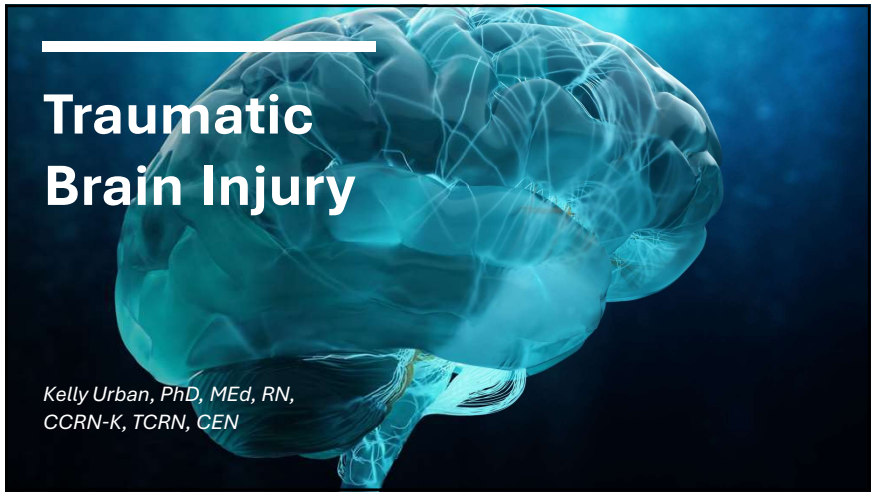

Traumatic Brain Injury

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TBI Epidemiology

- 1.7 million cases annually
- Contributes to 30.5% injury-related deaths in US annually

2

Mechanisms of Injury

Blunt

- Falls**
- MVCs
- Sports-related injuries
- Recreational vehicle injuries

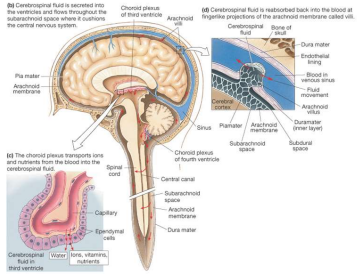
Penetrating

- Firearms
- Exploding objects or projectiles

3

Skull/Cranial Vault

- Non-compressible and filled to capacity with essential components
  - Brain (80%)
    - 75% water
  - CSF (10%)
    - ~ 100-125 ml
  - Blood (10%)
    - 80% is venous



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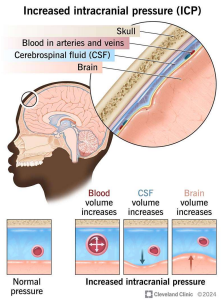
Intracranial Pressure

- Pressure exerted in the cranium by its contents:
  - Brain
  - Blood
  - Cerebrospinal Fluid (CSF)



- Compliance is the ability of the brain to adapt to increasing pressure without increasing the ICP

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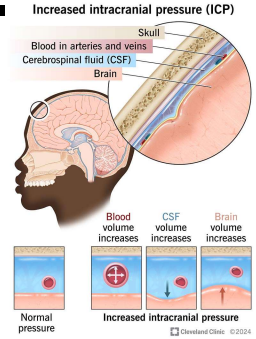
Intracranial Pressure

- Normal Pressures
  - Child – 0-5 mmHg
  - Adult – 5-12 mmHg
- Pressures > 20 mmHg
  - Elevated
  - Compromise blood flow → cerebral hypoxia



- Significantly high pressures → brain forced through any opening (herniation)

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Cerebral Perfusion Pressure

$$CPP = MAP - ICP$$

- Perfusion to cerebral tissue
- Average CPP is 80-100 mmHg
- Dependent on:
  - Mean Arterial Pressure (MAP)
  - ICP

7

Cerebral Perfusion Pressure

Cerebral Perfusion Pressure (CPP)	Consequences
> 70 mmHg	Ideal
> 60 mmHg	Required for consciousness
< 50 mmHg	Critical reduction in brain tissue oxygenation
< 40 mmHg	Cerebral flood flow to 25% of normal
< 30 mmHg	Irreversible brain ischemia

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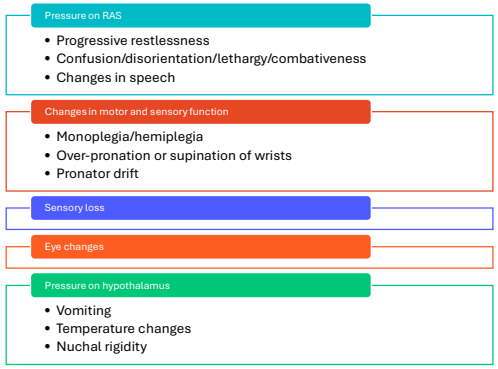
Signs of Increased Intracranial Pressure



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Increased ICP Signs/Symptoms

Early

- Headache
- Vomiting

Late

- Cushings Triad (pressure on brainstem)
  - Systolic hypertension with widened pulse pressure
  - Bradypnea or irregular respirations
  - Bradycardia

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Indications of Brain Herniation

- GCS ↓ by 2 or more points
- BP and ICP significantly ↑
- Heart rate ↓ by 30-40 bpm
- Respiratory pattern changes
- Unilateral or bilateral dilation of pupils
- Pupils fail to react to light
- Posturing

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Neurological Assessment

Determine	Determine LOC
Assess	Assess Motor Function
Assess	Assess Pupillary Function and Eye Movement
Assess	Assess Vital Signs

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Level of Consciousness

Alert
- Responds immediately to minimal external stimuli

Lethargic
- State of drowsiness or inactivity (patient needs increased stimulus to be awakened but is easily arousable)

Obtunded
- Very drowsy when not stimulated (follows simple commands when stimulated, duller indifference to external stimuli still exists, and response is minimally obtained)

Stuporous
- Minimal spontaneous movement, arousable only with vigorous and continuous external stimuli, motor responses to tactile stimuli are appropriate, verbal responses are minimal and incomprehensible

Comatose
- Vigorous stimulation fails to produce any voluntary neural response
- Arousal and awareness are absent, no verbal responses
- motor responses may be purposeful withdrawal to pain (light coma) or non-purposeful/absent movement (deep coma)

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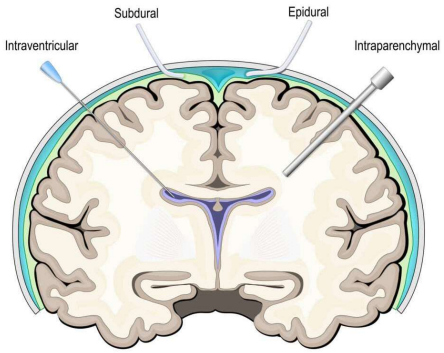
ICP Monitoring



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Treatment of Increased ICP

Goal: ↓ volume of skull constituents to reduce ICP

- Reduce volume of brain
- Reduce volume of blood
- Reduce volume of CSF

Other Treatment Measures

- BP management
- Airway/ Oxygenation management
- Cerebral metabolism reduction
- Euglycemia
- Anticonvulsant prophylaxis
- Normothermia

