FLUID THERAPY

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OVERVIEW

- ✓HUMAN BODY IS ESSENTIALLY A SOLUTION OF DISSOLVED PARTICLES WITH THE CELL AS THE FUNDAMENTAL FUNCTIONING UNIT.

- ✓THESE CELLS WORK TOGETHER TO MAINTAIN THE INTERNAL BALANCE IN THE PRESENCE OF A CONSTANTLY CHANGING EXTERNAL ENVIRONMENT – TERMED HOMEOSTASIS

WATER

- PRIMARY CONSTITUENT OF THE HUMAN BODY
- ACCOUNTS FOR 60% OF TOTAL BODY WEIGHT IN KG
- VARIES DEPENDING ON BODY SIZE, SEX, AGE, METABOLIC ACTIVITY AND FAT CONTENT.

VITAL PHYSIOLOGIC PROCESS

- TEMPERATURE REGULATION
- CIRCULATION
- DIGESTION

- **CHEMICAL REACTIONS THAT OCCUR TO SUSTAIN LIFE REQUIRE WATER**

WATER BALANCE

- INTAKE
  - APPROXIMATELY 2500 ML/DAY
- OUTPUT
  - APPROXIMATELY 2500 ML/DAY

BODY FLUID DISTRIBUTION/COMPARTMENTS

- THE TWO MAJOR COMPARTMENTS ARE:
  - INTRACELLULAR
    - FLUID WITHIN THE CELLS, INCLUDING RBC'S AND WBC'S
    - LARGEST COMPARTMENT
    - CONTAINS 2/3 OF THE TOTAL BODY WEIGHT (APPROX. 25 LITERS IN A 70 KG ADULT)
  - EXTRA-CELLULAR
    - FLUID OUTSIDE THE CELLS
    - MAKE UP 1/3 TOTAL BODY WEIGHT
    - 3 COMPARTMENTS
      - INTRAVASCULAR – FLUID WITHIN THE BLOOD VESSEL (PLASMA)
      - INTERSTITIAL – FLUID THAT SURROUNDS THE CELLS
      - TRANS-CELLULAR – INCLUDES CSF, PERICARDIAL, PLEURAL, DIGESTIVE, ETC

BASIC CONCEPTS OF FLUID DYNAMICS/THERAPY

- SOLUTIONS = SOLUTES & SOLVENT
- OSMOLARITY = PARTICLE CONTENT IN BODY FLUIDS AND SERUM
  - NORMAL SERUM OSM = 275-295 MILLIOSMOLS
  - DETERMINANTS OF OSM = NA, BS, BUN
  - MEASUREMENTS OF OSM = $(NA \times 2) + \frac{BS}{18} + \frac{BUN}{3}$

REGULATION OF FLUID AND ELECTROLYTES

- THIRST
  - ACTS AND REGULATES THE ECF CONCENTRATION
- ADH –
  - HORMONE
  - STORED IN THE PITUITARY GLAND
  - ACTS ON THE DISTAL TUBULE COLLECTING DUCT IN THE KIDNEY TO INCREASE REABSORPTION OF WATER
- ALDOSTERONE
  - HORMONE RELEASED BY THE ADRENAL CORTEX
  - PROMOTES NA REABSORPTION (WATER FOLLOWS PASSIVELY)

ASSESSMENT OF FLUID STATUS

- ASSESSMENT OF BLOOD VOLUME: B/P (EXTRA-CELLULAR)
  - CVP
  - PAWP
  - PERIPHERAL PULSES
  - NECK VEINS
  - URINE OUTPUT
  - SPECIFIC GRAVITY

- ASSESSMENT OF INTERSTITIAL FLUID SPACE
  - SKIN
  - RESPIRATORY STATUS
  - THIRST
  - EDEMA

- LABORATORY INFORMATION:
  - SERUM NA
  - SERUM OSM
  - HCT
  - TOTAL PROTEIN

ALTERATIONS IN FLUID BALANCE

- HYPOVOLEMIA
  - **CAUSES**
    - PROLONGED VOMITING
    - DIAPHORESIS
    - PROLONGED GI SUCTION
    - DKA
    - DIURETICS
    - "THIRD SPACE"
    - CARDIAC ARREST
    - DISEASES AFFECTING ALDOSTERONE

– **Signs and Symptoms**
- Decreased B/P
- Tachycardia
- Postural changes
- Weak arterial pulses
- Flat neck veins
- Decreased CVP/PAWP
- Decreased urine output
- Increased Specific Gravity
- Dry mucous Membranes
- Poor skin turgor
- Weight loss
- I<O

- TREATMENT:
  - REPLACE NA
  - REPLACE WATER

- HYPERVOLEMIA
  - **CAUSES**
    - EXCESSIVE ISOTONIC N/S INFUSIONS
    - CHRONIC RENAL FAILURE
    - LIVER DISEASE
    - PARTIAL HYPERTENSION
    - MALNUTRITION WITH PROTEIN DEFICIENCY
    - CHF WITH DECREASED CO

    (REFLEX VASOCONSTRICTION)

- **Signs and Symptoms**
  - Increased B/P
  - Tachycardia
  - Pulmonary Congestion
  - Orthopena
  - Crackles/Wheezes
  - Full, bounding pulses
  - Rapid venous filling
  - Neck vein distention
  - increased PAWP/CVP
  - Decreased Specific Gravity
  - Dependent Edema
  - Urine output varies

- TREATMENT
  - RID BODY OF ECESSIVE NA
  - RID BODY OF EXCESSIVE NA AND WATER

- NORMOVOLEMIA

  INTAKE + OUTPUT

TONICITY

- ISOTONIC
  - SAME OSM AS SERUM AND BODY FLUIDS
  - EXPANDS THE IV COMPARTMENT WITHOUT EFFECTING THE IC/IS COMPARTMENTS

  EXAMPLES: LR, NS, D5W

- HYPOTONIC
  - LOWERS SERUM OSM AND CAUSES FLUID SHIFTS OF IV INTO IC/IS COMPARTMENTS
  - HYDRATES THE CELLS

  EXAMPLES: 0.45% NS, 0.33% NS

- HYPERTONIC
  - RAISES SERUM OSM
  - SHIFTS (PULLS) FLUID FROM IC/IS COMPARTMENTS INTO IV COMPARTMENT
  - SHRINKS THE CELLS

  EXAMPLES: $D_5$1/2 NS, D_5NW, D_5RL

FLUID REPLACEMENTS

- CRYSTALLOIDS
  - CAPABLE OF PASSING THRU A SEMI-PERMEABLE MEMBRANE

  EXAMPLES: D_5W, NS, RL
  $D_5$1/2NS, $D_5$1/4NS

- COLLOIDS
  - NOT CAPABLE OF PASSING THROUGH A SEMI-PERMEABLE MEMBRANE

EXAMPLES: DEXTRAN, ALBUMIN, HETASTARCH

- BLOOD PRODUCTS
  - SAME AS A COLLOID

EXAMPLES: PRBC'S, WHOLE BLOOD, COGULATION FACTORS, (FFP, CRYO PLATLETS)

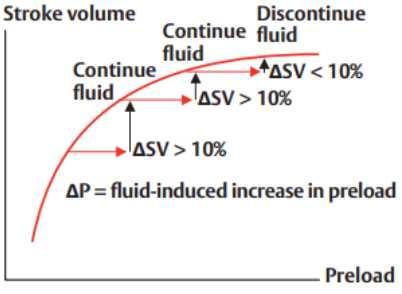
VOLUME THERAPY

- PURPOSE
  - INCREASE FLOW
  - MONITOR CENTRAL VENOUS PRESSURE

  - NO CHANGE – HYPOVOLEMIA
  - MODERATE CHANGE – NORMOVOLEMIA
  - GREAT CHANGE - HYPERVOLEMIA

FLUID CHALLENGE

Frank-Starling curve
Relationship between preload status and fluid-induced increases in stroke volume.



Increasing SV with fluid until the plateau of the Frank-Starling curve is reached has been shown to improve the outcome of high-risk surgery patients.

Lopes MR, et al. Goal-directed fluid management based on pulse pressure variation monitoring during high-risk surgery: a pilot randomized controlled trial. Critical Care. 2007 Oct;11(5):R100.

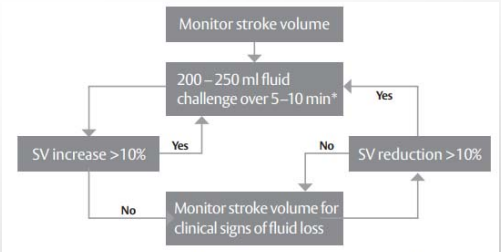
- CENTRAL VENOUS PRESSURE (CVP)

  7-3 RULE: IF AFTER 50 – 200CC FLUID

  - CVP INCREASES < 3 = CAN GO
  - CVP STAYS 3-7 = WAIT
  - CVP INCREASES > 7 = STOP

STROKE VOLUME

- STROKE VOLUME OPTIMIZATION
  - % CHANGE IN STROKE VOLUME IS A SENSITIVE METHOD FOR ASSESSING PRELOAD RESPONSIVENESS ON ALL PATIENTS



Oesophageal Doppler-guided fluid management during major surgery: reducing postoperative complications and bed days. NHS Technology Adoption Centre. January 2012.

*A passive leg raising maneuver over 1-2 min. can also be used as a fluid challenge *Monnet X, Teboul JL. Passive Leg Raising. Intensive Care Med. 2008 Apr; 34 (4): 659-63.*

FLUID RESUSCITATION

SOLUTION	DESCRIPTION	USES	ADVANTAGES	DISADVANTAGES
D5W 5% Dextrose in Water	50 Grams of Dextrose/L 200 calories/L Osmolality 280 mOSM/L	*Replace fluid when pt is dehydrated *Keep- open or flush	Rehydrate when serum Na and Cl are concentrated	*Excess can dilute electrolytes and plasma proteins → electrolyte imbalance, interstitial & cellular edema *Do not se in pts with head injury
Hypotonic Saline 0.45% NaCl	Sodium 77 mEq/L Chloride 77 mEq/L Osmolality 154 mOsm/L	Replace volume	Rehydrate without increasing serum Na or Cl	Interstitial and intracellular edema
Normal Saline 0.9% NaCl	Na = 154 mEq/L Cl = 154 mEq/L Osmolality 308 mOsm/L pH = 5.0	*Replace loss of body fluid (eg. bowel obstruction, peritonitis, GI suction) when red cell mass is adequate	Increase plasma volume without changing Na concentration or serum osmolality	*Dilutes RBC's & Plasma proteins. *Lowers osmotic pressure which can result in ulmonary edema *Can cause hypernatremia and hyperchloremic metabolic acidosis
Ringers Lactate (RL)	Na=130 mEq/L Cl=109 mEq/L K=4 mEq/l Ca = 4 mEq/L Lactate = 27 mEq/L Osmolality 274 mOsm/L pH = 6.5	Early stages of Hemorrhagic Shock	Isotonic, cheap, electrolytes are balanced to interstitial fluid, K+ and Ca++ promote myocardial contractility, Ca aids clotting lactate is converted by the liver to bicarbonate (buffer), doesn't increase serum Na or Cl	Given too fast → increases Na→ decreases K, dsrhythmias, and seizures

SOLUTION	DESCRIPTION	USES	ADVANTAGES	DISADVANTAGES
Hypertonic Saline 3%	Sodium 512 mEq/L Chloride 513 mEq/L Osmolality 1026 mOsm/L	*Increase plasma volume *Increase serum sodium	Hypertonic takes less to resuscitata	Given too fast → increases Na→ decreases K, dsrrhythmias, and seizures
Hypertonic Saline 5%	Sodium 855 mEq/L Chloride 855 mEq/L Osmlality 1710 mOsm/L	*Increase plasma volume *Increase serum sodium	Hypertonic takes less to resuscitate	
Albumin 5%	Albumin 50 g/L in NS Osmolality 300 mOsm/L	Rapidly expand volume Mobilize interstitial (pulmonary) edema	Increases osmotic pressure *stays intravascular longer *takes less too resuscitate	*Expensive *Can increase edema if patient has capillary leak syndrome *Curtails albumin synthesis
Albumin 25% (salt-poor)	Albumin 240 grams/L Osmolality 1500 mOsm/L	Increase plasma colloid osmotic pressure for patients with hypoproteinemia	*Rarely sociated with allergic reactions or transmission of hepatitis	*Risk of volume overload *Limit rate to < 4 ml/min

SOLUTION	DESCRIPTION	USES	ADVANTAGES	DISADVANTAGES
Dextran 40 (Rheomacrodex)	Synthetic colloid, with glucose polysaccharides in NS or D5W Molecular Weight = 40,000	Rapidly expand volume Prevent formation of microthrombi	Volume expands 100% *Can prevent microemboli by decreasing platelet adhesiveness Associated with les allergic reaction than Dextran – 70	*Anticoagulant properties *May cause allergic reaction in low flow states *70% is excreted unchanged in urine *Can cause osmotic Nephrosis and ATN *Expensive *Alters urine Specific Gravity and mOsm
Dextran 70 (Rhemacrodex)	Molecular weight of 70,000	Rapidly expand volume Mobilize interstitial edema	*Has identical weight to albumin, but much larger molecules *Increases plasma volume up to 24 hours	Higher incidence of allergic reactions
Hespan	Synthetic colloid, made from corn Molecular weight similar to albumin (70,000) Sodium 154 mEq/L Chloride 154 mEq/L Osmolslity 310 mOsm/L	Rapidly expand volume Mobilize interstitial edema	Volume expands 150% *Effects last 24 – 36 hrs *Cheaper and has lower risk for allergic reaction than Dextran or Albumin	*May increase serum amlyase, serum glucose, decrease factor VIII *May prolong PT, PTT and decrease platelets *Must have good renal function for renal elimination *Can cause circulatory overload and dilute RBC"s
Plasmanate (Plasma protein fraction)	5% solution of human plasma proteins in NS Albumin 44 g/L Osmolality 290 mOsm/L	Increase serum colloid osmotic pressure	*Like 5% Albumin- has low risk of transmitting Hepatitis	*Risk of allergic reaction *Hypotension if given > 10ml/min *Expensive

Sepsis

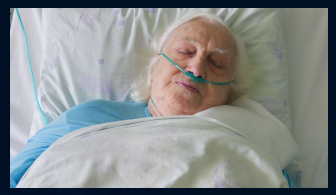
Surviving Sepsis Campaign

KELLY URBAN, PHD, MED, RN, CCRN-K, TCRN

1

Case Study

- 80 year old female with ESRD, DM, CHF, CAD (CABG 10 years ago), Paroxysmal A-fib, PVD, ITP, and recent COVID-19 infection
- Arrived to ED from SNF (rehab from recent hospitalization) due to fever and chills
- Initial VS :
  - Temp – 102.6
  - HR – 120
  - BP – 73/40
  - RR - 30



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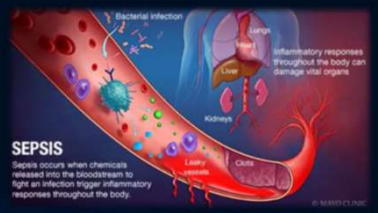
What is Sepsis?

