Endocrine System **BASIC Function**

The Endocrine System is a network of glands that produce hormones, which are chemical signals that regulate various physiological processes in the body. These hormones are released into the bloodstream to affect target cells and organs throughout the body. The system works in conjunction with the central nervous system to maintain homeostasis and respond to environmental changes.

- **Central Nervous System** sends a signal through the hypothalamus, which releases hypothalamic-pituitary hormones (e.g., TRH, CRF) to stimulate the anterior pituitary gland to release hormones (e.g., TSH). These hormones then stimulate the target glands (e.g., thyroid, adrenal) to produce their respective hormones (e.g., T3, T4, cortisol, aldosterone).

- **Hypothalamus** regulates the release of hormones from the pituitary gland.

- **Oxytocin** and **Antidiuretic Hormone** (ADH) are produced by the posterior pituitary gland in response to neural signals.

- **Blood sugar**, **Glucagon**, **Insulin**, and **Somatostatin** are produced by cells in the pancreas, each playing a role in glucose metabolism.

- **Epinephrine** and **Norepinephrine** are produced by the adrenal gland and regulate the body's response to stress.

- **Cortisol** and **Aldosterone** are produced by the adrenal cortex and regulate metabolism and sodium balance.

Understanding the Endocrine System is crucial for nurses in providing effective care, especially in critical care settings, where timely interventions can be life-saving.
The Endocrine System

- **Endocrine system**
  - System of glands that produce and secrete hormones

- **Hormones**
  - Molecules synthesized and secreted by special cells and released into the blood
  - Exert biochemical effects on target cells
  - Control metabolism
  - Transport substances across cell membranes
  - Control fluid and electrolyte balance
  - Control growth and development
  - Control adaptation
  - Control reproduction

The Process

Hypothalamus detects a system need

Pituitary

- Releasing Hormone
- Growth Releasing Hormone
- Thyroid Releasing Hormone
- Cortisol Releasing Factor

Stimulating Hormone

Target Organ

Secrete Hormone
The Glands

Hypothalamus
- Lower central part of the brain
- Regulation of satiety
  - Hunger
  - Rest
  - Sexual stimulation
- Water and electrolyte balance
- Emotions
- Regulation of body temperature
  - Sweat and shiver
- Stimulate or suppress release of hormones in the pituitary gland
Pituitary Gland
- Master Gland
- Size of a pea
- Beneath the hypothalamus
- Two lobes: anterior and posterior
- Anterior Lobe is 75% of gland

Anterior Lobe of Pituitary

CNS ➔ HYPOTHALAMUS ➔ ANTERIOR PITUITARY GLAND
- Growth Hormone
- Bone and Muscles
- FSH and LH
- Thyroid Stimulating Hormone
- Ovaries/Testes
- Sexual Function
- TH, T3 and T4

ACTH ➔ Adrenocorticotropin ➔ Adrenal Gland
- Cortisol
- Aldosterone
- Epi
- NorEpi
Posterior Lobe of Pituitary

- Not regulated by the Central Nervous System
- Controlled by nerve fibers in the hypothalamus
- Released after activation of cell bodies in the nerve tract
- Responds to changes in plasma osmolality, decreased BP, decreased volume
- Secreted hormones produced in the hypothalamus and stored in the pituitary

**Produces**

- **Antidiuretic hormone (vasopressin)**
  - Water conservation
- **Oxytocin**
  - Contraction of uterine walls
  - Ejection of breast milk

---

Thyroid Gland

- Immediately below larynx laterally and anterior to trachea
- Release of thyroid hormones controlled by pituitary

**Normal function**

- **Decreased levels of T3, T4** ► **Pituitary releases TSH** ► **Thyroid produces more T3, T4** ► **↓ production of TSH**
- Regulates body metabolism
- Stimulates carbohydrate, fat and protein metabolism
- Positive chronotropic and inotropic effect
- Bone growth and brain and nervous system development in children
- Helps maintain blood pressure, heart rate, digestion, muscle tone and reproductive functions
Thyroid hormones
- Thyroid takes iodine and converts to thyroid hormones
- Combine iodine with amino acid to make T3, T4
- T3 - Triiodothyronine
  - 20%
  - 4 – 10 times stronger than T4
  - More active form
- T4 - Thyroxine
  - 80%

T3, T4
- TSH
  - Elevated in hypothyroidism
  - Reduced in hyperthyroidism
- T3, Free T3
  - Routine evaluation not recommended
- T4, Free T4
  - Better indicator of hypothyroidism
  - T4 converts to T3 when T3 in deficit
  - Free T4 – unbound and available

Adrenal Glands
- Triangular shape
- Sit on top of each kidney
- Two part
  - Adrenal Cortex
  - Adrenal Medulla
- Cortisol Releasing Factor
  - Stimulated in response to stress, ↓ glucose, heat/cold extremes, trauma, surgery, immobility, ↓ cortisol levels
- Activates ACTH – Adrenocorticotropin
  - Response: Release of Adrenocotical Hormones
    - Glucocorticoids – Cortisol
    - Mineralcorticoids – Aldosterone
    - Medullary Hormones - Catecholamines
Adrenal Glands

- Adrenal Cortex
  - Glucocorticoids
    - Cortisol (corticosteroids)
    - Helps cope with stress
    - Carbohydrate, fat and protein metabolism
    - Increases blood pressure
    - Blocks allergic and inflammatory response
      - Blocks WBC response

- Addisons Disease
  - Primary adrenal insufficiency
  - Lack of Cortisol
  - Fatigue, lethargy, hypotension, fever, tachycardia
  - Treat with IV Cortisone

- Cushing’s Disease
  - Overproduction of cortisol / over treatment
  - Increased appetite, weight gain, fat deposits, slow wound healing, Na and H2O retention, HPTN
  - Over treatment with glucocorticoid / Adrenocortical Tumor
  - Gradually decrease glucocorticoid / Resect Tumor
Adrenal Insufficiency

Glucocorticoid insufficiency (mineral corticoid in sufficient)
- Adrenal cortex produces cortisol, aldosterone, and androgens
- Adrenal medulla secretes epinephrine and norepinephrine

Causes: Disease or suppression of hypothalamic-pituitary axis from chronic steroid use (most common cause) - hypopituitarism due to hypothalamic-pituitary disease needs to be ruled out
- Sepsis can cause exacerbation of chronic insufficiency. Other causes adrenal hemorrhage from septicemia or complications of anticoagulation.

Signs and Symptoms
- Hypoglycemia, dehydration, disorientation, hypotension, lethargy,
- Hyponatremia

Treatment
- Treatment with hydrocortisone (Solu-Cortef) 100 mg IV
- Dexmethasone allows for diagnosis.

Diagnostics
- Cortisol level
- Endocrine consult
Adrenal Glands

- Adrenal cortex
  - Mineralocorticoids
    - Aldosterone
    - Renin – angiotensin system

- Adrenal Medulla
  - Medullary Hormones
    - Catecholamines (adrenergic response)
    - Response to stress
    - Fight or flight
    - Increased HR, BP, RR, contractility, and vasoconstriction
      - Epinephrine
      - Norepinephrine
### Descriptions

<table>
<thead>
<tr>
<th>SIADH</th>
<th>Diabetes Insipidus</th>
</tr>
</thead>
</table>
| Impaired renal excretion of water resulting in:  
- **Oliguria**  
  100 to 400 ml / 24 hours  
- ↑ urine specific gravity  
- **Water intoxication**  
- **Hyponatremia**  
Caused either by:  
- Excess excretion of ADH  
- Increased responsiveness to ADH | Impaired renal conservation of water, resulting in:  
- **Polyuria**: 5-20 L/24 hours  
- Dehydration  
- **Hypernatremia**  
Caused by either:  
- Deficiency of ADH  
- Decreased renal response to ADH |
### Types

<table>
<thead>
<tr>
<th>SIADH</th>
<th>Diabetes Insipidus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurogenic SIADH</td>
<td>Neurogenic</td>
</tr>
<tr>
<td>↑ production and / or</td>
<td>Deficit in release or synthesis of ADH</td>
</tr>
<tr>
<td>release of ADH</td>
<td>Nephrogenic</td>
</tr>
<tr>
<td>Ectopic SIADH</td>
<td>Deficit in renal tubular response to ADH</td>
</tr>
<tr>
<td>Production of a substance</td>
<td>Psychogenic</td>
</tr>
<tr>
<td>indistinguishable from ADH by tissue</td>
<td>Psychogenic Polydipsia</td>
</tr>
<tr>
<td>Nephrogenic SIADH</td>
<td>Can mimic nephrogenic</td>
</tr>
<tr>
<td>Pharmacological agents that ↑ ADH</td>
<td>Hypotonic urine</td>
</tr>
<tr>
<td>secretion or ADH effect</td>
<td>Water intoxication</td>
</tr>
<tr>
<td></td>
<td>versus volume depletion</td>
</tr>
</tbody>
</table>

### Pathophysiology

<table>
<thead>
<tr>
<th>SIADH</th>
<th>Diabetes Insipidus</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ secretion of ADH or ADH like substance</td>
<td>Inadequate Antidiuretic Hormone</td>
</tr>
<tr>
<td>or increased renal responsiveness</td>
<td></td>
</tr>
<tr>
<td>Failure of negative feedback system:</td>
<td>Diuresis of large volumes of hypotonic</td>
</tr>
<tr>
<td>ADH secretion continues despite low</td>
<td>urine</td>
</tr>
<tr>
<td>serum osmolality</td>
<td></td>
</tr>
<tr>
<td>Renal reabsorption of water increases</td>
<td>Dehydration and hypernatremia</td>
</tr>
<tr>
<td>Water intoxication</td>
<td></td>
</tr>
<tr>
<td>Hyponatremia and hypoosmolality</td>
<td>Potential shock and / or neurologic</td>
</tr>
<tr>
<td></td>
<td>effects</td>
</tr>
</tbody>
</table>
## Presentation

### SIADH

**Early**
- Headache
- Weakness
- Anorexia
- Muscle Cramps

**Weight Gain**
- NO EDEMA

**Late**
- Lower sodium levels
- Personality changes
- Hostility
- Sluggish Deep tendon reflexes
- Nausea and vomiting
- Diarrhea
- Oliguria

### Diabetes Insipidus

- **Onset** may occur several days after insult if neurogenic
- **Polydipsia** – thirsty for cold liquids
- Fatigue, Weakness
- **Polyuria**
  - Suspect DI if UNEXPLAINED
  - Urine Output > 200 ml/hr x 2 hrs
- Signs of dehydration and volume depletion
- Neurological
  - Restless, confusion, irritability, lethargy, coma

### Impending Crisis

- Confusion
- Lethargy
- Cheyne-Stokes respirations
- Na level < 110 mEq/L
  - Cerebral Edema
    - Brain with higher osmolality draws fluid in
  - Seizures
- Coma
- Death

### Complications

- Coma
  - Increased sodium
- Shock
  - Decreased circulating volume
- Thromboembolism
- Dehydration
### Diagnosis

<table>
<thead>
<tr>
<th>SIADH</th>
<th>Diabetes Insipidus</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ serum sodium* (&lt;135mEq/L)</td>
<td>Serum Sodium &gt; 145 mEq/L</td>
</tr>
<tr>
<td>↓ serum osmolality &lt;280 mOsm/kg</td>
<td>↑ BUN</td>
</tr>
<tr>
<td>Urine osmolality &gt; serum osmolality</td>
<td>↑ Osmolality &gt; 295 mOsm/kg (Normal 280-295)</td>
</tr>
<tr>
<td>↑ urine osmolality &gt; 1200 mOsm/kg</td>
<td>↑ Hematocrit</td>
</tr>
<tr>
<td>↑ urine specific gravity &gt;1.030</td>
<td>↓ ADH (Neurogenic)</td>
</tr>
<tr>
<td>Normal 1.005-1.030</td>
<td>&lt;1 pg/ml</td>
</tr>
<tr>
<td>↑ urine NA - &gt; 18 mmol/L</td>
<td>Urine Specific Gravity &lt; 1.005 <em>(hallmark sign)</em></td>
</tr>
<tr>
<td>Rule out adrenal, renal and thyroid disorders.</td>
<td>Normal 1.005 – 1.030</td>
</tr>
<tr>
<td>Absence of peripheral edema or signs of dehydration**</td>
<td>Osmolality</td>
</tr>
<tr>
<td>&lt; Serum osmolality</td>
<td>&lt; 500 mOsm/kg</td>
</tr>
<tr>
<td>&lt; 200 mOsm/kg <em>(hallmark sign)</em></td>
<td>Normal 300-800 mOsm/kg</td>
</tr>
<tr>
<td>Extremes of normal 50-1200 mOsm/kg</td>
<td></td>
</tr>
</tbody>
</table>

### Treatment

<table>
<thead>
<tr>
<th>SIADH</th>
<th>Diabetes Insipidus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treat Cause</td>
<td>Treat Cause</td>
</tr>
<tr>
<td>Surgery to remove malignant lesion</td>
<td>Neurogenic DI</td>
</tr>
<tr>
<td>Stop drugs causing SIADH</td>
<td>Exogenous ADH - Vasopressin</td>
</tr>
<tr>
<td>Correct Fluid Volume Excess</td>
<td>Aqueous Pitressin - IV/SQ</td>
</tr>
<tr>
<td>Restrict Fluids (1,000 ml/d)</td>
<td>Lysine vasopressin – nasal</td>
</tr>
<tr>
<td>Diuresis: Lasix or mannitol</td>
<td>Desmopressin acetate – DDAVP</td>
</tr>
<tr>
<td>Correct electrolyte imbalance</td>
<td>(less vasoactive effects)</td>
</tr>
<tr>
<td>Encourage dietary sodium</td>
<td>Hypophysectomy – Removal of pituitary tumor</td>
</tr>
<tr>
<td>Fluid restriction</td>
<td>Nephrogenic DI</td>
</tr>
<tr>
<td>Hypertonic saline 3%</td>
<td>ADH Potentiator – Diabenese</td>
</tr>
<tr>
<td>If sodium &lt; 115 mEq/L – 250-500 ml at rate of 1-2ml/kg/h</td>
<td>(chlorpropamide)</td>
</tr>
<tr>
<td>If sodium &gt; 125 mEq/L Stop hypertonic saline</td>
<td>Thiazide diuretics and sodium restriction</td>
</tr>
<tr>
<td>Medications</td>
<td>Psycogenic DI</td>
</tr>
<tr>
<td>Declomycine (ADH antagonist)</td>
<td>Pharmacologic agents</td>
</tr>
<tr>
<td>Tetracycline derivative</td>
<td></td>
</tr>
<tr>
<td>Potentially nephrotoxic</td>
<td></td>
</tr>
<tr>
<td>Lithium (ADH antagonist)</td>
<td></td>
</tr>
</tbody>
</table>
# Sodium Administration

<table>
<thead>
<tr>
<th>Hypertonic</th>
<th>Hypotonic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid infusion of 3% saline can cause cerebral osmotic dimyelination syndrome</td>
<td>Rapid infusion of D5W can cause cerebral edema</td>
</tr>
<tr>
<td>Pulls fluid from the cells</td>
<td>Fluid moves into the cells</td>
</tr>
<tr>
<td>Caution with cardiac and renal patients</td>
<td>Caution with any patient with neurological injury</td>
</tr>
<tr>
<td>Shift in fluid from intracellular to extracellular</td>
<td>Shift in fluid from extracellular to intracellular</td>
</tr>
</tbody>
</table>

## Nursing Considerations

### SIADH
- VS q15 until stable
- I and O hourly
- Fluid restriction – 1000ml/24 hours
- Daily Weights
- Neuro Assessment – seizure precautions
- Urine Specific Gravity Q1-2 hours while NA is low
- Frequent mouth care to help with thirst
- Relieve pain and stress as both promote ADH release

### Diabetes Insipidus
- VS q15 until stable
- I and O hourly
- Cardiac monitoring
- Assess urine output and specific gravity hourly
- Daily weights
- Low sodium diet
- Monitor neuro status
Diabetic Disorders

➢ Diabetic Ketoacidosis (DKA)
➢ Hyperosmolar Hyperglycemiac Nonketotic Syndrome (HHNS)

Diabetes Mellitus

Diabetic disease characterized by hyperglycemia that results from deficits in insulin secretion, insulin action or both.