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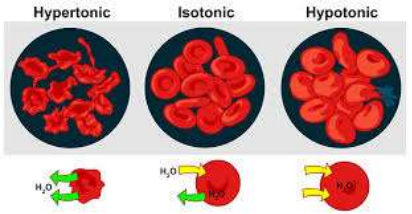
Reduce Volume of Brain

- Osmotic Diuretics (moves fluid out of tissues → blood stream)
  - Mannitol (preferred as bolus)
- Hypertonic Saline
  - 3-23.4% available
  - Advantages:
    - No diuretic effect
    - Does not accumulate in neural tissue
  - Risk Factors:
    - Bleeding
    - Prolonged coagulation times
    - Hypokalemia
    - Hyperchloremic acidosis



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Reduce Volume of Blood

Patient Positioning
- ↑ HOB
- Head midline
- Avoid hip flexion

Avoid Venous Compression
- Tracheostomy tubes not too tight
- C-collar not too tight

Ventilator Management
- Lower PEEP/Tidal Volumes

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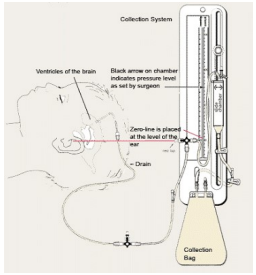
Reduce Volume of CSF

- Ventriculostomy
  - Removal 1-2 ml can temporarily ↓ ICP 1-2 mmHg



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Blood Pressure Management

- Systolic ↑ more than diastolic
- Optimal hemodynamic levels:
  - MAP > 90 mmHg
  - CPP > 70 mmHg
  - PAOP 10-15 mmHg
- Adequate BP accomplished with:
  - Fluids
  - Vasopressors
  - Inotropic agents

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Airway/Oxygenation Management

- Suctioning can ↑ ICP
  - Consider medicating prior to suctioning
  - Hyperoxygenate prior to suctioning
  - Limit suctioning to 2 passes of suction catheter not to exceed 10-15 seconds per pass

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Ensure Adequate Oxygenation

- Maintain adequate hemoglobin
- Ensure adequate PaO2

21

Reduce Cerebral Metabolism

- Maintain darkened room
- Quiet room (speak softly)
- Limit visitors
- Cluster nursing activities
- Limit dialogue, keep topics light-hearted

22

Euglycemia

- Serum glucose between 80-120 mg/dL
- Insulin drip may be necessary
- Avoid IV fluids and medications mixed with D5W

23

Anticonvulsant Prophylaxis

- Seizures are more likely in patients with:
  - GCS < 10
  - Depressed Skull Fractures
  - Subdural, Epidural, or Intracranial hematomas

May prevent early-onset (7 days) seizures but have little effect on late onset seizures

24

Normothermia

- Treat fevers with antipyretics
- Sponge baths
- Cooling blankets

- AVOID shivering

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Goals of Treatment

Pulse Oximetry ≥ 95%	ICP 20-25 mmHg	Serum Na 135-145
PaO_2 ≥ 100 mmHg	$PbtO_2$ ≥ 15 mmHg	INR < 1.4

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Head Injuries

Focal Injuries

- Skull Fractures
- Basilar Skull Fracture
- Meningeal Bleeds
- Cerebral Contusions/Intracerebral Hematoma

Diffuse Injuries

- Traumatic Brain Injuries (mild, moderate, severe)
- Second Impact Syndrome
- Postconcussive Syndrome
- Diffuse Axonal Injury

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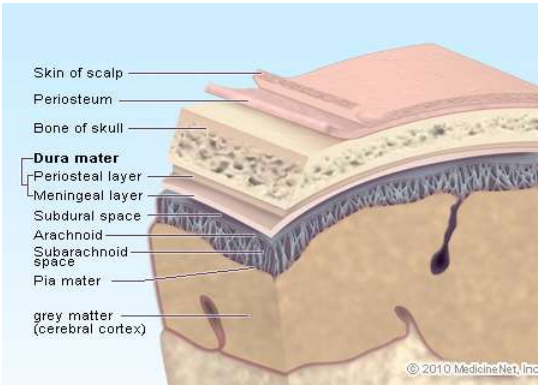

Layers of the Brain

- Scalp
- Skull
- Epidural Space (middle meningeal artery)
- Dura
- Subdural Space
- Arachnoid
- Subarachnoid
- Pia
- Brain

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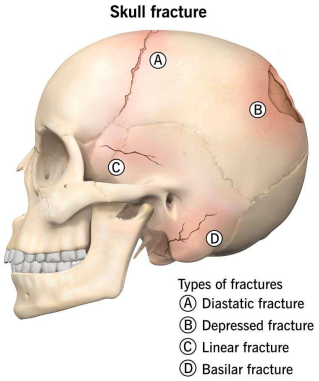
Skull Fractures

- Frontal/occipital bones are strongest
- Temporal bone is weakest (50% of skull fx)



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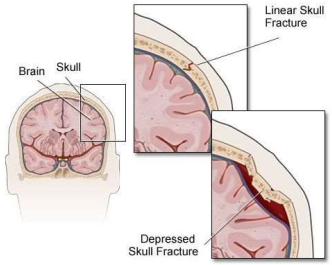
Skull Fractures

- Linear Fracture (most common)
  - Temporal/parietal most common
  - Epidural hematoma
- Comminuted Fracture
  - Multiple bone fragments (may penetrate through meninges)
- Depressed Skull Fracture
  - Inward displacement of bony fragments
- Open Skull Fracture
  - Depressed skull fracture with scalp laceration
  - May involve tear in dura



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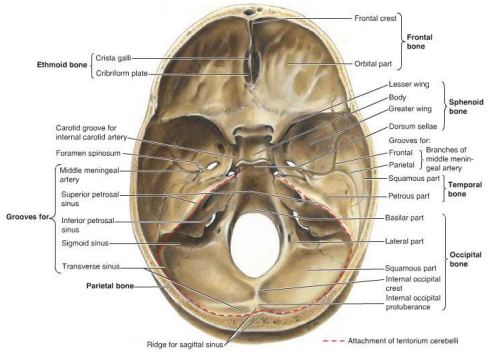
Basilar Skull Fracture



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Basilar Skull Fracture

Location	Symptoms	Nerve Involvement
Anterior Fossa Fracture	• Epistaxis • Rhinorrhea • Subconjunctival hemorrhage • Hemorrhage in the periorbital spaces • Salty taste in mouth	• Anosmia • Ptosis • Loss of sensation to forehead, cornea, & nares
Middle Fossa Fracture	• Otorrhea • Hemotympanum	• Loss of sensation to lower face • Tinnitus • Facial palsy • Nystagmus
Posterior Fossa Fracture	• Ecchymosis behind the ear • Impaired gag reflex	