- In a **NORMAL** response to an infection, the inflammatory and coagulation response is localized to the infection site as the immune system attacks the pathogen, eliminating it from the body.



3

A Worldwide Problem

Sepsis is a major, world-wide health care problem

- Affecting an estimated 30 million adults and children each year resulting in potentially six million deaths annually
- Accounts for ~20 percent of U.S. hospital admissions but is a factor in over 50 percent of U.S. hospital deaths
- Is the leading cause of U.S. hospital readmissions (20 percent)

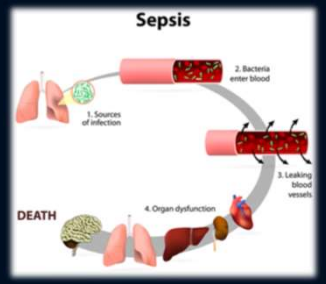
Although mortality has decreased in the last decade, it remains over 25 percent → 1 in 4

4

Sepsis

The inflammatory and coagulation response is rapid and widespread, causing a dysregulated response

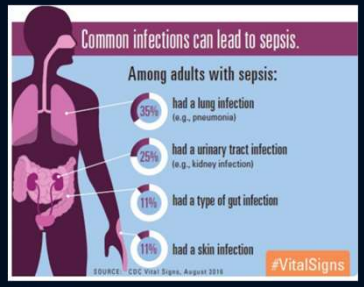
- The body's reaction to the pathogen may overwhelm all of the body's systems
- Immune systems that are too strong or too weak are unable to respond effectively to pathogen invasion



5

Causes of Sepsis

- Bacterial infections are the most common
- Fungal, parasitic or viral infections can also cause sepsis
- The infection can originate from anywhere in the body and can cause organ damage to any system of the body
- Unknown (1/3 of all sepsis cases)



6

Who is at Risk?

Host Factors

- Age, gender, genetics, comorbidities
- Elderly account for 60-85 percent of all cases of severe sepsis

Immunosuppression

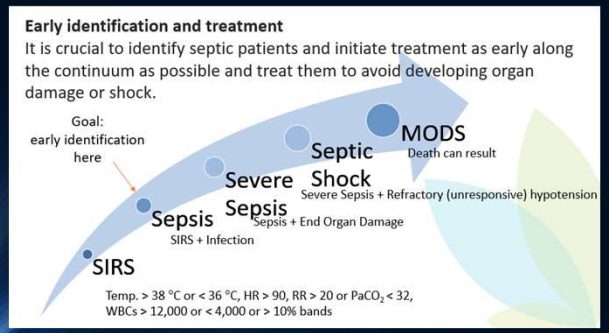
- Disease related, medications related

Exposure risk

- Community acquired: pneumonia, urinary, wounds, trauma
- Health care acquired: invasive devices, secondary infections and skin breakdown

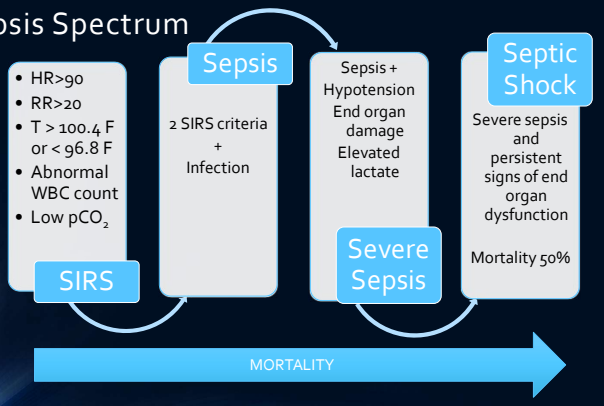
7

Sepsis Progression



8

Sepsis Spectrum



9

SIRS

SIRS is a nonspecific inflammatory response to an insult that results in activation of the immune system. This inflammatory response is the body's way of attempting to maintain homeostasis.

SIRS is defined as two or more of the following variables:

- Body temperature < 36°C or > 38°C
- Heart rate > 90 beats per minute
- WBC > 12,000/mm³ or < 4,000/mm³ or > 10% bands
- Respiratory rate > 20 breaths per minute or $PaCO_2$ < 32mmHg

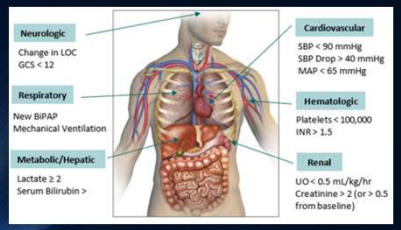
SIRS + infection = sepsis

10

Severe Sepsis

Sepsis + new organ dysfunction = severe sepsis

Organ dysfunction is defined as a condition in which an organ does not function as expected.



Acute Organ Dysfunction Related to Sepsis

11

Septic Shock

Severe sepsis + refractory hypotension OR lactate ≥ 4 mmol/L = septic shock

- Septic shock is a distributive shock
- Cytokine release leads to a large-scale inflammatory response
  - Massive vasodilation
  - Increased capillary permeability
  - Decreased systemic vascular resistance
  - Blood clots form in the microvasculature
  - Hypotension reduces tissue perfusion causing tissue hypoxia

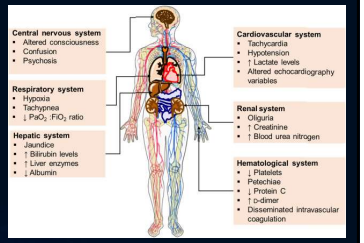
12

Multi-Organ Dysfunction Syndrome (MODS)

MODS is altered organ function in an acutely ill patient requiring medical intervention to achieve homeostasis. Can be the end result of septic shock.

Sepsis-related organ dysfunction → No organ system is immune

- Respiratory failure
- Liver failure
- Kidney failure
- Heart failure
- Gut permeability
- DIC (disseminated intravascular coagulation)
- Altered mental status
- Brain death



13

Surviving Sepsis 2021 Adult Sepsis Guidelines

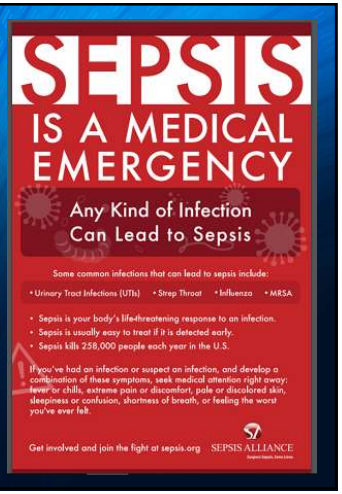
- SOFA
  - 5 point scale to rate presence and severity of indicators of organ failure in 6 categories
    - Respiration
    - Coagulation
    - Liver function
    - Cardiovascular function
    - CNS function
    - Renal function

qSOFA: RR ≥ 22/min, GCS, BP ≤ 100 mmHg

14

Sepsis and septic shock are medical emergencies!

Treatment should begin immediately!



15

Case Study continued

- Code Sepsis Initiated due to VS

- Initial Diagnosis
  - CXR – bilateral patchy opacities
  - Blood Cultures
  - Labs:
    - WBC – 26.99
    - Lactate – 2.5
    - Procalcitonin – 0.57

80 year old female with ESRD, DM, CHF, CAD (CABG 10 years ago), Paroxysmal A-fib, PVD, ITP, and recent COVID-19 infection
Arrived to ED from SNF (rehab from recent hospitalization) due to fever and chills
Initial VS :
Temp – 102.6
HR – 120
BP – 73/40
RR - 30

16

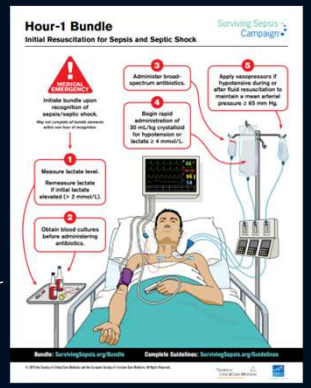
Treatment – Goal Directed Therapy

- Fluid Resuscitation
- Intensive antimicrobial therapy
- Vasopressors (MAP)
- Transfusion for bleeding complications
- Intensive patient monitoring

17

Hour-1 Bundle

- Measure lactate level
- Obtain blood cultures before administering antibiotics
- Administer broad-spectrum antibiotics
- Begin rapid administration of 30 ml/kg crystalloid for hypotension or lactate ≥ 4 mmol/L
- Apply vasopressors if hypotensive during or after fluid resuscitation to maintain a mean arterial pressure ≥ 65 mmHg



18

Lactate

- With sepsis, lactate is viewed as a marker of global tissue perfusion.
- Lactate has some predictive use:
  - Sustained > 6 hours, an elevated lactate foreshadows increased mortality
  - Mortality increases as lactate levels increase

Lactate Level	Mortality
0-2.5 mmol/L	4.9 percent mortality
2.5-4.0 mmol/L	9.0 percent mortality
> 4.0 mmol/L	28.4 percent mortality

Nguyen, et al., 2004; Shapiro, et al., 2005)

19

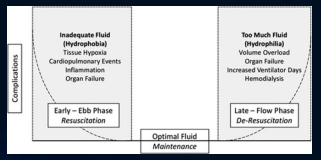
Fluid Administration Recommendations

- Begin rapid administration of 30 ml/kg crystalloid for hypotension or lactate ≥ 4 mmol/L
- Additional fluid should be given as small boluses
- To avoid over/under resuscitation, fluid administration (beyond initial resuscitation) should be guided by careful assessment of intravascular volume and organ perfusion
  - HR, CVP, SBP are poor indicators of fluid status
  - Dynamic measures should be used (passive leg raising combined with CO measurement, fluid challenges against SV, systolic or pulse pressure, and increases of SV in response to changes in intrathoracic pressure

20

Fluid Administration Recommendations

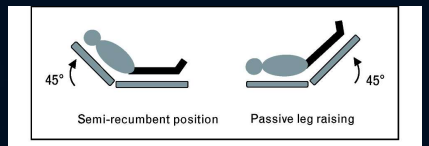
- Crystalloids are 1st-line fluid for resuscitation
  - Balanced crystalloids instead of normal saline
- Albumin can be used in patients who received large volumes of crystalloids
- Do not use starches or gelatin for resuscitation



21

Hemodynamic Monitoring

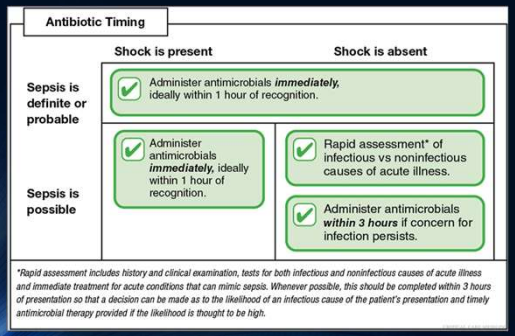
- Dynamic assessments to guide fluid administration may improve outcomes for septic shock



The passive leg-raising test consists of measuring the hemodynamic effects of a leg elevation up to 45°. A simple way to perform the postural maneuver is to transfer the patient from the semi-recumbent posture to the passive leg-raising position by using the automatic motion of the bed.