Type I
- Beta cell destruction leading to absolute insulin deficiency
- Usually Juvenile Diabetics
- IDDM

Type II
- Insulin resistance and a relative insulin deficiency
- Normal amounts of insulin inadequate
- Adult Onset
- NIDDM / IDDM
Hyperglycemic Crisis

- **Diabetic Ketoacidosis (DKA)**
  - Hyperglycemic crisis associated with metabolic acidosis and elevated serum ketones

- **Hyperglycemic Hyperosmolar Non-Ketotic Condition (HHNK)**
  - Hyperglycemic crisis associated with absence of ketone formation
Diabetic Ketoacidosis

Causes
- Undiagnosed Type I diabetics
- Illness or infection
- Omission of insulin
- Trauma
- Surgery
- Noncompliance: Too many calories
- Cushing’s Syndrome
- Hyperthyroidism
- Pancreatitis
- Pregnancy
- Drugs: Prednisone, HCTZ, dilantin, sympathomimetics

DKA Pathophysiology

- Insufficient insulin or cells ability to use insulin
- Hyperglycemia
- Osmotic diuresis
- Glycosuria, dehydration, electrolyte imbalance
- Impaired glucose uptake by adipose tissues
- Impaired triglyceride synthesis and liberation of free fatty acids
- Ketoacidosis
DKA Presentation

- Altered Mental Status (confusion to coma)
- Blurred Vision
- Excessive urination
- Enuresis – unable to control urine
- Abdominal Pain
- Nausea / Vomiting
- Polydipsia – excessive thirst
- Kussmaul’s ventilation – deep rapid breathing, gasping, air hunger
- Acetone fruity breath
- Weight Loss
- Signs of dehydration

DKA Diagnosis

Serum
- ↑ Glucose 300-800 mg.dl (average 600mg/dl)
- ABG’s
  - pH <7:30; HCO’s <18; PaCo2<35 (compensating)
- ↑ Ketones
- ↑ BUN/Creatinine Ratio > 10:1
- ↑ Osmolality - Usually 295 – 330 mOsm/kg
- ↑ Lipids
- ↑ HCT
- Anion Gap
Anion Gap

- (Na+) minus (Cl- + HCO3-)
- Normal gap 12

- Gap > 20
  - DKA
  - Lactic Acidosis

  - Means H+ have been added to the positive side

DKA Diagnosis

- Urine
  - + for Ketones
  - + for glucose

- EKG
  - Changes associated with hypokalemia
  - Flat T waves - dehydration
DKA Treatment

Increase circulating volume

- 1st hour
  - 10-30 ml/kg/h (1-2 L NS)

- After 1st hour
  - 500-1000 ml/h depending on volume status
  - .9NS if serum Na low OR if serum osmolality <320 mOsm/kg
  - >45NS if Serum Na normal or elevated OR if serum osmolality > 320mOsm/kg
  - Add dextrose after blood glucose levels <250 mg/dl

Decrease Blood Glucose

- IV Insulin
  - Bolus 10-20U (.15u/kg)
  - Drip 5-10U (0.1u/kg/h)

- Serum glucose decrease no greater than 75-100 mg/dl/hr (200 mg/dl/hr)
  - Cerebral edema, hypokalemia, hypoglycemia

- Drip discontinued 30 minutes ~2 hours after SQ dose given

- SQ started usually after BS < 250mg/dl, pH> 7.3 and HCO3 >18 and no further ketone production OR acidosis resolves and anion gap 10-12
DKA Treatment

Correct Electrolyte Imbalance
- K+ level represents extracellular potassium
- Only indirectly reflects intracellular potassium
- Intracellular potassium may be much lower
- Serum potassium <4.5mEq/L -> add potassium to IV Fluids
  - Give in combination of KCL and KPO4
  - Insulin will return potassium to the cell

DKA Treatment

Correct Electrolyte Balance
- Phosphorus Levels
  - Acidosis and osmotic diuresis cause a decrease in phosphorus -> decrease 2,3 DPG -> shift to the left
  - Phosphorus and Calcium function inversely
  - Replace phosphorus slowly and monitor Calcium
  - Too rapid replacement of Phosphorus -> rapid decrease in calcium -> tetany
DKA Treatment

- Prevent Complications
  - Hyperkalemia (initially)
  - Hypokalemia
  - Hypoglycemia
  - Cerebral Edema
  - Pulmonary Edema
  - Renal Failure

- Renal Disease
  - Dialysis

Nursing Considerations

- VS q 15 minutes until stable
- Hourly I and O
- Urine SG q 2 hours
- Labs initially Q1-2 hours
  - Glucose q1
  - Renal profiles q 2 hours
  - Labs for anion gap q2 hours
- Neuro checks
Hyperglycemic Hyperosmolar Non-Ketotic Syndrome (HHNS)

**Definition**
- Hyperglycemic crisis associated with the absence of ketone formation; most common severe metabolic disturbance in type 2 diabetes mellitus
HHNS

Causes
- Dehydration
- Pancreatitis
- Burns
- Infection
- Stroke
- Uremia
- Sepsis

Drugs
- Glucocorticoids
- Thiazide diuretics
- Loop diuretics
- Phenytoin
- Immunosuppressive drugs
- Beta Blockers
- Tagamet
- Calcium Channel Blockers
- Mannitol
- Sympathomimetics

HHNS
Pathophysiology

Insulin deficiency
↓
Hyperglycemia without ketosis
↓
Osmotic diuresis
↓
Serum hyperosmolality, cellular dehydration, decreased glomerular filtration rate
↓
Thrombosis, renal failure and neurologic changes
HHNS Presentation

- Weakness, fatigue
- Dehydration: dry mouth, polydipsia, dry skin
- Hypotension
- Tachycardia
- Changes in LOC
- Respirations rapid and shallow
- No ketosis
- No breath odor

HHNS Diagnosis

Serum

- ↑ Glucose
  - 600-2,000mg/dl (average 1,100 mg/dl)
- Ketones: Normal or mildly elevated
- pH: Normal
- Osmolality > 330m Osm/L
- Sodium: Normal or elevated
- Potassium: Low
- Bicarbonate: Normal
- Phosphorus: Low
- ↑ BUN / Creatinine: 10:1 ratio
- ↑ HCT
HHNS Diagnosis

- **Urine**
  - Glucose +
  - Ketones -

- **EKG**
  - Changes associated with K levels if K is abnormal
  - Sinus Tachycardia

HHNS Treatment

- **Increase circulating volume**
  - **1st hour**
    - 10-30 ml/kg/h NS
  - **After 1st hour**
    - 500-1000 ml/hr depending on volume status
    - 0.9% saline
      - If serum Na low OR
      - If serum osmolality < 320 mOsm/L
    - 0.45% saline
      - If serum Na elevated OR
      - If serum osmolality > 320 mOsm/L
    - 5% Dextrose (D5.9NS, D51/2 NS)
      - Added when serum glucose reaches <250 mg/dl
HHNS Treatment

- **Decrease Blood Glucose**
  - IV Insulin
    - Bolus 10 - 20U (0.15-0.30 U/kg)
    - Drip 5-10 U/hr (0.1 U/kg/hr)
    - Serum Glucose should not decrease > than 75-100 mg/dl/hr
  - SC Insulin
    - Started after glucose < 250 mg/dl
    - Insulin infusion stopped when SC initiated (overlap not necessary)

- **Correct Electrolyte Imbalance**
  - Potassium
    - Monitor hourly – usually severely low
    - No intracellular to extracellular shift
    - Replace with KCL or KPhos
  - Phosphorus
    - Often low
    - 1/3 to 1/2 of K+ is replaces with KPO4
    - To prevent hypocalcemia do not exceed 1.5 mEq/kg increase in 24 hours
  - Magnesium
    - Often low
    - 1-2 g 10% solution if renal function OK
HHNS Treatment

- Prevent Complications
  - Aspiration from paralytic ileus
  - Hyovolemic shock
  - Dysrhythmias
  - Embolism
  - MI
  - Pulmonary Edema
  - Cerebral Edema
  - Intracranial Hypertension
  - Hypoglycemia
  - Acute renal failure

Metabolic Syndrome

- A *multiplex risk factor* that arises from insulin resistance accompanying abnormal adipose deposition and function.

- Those who meet criteria have a greater risk for significant clinical consequences
  - Diabetes mellitus
  - Coronary heart disease – doubled the risk
    - CAD, AF, aortic stenosis, heart failure
  - Stroke
  - Fatty Liver
  - Obstructive Sleep Apnea
  - Cancer (breast, colon, gallbladder, kidney)
  - Acceleration of neurocognitive aging
Metabolic Syndrome

Diagnosed when the patient has at least 3 of the following:

- Fasting Glucose > 100 mg/dl
  Or receiving drug therapy for hyperglycemia
- Blood pressure > 130/85 mmHg
  Or receiving drug therapy for hypertension
- Triglycerides > 150 mg/dl
  Or receiving drug therapy for hypertriglyceridemia
- HDL-C < 40 mg/dl in men or < 50 mg/dl in women
  Or receiving drug therapy for reduced HDL-C
- Waist circumference
  > 40 in in men; > 35 in in women
  If Asian: ≥35 in in men; ≥32 in in women

TREATMENT
Risk Factor Modification
Renal and Electrolytes

Renal Anatomy: Nephron
Nephron

• Functional unit of kidney
• Consists of glomerulus and a tubular structure
• 85% of nephrons originate in superficial part of cortex (cortical nephrons)
• 15% of nephrons originate deeper in the cortex (juxtamedullary nephrons)
  – Longer, thinner loops of Henle

Glomerulus of Nephron

• Tuft of capillaries emerging from afferent arterioles
• Contained within Bowman’s capsule
• Blood flows out of glomerular capillaries via the efferent arterioles
• Space within Bowman’s capsule for the filtrate
• Basement membrane of the glomerular capillaries determines permeability
  – Not permeable to plasma proteins
Majority of what is in blood is filtered. Blood cells and majority of proteins are not filtered.

Glomerular Filtration

- Filtration of protein free plasma through the glomerular capillaries into Bowman’s space.
- The capillary filtration pressure is approximately 60 mm Hg. (higher pressure than other capillary beds)
- Glomerular filtration produces 180 L of filtrate per day
- Autoregulation.
- Constriction of the efferent arterioles increases glomerular pressure and filtration.
- Constriction of the afferent arterioles decreases glomerular pressure and filtration.
Chronic Kidney Disease

- Abnormal kidney function or structure present for > 3 months

- Classified by cause

- Staged by:
  - GFR category
  - Albuminuria category
GFR

<table>
<thead>
<tr>
<th>GFR Category</th>
<th>GFR (ml/min/1.73m²)</th>
<th>Terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td>&gt; 90</td>
<td>Normal</td>
</tr>
<tr>
<td>G2</td>
<td>60-89</td>
<td>Mildly decreased</td>
</tr>
<tr>
<td>G3a</td>
<td>45-59</td>
<td>Mildly to moderately decreased</td>
</tr>
<tr>
<td>G3b</td>
<td>30-44</td>
<td>Moderately to severely decreased</td>
</tr>
<tr>
<td>G4</td>
<td>15-29</td>
<td>Severely decreased</td>
</tr>
<tr>
<td>G5</td>
<td>&lt; 15</td>
<td>Kidney failure</td>
</tr>
</tbody>
</table>

Note: Estimated GFR uses serum creatinine. Cystatin C can be measured for confirmation if estimated GFR is thought to be inaccurate.

Strategies to Prevent Progression of CKD

• BP control and interruption of RAAS
  – < 140/90 if urine albumin excretion is < 30 mg/24 hours
  – < 130/80 if urine albumin excretion is ≥ 30 mg/24 hours
  – ACE-I or ARB in patients with diabetes, CKD, and urine albumin 30 to 300 mg/24 hours
  – ACE-I or ARB in all patients with CKD with urine albumin excretion > 300 mg/24 hours
• Other: Glycemic control / reduced sodium
Acute Kidney Injury

A sudden loss of the kidneys’ ability to excrete wastes, concentrate urine, and conserve electrolytes.

New definitions and stages proposed by Acute Kidney Injury Network.

Acute Kidney Injury (AKI): Entire Spectrum of Acute Renal Failure

• One or more of the following that occurs abruptly:
  – Increase in creatinine of ≥ 0.3 mg/dL within 48 hours
  – Increase in creatinine > 1.5 x baseline within last 7 days (known or presumed)
  – A reduction in urine output of < 0.5 ml/kg per hour for more than six hours

• Application of diagnostic criteria can only be applied if patient is optimally hydrated!
OPTIMAL VOLUME MANAGEMENT IS UPSTREAM OF ALL OTHER INTERVENTIONS!

Volume management becomes more and more challenging in critically ill patients.

Increased risk of adverse pulmonary function in patients MODS if excess volume.

Early in AKI

Volume Responsive Kidney

• Volume responsive kidney usually occurs in volume responsive patient
• Hypovolemia most important cause of volume responsive kidney.

Volume Responsive Patient

• Patient may respond to volume with an increase in cardiac output but not with an improvement in kidney function.
Stages of Acute Kidney Injury

<table>
<thead>
<tr>
<th>Stage</th>
<th>Creatinine Criteria</th>
<th>Urine Output Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Increase in serum creatinine of more than or equal to 0.3 mg/dL or increase of 1.5 to 1.9 x baseline</td>
<td>Less than 0.5 ml/kg per hour for 6 12 hours.</td>
</tr>
<tr>
<td>2</td>
<td>Increase in serum creatinine of 2.0 to 2.9 x baseline</td>
<td>Less than 0.5 ml/kg per hour for ≥ 12 hours.</td>
</tr>
<tr>
<td>3</td>
<td>Increase in serum creatinine to more than 3.0 x baseline, or ≥ to 4.0 mg/dL, or initiation of RRT</td>
<td>Less than 0.3 ml/kg per hour for ≥ 24 hours or anuria for ≥ 12 hours.</td>
</tr>
</tbody>
</table>

Diagnosis within 48 hours / Staging over a week.

Implications of AKI

- Often secondary to injury that causes functional or structural changes in the kidneys
- Antecedent to CKD
- Mortality is increased even with small changes in creatinine
- Occurs in approximately 13 to 20 % hospitalized patient
- Occurs is up to 67% of patients in ICUs
Etiology

Outside Hospital
- Glomerular nephritis
- Vasculitis
- Obstructive uropathy

Inside Hospital
- Renal hypoperfusion
- Drug toxicity
- Combination of hypoperfusion and drug effect

Classifications of Acute Kidney Injury

- Prerenal
  - 55-60%
- Intrarenal
  - 35-40%
- Postrenal
  - < 5%
Diagnostic Parameters for Prerenal AKI

- **Positive response to a fluid challenge** is diagnostic of pre-renal AKI.
- **Oliguria.**
- **Concentrated urine:** Urine specific gravity > 1.0150 and urine osmolality > 500 mOsm/L.
- **BUN: Creatinine Ratio > 10:1** (usually closer to 20:1). Increased proximal tubular reabsorption of BUN.
- **Urinary Sodium < 20 mEq/L.**
- **Fractional excretion of sodium (FENa) < 1%**
- **Fractional excretion of urea (FEurea) < 35%**
- **Urinary Sediment:** Normal or minimally abnormal; hyaline casts, no or finely granular casts.