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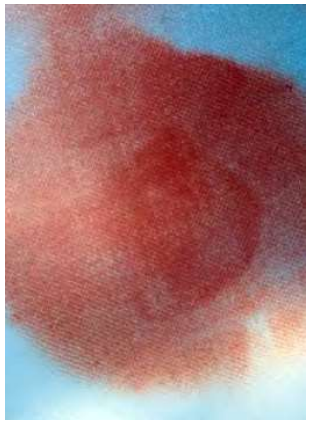


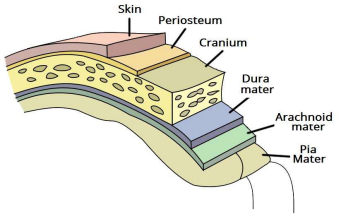
Basilar Skull Fracture

- Assess for CSF
  - Blood from head, nose, or ear
  - Perform "halo" test on blood
- Treatment
  - Monitor/treat signs of ↑ ICP
  - Do NOT pack ears or nose – allow free drainage
  - Do NOT place anything in nose (nasal cannula, packing, NGT, etc…)
  - Discourage nose blowing
  - Antibiotics may be considered

33

Meningeal Bleeds

- 3 Meninges
  - Dura
  - Arachnoid
  - Pia



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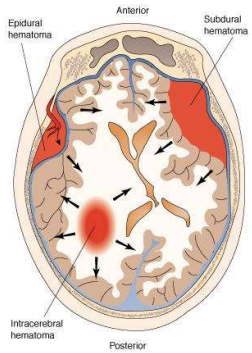
Meningeal Bleeds

- Epidural Bleed
- Subdural Bleed
- Subarachnoid Bleed



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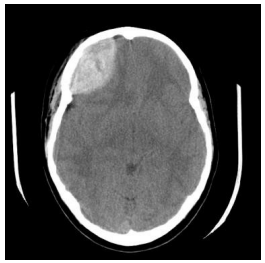
Epidural Bleed

- Bleeding between skull & dura
- Usually with blow to temporal region
- Disruption of middle meningeal artery
- Symptoms
  - LOC → lucid (headache) → rapid decline in LOC
  - "talk and die syndrome"
- Treatment
  - Evacuation of blood (burr hole or surgery)



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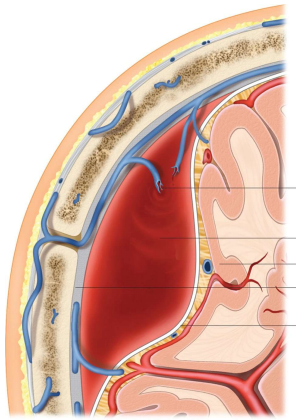
Subdural Bleed

- Bleeding between dura & arachnoid mater
- Venous (bridging veins common)
- Symptoms (similar to epidural bleeds)
  - Acute: symptoms within 48 hours
  - Subacute: symptoms btwn 48 hours & 2 weeks
  - Chronic: > 2 weeks
- Treatment
  - ICP reduction
  - Surgical intervention



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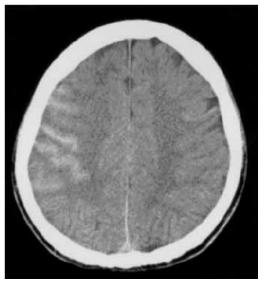
Subarachnoid Bleed

- Bleeding between arachnoid & pia mater
- Usually associated with other brain injuries (contusions)
- Symptoms:
  - Meningeal irritation (worst headache ever)
  - Decreased LOC
  - Motor Deficits
  - Pupillary Abnormalities
- Treatment:
  - Reduce ICP
  - Calcium channel blockers to reduce vasospasms



38

Cerebral Contusions/Intracerebral Hematoma

- Symptoms increase with time (may not be apparent for hours or days)
- Symptoms (dependent on location of lesion)
  - Personality changes
  - Nausea and vomiting
  - Deficits in memory, executive function, behavior and motor function (contralateral hemiplegia)
  - Language deficits
  - Visual disturbances
- Treatment
  - Monitor/treat ICP

39

TBI Definitions

Severity	GCS	Symptoms
Mild TBI	13-15	• Brief (< 30 min) or no loss of consciousness • No retrograde amnesia • No change on imaging studies
Moderate TBI	9-12	• Wide variety of symptoms • Alterations in LOC/confusion • Amnesia • Focal neurological deficits
Severe TBI	3-8	• Prolonged unconscious state • Abnormal pupillary response • Abnormal motor posturing

40

Second Impact Syndrome

- Occurs when the patient suffers a 2nd mild TBI before recovery from 1st
- Rare but fatal
- Symptoms:
  - Loss of autoregulation → cerebral edema

41

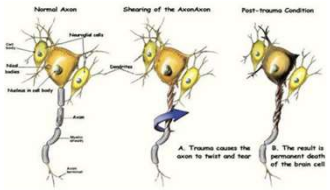
Postconcussive Syndrome

- Patients who suffer a mTBI may develop
- Generally manifests over several days or months after the head trauma
- Usually not permanent but may continue over a period of months
- Symptoms:
  - Nausea
  - Dizziness
  - Persistent Headache
  - Memory/judgement impairment
  - Attention deficits
  - Sleep disturbance
  - Anxiety, irritability, depression
  - Noise/light oversensitivity

42

Diffuse Axonal Injury

- Disruption of axons in the cerebrum leading to disconnection of cortex and brainstem reticular formation
- Clinical Manifestations frequently presents with posturing and autonomic dysfunction
  - Hyperpyrexia
  - Hypertension
  - Diaphoresis



Severity	Coma	Posturing	Outcome
Mild	6-24 hours	Transient (33% cases)	Minimal or no deficits
Moderate	> 24 hours	Transient	Amnesia & cognitive defects
Severe	Prolonged	Persistent	Profound cognitive effects (including persistent vegetative state)

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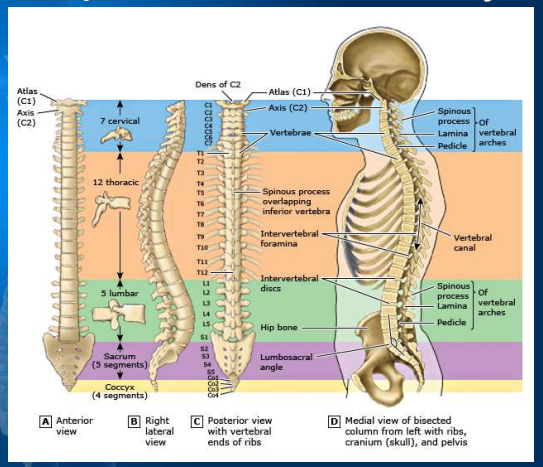
Spine Injuries