22

Antibiotic Timing



23

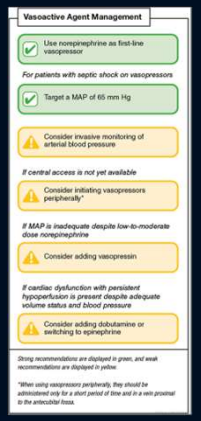
Antimicrobial Recommendations

- If at high risk of MRSA, recommendation to use empiric antimicrobials with MRSA coverage
- If at high risk for multidrug resistant organisms, suggest using 2 gram-negative agents for empiric treatment
- If at high risk for fungal infection, suggest using empiric antifungal therapy
- There is NO recommendation for use of antiviral agents

24

Vasoactive Agent Management

- An initial target MAP of 65 mmHg
- Norepinephrine is 1st-line agent
- Vasopressin is 2nd-line agent
- Epinephrine can be used if norepinephrine and vasopressin are inadequate
- May also consider using Dobutamine if suspect cardiac dysfunction with persistent hypoperfusion



25

Case Study continued

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Arrived to ED from SNF (rehab from recent hospitalization) due to fever and chills
Initial VS :
Temp – 102.6
HR – 120
BP – 73/40
RR - 30

- Initial Treatment
  - Blood Cultures prior to Antibiotic Administration
    - Vancomycin
    - Cefepime
  - LR 500 ml bolus (due to ESRD)
  - Acetaminophen for fever
  - Norepinephrine infusion (initially 4 mcg/min)
  - Oxygen – 2L nasal cannula

Patient was admitted to MICU

26

Surviving Sepsis Guidelines 2021

- Admission to ICU within 6 hours
- Invasive blood pressure monitoring when available
- Vasopressors can be started peripherally rather than delaying initiation until central venous access is obtained

27

Additional Therapies

- IV corticosteroids should be considered if there is an ongoing requirement for vasopressor therapy
  - 200 mg IV/day (typically 5-7 days)
- Restrictive transfusion strategy
- If high risk for GI bleeding, use stress ulcer prophylaxis
- Pharmacologic VTE prophylaxis is recommended unless a contraindication (LMWH)
- Recommend against using mechanical VTE prophylaxis in addition to pharmacologic prophylaxis

28

Additional Therapies

- Initiation of insulin therapy if blood glucose ≥ 180 mg/dL
- Sodium Bicarbonate therapy only if in a severe metabolic acidemia (pH ≤ 7.2) and AKI
- Early enteral nutrition (within 72 hours)

29

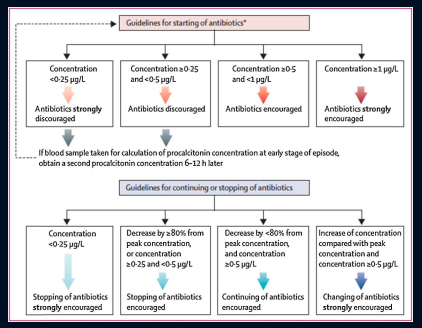
Additional Therapies

- Appropriate Source Control as soon as possible following initial resuscitation
  - Drainage of an abscess
  - Debriding infected necrotic tissue
  - Removal of a potentially infected device
  - Definitive control of a source of ongoing microbial contamination

30

Procalcitonin

- Biomarker that is undetectable in health states
- Rises rapidly in response to pro-inflammatory stimuli, especially bacteria



31

Case Study continued

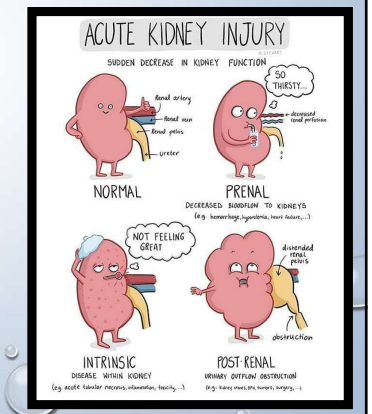
- Continued treatment with narrowing of antibiotics to cefepime for duration of treatment
- Norepinephrine weaned off 1st due to atrial fib
- Vasopressin weaned off on hospital day 3
- Patient transferred to hospitalist unit
- Discharged back to SNF on hospital day 10
  - Discharge Information
    - WBC 10.26
    - VS :
      - Temp – 97.3
      - HR – 109
      - BP – 105/61
      - RR - 20

80 year old female with ESRD, DM, CHF, CAD (CABG 10 years ago), Paroxysmal A-fib, PVD, ITP, and recent COVID-19 infection
Arrived to ED from SNF (rehab from recent hospitalization) due to fever and chills
Initial VS :
Temp – 102.6
HR – 120
BP – 73/40
RR - 30

32

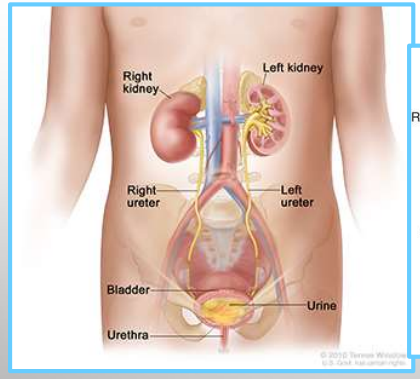
ACUTE KIDNEY INJURY

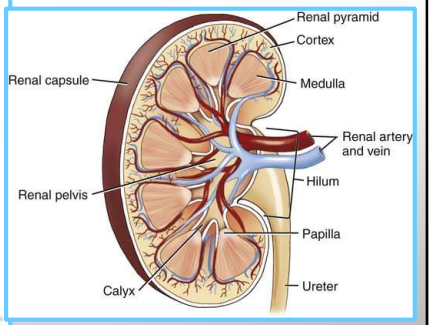
Kelly Urban



1

ANATOMY





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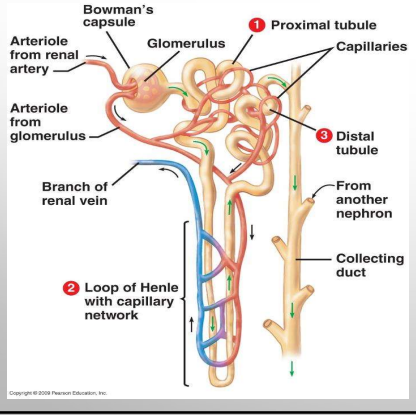
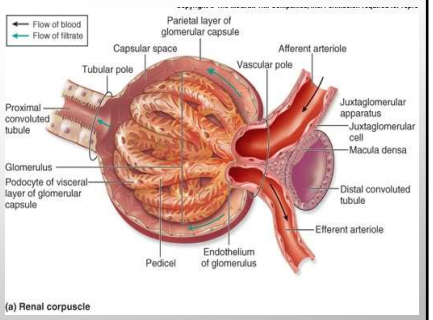
MICROSCOPIC ANATOMY





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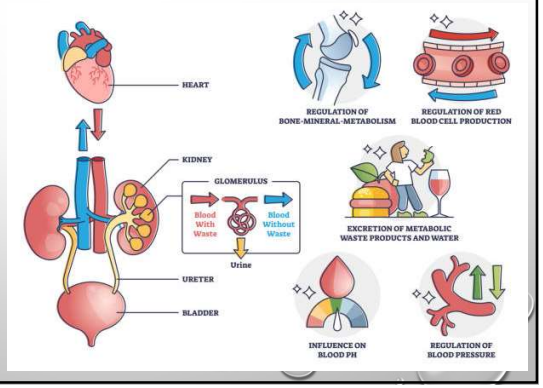
KIDNEY FUNCTIONS

- REGULATORY
- HORMONAL



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KIDNEY FUNCTIONS

REGULATORY FUNCTIONS

- REMOVES TOXINS FROM BODY THROUGH ELIMINATION
- FLUID AND ELECTROLYTE BALANCE
  - GLOMERULAR FILTRATION, TUBULAR REABSORPTION OR SECRETION
- ACID/BASE BALANCE

HORMONAL FUNCTIONS

- BLOOD PRESSURE REGULATION
  - RENIN, ANGIOTENSIN, ALDOSTERONE
  - PROSTAGLANDINS, BRADYKININS
- ERYTHROPOIETIN PRODUCTION
  - STIMULATES MARROW FOR RBC PRODUCTION
- VITAMIN D ACTIVATION

5

ACUTE KIDNEY INJURY (AKI)

- SUDDEN CESSATION OF RENAL FUNCTION → HOURS OR DAYS
- RESULTS IN INABILITY TO MAINTAIN PROPER FLUID/ELECTROLYTE BALANCE
- GENERALLY CHARACTERIZED BY:
  - RAPID ONSET OF DECREASING UOP (OLIGURIA), INCREASING BUN AND CREATININE (AZOTEMIA), DEVELOPING ELECTROLYTE IMBALANCES (ESPECIALLY HYPERKALEMIA)
- ASSOCIATED WITH MORBIDITY AND MORTALITY IF NOT DETECTED QUICKLY
  - UP TO 30% OF ICU PATIENTS MAY DEVELOP AKI
  - ASSOCIATED WITH MORTALITY RATE OF 60% (IN-HOSPITAL UP TO 1 YEAR)

6

PATHOPHYSIOLOGY IN RENAL FAILURE

Functions of the Kidney	***Manifestation in Renal Failure***
Excretes Water	*Fluid Overload* *Hemodilution*
Excretes Potassium	*HYPERkalemia*
Excretes Hydrogen ions (H+) Phosphate salts Titratable acids Reabsorbs and generates Bicarb	*Metabolic Acidosis*

7

PATHOPHYSIOLOGY IN RENAL FAILURE

Functions of the Kidney	***Manifestation in RF***
Excretes Metabolic wastes (Bun, creatinine, +200	*Azotemia or Uremia (organ irritation and disfunction)*
Converts Vitamin D	*Hypocalcemia* *Hyperphosphatemia*
Makes Erythropoietin	*Anemia: ↓ RBC production*
Excretes Renin	*Hypertension*
Excretes (secretes) medication	*↑Length of action* *↑Half-life* *Dosages need to be ↓*

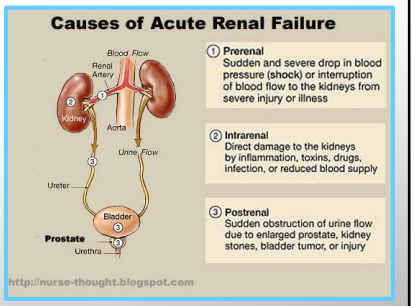
8

AKI RISK FACTORS

PRERENAL
- HYPOPERFUSION TO THE KIDNEY

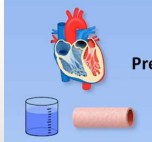
INTRARENAL
- DAMAGE TO THE RENAL TISSUE

POSTRENAL
- FROM OBSTRUCTION TO THE PASSAGE OF URINE

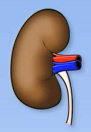


9

Acute Kidney Injury (AKI)
Prerenal vs. Intrarenal vs. Postrenal Paradigm



Prerenal
- Dehydration*
- Heart failure (a.k.a. cardiorenal syndrome)
- Liver failure (a.k.a. hepatorenal syndrome)



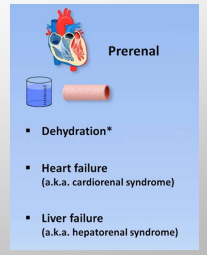
Intrarenal
- Intrinsic renovascular disease
  - Hypertensive emergency
  - Small vessel vasculitis
  - TTP / HUS
- Glomerular disease
  - Post-infectious glomerulonephritis
- Tubulointerstitial disease
  - Acute tubular necrosis (ATN)* (causes: sepsis, meds, contrast, rhabdo, prolonged prerenal AKI)
  - Acute interstitial nephritis (AIN)

Postrenal
- Ureteral obstruction (usually requires bilateral obstruction)
- Neurogenic bladder
- Urinary tract infection
- Medications
- Benign prostatic hypertrophy (BPH)

10

PRERENAL AKI

- MOST COMMON TYPE OF AKI
- INITIAL INSULT INTERFERES WITH BLOOD FLOW TO KIDNEYS
- **COMMON CAUSES**
  - SHOCK
  - HEART FAILURE
  - HYPOVOLEMIA
  - STENOSIS OF RENAL ARTERY
  - DISSECTING ABDOMINAL ANEURYSM



11

PRE-RENAL FAILURE

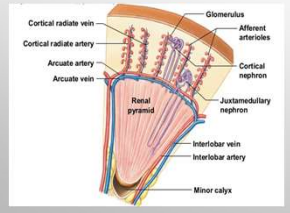
- SIGNS AND SYMPTOMS
  - SPECIFIC GRAVITY > 1.020
    - URINE OSMOLALITY > THEN SERUM OSMOLALITY
  - BUN ELEVATED > CREATININE (> 10:1 RATIO)
  - DECREASED URINE OUTPUT
  - URINE SODIUM < 10
- TREATMENT
  - FIX THE CAUSE

12

INTRARENAL AKI

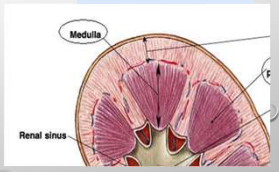
CORTICAL

- GLOMERULI OBSTRUCTION



MEDULLARY

- NEPHROTOXIC
- ISCHEMIC



13

INTRA-RENAL: CORTICAL

- OBSTRUCTION OF THE GLOMERULI
  - INFECTION
  - EDEMA
  - CELLULAR DEBRIS
  - SPECIFIC CAUSES
    - INFECTIONS
    - GOODPASTURE'S SYNDROME
    - SEVERE HYPERCALCEMIA
    - SYSTEMIC LUPUS ERYTHEMATOSUS
    - MALIGNANT HYPERTENSION
    - RABDOMYOLYSIS

14

MEDULLARY: ACUTE TUBULAR NECROSIS

- MOST COMMON CAUSE OF ARF
- TWO TYPES:
  - NEPHROTOXIC INJURY
  - ISCHEMIC INJURY

15

NEPHROTOXIC INJURY

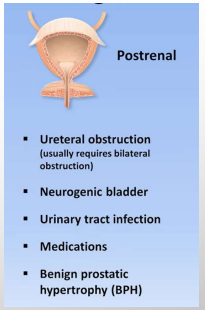
- DAMAGES EPITHELIAL CELLULAR LAYER
- CAUSES
  - ANTIBIOTICS
  - INFLAMMATORY MEDIATORS
  - X-RAY CONTRAST MATERIAL
  - ANESTHETICS
  - ANTI-INFLAMMATORIES (NSAIDS)
  - CHEMOTHERAPEUTIC AGENTS
  - PESTICIDES
  - FUNGICIDES
  - ORGANIC SOLVENTS
  - HEAVY METALS (LEAD, MERCURY, URANIUM)

16

POSTRENAL AKI

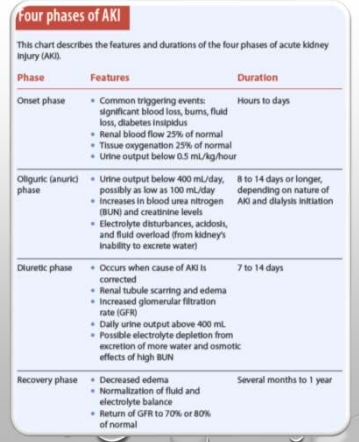
OBSTRUCTION IN OUTFLOW OF URINE POST-TUBULES CAUSES INJURY FROM BACKUP PRESSURES

- MECHANICAL CAUSES
  - CLOT, CALCULI, TUMOR, PROSTATE HYPERTROPHY OR CANCER, STRICTURE
- FUNCTIONAL CAUSES
  - DM NEUROPATHY, NEUROGENIC BLADDER DUE TO SCI



17

AKI STAGES



Four phases of AKI

This chart describes the features and durations of the four phases of acute kidney injury (AKI).