Glomerularnephritis (Cortical)

**Causes**
- Subacute bacterial endocarditis
- Post streptococcal infection
- Systemic lupus erythematosus
- Drug induced vasculitis
- Malignant hypertension

**Pathophysiology**
- Renal capillary swelling.
- Edema and cellular debris obstruct the glomeruli, resulting in a decrease the GFR.

**Diagnostic Parameters**
- Urinalysis will have RBC casts, protein, and leukocytes.
- BUN to creatinine ratio 10:1 and elevated.

**Treatment**
- Supportive
- Antibiotics for infection
- Glucocorticoids and cytotoxic agents in severe cases
Interstitial Nephritis (Cortical)

**Causes**
- Drug induced: Allergic nephritis.
  - Common but often unrecognized allergic event in the interstitium of the kidney.
  - Usually in response to a specific drug.
  - May have associated fever, rash, eosinophilia.
- NSAID induced
- Bacterial, viral, and other infections.
- Immune and neoplastic disorders.

**Diagnostic Parameters**
- Eosinophilia
- Urine
  - Proteinuria
  - WBC casts (no evidence of infection) with or without eosinophils.
- BUN to creatinine ratio 10:1 and elevated.

**Treatment**
- Remove the drug that is the causative agent.
- Steroids may be used.

---

Acute Tubular Necrosis (Medullary): Etiology

- Nephrotoxic agents
- Prolonged ischemic injury
- Hemolysis or rhabdomyolysis
- Endotoxin release in sepsis
- Hypercalcemia
- Any cause of prerenal AKI that is prolonged (clinical challenge)
ATN Pathophysiology

- Destruction of the tubular epithelial layer of cells
- Often reversible if treatment is promptly initiated.
  - If the tubular basement membrane is damaged from prolonged injury and ischemia, it cannot be regenerated.
- Oliguria develops when tubules become obstructed due to tissue swelling or cellular debris.
- A reabsorption (into circulation) of urine filtrate can occur through damaged tubular epithelium.
- Damaged tubular cells can leak ATP and potassium, and calcium can leak into the cell.
- Scar tissue can form over necrotic areas
ATN Diagnostic Parameters

- **Bun:Creatinine Ratio 10:1.**
- **Urine Sodium > 20 mEq/L.**
- **Fractional excretion of sodium (FENa) > 2-3%.**
- **Fractional excretion of urea (FEurea) > 50%.**
- **Urine osmolality < 400 mOsmol/L (loss of tubular concentrating ability).**
- **Minimal to moderate proteinuria.**
- **Urine Sediment: Muddy brown casts, granular / tubular casts, renal epithelial cells.**

Granular casts help differentiate between prerenal and ATN

Postrenal AKI

**Classifications**
- **Mechanical**
  - Urinary calculi
  - Tumor
  - Prostatic hypertrophy
  - Fibrosis
  - Blood clot
  - Retroperitoneal hemorrhage
- **Functional**
  - Neurogenic bladder.
  - Ganglionic-blocking agents.

**Pathophysiology**
- Obstruction can increase renal interstitial pressure causing an increased opposing force to GFR.

**Signs / Symptoms**
- Abrupt decrease in urine output
- Urinalysis may show hematuria.

Ultrasound can be used to rule out during initial assessment.
Treatment of AKI

• Rule out post-renal obstructive causes
• Reverse all pre-renal causes:
  – If no hemorrhagic shock: isotonic crystalloids rather than colloids to expand intravascular volume
  – Vasopressors and fluids if vasomotor shock
  – Goal directed hemodynamic and oxygenation protocols in the peri-operative setting

• Energy intake of 20-30kcal/kg/day
• Avoid restriction of protein
• Increased protein needs in catabolic patients and those on renal replacement therapy (RRT)
• Enteral nutrition
• Insulin therapy in critically ill patients to target glucose of 110 to 149 mg/dL.
Treatment of AKI: Not Recommended

- No diuretics for prevention or treatment: Only for volume overload
- No n-acetylcysteine in critically ill patients with hypotension
- No low dose dopamine to prevent or treat
- No fenoldopam to prevent or treat
- No atrial naturetic peptide to prevent or treat
- No recombinent human (rh) IGF-1 to prevent or treat

Treatment / Prevention of AKI: Contrast Nephropathy

- Consider optional imaging methods
- Use iso-osmolar or low osmolar contrast in smallest quantity possible
- Theophylline and fenoldopam are not recommended.

**Prevention in High Risk Patients**

- Oral n-acetylcysteine
  - 24 to 48 hours before contrast exposure
  - 600 mg BID

  **AND**

- Hydration with 0.9NS or sodium bicarbonate drip
  - 154 mEq of sodium bicarbonate/L at 3 ml/kg for 1 hour prior to procedure
  - Followed by 1 ml/kg/hr for 6 hours post procedure
  - Some evidence that sodium bicarbonate is superior to sodium chloride
Treatment and Prevention of AKI: Special Considerations in Surgical Population

- Goal directed hemodynamic and oxygenation protocols in the peri-operative setting
- No use of off pump CABG for sole purpose of preventing AKI
- No n-acetylcysteine for prevention of post operative AKI

Treatment / Prevention of AKI: Nephrotoxic Agents

- Aminoglycosides
  - Avoid unless no alternatives
  - Topical and local applications when possible
  - Single daily dose rather than multiple doses in patients with normal renal function
  - Drug level monitoring if more than 24 hours (multiple dose) and if more than 48 hours (single dose)
- Amphotericin B
  - Lipid formulations over conventional formulations
  - Azole antifungals and / or echinocandins instead of conventional formulation for systemic mycoses or parasitic infections
Outcomes of AKI

Full recovery

AKI

AKI on CKD

ESRD

Evaluate renal function 3 months after AKI.

Electrolyte Abnormalities in AKI

- Hyperkalemia – most common with oliguric AKI
- Hyperphosphatemia
- Hypermagnesemia
- Hypocalcemia
- Acidemia
  - The kidneys excrete acid.
  - Oral sodium bicarbonate is typically used to treat.
  - Negative hemodynamic effects have been associated with IV sodium bicarbonate bolus dosing
  - The treatment for severe metabolic acidosis remains controversial.
Signs and Symptoms:
AKI and CKD

- Fatigue
- Confusion
- Twitching or weakness related to metabolic acidosis
- Dry skin
- Edema
- Pallor
- Uremic frost/pruritis
- Flank pain
- Infection

Uremic Syndrome

- Seen in both AKI and in CKD
- All organs can be affected
- Signs and symptoms can include: nausea, vomiting, pruritis, bleeding, pericarditis, and encephalopathy
- Symptoms not related solely to elevated BUN or creatinine

Uremic symptoms warrants aggressive treatment with some type of renal replacement therapy!
Nursing: Meticulous Supportive Care / Avoid Complications

Infection
- Infection-related complications - most common cause of death
  - Nosocomial pneumonia
  - IV catheter infections
  - Intra abdominal sepsis
- Avoid corticosteroids (except for interstitial nephritis and some types of renal vasculitis)
  - Catabolic effect
  - Adversely affects immune function
- BUN > 80 to 100 mg/dL is associated with a high risk of infection.
- Low serum protein and albumin levels have an immunosuppressive effect

Fluid, electrolyte, and acid/base imbalances
- Limit fluid intake to avoid congestion
- Assess for hyponatremia
- Restrict potassium, phosphate, and magnesium intake
- Folate and pyridoxine are lost through dialysis

Nursing: Meticulous Supportive Care / Avoid Complications

Hematologic Abnormalities
- Uremic toxins inhibit platelets and factor VIII.
- Factor VIII may need to be replaced.
- Arginine vasopressin can also increase levels of factor VIII.

Drug toxicity from drugs metabolized or excreted from the kidney
- Base dose adjustment on an assumed GFR of zero.

Skin Integrity: Uremic effects -> high risk for breakdown
Criteria for Renal Replacement Therapy (RRT)

- Volume overload in presence of oliguria or anuria
- Uncontrolled hyperkalemia, hyperphosphatemia, hypermagnesemia
- Life threatening acidosis
- Life threatening drug overdoses or toxicity requiring dialysis
- Symptomatic uremia
  - Nausea and vomiting
  - Bleeding
  - Pericarditis
  - Seizures, coma
- BUN 80-100 mg/dL
- Creatinine 10 mg/dL

Renal Replacement Therapy: Peritoneal Dialysis

- Slow form of dialysis - exchange of fluids and solutes between the peritoneal cavity and peritoneal capillaries
- Utilizes diffusion
- Less efficient than hemodialysis
- No need for vascular access
- No significant hemodynamic effects
- Osmotic gradient for fluid removal
  - Hyperosmolar glucose concentrations
- 2 Forms
  - Continuous ambulatory peritoneal dialysis (CAPD)
    - 2 quarts, 3 to 5 times, dwell time 30 to 40 minutes
  - Automated peritoneal dialysis (APD)
    - Cycler machine delivers at night
- Complications
  - Abdominal distention and increased work of breathing
  - Pleural effusion
  - Hyperglycemia
  - Peritonitis
Renal Replacement Therapy (RRT):
Intermittent Hemodialysis

- Central venous access (emergency)
- Arteriovenous grafts or fistulas (chronic)
- Anticoagulation is generally required; non-heparin dialysis is also an option
- Blood pumped through an artificial kidney on one side of the dialysis membrane, while the dialysate (electrolyte) solution flows the opposite direction
- Combines adsorption, diffusion, osmosis, and ultrafiltration
  - Remove fluid and maximal amount of solute (electrolytes, metabolic products, drugs, and toxins)
  - Maximum removal allows for intermittent sessions
- Requires more hemodynamic stability than hemofiltration
- Hypotension is the most common problem
- Advances: Bicarbonate base instead of acetate based.
  - Improved cardiac stability / control of metabolic acidosis

Dialysis Equilibrium Syndrome

- From shifts in extracellular compartment
- Nausea, vomiting, confusion, seizures, coma.
- Most common in first dialysis session with high BUN.
- Treatment.
  - Decreased dialysis time.
  - Decreased dialysis flow rates.
  - Dialyzer with smaller surface area.
  - Sodium chloride, dextrose, mannitol.
RRT: SLEDD is alternative form of delivery

- Sustained low efficiency daily dialysis
  - Alternative to continuous renal replacement therapy due to the disadvantages
    - Patient immobility
    - Need for specialized equipment
    - Anticoagulation
    - Cost
  - More research needed

Criteria / Candidates for Continuous RRT (CRRT)

- Patients with criteria for RRT
  - Hemodynamically unstable
  - Increased ICP

- Nontraditional indications:
  - Hyperthermia
  - Rhabdomyolysis
  - Systemic inflammatory response syndrome
  - Fluid management in the hemodynamically unstable patient without renal failure
CRRT

Ultrafiltration

- **SCUF** – Slow continuous ultrafiltration
- Fluid moves through a semipermeable membrane via a pressure gradient (higher pressure gradient creates more fluid removal)
- **Results are primarily fluid removal**
- Hemofiltration, hemodialysis, and hemodiafiltration all use ultrafiltration as a component of therapy
- Adsorption is another principle involved in all 4 therapies
  - Clinging of positively charged molecules to the negatively charged membrane of the filter.
  - Filter can become clogged with molecules. The removal of these molecules from systemic circulation is a benefit of CRRT therapy.

CRRT

Hemofiltration

- **CVVH** – Continuous veno-venous hemofiltration
- **Uses convection to remove solutes**
  - Process of solute removal by solvent drag
  - More fluid through semi permeable membrane = more solute removed.
  - Replacement solution is used to create solvent drag
  - Faster rate of replacement solution = more solvent drag
- **Convection removes medium and large molecules**
- Solute removal is slow so the process must be continuous
- **Fluid removal still exceeds solute removal**
- Less likely than hemodialysis to produce hypotension
- Some medications are cleared via hemofiltration and require a dose adjustment
  - Dose adjusted based on an assumed creatinine clearance of approximately 14 ml/minute
CRRT

**Hemodialysis**
- CVVHD – Continuous veno-venous hemodialysis
- **Uses dialysate solution to create selective diffusion of electrolytes**
  - Excellent technique for the removal of small particles
- **Hemodialysis removes both solutes and fluid**
- Provides more hemodynamic stability than intermittent hemodialysis
- Allows fluid overloaded critically ill patients to receive a higher caloric intake

CRRT

**Hemodiafiltration**
- CVVHDF – Continuous veno-venous hemodiafiltration.
  - **Uses both hemodialysis and hemofiltration**
  - Allows for the removal of small, medium, and large molecules.
<table>
<thead>
<tr>
<th>Therapy</th>
<th>Principles</th>
<th>Replacement Solution</th>
<th>Dialysate Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCUF</td>
<td>Ultrafiltration</td>
<td>No</td>
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</tr>
<tr>
<td></td>
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</tr>
</tbody>
</table>

Effluent volume of 20 to 25 ml/kg/hr is recommended in AKI.
Sodium

- Dominant extracellular cation
- Primary determinant of serum osmolality
- Sodium is closely related to water (regulates ECF)
- Factors to consider when assessing sodium abnormalities:
  - Serum and urine osmolality
  - Intravascular volume status / presence of edema
  - Serum albumin, lipids, and glucose
  - Medications and IV fluids
  - Renal function

Hyponatremia:
Sodium < 135 mEq/L

- S&S related to rapidness of onset and severity
  - Signs and symptoms can also be related to fluid balance.
  - Primary effects are CNS related.
- Cognitive and motor function changes @ < 125 mEq/L
- Permanent changes at levels < 110 mEq/L
- 50% mortality when < 105 mEq/L

- Muscle cramps
- Twitching / tremors
- Muscle weakness
- N&V / abdominal cramps
- Headache
- Irritability / personality changes
- Confusion
- Lethargy progressing to coma
- Seizures
Hypotonic Hyponatremia

- Most common form of hyponatremia
- Occurs as a result of excess free water in relation to sodium
- Results in intracellular hypoosmolar state (creates S&S)

- Patients can be:
  - Hypovolemic
  - Isovolemic * most common form
    - SIADH
  - Hypervolemic

Treatment for Hyponatremia

- Free water restriction for levels > 125 to 130 mEq/L

- Sodium should be corrected to an initial level of 120 to 130 mEq/L
  - Over 12 to 24 hours
  - No more than 8-12mEq / 24 hours unless life threatening symptoms

- Correct at rate proportional to development
  (slower rate for chronic hyponatremia)

Caution: Osmotic demyelinating syndrome
Hypernatremia

- **Definition:** Sodium greater than 145 mEq/L with a serum osmolality > 295 mOsm/kg. Most cases of hypernatremia involve a hyperosmolar state.