1

Spinal Cord Anatomy



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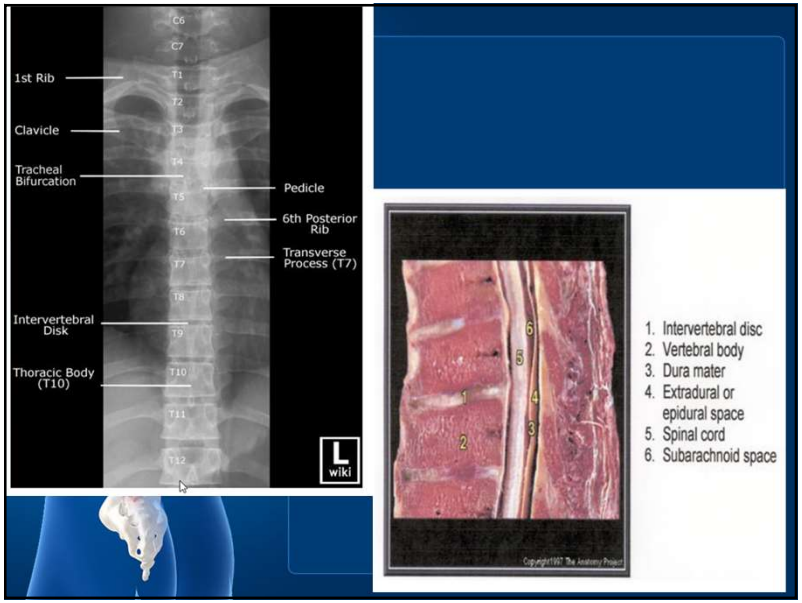

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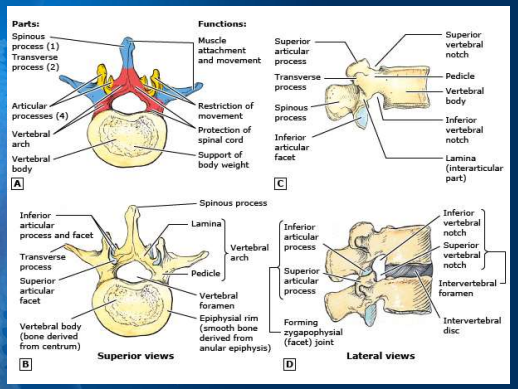
Vertebral Anatomy



4

Spinal Cord Injury

- Epidemiology
  - 12,400 annually (2010)
  - Common Causes
    - MVC (48%)
    - Falls (16%)
    - Violence (12%)
    - Sports (10%)
    - Other (14%)

5

Definitions

- SCIWORA
  - Spinal Cord Injury Without Radiographic Abnormality
- Tetraplegia
  - Paralysis of the arms and legs
- Paraplegia
  - Paralysis of the legs

6

Sites of Injury

- Area of greatest mobility most likely to be injured
  - C4-C7 is most mobile
  - C5 is most commonly injured
  - C4 is 2nd most common
  - C6 is 3rd most common

Mobility of the upper cervical spine ↓ with age of 65 → C1 & C2 are more commonly injured

7

Sites of Injury cont.

- Next most mobile area: T12-L1
- Lumbar fractures
  - Uncommon
  - Frequently associated with improperly worn seatbelts

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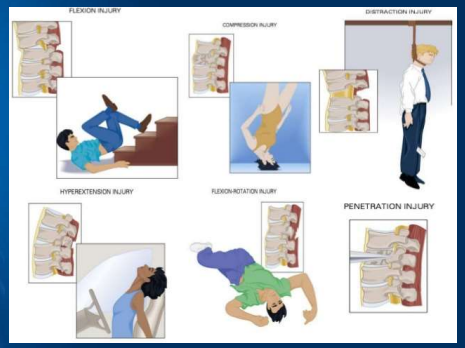
Mechanisms of Injury

- Hyperflexion
- Rotational
- Hyperextension
- Axial Loading/Vertical Compression
- Penetrating Injury



9

Hyperflexion

- Extreme flexion of the spine causing a disruption of the posterior ligament and anterior compression of the spine
- Usually sudden deceleration (diving, MVC)
- Tends to produce compression of the vertebral bodies

10

Rotational

- Extreme flexion rotation or lateral flexion of the spine disrupting the posterior ligament causing spinal instability

11

Hyperextension

- Usual mechanisms include falls or near impact MVC, external forces cause the lower cervical spine injuries, usually causes fractures of the posterior elements
- May be followed by forced flexion, increasing injury
- Treatment:
  - Traction
  - Decompression & Stabilization

12

Axial Loading/Vertical Compression

- Vertical force applied to the vertebral body, often causing burst fractures
- Common Mechanisms include diving, falling
- Treatment:
  - Realignment with traction
  - Surgical stabilization
  - For less severe injuries (< 30% compression): hard shell brace

13

Penetrating Injury

- Direct Cord Contact
- Low Velocity – stab wounds
- High Velocity – GSW

14

Injuries

- Soft Tissue Injury
- Vertebral Injuries
  - Simple Fractures
  - Compression Fractures
  - Teardrop Fractures
  - Atlas Fractures
  - Axis Fractures
  - Dislocations
  - Subluxations

15

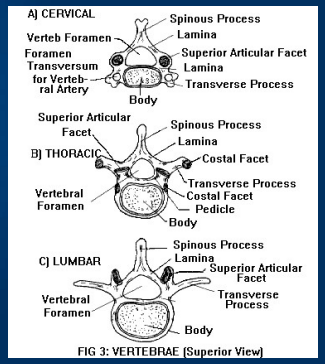
Soft Tissue Injury

- Muscle or ligament injuries
- May cause spinal instability
- Muscle spasms may cause temporary stability

16

Simple Fracture

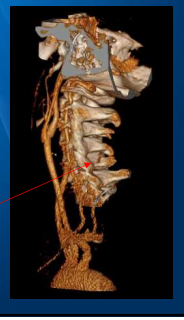
- Single break, usually affecting the spinal process, transverse process, pedicle or facets, but rarely causing neurological deficits

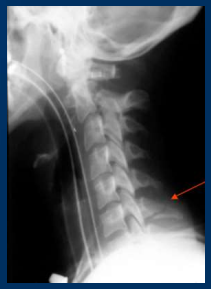


17

Spinous Process Fracture

- Clay shoveler's fracture
- Isolated fracture of 1 of the spinous processes of the lower cervical vertebrae
- Stable injury

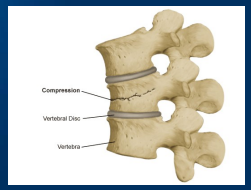




18

Compression Fractures

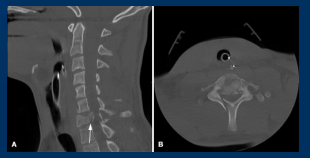
- Flattening or wedging of the vertebral body
- Usually stable but may cause bone or disk material to impinge on spinal canal requiring treatment



19

Burst Fractures

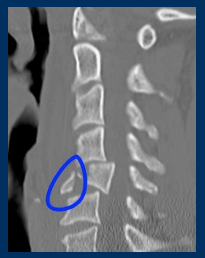
- Vertical compression injury in cervical or lumbar region
- Axially loading injury
- Typically stable unless posteriorly displaced fragments impinge on spinal cord → anterior cord syndrome
- Unstable if:
  - Associated neuro deficits
  - Loss of > 50% vertebral body height
  - > 20° of spinal angulation
  - Compromise > 50% of spinal canal