Phase	Features	Duration
Onset phase	• Common triggering events: significant blood loss, burns, fluid loss, diabetes insipidus • Renal blood flow 25% of normal • Tissue oxygenation 25% of normal • Urine output below 0.5 mL/kg/hour	Hours to days
Oliguric (anuric) phase	• Urine output below 400 mL/day, possibly as low as 100 mL/day • Increases in blood urea nitrogen (BUN) and creatinine levels • Electrolyte disturbances, acidosis, and fluid overload (from kidney's inability to excrete water)	8 to 14 days or longer, depending on nature of AKI and dialysis initiation
Diuretic phase	• Occurs when cause of AKI is corrected • Renal tubule scarring and edema • Increased glomerular filtration rate (GFR) • Daily urine output above 400 mL • Possible electrolyte depletion from excretion of more water and osmotic effects of high BUN	7 to 14 days
Recovery phase	• Decreased edema • Normalization of fluid and electrolyte balance • Return of GFR to 70% or 80% of normal	Several months to 1 year

18

AKI ASSESSMENT

HISTORY AND RISK FACTORS

- PAST: RENAL PROBLEMS, HTN, DM, MEDICATION USAGE
- RECENT: EXPOSURE TO TOXINS, AGENTS; HYPOTENSION
- PRESENT: BLOOD LOSS, SEPSIS, INFECTION, TUMOR, ETC.

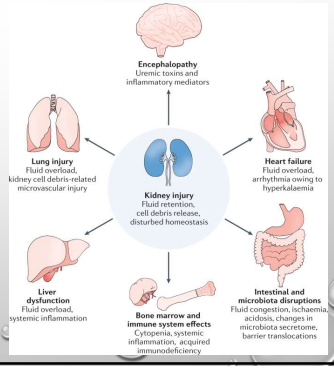
PHYSICAL ASSESSMENT

- MULTI-SYSTEM ASSESSMENT NECESSARY
- AKI AFFECTS MOST/ALL BODY SYSTEMS → UREMIA (CLINICAL SYNDROME)
  - HIGH LEVELS OF BUN CAUSING MULTI-SYSTEM COMPLICATIONS

19

AKI CLINICAL MANIFESTATIONS

- UREMIA
- CV/PULMONARY EFFECTS
- NEURO EFFECTS
- ACID/BASE IMBALANCE
  - METABOLIC ACIDOSIS



20

AKI ASSESSMENT APPROACH

Procedure	Rationale
Ultrasound	Obstruction?
Hemodynamic Evaluation	Hypotension in past few days as cause?
Drug History	Any medication that could have caused the AKI?
Urine Analysis	Acute Glomerulonephritis Interstitial nephritis Urine osmolality • < 350 mosmol/kg – ATN • > 500 mosmol/kg – pre-renal AKI
Labs (blood)	
Imaging	
Renal Biopsy	

21

AKI TREATMENT/NURSING CARE

TREATMENT
- MEDICATIONS
- HEMODYNAMIC MONITORING
- NUTRITION

NURSING CARE
- DAILY WEIGHTS
- FLUID RESTRICTION
- PATIENT/FAMILY EDUCATION

22

AKI PHARMACOLOGY

FLUIDS FOR PATIENTS WITHOUT FLUID OVERLOAD
- FLUID CHALLENGE TO PROMOTE KIDNEY PERFUSION
- REPLACEMENT OF VOLUME LOSS W/ PRERENAL INJURY

DIURETICS
- INCREASE URINE OUTPUT; DECREASES RETAINED FLUID
- HOW DO YOU MONITOR SUCCESS OF DIURESIS?

CALCIUM CHANNEL BLOCKERS
- IMPROVE RENAL BLOOD FLOW AND GFR

TREAT HYPERKALEMIA IF PRESENT
- INSULIN/D5W; CALCIUM; KAYEXALATE (GI TRACT), RRT

TREAT ACIDOSIS IF PRESENT AND SEVERE
- BICARBONATE , IVF, OR DIALYSIS

23

AKI HEMODYNAMICS

- MONITOR MAP
  - ENSURE MAP IS 65 OR GREATER
  - IMPORTANT FOR PERFUSION TO RENAL TISSUE
- MONITOR CVP
  - WILL BE ELEVATED IF IN FLUID OVERLOAD
  - MONITORING WILL HELP TO KNOW IF INTERVENTIONS ARE WORKING PROPERLY
- MANAGE HTN

24

AKI NUTRITION

- RESTRICT POTASSIUM, SODIUM – DEPENDENT ON UOP
- RESTRICT FLUID
- DIETICIAN WILL CALCULATE APPROPRIATE LEVEL OF PROTEIN IN DIET
- 20-30 CALS/KG/DAY OF BODY WEIGHT FOR ENERGY, DEMANDS OF STRESS
- POSSIBLE ENTERAL FEED RENAL FORMULAS OR TPN

25

AKI NURSING CONSIDERATIONS

MANAGE FLUID OVERLOAD

- CARDIOVASCULAR AND RESPIRATORY SUPPORT
  - O2, DIURETICS, I/O, **DAILY WEIGHT**
  - **STRICT FLUID RESTRICTIONS = TYPICALLY 500-1000 ML/DAY**
- MONITOR CHEMISTRY LAB VALUES
  - POTASSIUM
  - PHOSPHORUS
  - CALCIUM
  - SODIUM

26

RENAL REPLACEMENT THERAPY (RRT - DIALYSIS)

- INTERMITTENT RRT (HEMODIALYSIS)
- CONTINUOUS RRT (CRRT)
  - FOR HEMODYNAMICALLY UNSTABLE PATIENTS
- PERITONEAL DIALYSIS (PD)

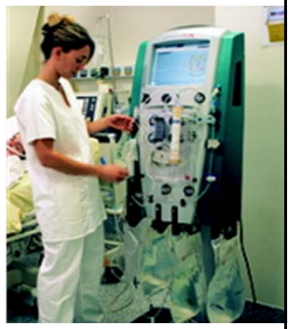
27

Basic Principles of CRRT

1

Continuous Renal Replacement Therapy (CRRT)

“Any extracorporeal blood purification therapy intended to substitute for impaired renal function over an extended period of time and applied for or aimed at being applied for 24 hours/day.”



2

Why CRRT?

CRRT closely mimics the native kidney in treating AKI and fluid overload

- Removes small and large solutes and fluid
- Replacement fluid fosters electrolyte and pH balance
- Appropriate therapy for hemodynamically unstable patients
- Sepsis

3

Goals of CRRT

- Maintain fluid, electrolyte, acid/base balance, and eliminate metabolic waste (mimic native kidney function)
- Prevent further damage to kidney tissue
- CRRT is a therapy to support abnormal kidney function

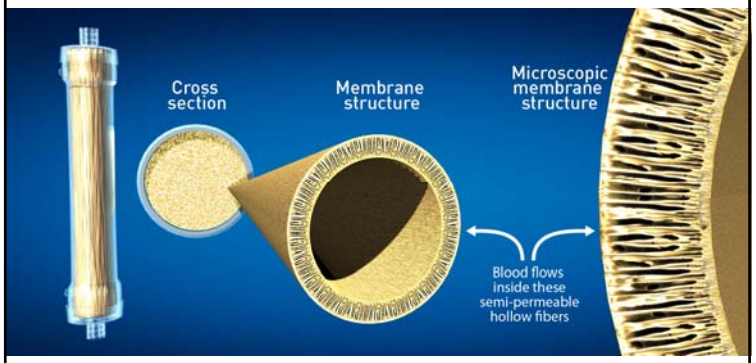
4

Anatomy of a Hemofilter

- 4 External ports
  - –Blood and dialysis fluid
- Potting material
  - –Support structure
- Hollow fibers
  - –Semi-permeable membrane
- Outer casing

Hemofilter: Semi-permeable membrane

Allows solutes (molecules or ions) up to a certain size to pass through



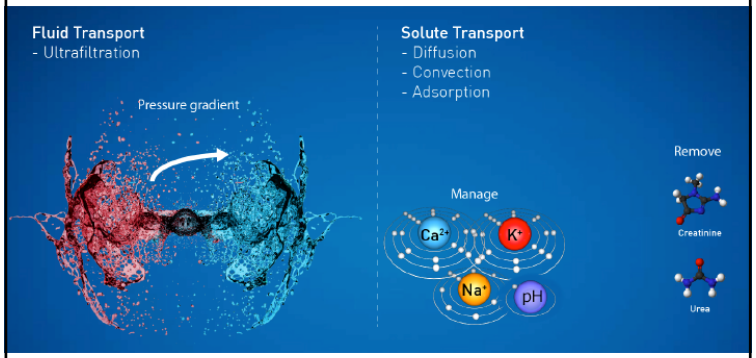
CRRT Transport Mechanisms

CRRT Modes of Therapy

- **SCUF:** Slow Continuous Ultrafiltration. Primary goal is to remove patient fluid
- **CVVH:** Continuous Veno-Venous Hemofiltration. Primary goal is to achieve small, medium and large molecule clearance, remove patient fluid
- **CVVHD:** Continuous Veno-Venous HemoDialysis. Primary goal is to achieve small molecule clearance, remove patient fluid
- **CVVHDF:** Continuous Veno-Venous HemoDiaFiltration. Primary goal is to achieve small, medium and large molecule clearance, remove patient fluid

All modes will assist in maintaining hemodynamic stability due to the slow fluid removal as tolerated by the patient MAP.

Molecular Transport Mechanisms



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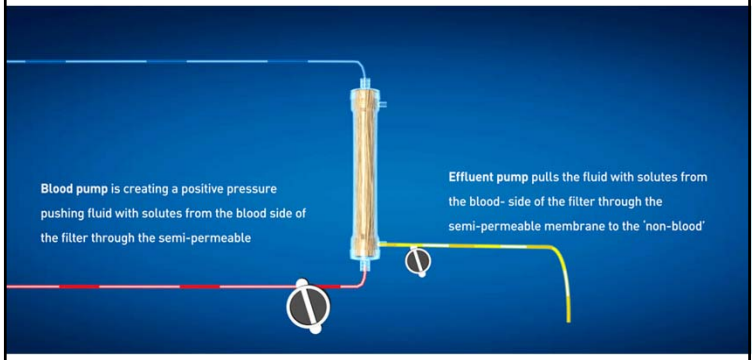
Ultrafiltration

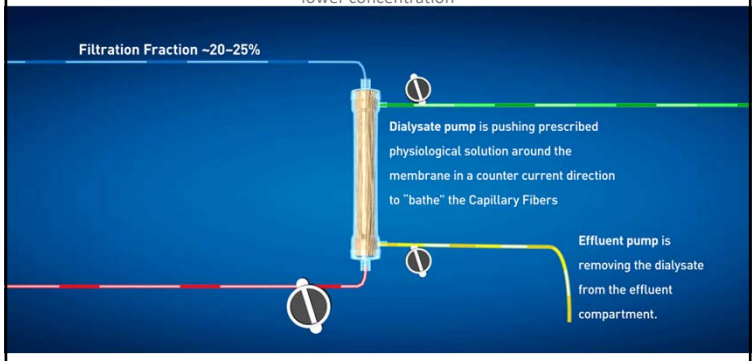
The movement of fluid through a semi-permeable membrane driven by a pressure gradient (hydrostatic pressure)



10

Diffusion = Hemodialysis

The movement of solutes only from an area of higher concentration to an area of lower concentration



* Filter has a 50K cut off

11

Major factors affecting diffusion

Solute removal by diffusion depends on:

- Concentration gradient blood / dialysis
- Dialysate flow rate
- Molecular size – diffusion clears small molecules
- Permeability of the membrane

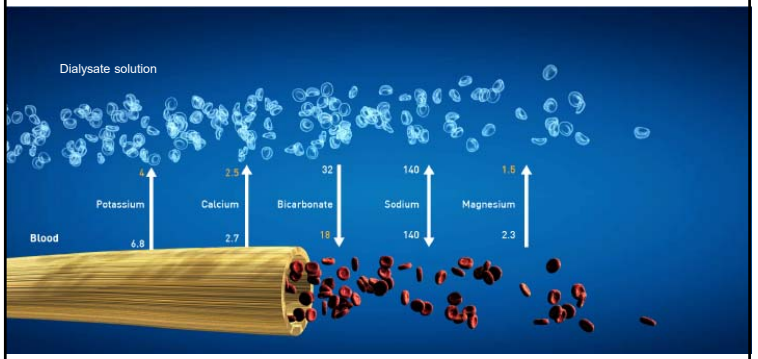
12

Dialysate Solutions

- Flow counter-current to blood flow
- Separated from blood by a semi-permeable membrane
- Drive diffusive transport-dependent on concentration gradient and flow rate
- Facilitate removal of small solutes
- Physician prescribed
- Solution choice adjusted to meet patient needs

13

How does diffusion work?