- **Rarely occurs in patients with:**
  - Normal ADH secretion
  - Thirst mechanism
  - Ability to consume free water

- **Almost always causes cellular dehydration.**

**Treatment**

- Correct underlying cause
- Decrease 0.5 to 1.0 mEq/L per hour
  - Replacement of free water with D5W or 0.2 or .45 NS
  - Normal saline may used if hemodynamically unstable (adequate circulating volume is priority)
  - Loop diuretics or dialysis rarely needed

*Caution – Cerebral Edema

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Hypernatremia

**Causes**

- Conditions with limited ability to consume free water (ICU setting)
- Hypertonic tube feedings
- Dehydration (burns, tachypnea, hyperthermia)
- Osmotic diuretics with excessive free water clearance.
- Diabetes mellitus
- Diabetes insipidus

**Signs / Symptoms**

- Thirst (early symptom)
- Urine output decreases and urine osmolality increases due to renal water conservation
- Dry mouth and skin
- Increased body temperature
- Muscle weakness
- Headache
- Irritability and agitation
- Seizures
- Coma
Potassium

• 95% or > of potassium is intra cellular
• Majority of potassium contained in muscle
  – Declines with age due to decrease in muscle mass
• Dietary intake is the major source / kidneys responsible for excretion
• Ratio of extracellular to intracellular important for electrical membrane potentials
• Major body systems impacted by abnormalities:
  – GI
  – Neuromuscular
  – Cardiac

Nerve impulse and muscular function transmission dependent on potassium.

Hypokalemia: Causes

• K+ less than 3.5 mEq / L (total body deficit of 5-10%)

• Causes:
  – Poor K+ intake
  – Increased GI loss (not usually cause of symptomatic imbalance)
  – Increased renal loss
    • Renal tubular acidosis
    • Diuretics
    • Excess mineral or glucocorticoids (aldosterone)
    • Low magnesium
    • Certain antibiotics

  • Extracellular to intracellular shifts
    – Alkalosis (potassium exchanged for hydrogen ions)
      * also causes increased renal loss
    – Insulin
    – Treatment of DKA or HHNK
      • Insulin
    – Beta adrenergic agonists

  • Note: Does not reflect total body potassium – Correct with caution

  • Caution with hypokalemia in presence of acidosis.

Urinary K+: High with renal loss; low with other causes
Hypokalemia: Signs and Symptoms

- Symptoms occur when $K^+ < 3.0 \text{ mEq/L}$
- Severity dependent on:
  - Rapidness of onset
  - Systemic pH
  - Calcium level
- S&S related to altered membrane potentials and impaired muscle contractility
  - Increase in resting membrane potential of neuronal and muscular cells
  - Reduces excitability
- GI
- Orthostatic hypotension
- Parasthesias, weakness, fatigue and muscle cramps
  - Lower extremities are typically impacted first
- Respiratory muscle weakness, dyspnea, paralysis and arrest ($< 2.5 \text{ mEq/L}$)
- Enhanced digitalis effect
- Severe hypokalemia can result in rhabdomyolysis

Hypokalemia: ECG Changes

- T wave flattening or decreased T wave amplitude
- ST sagging and T wave inversion
- Prominent U waves
  - U waves greater than T wave: $K^+ < 3.0 \text{ mEq/L}$
- Merger of T and U waves: pseudo QT prolongation
- Increased P wave amplitude
- Increased PR interval
- Supra and ventricular ectopic beats
- Atrial arrhythmias
- VT / VF / Torsades de Pointes
Hypokalemia: Treatment

- Treat cause
  - Correct alkalosis
  - Correct hypomagnesemia

- Increased potassium intake (dietary or supplement) if potassium ≥ 3.0 mEq/L
  - Foods high in potassium: orange juice, bananas, raisins, milk, green vegetables
  - Oral supplements up to 40 mEq can be used safely several times per day.

- Add potassium to maintenance IV fluid

- IV potassium bolus for severe deficiency (less than 3.0 mEq/L if on digoxin, symptoms related to hypokalemia, or less than 2.5 mEq/L without symptoms)
  - Non glucose solution
  - Safe dosage: 10 mEq/100 cc over 1 hour
  - May give at higher dose if life threatening
  - Concentration should not exceed 10 mEq per 100 ml via peripheral line or 20 mEq per 100 ml if central line

Note: Replace cautiously in those with impaired ability to excrete.

Hyperkalemia:

K+ greater than 5.0 mEq/L

- Rarely occurs in healthy people
- Impaired potassium management: Renal Disease / Diabetes

Decreased Excretion
- Renal disease
  - Decreased renal perfusion
  - Sickle cell disease
- Decreased aldosterone
  - Addison’s
  - Diabetes
  - Drugs inhibiting aldosterone (aldosterone antagonists (spironolactone / eplerenone, ACE-I, ARBs), Non steroidal antinflammatories, Heparin)

Increased Intake
- Salt substitutes
- Supplements
- High dose penicillin with K+
- Lactated ringers
- Transfusion of banked blood
Hyperkalemia: Causes

**Cellular disruption with leak of intracellular K+**
- Crush injuries
- Rhabdomyolysis
- Hemolysis (blood transfusion reaction)
- Early burns
- Trauma
- Large hematoma
- Severe catabolic state
- Lysis of tumor cells (chemotherapy)

**Intracellular to extracellular shift**
- Metabolic acidosis
- Hypertonic glucose with insulin deficiency
- Hyperosmolality
- Digitalis toxicity
- Depolarizing neuromuscular blocking agents
- Beta blockers

Hyperkalemia: Signs and Symptoms

**Symptoms when K+ > 6.0 mEq/L**
- Skeletal muscle effects when K+ > 7.0 mEq/L
- Neuromuscular effects complicated by acidosis, low sodium, low calcium, high magnesium
- Parathesias
- Lower extremity weakness
- Hypotension

**EKG Changes**
- Tall narrow peaked T waves
- Prolonged PR and flattened to absent P wave
- Wide and bizarre QRS
- Dysrhythmias
  - □ Bradycardia
  - □ High grade AV block
  - □ Blocks in His Purkinje system
  - □ Sine wave pattern
  - □ Asystole / Vfib / PEA
Hyperkalemia: Treatment

- Level > 6.0 mEq/L should be treated.
- Urgency based on clinical manifestations.

- Limit K+ intake / assess medication profile
- Volume expansion
- K+ > 6.5 or dysrhythmias
  - Stabilize cardiac membrane with calcium chloride
    - Not if digitalis toxic
  - Shift potassium into cell
    - 50% Dextrose and insulin (50 ml and 10 units)
    - High dose inhaled beta agonists (synergistic)
    - Sodium bicarbonate to correct acidosis

Hyperkalemia: Treatment

- Kayexalate is an exchange resin
  - Exchange sodium for K+ and moves K+ out via the GI tract
  - Can be given orally or as retention enema
- Oral dose is administered in sorbital
  - Sorbital orally acts as osmotic laxative
- Retention enema is administered in dextrose
  - Sorbital can cause intestinal necrosis when given by enema

- Loop diuretics if functioning kidneys
- Dialysis if renal dysfunction
Calcium

- Less than 50% of dietary intake is absorbed.
- The majority of the body's calcium is in the bone.
- Serum level regulated by parathyroid levels and vitamin D.
  - Also influenced by serum phosphate levels (inverse relationship), albumin levels, and blood pH.
- Calcium in bone can be exchanged to maintain extracellular levels.

There are 3 types of serum calcium:
- > 40% of calcium is protein bound (mostly albumin)
- 10% is chelated (non-ionized) with substances such as citrate or phosphate
- 50% is ionized (free to leave the extracellular fluid and participate in intracellular function)

Important for several key processes:
- Muscle contraction
- Transmission of nervous system impulses
- Hormone secretion
- Blood clotting and wound healing
- Cellular function

Hypocalcemia

- Calcium < 8.8 mg / dL or ionized calcium < 4.65 mg / dL.

Common disorder in critical care.

Generally asymptomatic if development is slow or if ionized calcium remains normal.

When evaluating hypocalcemia a corrected calcium must be calculated when the albumin is low.
Hypocalcemia: Causes

<table>
<thead>
<tr>
<th>Decreased calcium intake or absorption</th>
<th>Increased calcium excretion</th>
<th>Impaired ability to mobilize calcium from bone stores</th>
<th>Increased calcium binding; Increased calcium chelation (decreased ionized calcium)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low dietary intake</td>
<td>Loop diuretic therapy</td>
<td>Inadequate levels of parathyroid hormone</td>
<td>Alkalosis</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Chronic Diarrhea</td>
<td>(Decreased magnesium inhibits parathyroid release)</td>
<td>Acute Pancreatitis</td>
</tr>
<tr>
<td>Renal failure</td>
<td></td>
<td></td>
<td>Drugs</td>
</tr>
<tr>
<td>Vitamin D deficiency</td>
<td></td>
<td></td>
<td>Cimetidine</td>
</tr>
<tr>
<td>Liver disease</td>
<td></td>
<td></td>
<td>Heparin</td>
</tr>
<tr>
<td>Steroid therapy</td>
<td></td>
<td></td>
<td>Theophylline</td>
</tr>
<tr>
<td>Cushing’s disease</td>
<td></td>
<td></td>
<td>Aminoglycosides</td>
</tr>
</tbody>
</table>

Hypocalcemia: Signs and Symptoms

Most common symptoms due to neuromuscular irritability.

- Parathesias (common)
- Hyperreflexia
- Tetany (spasms of face, hands, and feet)
- Chvostek’s sign
  - Tapping of face over facial nerve located below the temple
  - Positive sign results in spasm of lip, nose or face
- Trouseau’s sign
  - Inflate blood pressure above systolic BP and hold for 3 minutes
  - Positive sign results in contraction of fingers or hand
- Stridor / wheezing / bronchospasm
- For severe deficit: laryngeal spasm, change in mental status, seizures
- Chronic: dry skin and hair and brittle nails; bone pain and risk of fracture

- Cardiovascular effects:
  - Decreased contractility
  - Hypotension
  - Prolonged QT (ST segment hugging baseline for extended period)
  - Torsades de pointes
  - Bradycardia / heart block
  - *Digitalis insensitivity*
  - Heart failure
  - Cardiac arrest
Hypocalcemia: Treatment

- Goal: Low-normal range
- High calcium, low phosphorous diet
- Vitamin D supplements if deficiency
- Phosphate binding antacids
  - Don’t give calcium in high phosphate state
- Treat hypomagnesemia
- Correct alkalosis (increases ionized Ca$^{2+}$)
- Thiazide diuretics (increase tubular calcium reabsorption)
- IV calcium chloride or calcium gluconate

IV Calcium Administration

**Calcium Gluconate**
- Give 10 ml
- 10 ml contains 4.5 mEq of calcium

**Calcium Chloride**
- Give 3-4 ml
- 10 ml contains 13.6 mEq of calcium

- Administer no faster than 1 ml per minute
- May cause sloughing or necrosis (central vein preferred)
Hypercalcemia

- Calcium > 10.4 mg / dL or ionized calcium > 5.26 mg / dL

- Causes:
  - Increased calcium intake (supplement or antacids)
  - Increased calcium absorption (hypophosphatemia, excessive vitamin D)
  - Increased mobilization of calcium from the bone (Vitamin D excess, immobility, hyperparathyroidism, thyroidtoxicosis, neoplasms)
  - Acidosis (increased ionized calcium)
  - Decreased calcium excretion (thiazide diuretics)

Hypercalcemia: Signs and Symptoms

- Hypophosphatemia
- Signs and symptoms related to dehydration
- Gastrointestinal symptoms (slowing of GI tract)
- Bone and flank pain / osteoporosis / pathological fractures
- Muscular symptoms: Hypotonicity / weakness / fatigue
- Neurological symptoms: Decreased mentation, agitation, coma, seizures
- Calcium salts form at high levels
  - Pruritis from skin deposits.
  - Renal calculi and potential kidney injury
  - Deposits on the aorta, cardiac valves, and coronary arteries.
Hypercalcemia: Signs and Symptoms

• Cardiac symptoms:
  – hypertension (may be offset by co-existing dehydration)
  – cardiac ischemia
  – shortened QT segments
  – arrhythmias (conduction abnormalities)
  – digitalis toxicity

• Life threatening signs and symptoms are rare unless calcium levels reach > 14 mg/dL

Hypercalcemia: Treatment

• **PRIMARY TREATMENT:** Rehydration with 0.9 NS

• Decrease calcium absorption
  • Low calcium, high phosphorous diet
  • Glucocorticoids

• Increase calcium excretion
  • Fluids (0.9NS)
  • Loop diuretics
  • Dialysis if renal failure or life threatening
  • Inhibit bone resorption (calcitonin, mithramycin, biphosphonates)

• Prevent cardiac effects
  • Calcium Channel Blockers

• Prevent renal calculi
  • Acidify urine
Low Magnesium: < 1.5 mEq/dL

- Common disorder in hospitalized patients
- Most common causes renal and GI loss
  - Transcellular shift after hypothermia
- Low magnesium = increased digitalis effect
- EKG: Prolonged QT and Torsades de pointes
- Neuromuscular irritability and decreased ability to relax neuromuscular tone
- May induce hypokalemia and hypocalcemia
- Signs and symptoms overlap with hypokalemia and hypocalcemia (often concurrent)
- Oral magnesium can cause diarrhea and further lower magnesium levels
- IV 1 to 2 grams over 10 – 60 minutes
  - More rapidly if life threatening
  - Decrease if kidney injury present

High Magnesium: > 2.5 mEq/L

- Uncommon disorder outside kidney dysfunction
- S & S
  - Hyporeflexia
  - Hypotension
  - Heart block/bradycardia
  - Muscle weakness
  - Change in mental status
  - Lethargy/coma
  - Cardiopulmonary arrest
- Treat with fluids and diuretics if normal renal function.
- Dialysis if renal failure.

Clinical application: Side Effect of IV magnesium administration is hypotension.
Low Phosphorous: < 2.5 to 3.0 mg/dL

- Easily lost from RBCs and skeletal muscle but levels well preserved in cardiac muscle
- Various neuromuscular and central nervous system effects related to depleted intracellular stores
- Enteral replacement preferred if not life threatening
- Parenteral sodium phosphate or potassium phosphate if severe
  - Dose .6mg/kg/hr to .9mg/kg/hr

High Phosphorous: > 4.5 mg/dL

- Causes:
  - Renal dysfunction
  - Hypocalcemia
  - Laxatives with phosphate
  - Increased cellular release
- Same clinical signs as hypocalcemia

Treat hypocalcemia
Aluminum antacids bind with phosphate
Acetazolamide to increase urinary excretion
Dialysis if due to renal failure
SODIUM

Signs and symptoms related to fluid status as well as sodium level.
Osmolality is determined by sodium.
Decrease sodium slowly to avoid cerebral edema.
Increase sodium slowly to avoid osmotic demyelinating syndrome.
Understand importance of thiazide diuretics in predisposing to hyponatremia.
Manifestations (neurological) of hyper and hyponatremia can be the same.

POTASSIUM

Potassium is primarily inside the cell (particularly muscle).
Oral kayexalate often contains sorbitol. Caution with using sorbitol with rectal administration.
The primary systems impacted by potassium are GI, neuromuscular, and cardiac.
Administer calcium chloride to prevent cardiac effects of hyperkalemia.
Shifts from extracellular to intracellular cause hypokalemia.
Shifts from intracellular to extracellular cause hyperkalemia.
Insulin moves potassium inside the cell.
Understand that hypomagnesemia may prevent correction of hypokalemia.
Manifestation of hyperkalemia and hypokalemia can include paresthesia and weakness.
Calcium

Calcium (serum) is found in three forms: protein bound, chelated, and ionized.
Assess for tetany from hypocalcemia with Chvostek’s sign and Trousseau’s sign.
Loss of calcium occurs with loop diuretics but not with thiazide diuretics.
Calcium gluconate differs from calcium chloride in amount of calcium per amp.
Ionized calcium is important: it can leave extracellular fluid to participate in intracellular function.
Understand that low magnesium affects the absorption of calcium and the release of parathyroid.
Make the association between low calcium and prolonged ST segment.