20

Teardrop Fracture

- Small chip of bone off the anterior/inferior edge of vertebral body
- Unstable fracture usually associated with hyperflexion
- May be displaced laterally, posteriorly, or wedged anteriorly



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Atlas Fracture (C1)

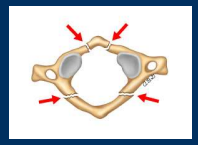
- C1 Burst
- Disruption of anterior and posterior arch of C1
  - Includes Jeffersonian Ring Fracture
- Rarely results in neurological injury
- Treatment
  - External orthosis

22

Specific Fracture: Jeffersonian Ring Fracture

- Rare fracture of C1 where the body splits into several parts
- Usually neurologically intact (fracture fragments may migrate → fatal)
- Associated with axial loading
- Treatment: immobilization until healed

Must have breaks in at least 2 places



23

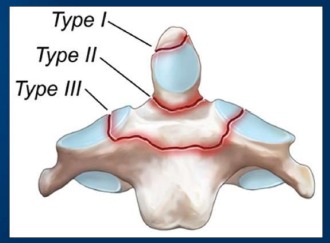
Axis Fracture (C2)

- Caused by extreme flexion, rotation, or extension
- Rarely associated with spinal cord injury
- Includes Odontoid & Hangman's Fractures

24

Specific Fracture: Odontoid (dens) Fracture

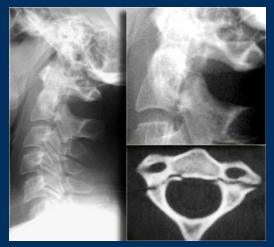
- Type I:
  - Avulsion fracture off the tip of the dens
  - Usually stable
- Type II:
  - Transverse or oblique fracture through the midsection of dens
  - Usually unstable
  - Often displaced anteriorly or posteriorly
- Type III:
  - Fracture through the base of the dens into the axis
  - Treated with light traction, halo orthosis



25

Specific Fracture: Hangman's Fracture

- Bilateral pedicles of C2 causing hyperextension of neck
- Rarely causes neurological impairment
- Treatment:
  - C1-C2 wiring
  - CTO brace vs rigid cervical collar for 6-8 weeks



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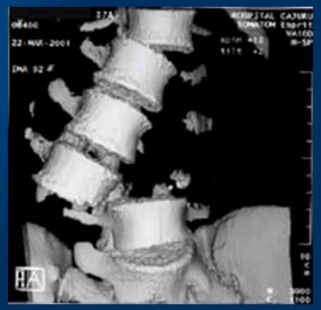
Fracture Dislocation

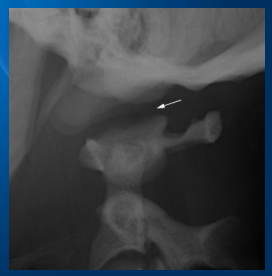
- Hyperextension or hyperflexion with or without rotation

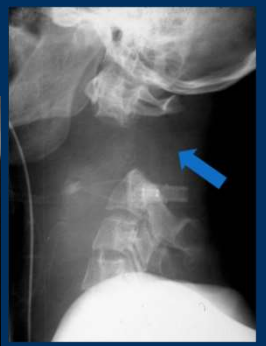


27

Specific Fracture: Atlanto-Occipital Dislocation

- Flexion injury involving the atlas (C1) and axis (C2)
- Unstable injury





Mortality Rate: 70%

28

Subluxation

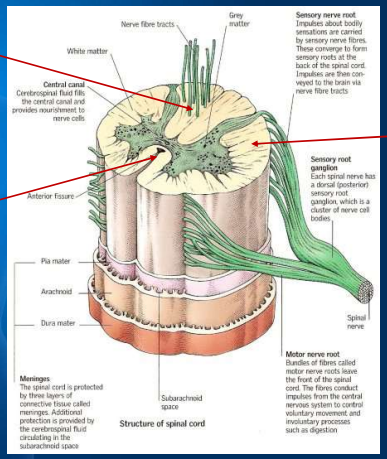
- Rotary subluxation: abnormal rotation of C1-C2
- Treatment:
  - Traction usually results in realignment
  - Surgery may be necessary

29

Injury to the Nervous System

Posterior Cord
Transmits:
- Light touch
- Proprioception
- Vibration

Anterior Cord
Transmits:
- Motor function



Lateral Cord
Transmits:
- Pain
- Temperature
- Crude touch

30

Classification of Spinal Cord Injuries (SCI)

- Cord Concussion
- Cord Contusion
- Cord Laceration
- Cord Transection
- Cord Hemorrhage
- Vascular Damage

31

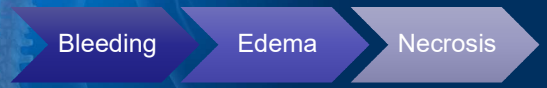
Classification of Spinal Cord Injuries (SCI)

- Cord Concussion
  - Caused by extreme vibration of cord
  - Temporary loss of function (24-48 hours)
  - No neuropathologic changes

32

Classification of Spinal Cord Injuries (SCI)

- Cord Contusion
  - Hemorrhage intramedullary associated with edema
  - Neurological involvement depends on severity of contusion



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Classification of Spinal Cord Injuries (SCI)

- Cord Laceration
  - Spinal cord is partially cut
- Cord Transection
  - Spinal cord is completely severed

34

Classification of Spinal Cord Injuries (SCI)

- Cord Hemorrhage
  - Hematoma can lead to cord compression
- Vascular Damage
  - Lack of perfusion, ischemia

35

Classification of SCI: Functional Loss

- Complete Injury
  - Loss of all voluntary motor and sensory function below the level of the injury
- Incomplete Injury
  - Some neurotransmission distal to the level of the injury
- SCI Syndromes

36

Complete Cord Injuries

- ASIA grade A
- Acute stage:
  - Reflexes are absent
  - No response to plantar stimulation
  - Flaccid muscle tone
  - Males – may have priapism
  - Bulbocavernosus reflex and sensation absent
    - Anal sphincter tone
  - Urinary retention/bladder distension

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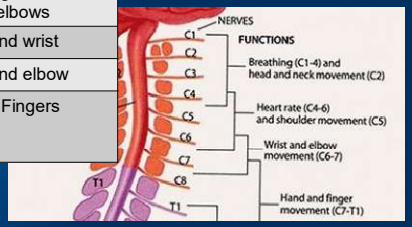
Complete Cord Injuries

Nerve Level	Muscles Innervated	Patient Response
C4	Diaphragm	Ventilation
C5	Deltoid, biceps, brachioradialis	Shrug shoulders, flex elbows
C6	Wrist extensor	Extend wrist
C7	Triceps	Extend elbow
C8	Flexor Digitorum Profundus	Flex Fingers



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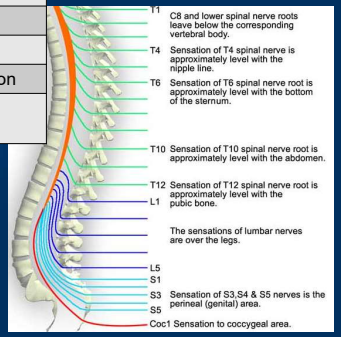
Complete Cord Injuries