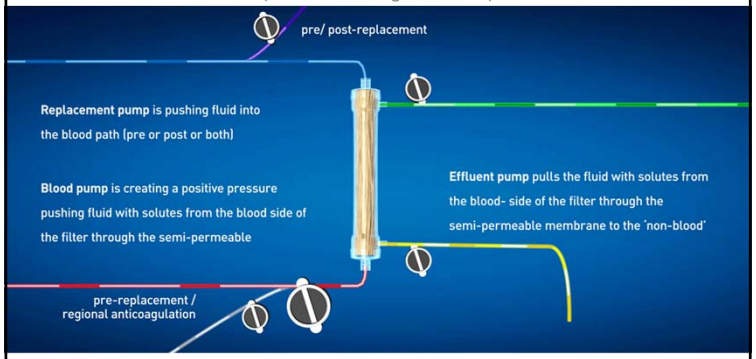


These numbers are for example only. Clinicians must use their judgment on prescribing the correct dialysate concentration.

14

Convection "Solute drag"= hemofiltration

The forced movement of fluid with dissolved solutes
(the fluid will drag the solutes)



15

Major factors affecting convection

Solute removal by convection depends on:

- High Membrane permeability
- Molecular size
- Degradation of filter membrane (can decrease performance)
- Replacement fluid flow rate (pressure gradient)

16

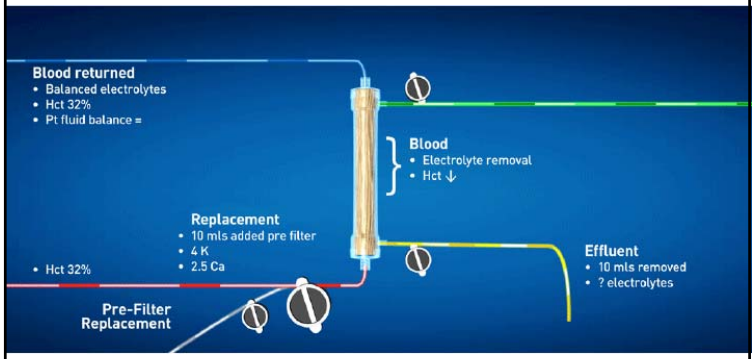
How Pre and/or Post Replacement work

- **Pre Replacement**
- Pre-filter replacement solution will deliver into the blood flow at set rate.
- Blood will be diluted ↓Hct.
- The replacement "fluid volume" will be removed by the effluent pump.

- **Post Replacement**
- The replacement "fluid volume" will be removed by the effluent pump.
- Blood will be concentrated ↑Hct.
- Post-filter replacement solution will deliver replacement solution to "replace" the removed "volume" and replenish lost electrolytes.

17

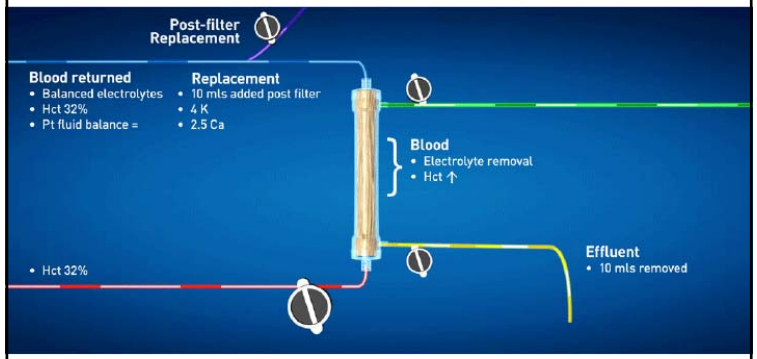
Pre-filter replacement



Cerda, J. & Ronco , C. (2010). Choosing a RRT in AKI. In J. A. Kellum, R. Bellomo, & C. Ronco (Eds.), *Continuous Renal Replacement Therapy* (pp. 79-92). New York, USA: Oxford University Press

18

Post-filter replacement



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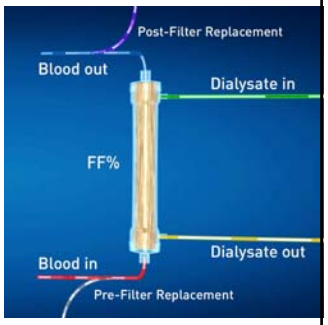
19

Why do we need to monitor Filtration Fraction percentage (FF%)?

- A FF > 25% can lead to premature filter degradation
- To decrease the FF%, prescribed fluid delivery strategies may need to be initiated such as a mix of pre- and post-dilution.
- For accurate Filtration Fraction percentage monitoring, the patient's hematocrit should be updated once a day.



Bellomo, R & Baldwin, I (2010) The circuit and the prescription. (pp. 101 & 130) In J.A. Kellum, R. Bellomo, & C. Ronco (Eds) *Continuous Renal Replacement Therapy* New York, USA Oxford University Press

20

Replacement Solutions

- Infused directly into the blood at points along the blood pathway
- Drives convective transport
- Facilitates the removal of small middle and large solutes
- Physician Prescribed
- Solutions choice adjusted to meet patient needs

21

Considerations for solution choice

Which mode of therapy?

- CVVH: Replacement Only
- CVVHD: Dialysate Only
- CVVHDF: Both Solutions

Which anticoagulant prescribed?

- Systemic, regional or none

22

Adsorption

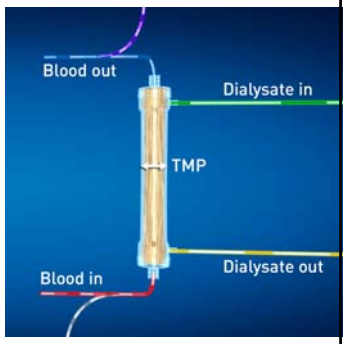
Molecular adherence to the surface or interior of the membrane.

23

Filter viability

Trans-membrane Pressure (TMP)

- Pressure exerted on filter membrane during operation
- Reflects pressure difference between fluid and blood compartments of filter
- Calculated by CRRT device



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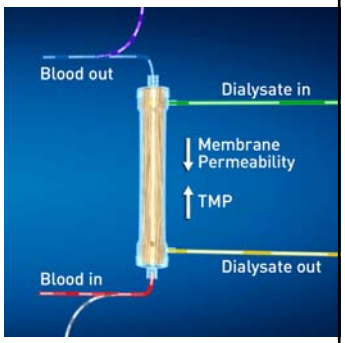
Trans-Membrane Pressure (TMP)

- Calculated and automatically recorded:
  - Entering Run mode - blood flow is stabilized
  - Blood flow rate is changed
  - Patient fluid removal rate is changed
  - Replacement solution rate is changed



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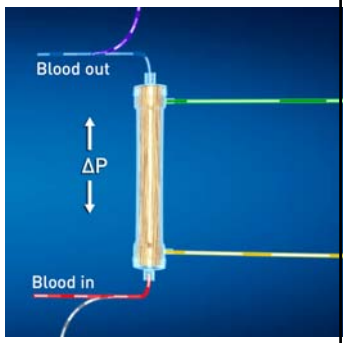
Filter viability

Filter Pressure Drop (ΔP Filter)

- Change of pressure from blood entering filter and leaving filter
- Determines pressure conditions inside hollow fibers
- Calculated and automatically recorded:
  - Entering Run mode
  - Blood flow rate is changed
- Calculated by CRRT device



26

27

CARING FOR THE TRAUMA PATIENT IN THE ICU

Kelly Urban, PhD, MEd, RN, CCRN-K, TCRN
UAMS Medical Center

MECHANISMS OF INJURY

Blunt

- Acceleration
- Deceleration
- Shearing
- Crushing/Compression

Penetrating

- GSW
- Stab wounds
- Impalement
- Avulsion/Degloving

TISSUE CHARACTERISTICS

Solid Structures → Crack — Bone, Solid Organ, Semi-solid organs

Hollow Structures → Pop — Air Filled, Fluid Filled, Air/Fluid Filled

Fixed Points → Tear — Ligaments, Tendons

INITIAL ASSESSMENT

Primary Survey

- A – Airway and Alertness with cervical spinal embolization
- B – Breathing and ventilation
- C – Circulation and control of hemorrhage
- D – Disability (neuro status)
- E – Exposure and Environmental control
- F – Full set of vitals and Family presence
- G – Get resuscitation adjuncts

Secondary Survey

- H – History and Head-to-Toe assessment
- I – Inspect posterior surfaces

AIRWAY & ALERTNESS

(WITH CERVICAL SPINAL IMMOBILIZATION)

Is the Airway patent?

- Is the patient alert?
- Can the patient speak/protect airway?
- Inspect for obstructions
- Auscultate for airway sounds
- Palpate for bony deformities

- Not patent:
  - Suction
  - Remove debris
  - Airway adjunct
  - Definitive airway

Definitive Airway (Intubation) Assessment

- 5 point auscultation
- Observe rise and fall of chest
- Capnometer results in color change or etCO2 results
- Observe skin color

BREATHING

Inspect for:

- Spontaneous breathing
- Symmetrical rise and fall of chest
- Depth, pattern and rate of respirations
- Signs of respiratory difficulty
- Skin color
- Wounds, contusions, abrasions, or deformities

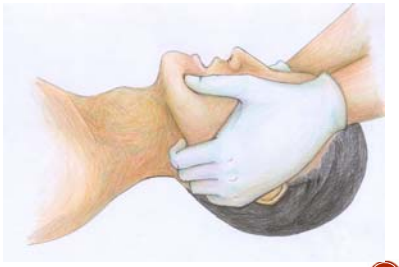
Auscultate/Palpate

- Auscultate
  - Breath sounds
- Palpate
  - Bony Structures
  - Subcutaneous Emphysema
  - Soft Tissue Injury

BREATHING CONT.

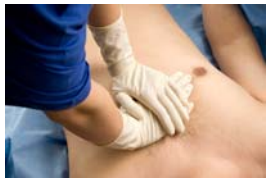
- Absent
  - Open airway (insert adjunct)
  - Assist ventilations
  - Prepare for definitive airway

- Ineffective
  - Assist ventilations
  - Prepare for definitive airway



CIRCULATION

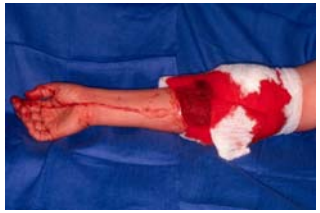
- Assess for any sign of external hemorrhage
- Assess skin color/moisture/temperature
- Palpate presence of central pulse



- Trauma patients typically need 2 large bore Ivs
- Fluid of choice typically isotonic crystalloid solution (warm) unless evidence of bleeding….need blood products

CIRCULATION CONT.

- Control of Hemorrhage:
  - Apply direct pressure
  - Elevate hemorrhage extremity
  - Apply pressure over arterial sites
  - Consider a tourniquet



MASSIVE TRANSFUSION PROTOCOL

- PRBC: FFP: Platelets = 1:1:1

(1 bag of platelets typically = 6 units of apheresed platelets)

Which product do I give 1st?

TXA ADMINISTRATION

- Bolus: 1 gm IV over 10 minutes
- Infusion: 1 gm IV over 8 hours

- Tranexamic Acid (TXA): anti-fibrinolytic that inhibits activation of plasminogen to plasma → prevents fibrinolysis and breakdown of clots
- Used for prompt control of hemorrhage

Indications	Contraindications
• Systolic blood pressure < 90 mmHg • Heart rate > 110 • Considered at risk for significant hemorrhage • < 3 hours since time of injury	• More urgent critical resuscitation interventions needed (do not delay to give TXA) • Isolated head injury • Time of injury > 3 hours

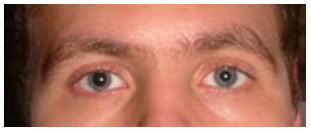
PERFUSION GOALS – WHAT ARE THEY?

- Systolic Blood Pressure
- Diastolic Blood Pressure
- Mean Pressure

- What does pulse pressure tell me?

DISABILITY (NEUROLOGIC STATUS)

- Inspect pupils



- Assess GCS
- Also a good time to consider if patient is going to need a head CT, ABGs, or a glucose

TRAUMATIC BRAIN INJURY (TBI)

- TBI is a primary injury.
- One goal of treatment is to prevent a secondary injury which may occur as a result of:
  - Hypotension
  - Hypoxia
  - Hypocapnia
  - Increased ICP
  - Anemia
  - Hyperthermia

TBI GOAL DIRECTED THERAPY

- Adequate Oxygenation
- Normothermia
- Avoid hypotension
- Normal electrolytes
- Seizure prophylaxis 7 days
- DVT ppx (24 hrs after stable head CT or 48 hrs after crani)
- Early enteral nutrition

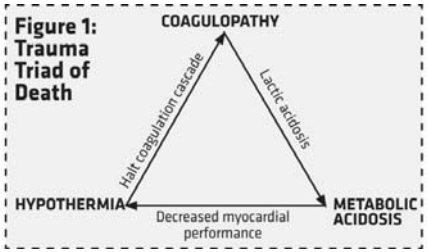
Pulse Oximetry $\geq$ 93%	ICP < 22 mmHg	Serum Na 135-165
$PaO_2 \geq 60$ mmHg	CPP $\geq$ 60 mmHg	Hgb $\geq$ 7 g/dl
$PaCO_2$ 35-42 mmHg	CVP > 5 mmHg	Glucose 80-150 mg/dL
SBP $\geq$ 100 mmHg	Temp 36.0-38.0°C	Serum Osm 280-325

TBI GOAL DIRECTED THERAPY CONT.

- Maintain head in midline position
- HOB elevated 30°
- Continuous $ETCO_2$ monitoring for brain injured ventilated patients
- ICP should be monitored in all salvageable patients with a severe TBI (GCS 3-8 after resuscitation) and an abnormal CT scan

EXPOSURE/ENVIRONMENTAL

- Remove all clothing to assess patient
- Keep the patient warm!!!!