Magnesium

Magnesium is the drug of choice in treatment of torsades de pointes.
A low level of magnesium can be associated with hypokalemia and hypocalcemia.
Give IV replacement 1 to 2 grams over 10 to 60 minutes.
Neuromuscular tone cannot relax with low levels of magnesium.
Especially be concerned when giving IV magnesium to patient with renal dysfunction.
Signs of high magnesium include hyporeflexia and hypotension.
IV magnesium administration has potential side effect of hypotension.
Understand that low magnesium will result in an enhanced digitalis effect.
Magnesium given orally can cause diarrhea and further lower magnesium levels.
HEMATOLIC SYSTEM

Hematologic System

- Spleen (Red Pulp)
  - Stores and releases RBCs
  - 100 cc in response to SNS
  - Filtering and destruction of damaged or old erythrocytes
    - Pitting
    - Catabolizes hemoglobin / iron returned to bone marrow
  - Storage and release of platelets
  - Destruction of damaged platelets
- Spleen (white pulp)
  - Production of lymphocytes
Hematologic System

• Liver
  – Filtering of blood from GI tract
  – Conversion of bilirubin (destruction of RBCs) to bile
  – Detoxification of toxic substances that enter blood
  – Manufacturing of some clotting factors and of thrombin
  – Storage of blood
  – Storage of iron

Hematologic System

Lymph

• Composition
  – Lymphocytes, granulocytes, macrophages, enzymes, antibodies
  – Deficient in platelets and fibrinogen

• Function
  – Returns excess interstitial fluid (hormones) to blood
  – Returns protein and fat from GI tract

Lymph Circulation

• Capillaries – larger than blood capillaries
• Ducts – drain to subclavian vein

Lymph Nodes

• Function – Filter / allow WBCs to phagocytosize bacteria
Blood

- Plasma 55%
  - Protein 7%
  - Water 91%
  - Albumin
  - Globulins
  - Fibrinogen
- Cells 45%
  - Platelets
  - WBCs
  - RBCs
- Other solutes 2%
  - Ions
  - Nutrients
  - Waste products
  - Gases
  - Regulatory substances

RBCs, WBCs and Platelets produced in the bone marrow.

- Cells
  - RBCs (Erythrocytes)
  - WBCs (Leukocytes)
  - Platelets (Thrombocytes)

- Indices
  - MCV
  - MCH / MCHC
  - RDW
RBCs (Erythrocytes)

Production regulated by erythropoietin (hormone produced by kidney)
If blood oxygen decreases kidneys release erythropoietin to stimulate RBC production in the kidneys.

↑ RBCs
- High altitude
- Prolonged exercise
- Reflects body’s attempt to compensate for hypoxia
- Polycythemia vera
  - Bone marrow disorder
  - Weekly phlebotomy

↓ RBCs
- Anemia
- Iron Deficiency
- Blood Loss
- Hemolysis
- Bone Marrow Suppression

Blood oxygen level decreases

Blood oxygen level increases

Erythropoiesis

Secretion of erythropoietin
Erythrocyte Components

Hemoglobin

- Protein molecule in RBC carrying oxygen from the lungs to the tissues.
- Helps RBC maintain itself
- Low HGB
  - Anemia
  - Bleeding
  - Bone marrow suppression
  - Kidney failure
  - Abnormal structure (sickle cell)
- High HGB
  - Dehydration
  - High altitudes
  - Chronic Smokers
  - Polycythemia Vera
  - Abuse of erythropoietin in athletes

Hematocrit

- Portion of blood that consists of RBCs
  - HCT 25% means there are 25 million RBCs in 100 mL of blood
- Low HCT
  - Anemia
  - Blood loss
  - Bone marrow suppression in chemotherapy
  - Hypervolemia
- High HCT
  - Dehydration
  - High altitudes
  - Chronic Smokers

Reticulocytes

- Number of young RBCs circulating in the blood
- Count assesses bone marrow function
- Usually less than 1% of total
- Elevation
  - Bleeding
  - Hemolytic anemia
- Reduced
  - Bone marrow failure
  - Cirrhosis
  - Folate or iron deficiency
  - Kidney disease with decreased erythropoietin production
**MCV: Mean Corpuscle Volume: RBC Size**

- **↑ MCV**
  - Macrocytic anemia (large cells)
  - Megoblastic anemia
  - Folate or B12 Deficiency
  - Liver disease
  - Post Spleenectomy
  - Chemotherapy
  - Hypothyroidism

- **↓ MCV**
  - Microcytic anemia (small cells)
  - Lead poisoning
  - Thalassemias
WBCs (Leukocytes)

Developed in stem cell of bone marrow.
Function: Response to inflammatory process or injury
Differential is needed when leukocytosis is present.

Granulocytes
• Granules in the cytoplasm
• Neutrophils
• Eosinophils
• Basophils

Agranulocytes
• No granules in the cytoplasm
• Monocytes
• Lymphocytes

Leukocytes

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WBC</strong></td>
<td>5,000-10,000</td>
<td>100%</td>
</tr>
<tr>
<td><strong>Neutrophils</strong></td>
<td>2,500-8,000</td>
<td>55-70%</td>
</tr>
<tr>
<td><strong>Lymphocytes</strong></td>
<td>1,000-4,000</td>
<td>20-40%</td>
</tr>
<tr>
<td><strong>Monocytes</strong></td>
<td>100-700</td>
<td>2-8%</td>
</tr>
<tr>
<td><strong>Eosinophils</strong></td>
<td>50-500</td>
<td>1-4%</td>
</tr>
<tr>
<td><strong>Basophils</strong></td>
<td>25-100</td>
<td>0.5-1%</td>
</tr>
</tbody>
</table>
Neutrophils

- Last 1-2 days
- Increase immediately after injury or inflammatory process
- Destroy and ingest bacteria (phagocytosis)
- 1st responder to site of inflammation - 30 minutes to reach site of infection
- Turn into pus and die!

**Neutropenia: Too low**
- Leukemia (decrease production)
- Acute significant infection
- Aplastic anemia
- B12, folate deficiency
- Splenomegaly
- Viral infections

**Neutrophilia: Too High**
- Infection / Inflammation
- Stress
- Burn injuries
- AMI
- Hemolytic anemia
- Steroids use

**Neutrophils**

**Bands**
- Immature neutrophils
- Released after injury or inflammation
- Indicative of acute bacterial infection
- Bandemia: > 6% of immature neutrophils

**Segs**
- Segmented neutrophils
- Mature bands
- Indicative of a more chronic bacterial infection

- Shift to the left
- Shift to the right
### Leukocytes

#### Eosinophils
- Granulocyte
- Found not only in blood but also tissue (GI)
- Increase with
  - chronic seasonal allergies
  - parasitic infection
  - asthma
- Allergic food reactions
- Inhibits histamine

#### Basophils
- Granulocyte
- Part of innate immunity
- Allergies such as asthma
- Inflammatory response
- Release histamines
  - Responsible for watery eyes, itchy skin, runny nose
- No phagocytosis
- Called “Mast Cells” when found in the tissue

#### Leukocytes

#### Monocytes
- Inflammatory process
- Move quickly to infected tissue
- Increased with viral and fungal infections
- Becomes macrophage when enters the tissue to begin phagocytosis
- Phagocytosis
  - Remove foreign materials and cellular debris

#### Lymphocytes
- Involved in all immunity
- B Cells
  - Mature in the bone marrow
  - Humoral, adaptive immunity
  - Make antibodies that attack bacteria
- T Cells
  - Mature in the Thymus gland
  - Cellular, adaptive immunity
  - Attack bodies own tissues that have been invaded or changed
- Natural killer cells
  - Innate immunity
  - Attacks human cells that have become abnormal
**Immune System**

**Acquired (Adaptive) Immunity**

<table>
<thead>
<tr>
<th>B Lymphocytes</th>
<th>T Lymphocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bone Marrow Cells</td>
<td>• Thymus Cells</td>
</tr>
<tr>
<td>• Humoral mediated immunity</td>
<td>• Cell mediated immunity</td>
</tr>
<tr>
<td>• IgG</td>
<td>• T Helper cells (T4 Cells)</td>
</tr>
<tr>
<td>– Chronic invasions</td>
<td>– Direct cells that can kill</td>
</tr>
<tr>
<td>– AIDS, Tumors, Multiple myeloma</td>
<td>• T Cytotoxic cells</td>
</tr>
<tr>
<td>• IgA</td>
<td>– Kills cell</td>
</tr>
<tr>
<td>– Mucosal immunity</td>
<td>• T Memory cells</td>
</tr>
<tr>
<td>• IgM</td>
<td>– Keeps record</td>
</tr>
<tr>
<td>– New infection</td>
<td>• T Suppressor cells (T8 Cells)</td>
</tr>
<tr>
<td>– Nephrotic Syndrome</td>
<td></td>
</tr>
<tr>
<td>– Parasitic infections</td>
<td></td>
</tr>
<tr>
<td>• IgE</td>
<td></td>
</tr>
<tr>
<td>– Allergies</td>
<td></td>
</tr>
<tr>
<td>– Parasites</td>
<td></td>
</tr>
<tr>
<td>• IgD</td>
<td></td>
</tr>
</tbody>
</table>

- **RBC** (Erythrocytes)
- **WBC** (Leukocytes)
- **Platelets** (Thrombocytes)

- Platelet Count
- Platelet Function
Platelets

- Thrombopoietin stimulates bone marrow to produce megakaryocytes
- Megakaryocytes shed platelets from their cytoplasm
- Thrombopoietin is produced in the liver
- Platelets circulate for 7-10 days
- 1/3 are sequestered in the spleen
- Normal 140,000 to 440,000
Figure 14.7: Platelet activation and aggregation


Figure 14.8: A, pathways of platelet activation. B, site of action of antiplatelet drugs

Platelet Disorders

- Thrombocytopenia – to few
- Thrombocythemia – to many
- Platelet dysfunction – abnormal function

### Thrombocythemia

- **Essential**: myeloproliferative disorder
  - Abnormality of hematopoietic stem cell
  - Associated with thrombosis
  - Polycythemia vera (over production of RBCs)
  - Essential thrombocythosis: over production of platelets
- **Secondary** (reactive): overproduction in response to another disorder
  - Acute infection
  - Chronic inflammation
  - Iron deficiency
  - Malignancy
  - Hyposplenism (post-splenectomy)
  - Not typically associated with thrombosis

### Thrombocytopenia

- Decreased platelet production
- Increased splenic sequestration (splenomegaly)
- Increased platelet destruction or consumption
- Dilution of platelets
- Drug induced (heparin)
- Idiopathic thrombocytopenic purpura
- Thrombotic thrombocytopenic purpura (RX with plasmapheresis)
- Increased bleeding risk
Platelet Dysfunction
Abnormal Function

**Congenital**
- Disorders of adhesion
- Disorders of activation
- Disorders of aggregation

**Aquired**
- Disorders of adhesion

ANTIPLATELET THERAPY
P2Y\textsubscript{12} Receptor Inhibitors / ADP Receptor Blockers

- **Thienopyridines**
  - Prodrug: requires activation by the liver
  - Clopidogrel
  - Prasugrel

- **Non thienopyridine**
  - Non-prodrug: does not require activation by the liver
  - Ticagrelor

---

P2Y\textsubscript{12} Receptor Inhibitors / ADP Receptor Blockers

- **Clopidogrel** (Plavix)
  - 600 mg initial dose
  - 75 mg daily

- **Prasugrel** (Effient)
  - 60 mg initial dose
  - 10 mg daily
  - Contraindicated: > 75, < 60 kg, previous TIA, CVA

- **Ticagrelor** (Brilinta)
  - 180 mg initial dose
  - 90 mg twice daily
  - Not to be given with ASA doses > 100 mg

- Adenosine Diphosphate (ADP) - Stored in platelets and released upon platelet activation

- ADP interacts with P2Y\textsubscript{12} chemoreceptors to enhance adhesiveness and aggregation of platelets through the activation of the GP IIb/IIIa pathway
Take Away Prasugrel Points

- Less concern with PPI administration
- Less concern regarding non responders
  - Prodrug but not as dependent on *CYP2C19* isoenzyme
- Only used in patients with planned PCI
  - No benefit to administration before the time of angiography
- Increased bleeding risk
  - ≥ 75 years old
  - <60 KG
- Cannot be used:
  - Previous CVA / TIA

Ticagrelor

- Faster onset and shorter duration than clopidogrel (known as reversible mode of action)
- BID dosing is a potential concern for compliance
- North American effect – thought to be due to higher dose ASA
  - Must not be given with maintenance ASA doses > 100mg
  - Contraindicated in history of intracranial bleeding, active pathological bleeding, severe hepatic impairment
- Although shorter ½ life – recommendation to be held 5 days before surgery.

COAGULATION

Clot Formation: Clotting Cascade

**Intrinsic Pathway**
- Initiated by vascular injury and direct exposure to collagen
  - Site of activated platelet
  - Site of endothelial damage
  - Subendothelial layer where collagen is exposed
  - From initiation to a clot is 2-6 minutes
  - Measured by APTT

**Extrinsic Pathway**
- Initiated by endothelial release (secondary to tissue injury) of thromboplastin tissue factor
  - From initiation to clot is 15 to 20 seconds
  - Measured by Protime

A clot can be produced by activation of either the intrinsic or extrinsic pathway.
The Clotting Cascade

**Intrinsic Pathway**
- XII → XIIa
- XI → Xla
- IX → IXa
- X → Xa
- II Prothrombin → IIa Thrombin

**Extrinsic Pathway**
- Thromboplastin Tissue Factor III → VII
- VIIla
- Phospholipid
- Calcium
- Factor VIIIa
- Activated platelets
- Endo / tissue damage

**Common Pathway**
- Va
- Fibrinogen → Fibrin → Fibrin Stable Clot

**Anticoagulants**
- Warfarin
- Fondaparinux
- Rivaroxaban
- Apixaban
- Edoxaban
- UFH
- LMWH
- UFH
- LMWH
- Bivalrudin
- Dibigitran
- Lirudin
- Argatroban
- Fondaparinox
- Rivaroxaban
- Apixaban
- Edoxaban
- Bivalrudin
- Dibigitran
- Lirudin
- Argatroban
- Fibronolytics
Unfractionated Heparin

- Works in the intrinsic and common pathway
- Antithrombin activator that inhibits factors Xa and IIa (thrombin)
  - Antithrombin III lyses factor Xa and thrombin and inhibits clotting
  - When heparin binds with antithrombin III the inhibition is increased 1000 times
- Concern that unfractionated heparin results in platelet activation - although thrombin is a strong platelet activator and heparin is an antithrombin drug
- Anticoagulation is almost instant
- ½ life relatively short
- Antidote: Protamine 1 mg per 100 units
- In NSTEMI: continue for 48 hours or until PCI

More About Heparin

- Different dose and aPTT for ACS versus venous thrombotic event
- aPTT (activated partial thromboplastin time) is used to monitor effectiveness and safety
  - Goal is aPTT 1.5 -2Xs the control
  - Weight based heparin dosing reaches goal 90% of time compared to 77% with standard therapy
- OR – Anti factor Xa levels
  - 0.3-0.7 IU/ml
- Baseline aPTT, PT/INR, platelets and CBC
- Increased bleeding can occur with renal failure
  - Heparin has dual clearance mechanism
Heparin Induced Thrombocytopenia