Nerve Level	Muscles Innervated	Patient Response
T1	Hand intrinsic muscles	Spread fingers
T2-L1	Intercostals	Vital capacity
L2	Iliopsoas	Hip flexion
L3	Quadriceps	Knee extension
L4	Tibialis anterior	Ankle dorsiflexion
L5	Extension halluces longus	Ankle eversion



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Incomplete Cord Injury

- ASIA Impairment Scale

Grade	Term	Description
A	Complete	No motor or sensory function is preserved in the sacral segments S4-S5
B	Incomplete	Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5
C	Incomplete	Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade < 3
D	Incomplete	Motor function is preserved below the neurological level, and at least ½ of key muscles below the neurological level have a muscle grade of ≥ 3
E	Normal	Motor and sensory function are normal

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Spinal Cord Injury Syndromes: Anterior Cord

- Symptoms

Loss of Descending Motor Nerves

  - Loss of:
    - Motor function
    - Pain, temperature, crude touch/pressure
  - Intact:
    - Proprioception
    - Fine touch/pressure
    - Vibration

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Spinal Cord Injury Syndromes: Posterior Cord

- Symptoms

Loss of Ascending Sensory Nerves

  - Loss of:
    - Proprioception
    - Vibration
    - Fine touch/pressure
  - Intact:
    - Motor function
    - Pain, temperature, crude touch/pressure

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Spinal Cord Injury Syndromes: Central Cord

- Symptoms

Loss of Upper Body Strength

  - Proportionally greater loss of motor function in upper extremities than lower extremities
  - Variable sensory sparing

43

Spinal Cord Injury Syndromes: Brown-Sequard

- Symptoms

Loss of Lateral Nerves

  - Loss of (on side of injury - ipsilateral):
    - Motor function
    - Proprioception
    - Vibration
  - Loss of (opposite side of injury - contralateral)
    - Pain
    - Temperature

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Spinal Cord Injury Syndromes: Cauda Equina

- Caused by landing on buttocks: damage to lower spinal cord
- Symptoms
  - Results in varying degrees of motor and sensory loss in lower body
  - Problems with bowel/bladder control
  - Problems with sexual function

45

Spinal Cord Injury Syndromes: Horner's

- High cervical lesions
- Related to damage to the cervical portion of the sympathetic chain
- Marked by ipsilateral:
  - Ptosis
  - Pupillary constriction (miosis)
  - Anhydrosis (inability to sweat)

46

Spinal Shock

- Temporary local neurological condition that occurs immediately after the spinal cord injury
- Swelling and edema of the cord create the effect of a physiologic transection with disruption of nerve conduction
- Completeness of SCI is indeterminable until this shock state abates

47

Spinal Shock

- Symptoms
  - Severe pain just above the injury
  - Flaccid paralysis with absent reflexes
  - Lack of sensory function
  - Impaired thermoregulation
  - Bowel distension/ileus

48

Spinal Shock

- Outcomes
  - Return of the bulbocavernosus reflex indicates the resolution of spinal shock
  - Usually subsides in hours to weeks
  - Spasticity usually supersedes the flaccid state after several weeks in areas where no function has returned

49

Neurogenic Shock

- Bradycardia & hypotension are secondary to autonomic dysfunction that occurs with injuries at or above T6
  - Interrupts the normal sympathetic outflow from T1-L2 resulting in unopposed vagal tone
  - Lack of sympathetic outflow causes peripheral vasodilation with decreased SVR
  - Blood pools in extremities, decreasing venous return to heart → ↓ CO & Systolic Blood Pressure

50

Assessment of Patient with SCI

- Sensation
- Motor
- Reflexes

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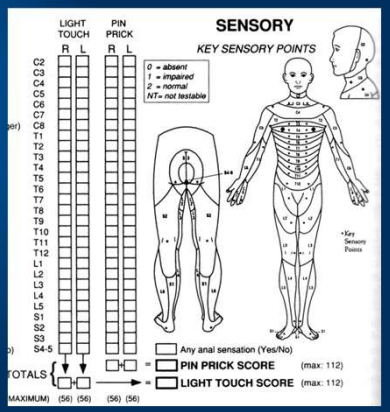
Sensory Assessment

Table 26-6 Key sensory landmarks

Level	Dermatome
C4	shoulders
C6	thumb
C7	middle finger
C8	little finger
T4	nipples
T6	xiphoid
T10	umbilicus
L3	just above patella
L4	medial malleolus
L5	great toe
S1	lateral malleolus
S4-5	peri-anal



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Sensory Assessment

- May have radicular pain at point of injury
  - Waves of stabbing or sharp pain
  - Band of burning pain at point where feeling stops

Radicular Pain: pain radiated along a dermatome

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Sensory Assessment

- To Test Posterior Cord
  - Use a cotton wisp to test for light touch
  - Use a tuning fork to test for vibration
  - Use finger placement to test for proprioception (position) – 2 point discrimination

Always start from areas of decreased sensation and move towards areas of increased sensation when assessing sensation

54

Sensory Assessment

- To Test Lateral Cord
  - Use a needle or the broken end of a wooden stick to test for crude touch

Always start from areas of decreased sensation and move towards areas of increased sensation when assessing sensation

55

Sensory Assessment

- Look for indications of partial cord syndrome
  - “islands of sparing” within a dermatone
  - Sacral sparing (maintenance of perianal sensation, rectal sphincter tone, and flexor toe movement)

56

Motor Assessment

- Muscle strength grading scale (0-5)

Table 26-7 ASIA motor scoring system
(EXTREMITIES)

RIGHT grade	Segment	Muscle	Action to test	LEFT grade
0-5	C5	deltoid or biceps	shoulder abduction or elbow flexion	0-5
0-5	C6	wrist extensors	cock up wrist	0-5
0-5	C7	triceps	elbow extension	0-5
0-5	C8	flexor digitorum prof	squeeze hand	0-5
0-5	T1	hand intrinsics	abduct little finger	0-5
0-5	L2	iliopsoas	flex hip	0-5
0-5	L3	quadriceps	straighten knee	0-5
0-5	L4	tibialis anterior	dorsiflex foot	0-5
0-5	L5	EHL	dorsiflex big toe	0-5
0-5	S1	gastrocnemius	plantarflex foot	0-5
50	← TOTAL POSSIBLE POINTS →			50
GRAND TOTAL: 100				

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Motor Assessment

- Muscle strength grading scale (0-5)

Grade	Strength	
0	no contraction	
1	flicker or trace contraction	
2	movement with gravity eliminated	
3	movement against gravity	4- slight resistance
4	movement against resistance	4 moderate resistance
5	normal strength	4+ strong resistance

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Reflexes

- Deep Tendon grading scale (0-4)
- Priapism: prolonged penile erection from unopposed parasympathetic stimulation (vasodilation/vascular enlargement)