FULL SET OF VITAL SIGNS/FAMILY

- Vital Signs
- Facilitate Family Presence

GET RESUSCITATION ADJUNCTS

- Laboratory studies
- Monitor for cardiac rhythm and rate assessment
- Naso/orogastric tube
- Oxygenation or ventilation analysis
- Pain assessment and management

INTERPRETATION OF LAB RESULTS

- HCT/Hgb
- Creatinine/BUN
- CK (total)
- Troponin
- K
- Lactate
- ABG (including base deficit)

$SPO_2/ETCO_2$

SPO_2

- Oxygenation status
- Goal ≥ 93%

$ETCO_2$

- Ventilation status
- Goal 35-45 mmHg

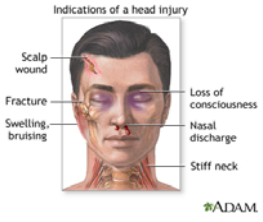
HISTORY/HEAD-TO-TOE

Inspect
Auscultate
Percuss?
Palpate

- History:
  - Mechanism of Injury
  - Injuries sustained
  - Signs and symptoms
  - Treatment
- Exam:
  - Head & Face
  - Neck
  - Chest
  - Abdomen/Flanks
  - Pelvis
  - Extremities

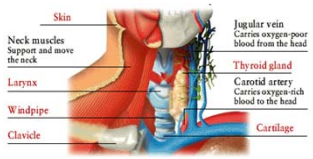
HEAD & FACE

- Assess for soft tissue and bony injuries
  - Inspect for wounds, edema, ecchymoses, asymmetry
  - Palpate for tenderness, step-offs, crepitus
- Eyes: Gross visual acuity, pupils, EOM
- Ears: Drainage or ecchymoses, wounds
- Nose: Nasal septum position, bony deformity, drainage



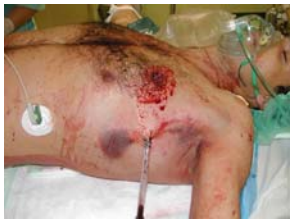
NECK

- Cervical Spine:
  - Immobilize until injury is excluded
- Neck: wounds, edema or contusions
  - Assess position of trachea and note any jugular venous distention

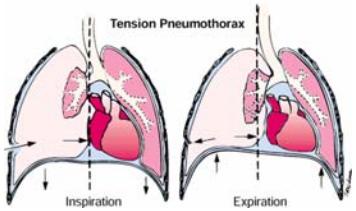


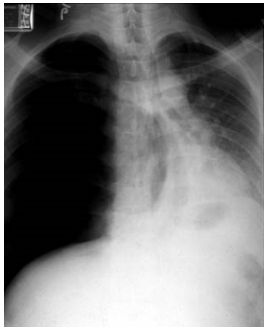
CHEST

- Chest: respiratory rate, depth, effort
  - Assess for wounds, ecchymoses, edema
  - Listen to breath sounds and heart sounds



COMPLICATIONS OF CHEST TRAUMA

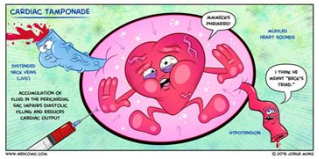




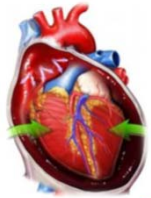
COMPLICATIONS OF CHEST TRAUMA — CARDIAC TAMPONADE

Collection of excess fluid between visceral pericardium and parietal pericardium

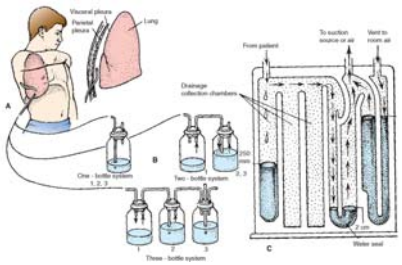
- Signs/Symptoms
- Beck's Triad
  - Muffled Heart Tones
  - Systemic Hypotension
  - Distended Neck Veins



- Cardiovascular Effects
- Elevated CVP
- Narrowed Pulse Pressure
- Rapidly falling cardiac output
- Tachycardia
- Pulsus Alternans
- Blunting of QRS complex
- PEA
- Pulsus Paradoxus

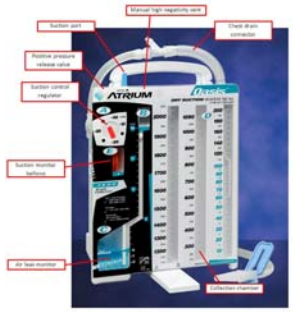


CHEST DRAINAGE (CHEST TUBES)



FOCA

- F (fluctuation)
- O (obstruction)
- C (color)
- A (air leak)



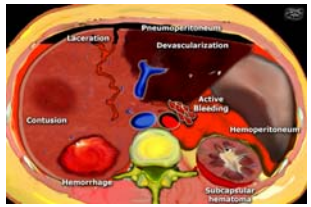
Notify physician
- Initial output is > 500 mL
- More than 200 mL/hour for 2–4 hours

Clamping chest tube is contraindicated

https://www.youtube.com/watch?v=RzzQMLpWE0M

ABDOMEN/FLANK

- Abdomen/Flanks
  - Inspect/palpate wounds, ecchymoses, pain
  - Auscultate bowel sounds



SERIAL ASSESSMENTS OF THE ABDOMEN

- Labs
- Abdominal Girth
- Bladder Pressures?

ABDOMINAL INJURIES

- Liver
- Spleen
- Bowel
- Kidneys
- Pancreas
- Diaphragm

Penetrating	Blunt
1. Small intestine	1. Spleen**
2. Colon	2. Liver
3. Liver	3. Small bowel

** In 66% of abdominal trauma, spleen is the only structure damaged

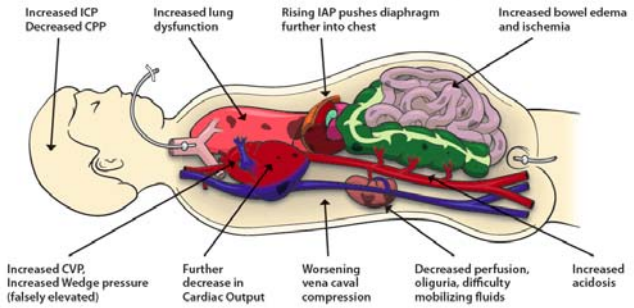
ABDOMINAL TRAUMA GENERAL TREATMENT

Indications for Surgical Intervention	Indications for Non-operative Management
• Signs of hypovolemia with persistent hypotension • Ongoing hemodynamic instability • > 2 units PRBCs required • Clear and persistent signs of peritoneal irritation • Radiological evidence of pneumoperitoneum consistent with viscus rupture • Evidence of diaphragmatic rupture • Persistent, significant GI bleeding • Evisceration	• Solid organ injuries are less likely to require surgical intervention than hollow organ injuries • Hemodynamically stable • Stable hemoglobin levels during first 12-48 hours • < 2 units PRBCs administered • < 55 years old • Alert and able to interact during exam

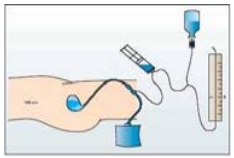
ABDOMINAL COMPARTMENT SYNDROME

- APP = MAP – IAP
- Normal is 0–5 mm Hg
- All body systems affected
  - Abdominal
  - Pulmonary
  - Cardiovascular
  - Neurologic
- Assessment findings
  - IAP measurement
  - Low UO and hypotensive shock
  - Tense, rigid, abdomen
  - Increased peak airway pressures without thoracic injury
  - Increased ICP without head injury
  - Increased IAP

Occult Organ Ischemia
IAP 16 – 20 mmHg



HOW TO MEASURE ABDOMINAL COMPARTMENT PRESSURE?



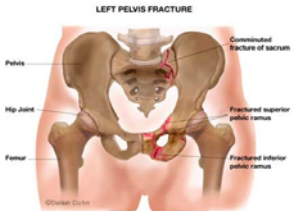
- Supine position
- End-expiration
- Transducer zeroed at mid-axillary line (some sources level of bladder)
- Instill fluid (25-50 ml)
- Clamp foley
- Measure pressure 30-60 seconds after fluid instillation in absence of active abdominal muscle contractions

TREATMENT OF ABDOMINAL COMPARTMENT SYNDROME

- Avoid elevating HOB
- Maintain MAP (avoid over-resuscitation)
- Consider diuresis
- Percutaneous catheter decompression for patients with intraperitoneal fluid, abscess, or blood
- Bedside or operative decompression

PELVIS

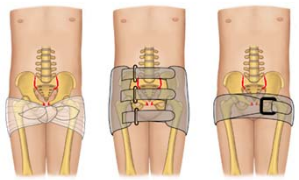
- Pelvis: check for stability
  - Apply gentle pressure over iliac crests
  - Apply gentle pressure on symphysis pubis



- If pelvis fracture has been ID, DO NOT perform these assessment maneuvers!

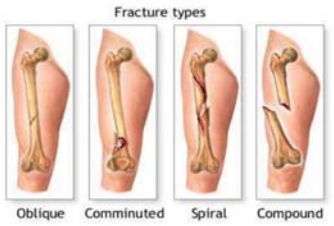
TREATMENT – PELVIC STABILIZATION

- Goal: reduce pelvic volume to decrease hemorrhage (earlier tamponade)
- Internal rotation of lower extremities
- Sheet or commercial binder
  - Placed at level of greater trochanter and symphysis pubis



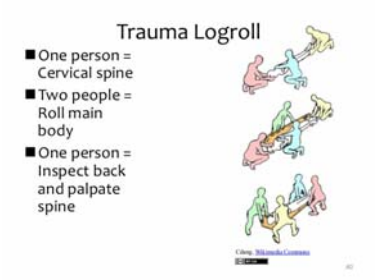
EXTREMITIES

- Extremities:
  - Assess for soft tissue injuries
  - Palpate temperature, moisture, pulses
  - Check neurovascular status



INSPECT POSTERIOR

- Logroll patient
- Inspect for blood, edema, wounds
- Palpate for deformity, tenderness



RHABDOMYOLYSIS

- Breakdown of muscle tissue releasing muscle fiber contents into the blood resulting in kidney damage

- Symptoms:
  - Dark red or cola-colored urine
  - Decreased urine output
  - General weakness
  - Muscle stiffness/tenderness

DVT/PE PROPHYLAXIS

Deep Vein Thrombosis

- 50% higher risk in trauma
- Classic triad
  - Stasis
  - Endothelial damage
  - Hypercoagulability
- Goal of treatment
  - Limit clot development
  - Prevent PE

Pulmonary Embolism

- Pleuritic chest pain
- Dyspnea/Orthopnea
- Hypoxemia
- Cough/Hemoptysis
- Decreased lung sounds
- Hypotension
- JVD
- Symptoms depend on size
  - Massive PE; hemodynamic instability
  - Pulmonary infarction/ischemia
  - Right ventricular failure

FAT EMBOLISM

- Manipulation of long bones
- Classic triad
  - Decreased mental status
  - Respiratory distress
  - Petechial rash
- Onset of symptoms
  - 24 to 72 hours
- Diagnosis
  - Helical CT
- Treatment
  - Supportive care

ALI/ARDS

Risk Factors

- Trauma; thoracic/general
- Aspiration
- Pulmonary contusion
- Fat embolism or PE
- Near-drowning or inhalation
- Massive transfusion
- Oxygen toxicity
- DIC
- Shock/Pneumonia/Sepsis

Treatment

- Supportive care
- Positive end expiratory pressure with lower tidal volumes

WHAT ABOUT THE GERIATRIC TRAUMA PATIENT?

- Anticoagulants/Antiplatelets?
- Comorbidities?
- Hypoperfusion may be underappreciated
- What about a geriatric consult?
- At particular risk for med-related adverse events
  - Beers criteria

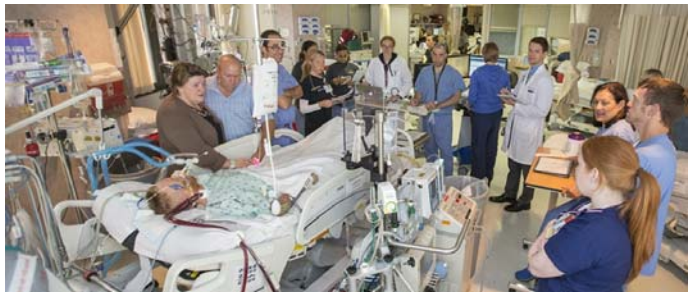
ANTICOAGULATION REVERSAL

- Warfarin: PCC (4 factor concentrate of II, VII, IX, & X)
- Dabigatran: none
- Rivaroxaban: none (may be partially reversed by PCCs)
- Clopidogrel, aspirin: none (may consider DDAVP or platelet transfusion)

BURN PATIENTS

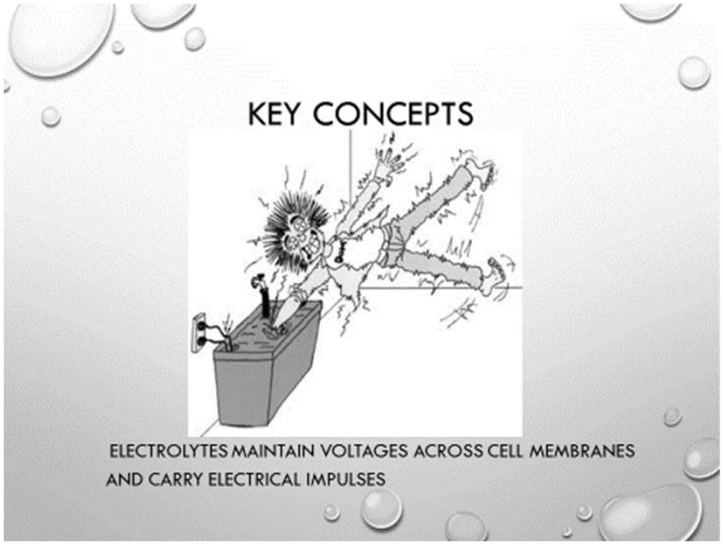
- What kind of burn?
- How much?
- Parkland Burn Formula
  - Adults: 2 ml x kg x TBSA burned
  - Electrical: 4 ml x kg x TBSA burned
  - ½ in 8 hours and ½ in remaining 16 hours
  - Only a starting point for resuscitation – use urine output as a guide

QUESTIONS?