Mild Thrombocytopenia
- Non-immune HIT
- Mild thrombocytopenia
- Occurs in 10-20% of patients
- Platelet count usually remains above 100,000

Severe Thrombocytopenia
- Immune induced HIT
- Occurs in 1-3% of patients
- Platelet aggregation resulting in venous or arterial thrombosis (HITT – Thrombocytopenia with thrombosis)
- Onset 5 to 14 days after exposure to heparin
- Platelet count usually < 100,000
- Usually a drop of > 20,000

More on Immune Mediated HIT

- Heparin can cause increase blood concentration of platelet factor 4 (PF4)
- PF4 can combine with heparin and create a complex
- Heparin/PF4 complex stimulates production of antiheparin/PF4 complex antibody
- Antibodies cause platelet activation leading to a hypercoagulable state -> thromboembolic complications
- Assess platelets every 2-3 days from day 4-14
- ELISA: Initial test
  - 99% sensitive, poor specificity
  - High negative predictive value
- SRA: Serotonin release assay
- Heparin-induced platelet aggregation assay (HIPA)
  - Gold standard for diagnosis (highly specific and very sensitivity)
- HIT antibodies are usually IgG class
  - Take 5 days to form
  - IgG antibodies associated with platelet activation and increased thrombin generation
  - Antibodies not necessarily associated with thrombotic risk
  - Can disappear 3 months after exposure
Treatment of HIT

1. Discontinue and avoid all heparin.
   1. Platelet count <100,00 or > 50% drop from baseline
2. Give a non-heparin alternative anticoagulant
   Direct thrombin inhibitors (argatroban, bivalirudin)
3. **Postpone warfarin pending substantial platelet count recovery (give vitamin K if warfarin has already been started).** Warfarin is associated with protein C deficiency and increased risk for microthrombosis
4. **Avoid platelet transfusions – leads to platelet activation.**
5. Test for HIT antibodies

Low Molecular Weight Heparin

- **Enoxaparin, dalteparin, tinzaparin, and nadroparin**
- **Smaller in size**

- Antithrombin by inhibiting factor Xa
- Causes less inactivation of thrombin and less bleeding than standard heparin

- More predictable anticoagulant response
- No need to monitor APTT
  - Anti Xa levels can be drawn 4 hours after SQ dose

- Lower incidence of heparin induced thrombocytopenia
- Less platelet activation concern than with UFH
Low Molecular Weight Heparin

- Can be self administered with Sub – Q administration

- ½ life 4-6 hours
- Protamine reverses 60% of drug effect

- Renal failure results in increased risk of bleeding because LMWH is renally cleared
  - Special dosing for chronic renal insufficiency with enoxaparin

NOVEL ORAL ANTICOAGULANTS

NOAC
Anticoagulants as Alternative to Warfarin

Valvular Atrial Fibrillation
- Warfarin
  - Vitamin K antagonist

Non-Valvular Atrial Fibrillation
- Dabigatran
  - Direct thrombin inhibitor
- Rivaroxaban
  - Factor Xa inhibitor
- Apixaban
  - Factor Xa inhibitor
- Edoxaban
  - Factor Xa inhibitor

Newer Oral Agents

<table>
<thead>
<tr>
<th>Generic</th>
<th>Peak Plasma Level</th>
<th>Elimination Half-life</th>
<th>Clearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dabigatran</td>
<td>1.5 hours</td>
<td>12 to 18 hours</td>
<td>Mostly by kidneys</td>
</tr>
<tr>
<td>Rivaroxaban</td>
<td>3 hours</td>
<td>5-9 hours (up 11 to 13 hours if &gt; 75 years old)</td>
<td>Hepatic and renal excretion</td>
</tr>
<tr>
<td>Abixaban</td>
<td>3-4 hours</td>
<td>8 to 15 hours</td>
<td>25% cleared by the kidneys</td>
</tr>
<tr>
<td>Edoxaban</td>
<td>1-2 hours</td>
<td>10-14 hours</td>
<td>Concern in patients with normal renal function</td>
</tr>
</tbody>
</table>
### Dosing for Atrial Fibrillation

<table>
<thead>
<tr>
<th>Generic</th>
<th>Trade Name</th>
<th>Class</th>
<th>Dosing</th>
</tr>
</thead>
</table>
| Dabigatran | Pradaxa | Direct thrombin inhibitor | 150 mg PO BID  
75 mg PO BID with Cr. Cl. 15 to 30 mL/minute  
CrCl: < 15 mL/m – not recommended |
| Rivaroxaban | Xarelto | Factor Xa inhibitor | Dose 20 mg PO daily  
CrCl: 15-50 mL/m  
CrCl: < 15 mL/m – not recommended  
ESRD: Avoid |
| Abixaban | Eliquis | Factor Xa inhibitor | Dose: 5 mg BID  
Dose: 2.5 mg BID with any two of the following  
> 80 years of age  
Creatinine > 1.5 mg/dL  
Weight < 60 kg |
| Edoxaban | Savaysa | Factor Xa inhibitor | 60 mg daily for Cr. Cl. > 50 to ≤ 95  
30 mg daily for Cr. Cl. 15 to 50  
CrCl: > 95 ml/m do not use – ENGAGE AF-TIMI 48 |

### Dosing VTE / DVT Prophylaxis

<table>
<thead>
<tr>
<th>Generic</th>
<th>Trade Name</th>
<th>Class</th>
<th>Dosing VTE / DVT Prophylaxis</th>
</tr>
</thead>
</table>
| Dabigatran | Pradaxa | Direct thrombin inhibitor | VTE: 150mg BID AFTER 5-10 days of parenteral anticoagulation  
DVT Prophylaxis: 110mg initial dose then 220 mg daily |
| Rivaroxaban | Xarelto | Factor Xa inhibitor | VTE: 15 mg BID x 21 days then 20mg daily  
DVT Prophylaxis: 10mg daily  
CrCl: < 30 mL/min  
CrCl: > 30 mL/min and > 65 years decrease dose |
| Abixaban | Eliquis | Factor Xa inhibitor | VTE:10 mg BID x 7 days then 5mg BID after 6 months  
2.5mg BID.  
Patients with serum creatinine > 2.5 mg/dL or CrCl <25 mL/m were excluded from clinical trials |
| Edoxaban | Savaysa | Factor Xa inhibitor | VTE: 60 mg daily AFTER 50-10 days of parenteral anticoagulation  
< 60 kg 30mg daily  
CrCl: 15-50 mL/m 30 mg daily  
CrCl: < 15 mL/m – not recommended  
CrCl: < 30 mL/min in age > 65 years not recommended |
## Hold Times for Newer Oral Agents

<table>
<thead>
<tr>
<th>Drug</th>
<th>Surgery with high risk for bleeding (i.e. CABG)</th>
<th>Surgery Low Bleeding Risk</th>
</tr>
</thead>
</table>
| Dabigatran    | ▪ 3 to 5 days.  
▪ For urgent cases until clotting times are normal or until four half-lives has passed  
▪ Hold times for surgery are dependent on renal function  
▪ DO NOT USE INR. Can be falsely elevated                                                               | Minimum hold time for low risk surgery and normal renal function is ≥ 24 hours          |
| Rivaroxaban / Apixaban | **Renal impairment**  
Cr. Cl.: ≥ 50 = 3 days  
Cr. Cl. < 50 = 4 days  
**Liver impairment**  
Mild: 2 days  
Mod: At least 4 days  
Severe: At least 7 days | **Renal impairment**  
Cr. Cl.: ≥ 50 = 1 day  
Cr. Cl. < 50 = 3 days  
**Liver impairment**  
Mild: 1 day  
Mod: At least 2 days  
Severe: At least 5 days |
| Edoxaban      | Not specifically addressed in product information.                                                            | Minimum hold time of at least 24 hours                                                   |

---

**Important Nursing Consideration in Oral Anticoagulation:**

**HTN Control to Reduce the Risk of Cerebral Hemorrhage**
Transfusions

- Most common cause for transfusion reaction is human error
- One unit of packed RBCs
  - Raise HGB by 1 g/dL
  - Raise hematocrit 3%.
- Platelet transfusion
  - Raises platelet count by 6,000-10,000/mm³

Transfusion Reaction

**Allergic**
- Sensitivity to plasma protein of donor antibody – reacts with recipient
- Monitor for
  - Flushing
  - Hives
  - Pruritis
  - N/V
  - Laryngeal edema
  - Dyspnea

**Febrile, Non-Hemolytic**
- Hypersensitivity to donor white cells, platelets or plasma proteins.
- Monitor for:
  - Sudden chills and fever
  - Flushing
  - Headache
  - Anxiety
  - Malaise
  - Tachycardia
Transfusion Reaction

**Septic Reaction**
- Caused by transfusion of blood or blood components contaminated with bacteria
- Monitor for:
  - Rapid onset of chills
  - Vomiting
  - Marked Hypotension
  - High Fever
  - Abdominal and extremity pain
  - Blood diarrhea

**Hemolytic Reaction**
- Caused by infusion of incompatible blood
- Monitor for:
  - Low back pain (first sign). This is due to inflammatory response of the kidneys to incompatible blood.
  - Chills
  - Feeling of fullness
  - Tachycardia
  - Flushing
  - Tachypnea
  - Hypotension
  - Bleeding
  - Vascular collapse
  - Acute renal failure

**Treatment of Transfusion Rx**
- STOP transfusion
- Change Tubing
- Start NS
- Notify blood bank – send rest of blood back to blood bank
- Monitor urine output for oliguria an anuria (hemolytic reaction)
- Send urine to test for hemoglobinuria (hemolytic reaction)
- Epinephrine for allergic reaction
- Antibiotic for septic reaction
- Support vital signs
GI System

- Upper GI Tract
  - Mouth to stomach
- Mid GI Tract
  - Small intestines
  - Absorption of nutrients
- Lower GI Tract
  - Large intestine / colon
  - Absorption of water an electrolytes

GI System Overview
GI System Overview

• Protective Mechanisms
  – Gastroduodenal mucosa is coated by glycoprotein mucus
    • GP mucus forms a gel
    • Gel prevents the back diffusion of acid and pepsin and helps maintain mucosal-luminal pH gradient
  – Gastroduodenal epithelial cells secrete bicarbonate
    • Augments the actions of the glycoprotein mucus in maintaining pH

• Protective Mechanisms (Cont)
  – Gastroduodenal epithelial cells are protected structurally against damage from acid and pepsin
    • Connected by tight junctions that help prevent acid penetration
  • Maintain mucosal pH above 6
  • Normal gastric pH 1-3 (4)
Intra abdominal Pressure (IAP)

- Intra abdominal hypertension = elevation of IAP above normal
- Normal abdominal pressure = 0 mmHg to subatmospheric
- Mildly elevated = 10-20 mmHg
- Moderately elevated = 21-40 mm Hg
- Severely elevated = > 40 mmHg
- IAP > 25 mmHg is considered high -> abdominal compartment syndrome

Who is at risk for Increase IAP?

- Abdominal or pelvic trauma
- Abdominal surgery
- Intra abdominal infection
- Rupture abdominal aortic aneurysm
- Pancreatitis
- Intra abdominal neoplasm
- Intestinal obstruction
- Ascites
- Obstetric conditions
How might you recognize IAP?

- Abdominal pain and distention
- Increased abdominal girth
- Shortness of breath
- Decreased urine output
- High peak and plateau pressures on vent
- Other organ failure

Consequences of Increased IAP

- ↑ HR, RR, intrathoracic pressure, CVP, PAOP, SVR, Intacranial Pressure
- ↓ SaO₂, SpO₂, CO/CI, lung compliance, GFR, cerebral perfusion pressure, portal, celiac and mesenteric blood flow
- Metabolic acidosis
- Increased mortality
Measurement Considerations

- The transducer should be leveled and zeroed at the iliac crest in the midaxillary line
- IAP pressure should be measured at the end of expiration
- IAP should be expressed in mm Hg keeping in mind that 1 mm Hg is equal to 1.36 cm H₂O
Treatment of Increased IAP and Abdominal Compartment Syndrome

- ↑ HOB, encourage deep breathing
- Paracentesis may be required
- Decompress the GI tract
  - Medications – prokinetic agents (metoclopramide - reglan)
  - Gastric or colonic tube
  - Enemas
- Reduce edema
  - Diuretics
  - Dialysis / ultrafiltration
  - Paracentesis
- Surgical decompression may be needed
Malnutrition

- Dietary intake insufficient to meet metabolic demands
- Nutritional needs:
  - Carbohydrates
  - Proteins
  - Fats
- Evaluate Protein
  - Albumin (abnormal if < 3.4 mg/dl)
  - Prealbumin (abnormal if <15 mg/dl)
  - Transferrin (abnormal if < 200 mg/dl)
Management of Malnutrition

- Prevent malnutrition in ICU
- Start nutrition within 48 hours
- Calories
  - Minimal 25 kcal/kg/day
  - Moderate illness: 35 kcal/kg/day
  - Sepsis: 45 kcal/kg/day
  - Burns: 80 kcal/kg/day
- Balance
  - Protein: 15-20%
  - Carbs: 50-60%
  - Fats: 20-30%
- Fluids
  - 25-36 mL/kg/d
  - Add 150 mL/day for each degree of body temperature > 37°C

Enteral Feedings

- If the gut works use it!
- Intermittent or continuous
- X-ray is only reliable method of assessing location of tube - ✓ before starting feedings
- I and O
- Daily weights
- Blood glucose testing
- Electrolytes daily
- BUN daily
Enteral Feedings

- NO FOOD COLORING!!!
- Elevate HOB 30-40 degrees
- ✔ residual every 4-6 hours or before bolus feeding
  - >200 hold feeding for 1 hour
  - Check again in 1 hour
  - If continue may need to change to parenteral if cannot achieve caloric needs with enteral feedings
- Do not use gastric residual volumes as part of routine care to monitor ICU patients on EN. (New 2016)
- Administer free water (1mL/kcal)
  - Prevents hyperosmolality

Diagnostic Tests

- Abdominal flat plate
  - Detects dilated bowel loops, free air (-> perforation), fluid accumulations, intramural bowel gas
- KUB – kidneys, ureter, bladder
- Upper GI Series
  - Uses contrast
  - Can detect ulcers, tumors, strictures, obstructions
- Barium Swallow
  - Detects swallowing, motility and esophagus emptying
- Small Bowel
  - Detects ulcers, tumors, diverticula, polyps and inflammatory bowel disease
- Lower GI Series
  - Barium enema
  - Detects ulcers, tumors, strictures, obstructions, diverticula, polyps and inflammatory bowel disease
- Esophagogastroduodenoscopy (EGD) or upper endoscopy
  - Detects ulcers, tumors, strictures, obstructions
  - Evaluate bleeding, nausea, vomiting or unexplained abdominal pain
  - Perform biopsy
- Angiography
  - Catheterization of visceral arterial system and portal venous system
  - Vasopressin administration
KUB – Flat Plate of Abdomen

- Determine the cause of acute abdominal pain or palpable mass
- Evaluate the effects of lower abdominal trauma, such as internal hemorrhage
- Evaluate known or suspected intestinal obstructions
- Evaluate the presence of renal, ureter, or other organ calculi
- Evaluate the size, shape, and position of the liver, kidneys, and spleen
- Evaluate suspected abnormal fluid, air, or metallic objects in the abdomen

Abdominal Radiograph

- Abdominal X-rays are only useful for certain defined pathology such as abnormal ‘gases, masses, bones and stones’.
- May be useful in undifferentiated abdominal pain with a provisional diagnosis of:
  - Toxic megacolon - Colonic diameter >6 cm
  - Bowel obstruction (50% sensitive for acute obstruction)
  - Bowel ischemia
  - Perforation of a viscus with abdominal free air
  - KUB for renal tract calculi: 80–90% sensitivity if radiolucent stone >3 mm diameter.
  - Foreign body—following ingestion
THE ACUTE ABDOMEN

Abdominal Pain: Persistent and Severe
Abdominal Distension
Abdominal Guarding: Rigid and board like abdomen
Rebound tenderness
Diminished or absent bowel sounds
Nausea, vomiting
Fever
Leukocytosis

Acute Abdomen

Sudden onset of abdominal pain, associated with inflammation of peritoneal cavity and most often requiring surgery.