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Reflexes

- Deep Tendon
  - Arm
    - Bicipital: C5
    - Styloradial: C6
    - Tricipital: C7
  - Leg
    - Patellar: L3 (some L4)
    - Achilles: S1

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Level of Injury Determination

- Motor Level:
  - last level with at least 3/5 (against gravity) function
- Sensory Level:
  - last level with preserved sensation
- Radiographic Level:
  - level of fracture on plain X-Rays/CT Scan/MRI

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SCI Diagnostics

- X-Rays
- CT
- MRI
- Myelogram
- Angiography
- Somatosensory Evoked Potentials (SSEP) - OR

62

CT Scan

- Good in acute situations
- Shows bone well
- Sagittal reconstruction is mandatory
- Soft tissues (discs, spinal cord) are poorly visualized
- Do NOT give contrast (mimics blood)

63

MRI

- Shows tumors and soft tissues much better than CT scan
- May be used to clear c-spine in comatose patients
- Usually performed without contrast (unless tumor is suspected)

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Treatment

- Airway and cervical spine
  - C-spine collar
  - Upper cervical injuries may develop hematomas which impede the airway
  - Cervical spine injuries cause loss of ability to cough and clear the airway
    - If suction required, limit pass to 10 seconds to ↓ vagal stimulation
  - Do NOT use succinylcholine without a defasciculating agent

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Treatment

- Spinal Precautions
  - Proper position of cervical collar, TLSO Brace, or lumbar corset as ordered
  - Maintain spine and neck in straight and neutral position
  - Maintain patient flat in bed
  - Log Roll only
  - Restrict use of overly or specialty mattresses
    - Firm mattress surface
  - Transfer patients on spine boards only

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Treatment

- Breathing
  - Phrenic nerve innervates the diaphragm and will be negatively impacted with injuries between C3-C5 ("C3-5 keep the diaphragm alive")
    - Injury above C4: diaphragmatic paralysis/respiratory arrest
    - Injury below C4: diaphragm can support breathing
  - Injury between T1 & T8: loss of intercostal innervation
    - responsible for 35% of respiratory effort
  - Injury above T7: decreased ability to cough deep breath
  - Injury above T12: Loss of abdominal muscles
    - ↓ forceful expiration and coughing

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Treatment

- Circulation
  - Monitor and treat neurogenic shock
    - ICU placement for monitoring (especially with severe cervical level injuries)
  - Patient may have limited or absent peripheral tone, avoid sudden patient movements
  - Hypotension should be avoided
    - MAP of 85-90 mmHg for 1st 7 days recommended
    - Fluid (judicious use to prevent further cord edema)
    - Vasopressors

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Treatment

- Medications
  - Insufficient evidence to support use of corticosteroids for either 24-48 hours

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Log Roll

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Cervical Traction

- Provides temporary stability of the cervical spine
- Weight depends on the level
- Cervical collar can be removed while patient in traction
- Pin Care:
  - ½ Normal Saline & ½ Peroxide
  - Triple antibiotic ointment
  - Every 4-6 hours

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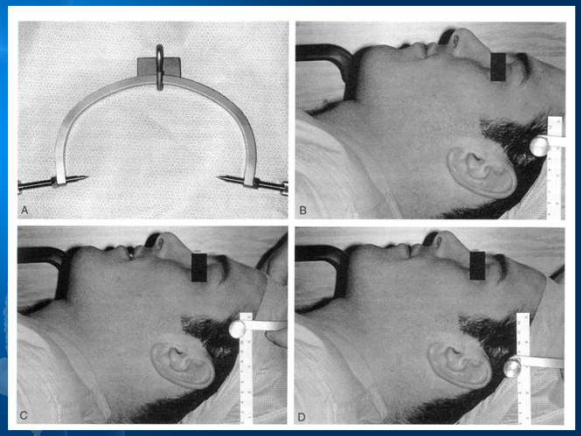
Cervical Traction



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Surgical Decompression/Fusion

- Indications
  - Decompression of the neural elements (spinal cord/nerves)
  - Stabilization of the bony elements (spine)
- Timing
  - Emergent
    - Incomplete injuries with progressive neuro deficit
  - Elective
    - Complete injuries (3-7 days post injury)
    - Central Cord Syndrome (2-3 weeks post injury)

73

Halo Vest

- Stabilize the neck by attaching a thoracic vest to a ring that is secured to the skull via pins
- Indications include:
  - Adjunct or alternative to internal fixation for cervical spine stabilization

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Halo Vest

- Precautions
  - Keep wrench accessible for emergency removal of vest
  - Perform pin care
  - Assess skin under vest
  - Educate patient and family
- Potential Complications
  - Pin loosening
  - Pressure ulcer
  - Pin site infection

75

Long Term Care

- Rehab
  - Maximizing motor function
- Bladder/bowel Training
- Psychological and Social Support

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Potential Complications

- DVT
- Autonomic Dysreflexia
- Pressure Sores
- GI ulcers
- Paralytic Ilieus
- Poikelothermy

77

Deep Vein Thrombosis

- DVT Prophylaxis
  - Use of low molecular weight heparin is treatment of choice
  - Low dose heparin in combination with pneumatic compression stockings is recommended (neither should be used alone)
  - 3 months
  - Inferior vena cava filters recommended for patients who fail anticoagulation or who are not candidates

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GI/Bladder

- Adjuncts
  - Bladder catheterization
    - Lesion above S2/S3/S4 will have areflexic bladder and may develop autonomic dysreflexia
  - GI Stress Ulcers
    - Prophylaxis with proton pump inhibitors is recommended for 4 weeks
  - Gastric tube
    - High cervical lesions have ileus for the 1st 24 hours with an ↑ risk of vomiting

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Autonomic Dysreflexia

SCI at or above T6 (after spinal shock has resolved)

Noxious Stimuli below level of injury

↓

Sympathetic response

↓

Severe Vasoconstriction (beneath level of lesion)

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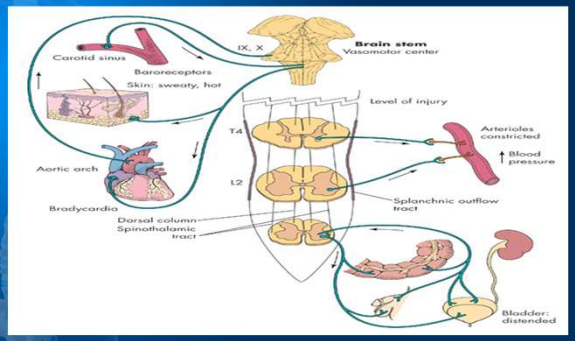
Autonomic Dysreflexia

Precipitating Factors – noxious stimuli below level of SCI



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Autonomic Dysreflexia

Common Etiologies

- Urinary Tract Abnormalities
- Painful/Uncomfortable Diagnostic Interventions
- Lower GI Tract Abnormalities
- Intra-abdominal Abnormalities
- Pressure Ulcers
- Ingrown Toenail
- Sunburn

Characteristics

- ↑ BP
- Bradycardia
- Dysrhythmias
- Flushing, Diaphoresis (above level of SCI)
- Pallor, Cooler skin (below lesion)
- Anxiety
- Headache
- Visual disturbances (blurred vision, photophobia)

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Autonomic Dysreflexia

- Interventions
  - Prevention – monitor bowel/bladder functions, skin
  - Management
    - ID and remove precipitating stimuli
      - Bladder catheterization
      - Loosen tight clothes
    - Decrease BP
    - Surgery
      - Hemorrhoids?