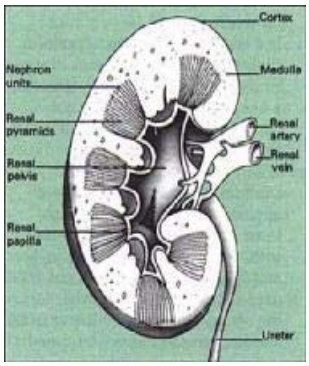
Overview of Electrolytes

Patti Esmail MSN/ED RN CCRN-K
Clinical Educator
Chi St Vincent



Key Concepts

- Fluid balance cannot be maintained without electrolytes balance
- The kidneys keep electrolytes concentrations constant



Daily Electrolyte requirements.

- Potassium – 40 – 60 mEq per day
- Sodium – 500 mg
- Chloride – 6 – 15 mg
- Calcium – 800 mg
- Magnesium – 250 mg
- Phosphate – 800 – 1300 mg
- Bicarbonate

•An electrolyte is a term for salts, or ions and is expressed as millequivalents per liter

•When immersed in water, electrolytes dissociate into charged particles, either positively charged ions (cation) or as a negatively charged ion (anion)

•In normal circumstances total cations = total anions (if not, a "gap" occurs)

Anion Gap

Anions (--)

- Bicarbonate 27
- Chloride 106
- Phosphorus 2
  - Sulfate 1
  - Proteinate 15.5
  - Organic acids 3

154.5

Cations (+)

- Calcium 5
- Magnesium 2
- Potassium 4.5
- Sodium 143

154.5

Electrolytes of Major concern

- **Potassium**
- **Sodium**
- **Magnesium**
- **Calcium**
- **Phosphorous**

Laboratory ELECTROLYTE values

ELECTROLYTE	NORMAL	HYPO-LOW	HYPER-HIGH
POTASSIUM	3.5 – 5.0	<3.5	>5.5
SODIUM	135 – 145	<135	>145
CALCIUM	8.5 – 10.00	<8.5	>10.00
MAGNESIUM	1.5 – 2.5	<1.5	>2.5
PHOSPHOROUS	2.5 – 4.5	<2.5	>4.5

Problems with electrolytes – K U R I O

- **K -** know normal levels – then can identify abnormal
- **U -** understand what role/function the electrolyte plays in the body/cell
- **R -** recall the clinical symptoms associated with the electrolyte disturbance
- **I -** implement the appropriate course of treatment

O - observe patient for resolution of symptoms and normalization of electrolyte level

Potassium (K^+)

- Normal Values (mEq/L)
  - 3.5-5.5
- Daily Requirements
  - 50-125mEq
- Action in Body
  - Maintains cellular osmolarity.
  - Necessary for transmission of nerve impulses and for muscle contraction.
  - Helps transform CHO into energy.
  - Assists with reassembling amino acids into proteins.
  - Maintains acid-base balance.

Potassium (K^+)

- Regulated by
  - Kidney through glomerular filtration rate, aldosterone
- Comments
  - Kidney cannot conserve K^+, so it must be consumed daily.
  - K^+ and H^+ move together in the kidney.
  - If cellular K^+ is lowered, Na^+ enters the cell, making it more irritable.

Potassium (K+)

Hypokalemia

- Less than 3.5mEq/L
- ETIOLOGY
  - Alkalosis: K^+ shifts into cell
  - Severe stress: K^+ shifts into the cell
  - Diuretic therapy
  - Abnormal GI losses
  - Starvation or malnutrition
  - Metabolic disease
  - Increased adrenal corticosteroid secretion or corticosteroid therapy
  - Liver disease
  - Bartter's syndrome

Hyperkalemia

- > 5.5 mEq/L
- ETIOLOGY
  - Injured cells
  - Early burns
  - Hemolysis
  - Renal disease
  - Adrenal insufficiency
  - Low cardiac output syndrome
  - Certain drugs
  - Too much intake of K+

Clinical sYMPTOMs

Hypokalemia

- Skeletal muscle: weakness, fatigue, decreased reflexes.
- Heart muscle: weak pulse, low voltage T-waves, S-T depression, predominant U-waves, faint heart sounds, dysrhythmias.
- GI disturbances: vomiting
- Respiratory: shortness of breath
- Neurological: depression, mental clouding.

Hyperkalemia

- Skeletal Muscle: weakness, cramps and pain, flaccid muscle paralysis
- Heart muscle: high peaked T-waves on EKG, wide QRS
- GI disturbances: intestinal colic, diarrhea, nausea
- Neurological: irritability, dizziness

Goals of nursing Management

Hypokalemia

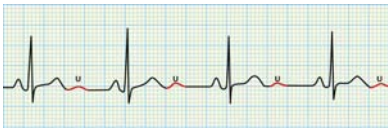
- Return serum potassium level to normal
  - Replace potassium IV or PO
- Cardiac function is WNL

Hyperkalemia

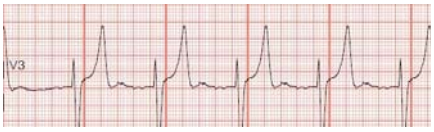
- Return serum potassium level to normal by:
  - Reducing K^+ intake
  - Giving oral or IV hydrating solutions
  - Giving dextrose and insulin infusions (20% dextrose solution with 1 unit of insulin for each 2gm of dextrose).
  - Using extrarenal dialysis
  - Giving binding resins (e.g., Kayexalate)
  - Giving osmotic diarrheal

Rhythm changes

hypokalemia



hyperkalemia



Electrolyte Disturbances and Voltage Abnormalities

- **Potassium Disturbances**

Hyperkalemia

- » K = 5.5: T-waves became tall and peaked
  » K = 6.5: QRS changes start
  » K > 7.0: P-waves decreased pwave amplitude
  » K > 8.0: P-wave becomes invisible
  » K > 12: Vfib or asystole

Hypokalemia

- » K < 2.7: U wave (Large U-wave or TU complex)
  » Depress ion of ST segment > 0.5mm
  » U-wave amplitude > 1.0 mm
  » U-wave > T-wave

Sodium (Na+)

- Action
  - Maintains osmotic pressure and serum osmolarity. Helps maintain acid-base balance, along with bicarbonate ion. Regulates fluid volume. Controls muscle contraction
- Regulated by
  - Kidney through aldosterone, ADH, glomerular filtration rate, 3rd factor in kidney, tubular enzymes.
- Normal Values
  - 135-145mEq
- Comments
  - Na^+ competes with H^+ and K^+ in the renal tubule for excretion and absorption. 99% filtered Na^+ is reabsorbed in the kidney.

Sodium (Na+)

Hyponatremia

- **Serum Na is <136mEq/L**
- **Etiology**
  - Excess water relative to the amount of sodium
  - Sodium depletion
    - Abnormal losses
    - Hyperglycemia
    - Salt-losing renal diseases
    - Bartter's syndrome
  - Heart failure and cirrhosis

Hypernatremia

- **Serum Na >145mEq/L**
  - Elevated hematocrit with volume depletion
  - Specific gravity of urine >1.030, except with diabetes insipidus where SG may be as low as 1.005
- **Etiology**
  - Impaired renal function
  - Cushing's syndrome
  - Inhalation or ingestion of sea water.

Clinical symptoms

Hyponatremia

- Skeletal muscle: convulsions
- Heart muscle: reduced blood volume, increased blood viscosity, low blood pressure, pallid and clammy skin
- GI disturbances: anorexia, nausea
- Neurological: mental confusion, giddiness, apprehension

Hypernatremia

- Heart muscle: weight gain, edema, increased blood pressure
- GU disturbances: increased urine output
- Neurological: confusion, obtunded, coma
- Increased temperature

Goals of nursing management

Hyponatremia

- Restore sodium concentration to normal levels or at an asymptomatic level
- Normal Fluid status is maintained
  - Na+ and water loss – high sodium and adequate fluid intake
  - Water intoxication: restrict fluid intake - 500ml/day
  - Water intoxication related to SIAHD: restrict water intake, because low sodium is R/T inability to excrete water

Hypernatremia

- Serum sodium is WNL and patient is asymptomatic
- Hydration status normal

Rhythm changes

- Changes in Sodium levels rarely cause electrocardiographic changes
- Most often the noticeable change is neurological

Calcium (Ca_2^+)

- Action in Body
  - Serves as framework for bones and teeth. Essential for blood clotting, for normal functioning of the central nervous system, and for muscle contraction and neuromuscular stability. Stabilizes cell membranes.
- Regulated by
  - Parathyroid hormone, thyrocalcitonin, vitamin D, kidney function
- Normal Values
  - 8.5-10.0 total; 4.5-5.5 unbound. E
- Comments
  - Major concentration is in the bone. 50% of serum Ca_2^+ is bound to protein. Normal gastric acidity is necessary for absorption of Ca_2^+ in the gut. Acts as a sedative on body.

Calcium (Ca_2^+)

Hypocalcemia

- Less than 8.5mEq/L
- Etiology
  - Hypoparathyroidism
  - Acute pancreatitis
  - Peritonitis
  - Dietary lack of Ca_2^+
  - Deficiency of vitamin D
  - Burns
  - Renal failure
  - Hyperphosphatemia
  - Osteomalacia
  - Diuretic therapy

Hypercalcemia

- Greater than 10.5mEq/L
- Etiology
  - Malignancy
  - Hyperparathyroidism
  - Renal disease
  - Immobilization
  - Vitamin D intoxication
  - Neoplastic disease of bone, breast, and lungs
  - Paget's disease
  - Addison's disease
  - Milk-alkali syndrome
  - Sarcoidosis

Clinical symptoms

Hypocalcemia

- Skeletal muscle: , muscle cramps, tetany, positive Chvostek's and Trousseau's signs
- Heart: tingling of fingertips and circumoral area, ECG changes-prolonged QT interval
- GI disturbances: abdominal cramps
- Respiratory: laryngeal stridor
- Neurological: positive Chvostek's and Trousseau's signs, confusion, numbness

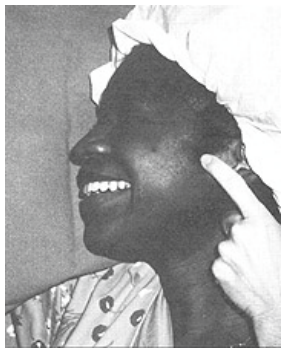
Hypercalcemia

- Skeletal muscle: muscle hypotonicity
- Heart: ECG-AV block
- GI disturbances: anorexia, nausea, weight loss
- Bone loss: resulting in deep bone pain
- GU disturbances: kidney stones, azotemia
- Neurological : Lethargy

Too Low…..Hypocalcemia

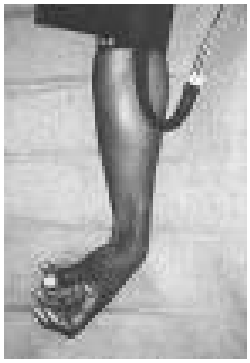
- Chvostek's Sign

Tapping on the facial nerve as it passes through the parotid gland causes ipsilateral contraction of the facial muscle



Too Low…..Hypocalcemia

- Trousseau's Sign
- A positive Trousseau's sign is carpal spasm induced by arterial occlusion of the arm with a blood pressure cuff



Goals of nursing management

Hypocalcemia

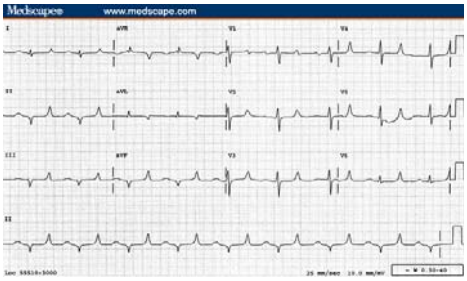
- As serum ionized calcium is lowered, there is increased neuromuscular activity (Ca_2^+ has a sedative effect on the body), producing hyperaction of motor and sensory nerves to stimuli
- Return calcium level to normal by administering:
  - Isotonic 0.9% NaCl solution with calcium additives (calcium gluconate, calcium chloride)
  - Oral Ca_2^+ with additional vitamin D
  - Parathyroid hormone (100-200 units given every 4-6 hr during

Hypercalcemia

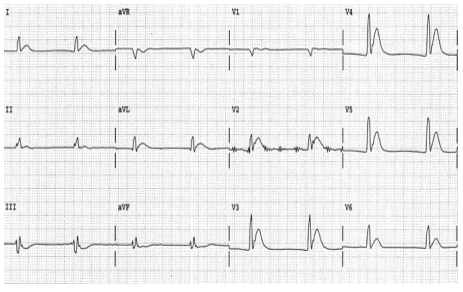
- As serum calcium rises, there is depression of all neuromuscular activity and increased myocardial irritability
- Increased calcium ions in sympathetic ganglia impede transmission of impulses within GI system
- Return calcium level to normal levels or in an asymptomatic range
- Cardiac and neurologic functions are normal

Rhythm changes

Hypocalcemia - long QT



Hypercalcemia – short QT



Electrolyte Disturbances and Voltage Abnormalities

- **Calcium Disturbances**

Hypercalcemia

- » Foreshortened ST segment
  » QT inverval shortened (Qtc of less than 270ms is 90% of the time related to hypercalcemia)

Hypocalcemia

- » Long ST
  » Long QTc

Magnesium (Mg+)

- Action in Body
  - Regulates nerve and muscle tone by preventing their activation by Ca
  - Required for over 300 enzymes to work including protein, carbohydrate and fat metabolism
- Regulated by
  - Kidney function parathyroid hormone
- Normal Values (mEq/L)
  - 1.5-2.5
- Comments
  - Has a higher concentration in cerebrospinal fluid than in serum.
  - 35% is bound to protein.
  - Stored in bone, muscle, and soft tissue.