- Perforation of abdominal viscera
  - Any abdominal organ
  - Appendix most common
- Perforated/ruptured blood vessel
  - Ulcer
  - Abdominal aortic aneurysm
- Bowel Ischemia
  - Acute or chronic (venous or arterial)
  - Occlusive or Non Occlusive
    - Shock, dehydration, vasoconstrictors, CHF, CABG
- Bowel Obstruction
  - Acute, subacute, chronic or intermittent
  - Acute leads to infarction or strangulation
Intestinal Perforation

**Treatment:** Surgical repair, antibiotics, hemodynamic support.

- Abdominal pain – periumbilical area and spreading throughout
- Preference for flat and still
- Diffuse tenderness
- Board like abdomen
- Muscle rigidity
- Absent bowel sounds
- Rebound tenderness may be present
- Abdominal symptoms may be localized to one area
- Feverish
- Hypovolemia / hypotension
- Leukocytosis – shift to left
- Free air under diaphragm on x-ray / ileus pattern may be present
- X-ray with contrast will confirm perforation
- Exploratory laparotomy to diagnose cause

Mesenteric Ischemia

- **Acute:** Arterial embolism
  - Pain (gut attack)
  - Gut emptying (vomiting / diarrhea)
  - Cardiac etiology
- **Acute:** Arterial thrombosis
  - Severe abdominal pain
  - History post prandial pain (abdominal angina); weight loss; food fear
  - Atherosclerotic disease
Mesenteric Ischemia

• Non occlusive mesenteric ischemia
  — Older adults in ICU
  — Shock, vasopressor use
  — Symptoms
    • Prodrome over several days (malaise and vague abdominal discomfort).
    • Infarction: Patient deteriorates with no apparent reason.
      — Increased pain associated with vomiting
      — Bloody loose stool
      — Hypotension and tachycardia

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Intestinal Obstruction

- Presentation
  - Crampy abdominal pain (most often with small bowel obstruction)
  - Vomiting
  - Decreased passing of stool / flatus
  - Distended abdomen

- Most Common Causes
  - Adhesions – previous surgery
  - Ulcers
  - Crohn’s Disease
  - Inflammatory bowel
  - Hernia’s containing bowel
  - Foreign body
Intestinal Obstruction

- **Physical exam**
  - Distended abdomen - diffusely - but not severely tender
  - **Hyper resonance or tympani on percussion**
  - Increased, high pitched bowel sounds
- **Vomitus**
  - Yellow – green for proximal obstructions of small bowel
  - Darker / fecal smelling for distal small bowel or colon obstructions

- **Diagnosis**
  - Abdominal x-ray
  - Barium contrast studies
  - CT (oral contrast)
  - Abdominal ultrasound

- **Treatment**
  - Fluid and electrolyte correction
  - **NG to suction**
  - Manual reduction of hernia
  - Surgery
    - Almost always for tumors
    - Maybe for adhesions

---

**GI Complications of Bariatric Surgery**

- **Mortality** is low but is usually associated with anastomotic leaks with peritonitis.
  - Obvious signs or vague signs and symptoms.
  - Vague: mild abdominal, shoulder, or back pain, altered urination and BM frequency, tachycardia.
- **Acute gastric distention** secondary to edema and obstruction at the enteroenterostomy.
  - May lead to staple line dehiscence or gastroenterostomotic leaks.
GI Hemorrhage

- Bleeding in upper or lower GI tract
- Erosion in mucosa
- May involve submucosa and muscular layers of esophagus, stomach, duodenum

Upper GI Bleed
- Peptic ulcer
- Stress Ulcer / Erosive Gastritis
- Esophageal Varices
- Mallory-Weiss Tears
- Angiomas of stomach or small bowel
- AV Malformations

Lower GI Bleed
- Bleeding from the colon, rectum or anus (after duodenum)
- Diverticulitis
- Inflammatory bowel disease
- Polyps
- AV malformation
- Internal hemorrhoids

Upper GI Bleed more common
- 75% of those presenting with GI bleed have upper GI source
- Just under 25% of patients who develop upper GI bleeding are already hospitalized for with another condition
- 5% percent who develop lower GI bleed are already hospitalized with another condition
GI Hemorrhage
Assessment

• **Hematochezia:** Bright red blood from the rectum
  – Typically: Lower GI bleed
  – HOWEVER: May suggest upper GI bleed if rapid enough
  – Blood in GI tracts -> increase peristalsis and diarrhea

• **Melena:** Black, tarry, sticky stools
  – Usually an upper GI bleeding source
  – HOWEVER: May result from bleeding in small bowel or proximal colon
  – May take several days after bleeding stops to clear

GI Hemorrhage: Assessment

Hematemesis – bright red blood or coffee ground emesis
  – Usually upper GI bleeding source

Important: Gastric acid converts bright red hemoglobin to brown hematin (coffee ground)
THEREFORE: Hematemesis that is bright red results from profuse bleeding with little contact with gastric secretions
**GI Hemorrhage Assessment**

- Symptoms based on:
  - Extent of blood loss
  - Rate of bleeding
  - Patients status before bleeding
  - Cardiac patient
  - Chronic GI bleed

<table>
<thead>
<tr>
<th>Blood Volume Loss</th>
<th>Clinical Signs and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;15% total blood volume (&lt;1000 ml)</td>
<td>HR normal or &lt;100 bpm supine</td>
</tr>
<tr>
<td>15%-30% total blood volume (1000 ml)</td>
<td>Capillary refill WNL</td>
</tr>
<tr>
<td>31%-40% total blood volume (1500-2000 ml)</td>
<td>Urine output &gt;30 ml/hr</td>
</tr>
<tr>
<td>&gt;40% total blood volume (&gt;2000 ml)</td>
<td>Anxiety may be present</td>
</tr>
<tr>
<td></td>
<td>Systolic BP &gt;90 mm Hg</td>
</tr>
<tr>
<td></td>
<td>Decreased pulse (pressure)</td>
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<tr>
<td></td>
<td>HR 100-120 bpm</td>
</tr>
<tr>
<td></td>
<td>Capillary refill &gt;3 sec</td>
</tr>
<tr>
<td></td>
<td>Increased respiratory rate</td>
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<tr>
<td></td>
<td>Urine output 25-30 ml/hr</td>
</tr>
<tr>
<td></td>
<td>Weakness</td>
</tr>
<tr>
<td></td>
<td>May have mental status changes</td>
</tr>
<tr>
<td></td>
<td>HR &gt;120 bpm</td>
</tr>
<tr>
<td></td>
<td>Systolic blood pressure 70-90 mm Hg</td>
</tr>
<tr>
<td></td>
<td>Cool, pale skin</td>
</tr>
<tr>
<td></td>
<td>Increased respiratory rate</td>
</tr>
<tr>
<td></td>
<td>Urine output 1-15 ml/hr</td>
</tr>
<tr>
<td></td>
<td>Mental status changes</td>
</tr>
<tr>
<td></td>
<td>HR &gt;140 bpm</td>
</tr>
<tr>
<td></td>
<td>Mean arterial pressure &lt;50 mm Hg</td>
</tr>
<tr>
<td></td>
<td>Confused/lethargic</td>
</tr>
<tr>
<td></td>
<td>Cold, clammy skin</td>
</tr>
<tr>
<td></td>
<td>Urine output minimal</td>
</tr>
</tbody>
</table>

Acute GI Bleed: Typically 1200-1500ml

H&H 48-72 hours to equilibrate
Peptic Ulcer

- Gastric and Duodenal Ulcers
- Leading cause of upper GI hemorrhage
- Occurs when normal protective mechanisms cease to function
- Once protective lining is penetrated -> gastric secretions autodigest the layers of the stomach -> damage to mucosal and submucosal layers -> damage to blood vessels -> hemorrhage

Erosive Gastritis: Stress Ulcer

- Hemorrhagic erosive gastritis
- Usually limited to the stomach
- Superficial erosions to deep ulcerations
- Can develop within hours of admission
- Same mechanism as with peptic ulcer
- Contributing factor: decreased mucosal blood flow -> ischemia and degeneration of mucosal lining

- Hemorrhage in 2-6% of patients
- 50-80% mortality
- 2nd leading cause of GI hemorrhage
Stress Ulcer

• High physiologic stress situations
• Burns, head trauma, extensive surgery, shock
  — Curling’s Ulcer – burns
  — Cushing’s Ulcer – brain-injury
  • Over stimulation of PNS
    — (Increase activity of GI tract)

GI Hemorrhage
Signs and Symptoms

• Cool, clammy
• Pallor
• Restless, apprehension
• Tachycardia
• Diaphoresis
• Hypoactive bowel sounds
• Hypotension
• Syncope
GI Hemorrhage Treatment

**FOCUS:** Stop the bleeding and provide fluid resuscitation while maintaining vital functions.

- **Fluid Resuscitation**
  - Crystalloids
    - Ringers Lactate preferred – if no liver diseases
    - Maintain urine output 0.5 to 1.0ml.kg.hour
    - Maintain PAOP or 12-15mmHg
  - Blood and blood products
    - Packed cells (if still hemodynamically unstable after 2 L of fluid)
    - Keep HCT > 30%
    - 1 unit = 3% increase in HCT

- **Control Bleeding**
  - Vasopressin IV – constricts splenic arteriolar bed and decreases portal venous pressure
    - Can be given intra-arterial during angiography
  - Endoscopic procedures
    - Thermal therapy
    - Endoscopic injection
      - Sclerosing Agent
      - Epinephrine
GI Hemorrhage
Other Treatment

• Gastric lavage
  • Room temperature saline
  • Does not control bleeding
  • Monitors bleeding and removes nitrogenous materials (blood) from gut

• Magnesium sulfate and saline enemas
  • Removes nitrogenous material from colon

GI Hemorrhage
Other Treatment / Care

Increase gastric pH
  – Maintain pH 3.5 to 5.0 (normal 1-3)
  – Agents that decrease acidity and/or protect gastric mucosa
    • Antacids: Buffer gastric acid
    • Histamine blockers (Tagamet, Zantac, Pecid, Axid): Block histamine and decrease volume and concentration of gastric secretions
    • Proton Pump Inhibitors (Prilosec, Prevacid, Aciphex): Prevention of formation of hydrochloric acid
    • Mucosal protectants (Cytotec): Decreases gastric acid secretion; increases mucosal blood flow; enhances normal body protective mechanisms
    • Mucosal Inhibitors (Carafate): Forms an adhesive protective coating over an ulcer crater; absorbs pepsin
GI Hemorrhage
Surgical Treatment

• Indications
  – Continuation of bleeding despite treatment
  – Administration of > 8 Units of blood in 24 hours
  – Hemorrhage to the point of hypotension or shock
  – Rebleeding after hemostasis is achieved

Esophageal Varices

• Result of portal hypertension secondary to hepatic cirrhosis
• Engorged and distended blood vessels of esophagus and proximal stomach
• Damage to liver sinusoid results in:
  – Resistance to portal blood flow increases elevating the pressure in the liver -> increased portal venous pressure -> diversion of portal blood from areas of high pressure to areas of low pressure -> engorged and dilated vessels
Esophageal Varices

- Rupture occurs in 19%-40% of patients
- Mortality 40-70%
- 3rd leading cause of GI hemorrhage

Esophageal Varices
Treatment

- Blood and fluid replacement as with GI Bleed
- Insert NG tube: Gastric lavage WITH CAUTION
- Maintain Gastric pH at 3.5 to 5.0
- Vasopressin – decrease portal venous pressure
- Betablockers – decrease portal venous pressure
- Somatostatin – decreases splanchnic blood flow
- Assess for alcohol withdrawal
- Nutrition
  - Begin with clear liquids, progress as tolerates
  - Encourage to chew foods, especially hard, sharp foods
  - Avoid alcohol-containing mouthwashes
Esophageal Varices
Treatment

Sclerotherapy
– Injection of sclerosing agent
– Induces vasoconstriction and results in formation of venous thrombosis

Esophageal Variceal Ligation
Rubber bands or O-rings are placed on the target vessels at the gastroesophageal junction

Balloon Tamponade

- Halt hemorrhage by applying direct pressure
- Elevate HOB to 45 degrees
- Suction equipment at bedside
  - Suction as needed – patient cannot swallow
- Maintain ordered balloon pressures
- Maintain traction
- Deflate as ordered
- Scissors at bedside
  - Cut balloon for acute respiratory distress
- Good oral hygiene
C Difficile

- Disturbance of normal bacteria of colon
- Hyper virulent *C. difficile* strain
- *C. difficile* colitis associated most strongly with the following antibiotics: fluoroquinolones, cephalosporins, and clindamycin
- H2Blocker and PPI use
- Metronidazole or vancomycin
- Fecal bacteriotherapy

- Probiotic prophylaxis

ABDOMINAL TRAUMA
Abdominal Organs Frequently Injured

- Spleen
- Liver
- Retroperitoneum
- Small bowel
- Kidneys

- Bladder
- Colorectum
- Diaphragm
- Pancreas

Abdominal Components

Intrathoracic Abdomen
- Diaphragm
- Liver
- Spleen
- Stomach

The rib cage limits palpation and complete examination.

Pelvic Abdomen
- Bladder
- Urethra
- Rectum
- Small intestine
- Female organs

May be extraperitoneal and difficult to diagnose.

Retroperitoneal Abdomen
- Kidneys
- Ureters
- Pancreas
- Aorta
- Vena cava.

Evaluation may require CT, angiography, and IVP.

True Abdomen
- Small and large intestines
- Uterus (if gravid)
- Bladder (when distended)

Perforation associated with significant physical findings.
Key Complications

<table>
<thead>
<tr>
<th>Liver</th>
<th>Spleen</th>
<th>Pancreas</th>
<th>Stomach</th>
<th>Intestine</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Shock</td>
<td>● Shock</td>
<td>● Shock</td>
<td>● Gastric fistula</td>
<td>● Ileus</td>
</tr>
<tr>
<td>● Clotting abnormalities</td>
<td>● Infection, abscess, sepsis</td>
<td>● Pancreatitis</td>
<td>● Abscess, fistula</td>
<td>● Abscess, fistula</td>
</tr>
<tr>
<td>● Hepatic failure</td>
<td></td>
<td>● Abscess, fistula</td>
<td></td>
<td>● Peritonitis, Sepsis</td>
</tr>
<tr>
<td>● Infection, abscess, sepsis</td>
<td></td>
<td>● Diabetes</td>
<td></td>
<td>● Intestinal ischemia or infarction</td>
</tr>
<tr>
<td>● Respiratory complications</td>
<td></td>
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<td></td>
<td>● Obstruction</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>● Perforation</td>
</tr>
</tbody>
</table>

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FAST

Focused Abdominal Sonography for Trauma (FAST) allows rapid and noninvasive determination of the presence of free intra-abdominal fluid.