83

Poikelothermy

- Decline in body temperature – core temp will match environment
- Undertake efforts to keep the patient warm

84

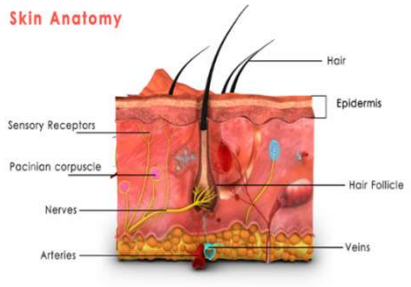
Integumentary

Patti Esmail MSN/ED RN CCRN

1

Integumentary

- Largest organ
- Major functions
  - Barrier to environment
  - Absorption of vitamins (Vit D)
  - Temperature regulation
  - Sensory Perception
  - Shock absorber
  - Appearance and identity
  - Assists with blood pressure regulation



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Factors impacting the health of the integumentary system

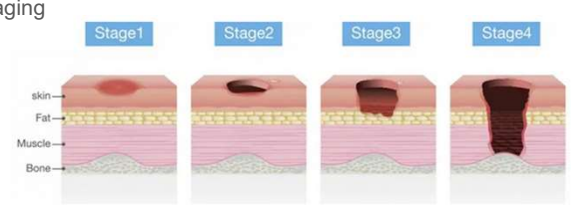
- Overall general health and wellness
- Diabetes Mellitus
- Infection
- Nutrition
- Activity
- Age and obesity

3

Pressure Injury

Pressure Injury Staging

- Stage 1
- Stage 2
- Stage 3
- Stage 4
- Unstageable



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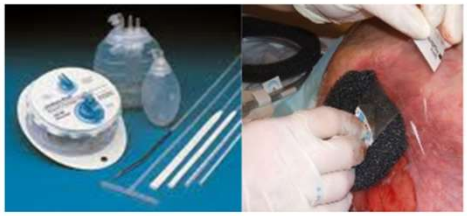
Drains



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Question 5

A nurse turns a patient and notices that the sacrum is red. The skin is not broken or blistered. This assessment is consistent with what stage pressure injury?

A. Stage 1

A. Stage 2

A. Stage 3

A. Stage 4

6

Question 5 - Rationale

A nurse turns a patient and notices that the sacrum is red. The skin is not broken or blistered. This assessment is consistent with what stage pressure injury?

A. Stage 1

A. Stage 2 - skin is not broken

A. Stage 3 - skin is not broken

A. Stage 4 - skin is not broken

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Question 6

A stage 4 pressure injury typically requires:

A. Debridement

A. Packing

A. Skin Grafting

A. Positioning off the area and monitoring

8

Question 6 - Rationale

A stage 4 pressure injury typically requires:

A. Debridement - Possible treatment for stage 2

A. Packing - Possible treatment for stage 3

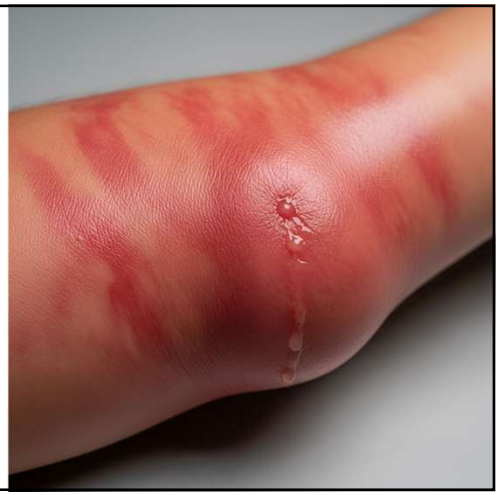
A. Skin Grafting

A. Positioning off the area and monitoring - Possible treatment for stage 1

9

Cellulitis: Key Features

- Cellulitis is an acute bacterial skin infection of the dermal and subcutaneous tissue
- It causes redness, swelling, and tenderness
- It typically affects the deeper layers of skin
- Cellulitis can spread rapidly if untreated
- One of the most common skin infections seen in primary care and the hospital



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Cellulitis Treatment

- Superficial bacterial skin infection
- Typically Streptococcus or Staphylococcus
- Bacteria gain entry through skin opening (cut, surgery, wound, ulcer, dermatitis, animal bite)
- Signs and symptoms: edema, red, tender,warm, red spots, blisters, fever
- Highest incidence in lower extremities
- Vulnerable population at highest risk for progression to systemic infection/sepsis

- **Prevention**
- **Culture and ABX**
- **Warm compresses**
- **Irrigation and drainage**
- **Infected wound management**

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Necrotizing Soft Tissue Infections

- Superficial bacterial infection of the skin that extends all the way to the sheath covering the muscle (subcutaneous fat, soft tissue, facial plane)
- Necrotizing fasciitis is a serious, uncommon infection
- Group A β-hemolytic Streptococcus (flesh-eating bacterium - GAS)
- May progress rapidly
- GAS is hard to phagocytosis
- When systemic, progresses to septic shock rapidly
- High mortality rate

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- **Prevention!!**
- **Prompt identification and debridement**
- **Empiric antibiotics**
- **Hemodynamic monitoring and shock management**
- **Hyperbaric oxygen**
- **Aggressive management of organ dysfunction**
- **Wound management**
- **Possible amputation of extremity**

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Question 7

A 72 year old diabetic patient is admitted for COPD exacerbation is found to have "a week old cat scratch" on the left lower leg that is red, tender, and has blisters that are draining a yellow fluid. A priority intervention during the admission will be to:

A. Document the finding on the skin assessment tool
B. Culture the drainage to send to the lab
C. Place a warm compress on the area for comfort
D. Evaluate the white blood cell count

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Question 7 - Rationale

A 72 year old diabetic patient is admitted for COPD exacerbation is found to have "a week old cat scratch" on the left lower leg that is red, tender, and has blisters that are draining a yellow fluid. A priority intervention during the admission will be to:

A. Document the finding on the skin assessment tool - documentation will certainly be done
B. Culture the drainage to send to the lab - Older adults with DM and COPD (impaired O2 delivery and possibly on steroids) are immunosuppressed and at higher risk of poor wound healing
C. Place a warm compress on the area for comfort - Warm compresses will decrease pain
D. Evaluate the white blood cell count - Evaluating the WBC is needed and will be done

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Summary of Integumentary Test Plan Topics

- Cellulitis
- IV infiltration
- Necrotizing fasciitis
- Pressure Injury
- Wounds
  - Infectious
  - Surgical
  - Trauma

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Questions??

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