- **Stable**
  - Positive
    - Computed Tomography Abdomen
    - Or laparotomy if indicated for other reasons (e.g., free gas)
  - Negative
    - Serial examinations and follow up ultrasound
    - Or CT depending on clinical scenario

- **Unstable**
  - Positive
    - Laparotomy
    - Continued search for and management of other causes of hypotension
  - Negative
    - Search for other causes of hypotension
    - Repeat FAST and consider Diagnostic Peritoneal Lavage
Key Nursing Considerations

- Maintain airway, ventilation, and oxygenation
- Detect bleeding, control bleeding, and support circulating volume
- Decompress abdomen
- Prevent infection
- Fluid and electrolyte balance
- Assess for complications

Diabetic Gastroparesis

- Delayed gastric emptying
- Slows or stops movement of food from stomach to small intestine.
- Damaged Vagus nerve is damaged and stomach muscles stop working resulting in delayed emptying
- Diabetes mellitus most common cause of gastroparesis
- High blood sugars ultimately damage Vagus nerve.
Diabetic Gastroparesis

**Symptoms**
- Nausea
- Feeling of fullness after eating only small amounts of food
- Vomiting undigested food
- Gastroesophageal reflux
- Abdominal pain
- Abdominal bloating
- Lack of appetite

**Aggravated By:**
- Greasy or rich foods
- Large quantities of foods high in fiber
  - Raw fruits and vegetables
- Drinks high in fat or carbonation

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Diabetic Gastroparesis

**Outlook**
- Not curative in most cases
- Long-term chronic illness
- Symptoms come and go

**Dietary Changes**
- Eat six small meals a day
- Chew foods well
- Noncarbonated beverages with meals
- Avoid foods with fibrous parts: oranges, broccoli
- Walking or sitting for 2 hours after eating – avoid lying down to enhance gastric emptying
- Liquid or pureed foods with extreme symptoms

**Other Treatment**
- Reglan (Metoclopramide)
- Erythromycin
- Antiemetics
- Gastric Electrical Stimulation
- J Tube
- TPN
Irritable Bowel Syndrome

• Recurrent abdominal pain or discomfort at least 3 days/months in the past 3 months associated with >2 of the following:
  – Improvement with defection
  – Onset associate with a change in frequency of stool
  – Onset associated with a change in form (appearance) of stool

Subtypes

• IBS with constipation
• IBS with diarrhea
• IBS mixed type
• IBS unclassified

• Chronic relapsing disease
• Symptoms vary over time
• Worse outcomes in those with
  • Previous Surgery
  • Longer duration of the disease
  • Higher somatic scores
  • Comorbid anxiety
  • Depression

Irritable Bowel Syndrome

Medication Triggers

• Antihistamines
• Calcium
• Iron
• Magnesium
• NSAIDS
• Wheat Bran
• Antibiotics
• Antidepressants
• Antipsychotics
• CA channel blockers
• Diuretics
• Mefformin
• Opiods
• Sympathomimetics

Food Triggers

• Rapidly fermentable, osmotically active, short-chain carbohydrates
  – Fructose, lactose, fructans, galactans, sugar alcohols
IBS - Treatment

### NonPrescription
- Fiber: Psyllium (IBS-C)
- Laxatives (IBS-C)
- Antidiarrheals (IBS-D)
- Probiotics
- Antispasmodics: Peppermint Oil
- Exercise
- Diet
  - Gluten free
  - Low in fermentable carbohydrates (wheat, some fruits and veggies, sorbitol, some dairy)

### Prescription
- Antidepressants
  - IBS-D: TCAs
  - IBS-C: SSRIs
- Antispasmodics
- Prosecretory Agents (IBS-C)
- Antibiotics: Rifaximin
  - Nonconstipated IBS

### Psychological / Behavioral Therapy

Pancreatitis

Pancreatitis

- Inflammatory process (caused by edema, necrosis or hemorrhage) in which pancreatic enzymes auto digest the gland
- Acute: within 1 week
  - Characterized by SIRS and/or organ failure
- Late: Greater than 1 week
  - Local complications: peripancreatic fluid collections, pancreatic and peripancreatic necrosis, pseudocysts, and walled-of necrosis.

Edematous    Necrotizing    Hemorrhagic

Pancreatitis

- Causes
  - Biliary disease (gallstones) – 40-70% of cases
    • Abdominal ultrasound to rule out
  - Alcoholism- 25-35% of cases
  - Hypertriglyceridemia – 1-4% of cases
  - Pancreatic cyst
  - Pancreatic tumor (age > 40)
  - Drugs
  - Trauma
  - ERCP – endoscopic retrograde cholangiopancreatography
  - Idiopathic
• Function
  – Secretes pancreatic juice for digestion of carbohydrates, proteins and fats
  – Secretes bicarbonate to neutralize chyme
  – Secretion of insulin and glucagon

Pancreatitis

• Pathophysiology
  – Normally inactive digestive enzymes become prematurely activated within the pancreas
  – Trypsin activation causes edema, necrosis and hemorrhage
  – Elastase activation causes hemorrhage
  – Kinin activation results in decreased peripheral vascular resistance, vasodilatation and increased vascular permeability
  – Inflammatory process causes necrosis of fat in pancreas and exudates with high albumin content leading to increased albumin and ascites
  – Hypovolemia -> shock
Pancreatitis: Presentation

- Abdominal pain
  - Epigastric or left upper quadrant
  - Radiates to back or flanks
  - Pain is dull, constant and severe
  - Pain described as dull, colicky
  - Tenderness or guarding
- Nausea, vomiting, dyspepsia, flatulence
- Low grade fever

- Tachycardia, hypotension
- Decreased or absent bowel sounds
- Hematemesis
- Abdominal distension
- Ascites
- Patient may to prefer to sit up or lie in fetal position
- Respiratory distress leading to ARDS

Grey Turner’s Sign
Grey Flank

Cullen’s Sign
Umbilical Discoloration
Pancreatitis

• Diagnosis based on presence of 2 or more of the following:
  – Abdominal pain consistent with the disease
  – Serum amylase and/or lipase greater than 3 times the upper limit of normal
  – Characteristic findings from abdominal imaging
    • Magnetic-enhanced computer tomography (CECT)
      – 90% sensitivity and specificity
    • Magnetic resonance imaging (MRI)
    • Reserved for when diagnosis is unclear or who fail to improve clinically within the 1st 48-72 hours after admission or to assess for complications

Laboratory Findings

• Elevated serum amylase
  – Peaks 4-24 hours after onset of symptoms
  – Returns to normal within 3-5 days
  – May be normal in alcohol induced pancreatitis and hypertriglyceridemia.
  – May be elevated in patients with decreased GFR, acute appendicitis, cholecystitis, intestinal obstruction / ischemia, peptic ulcer, or gynecological diseases.

• Elevated serum lipase
  – More specific to pancreatitis
  – Peak 24-48 hours; return to normal 5-7 days
  – In DM needs to be 3-5 times the upper limit of normal
  – May also be elevated with same processes as amylase
Pancreatitis

• Medical Management
  – Fluid resuscitation 250-500 ml / per hour
    • Crystalloids (Ringer’s lactate)
    • Caution in cardiovascular and renal disease
    • Goal of aggressive hydration is to reduce HCT /BUN while maintaining a normal creatinine
    • Reassess fluid need q6hours
  – Minimize pancreatic function
    • NPO
    • Mouth care with water and NS only
    • NG tube
    • IV or SC actreotide acetate (Sandostatin)
  – Correct metabolic alterations
  – Antibiotics
    • Only if infection present

Pancreatitis

• Dietary Considerations
  – NPO
  – Gradually reintroduce food when N/V and pain has resolved
  – Fats, cholesterol and triglycerides are kept to a minimum – NO ALCOHOL
  – If pain returns -> NPO
  – Enteral feedings
    • Nasogastric feeding tube or
    • Nasojejunal tube (after the ligament of Treitz)
  – TPN should be avoided but if needed – no lipids
Pancreatitis

• Pain Management
  – MS vs. Demerol
  – Historically: MS – May cause biliary colic and spasms of the sphincter of Oddi
  – Demerol does not
  – Recent studies do not support this
  – Position – knee to chest most comfortable

Pancreatitis

• Complications
  – Hypovolemic Shock
    • Leakage of protein and plasma causing third spacing.
  – Pulmonary
    • Atelectasis
    • Pleural effusion
    • Lung injury
    • ARDS
  – Infection
    • Infected necrosis, pneumonia, cholangitis, bacteremia, UTI etc.
Pancreatitis

- Cholecystitis
  - Cholechstectomy
- Infected pancreatic necrosis
  - Antibiotics
  - If illness continues after antibiotic - minimally invasive necrosectomy
  - If unstable then urgent surgery for debridement
- Pseudocysts
  - Collection of inflammatory debris, pancreatic secretions and necrotic tissue
  - Causes compression of portal vein or bile duct or rupture and peritonitis and sepsis
  - No treatment of asymptomatic
  - If symptomatic Surgical drainage or CT guided needle aspiration
- Pancreatic Abscess
  - Accumulation of pancreatic enzyme-rich fluid in peritoneal cavity
    - Surgical Drainage
Liver
The Liver

• Largest gland and solid organ in body
• Holds 13% of total blood supply at any moment
• Has approximately 500 functions
• Plays critical role in metabolism
  – Fuel management
  – Nitrogen excretion
  – Regulation water distribution between the blood and tissues
  – Detoxification of foreign substances.

The Liver

• Lobes of liver contain about 100,000 lobules
• Lobules serve as the structural and functional units of the liver
• Structures located between the lobules (portal triad):
  – branch of hepatic artery bringing oxygenated blood to the liver
  – branch of hepatic portal vein bringing nutrients from the intestines
  – bile duct to take bile away from the liver.
5 Main Liver Functions

- Maintenance of acid-base balance through the metabolism of lactate
- Detoxification
- Fuel Management
  - Fat, Carbo and protein Metabolism
- Glucose and lipid metabolism
- Protein synthesis (including immunoglobulins, clotting factors, and albumin)
- Phagocytic clearance of organisms and circulating debris

Liver Functions

- Secretion of bilirubin, bile salts, cholesterol, fatty acids, calcium and other electrolytes in to bile
- Storage of Amino Acids, glucose, vitamins, minerals and blood
- Conversion of complex carbohydrates to fats
- Conversion of stored glucose (glycogen) to glucose
- Conversion of amino acids and fats to glucose
- Conversion of amino acids to fatty acids and triglycerides
- Formation of phospholipids and cholesterol
- Formation of lipoproteins from triglycerides and peptides
- Conversion of amino acids to plasma proteins
Liver Functions

• Phagocytosis of old RBC’s
• Formation of clotting factors and heparin
• Conversion of ammonia to urea
• Conversion of creatine to creatinine
• Conversion of vitamin D3 to 25-hydroxycholecalciferol
• Detoxification of bacteria
• Biotransformation of drugs to active and / or inactive metabolites
• Deactivation of certain hormones
• Produces bile

Hepatitis

• A gastrointestinal disease featuring inflammation of the liver
• Causes:
  – Viral
  – Alcoholic induced
  – Drug induced
  – Metabolic disorders
  – Obstructive processes
  – Autoimmune processes
Key Definitions

Hepatic Failure: The liver’s inability to perform its key functions

- Acute Hepatic Failure: Coagulation abnormalities without encephalopathy
- Fulminant Hepatic Failure: Encephalopathy develops within certain time frame (8 weeks) or 2 weeks after development of jaundice from initial presentation

Key Definitions

- Hepatic Encephalopathy
  - The nonfunctioning liver permits toxins to circulate freely to the brain
  - Ammonia: main toxin causing encephalopathy
- Hepatorenal syndrome
  - Renal failure concurrent with liver failure
  - Kidneys abruptly stop functioning
Hepatotoxic Drugs

- Acetaminophen
- Halothane (general anesthetic – volatile liquid)
- Methyldopa – antihypertensive – aldomet
- Isoniazid (antituberculosis agent)
- Toxins: Amanita mushrooms, Industrial substances (chlorinated hydrocarbons)

Pathophysiology of Hepatic Dysfunction

- **Cirrhosis**
  - Liver cells progressively destroyed and replaced with fibrotic tissue
  - Distortion, twisting and constriction of central sections cause impedance of portal blood flow and portal hypertension

- **Fulminant Hepatitis**
  - Liver cells fail to regenerate and necrosis occurs
Pathophysiology of Hepatic Dysfunction

• Impairment of protein, fat and carbohydrate metabolism
• Inability to store vitamins
  – Responsible for absorption of fat soluble vitamins
• Inability to manufacture plasma proteins (i.e. albumin)
  – Decreased capillary oncotic pressure
  – Shifting of fluid in third space (edema, ascites, pleural effusions)
• Liver produces inadequate amounts of bile
  – Unable to conjugate bilirubin to make bile

Pathophysiology of Hepatic Dysfunction

• Inability to inactivate hormones
  – ↑ circulating aldosterone → retention of Na and water
• Inability to detoxify toxins
  – Drugs
  – Ammonia (by product of protein metabolism)
• Inability to remove bacteria
  – ↑ risk of infection / sepsis
• Inability to make clotting factors
  – ↑ coagulopathies
Hepatic Dysfunction Manifestations

• Portal Hypertension
  – Elevated portal vein pressure which obstructs or impedes blood flow through the portal venous system and vena cave
  – Esophageal varices
  – Spleenomegaly
  – Ascites

Signs and Symptoms of Liver Failure

• Anorexia
• Nausea / Vomiting
• Right upper Quadrant pain
• Abdominal fullness
• Weight loss / emaciation
• Excessive fat in stools (steatorrhea)
• Poor wound healing
• Apraxia
• JVD, Ascites
• Peripheral edema
• Distended superficial vessels on abdomen
• Esophageal varices
• Jaundice
• Abnormal bruising / bleeding
• Petechiae
Hepatic Failure
Diagnosis

- Elevated liver enzymes
- Elevated serum bilirubin
- Elevated serum ammonia
- Increased clotting times
- Decreased blood glucose
- Decreased platelets
- Decreased albumin
- Hyponatremia, hypokalemia

Treatment of Hepatic Failure

- Ascites Management
  - Promote ventilation – sit up
  - Paracentesis may be needed
  - Administer aldosterone antagonists (aldactone); may also need loop diuretic
  - Restrict sodium (500 mg / day)
  - Restrict Fluid (1500cc/day)
  - Shunt may be needed
Treatment of Liver Failure (continued)

- Maintain adequate circulating volume
  - Colloids not crystalloids (not albumin - contains protein)
  - No lactate
- H2 Receptor blockers / antacids to reduce risk of GI bleeding
- Electrolyte replacement therapy
  - Magnesium deficiency related to malnutrition
  - Observe K+ related to aldosterone
- Empty bowel of nitrogen material
  - Neomycin (kill bacteria that convert nitrogen to ammonia)
  - Lactulose (promote ammonia excretion)
  - Magnesium Citrate (removes nitrogen wastes)

Treatment of Liver Failure (Continued)

- Monitor Encephalopathy
  - Avoid sedatives
- Decrease Portal Hypertension
  - Beta blockers
- Nutritional Support
  - Low-protein, high carbohydrate diet
  - Fat soluble vitamins (A,D,E,K)
  - B vitamins, thiamine
- Assess for Bleeding
  - Vitamin K
  - Fresh Frozen Plasma
  - Platelets
Other Treatment Issues

• Esophogeal Varicies

• Alcohol withdrawal (chronic liver failure)

• Transplant

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