Magnesium (Mg+)

Hypomagnesemia

- Less than 1.5mEq/L
- Etiology
  - Impaired absorption or intake-alcoholism
  - Acute or chronic pancreatitis
  - Malnutrition
  - Increased losses
  - Chronic alcoholism
  - Diuretic therapy
  - Renal disease

Hypermagnesemia

- Greater than 2.5mEq/L
- Etiology
  - Renal disease
  - Overuse of magnesium-containing antacids

Clinical symptoms

Hypomagnesemia

- Skeletal muscle: positive Chvostek's and Trousseau's signs
- Heart: tachycardia, increased BP, ventricular dysrhythmias, ECG changes: depressed ST, prolonged QT
- Neurological: CNS agitation

Hypermagnesemia

- Skeletal muscle: hyporeflexia
- Heart: hypotension, cardiac dysrhythmias
- Neurological: weakness, coma
- Respiratory: respiratory arrest

Goals of nursing management

Hypomagnesemia

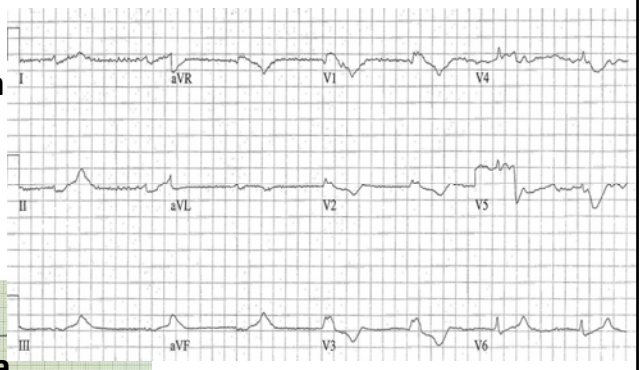
- Pathophysiology
  - Diminishes ability to relax muscular and neural tone
  - Disrupts numerous physiological and metabolic enzyme reactions
- Serum Magnesium level returns to normal
- No significant cardiac or neuromuscular symptoms

Hypermagnesemia

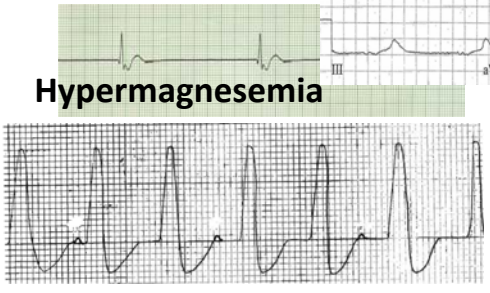
- Pathophysiology
  - Causes excessive relaxation of nerves and muscles including myocardium and respiratory muscles
  - Can cause numerous metabolic interactions
- Return serum level to normal
  - If renal function is normal - diurese
  - Peritoneal or hemodialysis
  - Elimination of magnesium containing antacids. For magnesium toxicity, administer 10% calcium gluconate slowly IV

Rhythm changes

Hypomagnesemia



Hypermagnesemia



Phosphate

- Action in Body
  - Acts as buffering agent for urine. Responsible for bone growth. Interacts with Hg in the red blood cell so as to promote oxygen release to tissues. Essential for metabolic processes such as ATP production. Promotes white blood cell's phagocytic action. Important to platelet structure and function.
- Regulated by
  - Kidney function, parathyroid hormone, vitamin D
- Normal Values (mEq/L)
  - 2.5-4.5
- Comments
  - Stored in bone. Calcium and phosphate have a reciprocal relationship

phosphate

Hypophosphatemia

- Less than 2.5mEq/L
- Etiology
  - Familial disease
  - prolonged hyperalimentation with phosphate-free solution
  - Osteomalacia
  - Rickets
  - Alcoholism
  - Alkalosis

Hyperphosphatemia

- Greater than 4.5mEq/L
- Etiology
  - Renal failure; acute/chronic
  - Hypoparathyroidism
  - Cathartic abuse
  - Cytotoxic agents for neoplasms
  - Over administration of IV or oral phosphates

Clinical symptoms

Hypophosphatemia

- Hemolytic anemia
- Lethargy
- Weakness - especially respiratory muscles
- Mental confusion

Hyperphosphatemia

- Skeletal muscle: , muscle cramps, tetany, positive Chvostek's and Trousseau's signs
- Heart: tingling of fingertips and circumoral area, ECG changes- prolonged QT interval
- GI disturbances: abdominal cramps
- Respiratory: laryngeal stridor
- Neurological: positive Chvostek's and Trousseau's signs, confusion, numbness
- Skin: coarse dry skin, alopecia

Goals of nursing management

Hypophosphatemia

- Pathophysiology
  - As extracellular HPO24+ is reduced there is less formation of nuclear acid within cells and diminished ATP and 2,3-DPG
- Return serum phosphate level to normal
- No neuromuscular signs of hypophosphatemia present

Hyperphosphatemia

- Pathophysiology
  - Hyperphosphatemia occurs when the phosphorus load (from GI absorption, exogenous administration, or cellular release) exceeds renal excretion and tissue uptake, an imbalance that can result from any of the following three pathogenic mechanisms:
- Return serum phosphate level to normal
- No neuromuscular signs of hyperphosphatemia present

Rhythm changes

Hypophosphatemia

- May show the same EKG changes as Hypercalcemia

Hyperphosphatemia

- May show the same EKG changes as Hypocalcemia

Ekg cHANGES

CARDIAC WAVEFORM	LOW POTASSIUM	HIGH POTASSIUM	LOW CALCIUM	HIGH CALCIUM	LOW MAGNESIUM	HIGH MAGNESIUM
p flat		X				
pr prolonged		X				X
qrs widened		X				X
qt prolonged			X			
st prolonged			X			
st shortened				X		
st depressed	X				X	
t widened				X		
t tall		X			X	
t inverted shallow, flat	X					
u prominent	X					

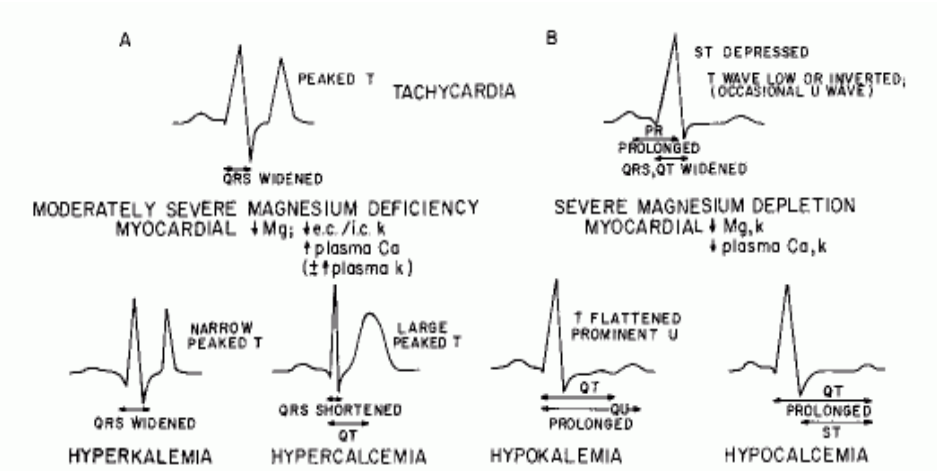
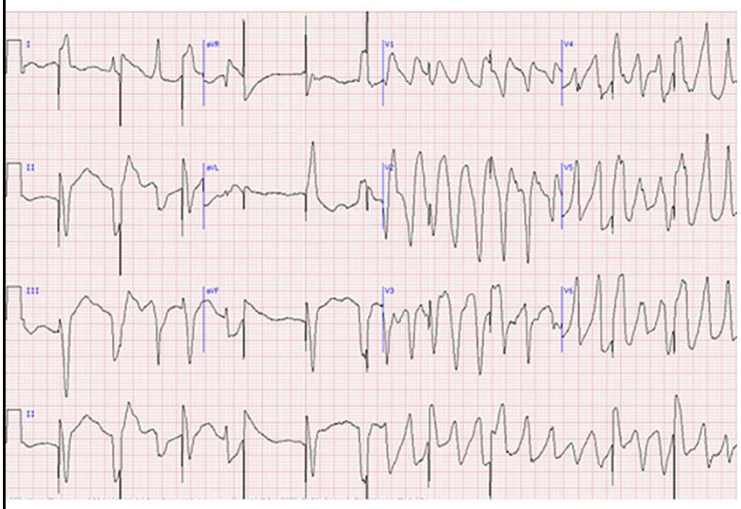
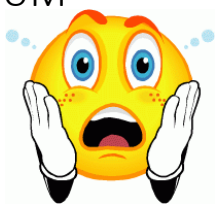


FIGURE 9-1. Schematic representations of electrocardiograms of moderate and severe hypomagnesemia, compared to those of hyper- and hypokalemia and -calcemia. (From Seelig, 1969.)

LOW POTASSIUM AND LOW MAGNESIUM





?Questions?

LRCCP Basic Critical Care Course

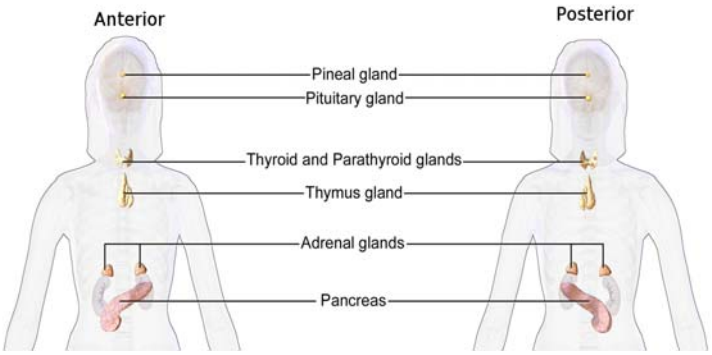
Endocrine Disorders

Patti Esmail MSN/ED RN CCRN-K
LRCCP Coordinator – CHI St Vincent

Endocrine Disorders

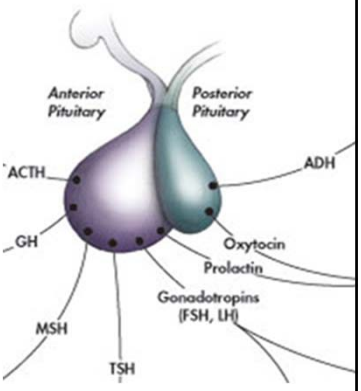
- *Endocrine System Assessment*

- 5 glands for discussion:
  - Pituitary
  - Thyroid
  - Parathyrc
  - Adrenal
  - Pancreas



Endocrine Disorders – Pituitary Gland
• *Antidiuretic Hormone – ADH*

- Secreted from the Posterior Pituitary
- aka Vasopressin
- Regulates water and Na^+ balance in the body
- Disrupt the levels of ADH and fluid balance is disrupted



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Endocrine Disorders – Pituitary Gland
• *ADH – Too Little or Too Much*

- Diminished ADH
  - Patient urinates constantly
  - Serum Na^+ is elevated
  - Serum Osmolality is elevated
- Excessive ADH
  - Urine output drops off
  - Urine becomes concentrated
  - Water is retained
  - Serum Na^+ is decreased
  - Serum Osmolality is decreased

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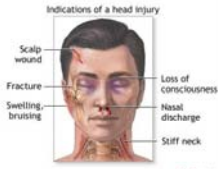
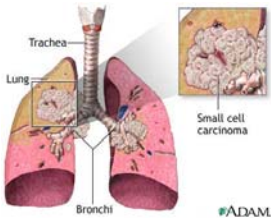
Endocrine Disorders

• Pituitary gland – ADH level imbalances

- Patient "A" – traumatic brain injury
  - Short term memory loss, disoriented to place, GCS – 13
  - c/o dry mouth, H/A, nausea, constant need to urinate
  - Refuses to eat or ambulate due to nausea and fatigue
  - c/o runny nose, clear fluid dripping from nose
  - Lungs clear, skin w/d without edema



- Patient "B" – bronchogenic small cell carcinoma
  - c/o dyspnea, fatigue, generalized pain
  - Refuses to eat; nausea and lack of appetite
  - Can't remember the last time he voided
  - Non-pitting edema of extremities; crackles auscultated through lung fields



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Endocrine Disorders

• ADH Effects on Osmolality

Decreased ADH Release	Increased ADH Release
Decreased water reabsorption/ diuresis	Increased water reabsorption/ conservation
High serum osmolality High urine volume Decreased urine osmolality	Low serum osmolality Low urine volume Increased urine osmolality

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6

Endocrine Disorders

• Lab Values used to assess fluid volume status

Serum Osmolality 285 – 295 mOsm/kg H2O

- Elevated numbers of solutes in the blood (sodium, glucose and urea) indicate greater concentration and less water, and suggest volume depletion

Urine Osmolality 50 – 1200 mOsm/kg H2O

- Urine concentration varies based on fluid intake. To evaluate urine osmolality you must know the patient's volume status.

Serum Sodium 136 – 145 mg/dL

- Normal Serum sodium has narrow normal limits making trends in serum sodium essential information.

Endocrine Disorders

• Lab assessments for Hormone levels

Plasma Adrenocorticotropic Hormone (ACTH) 0800 10-60 pg/ml
1600 <20 pg/ml

- ACTH is usually assessed with adrenal cortex cortisol levels.
- In healthy people, ACTH and cortisol show significant diurnal variation in levels
- The diurnal variation disappears in critically ill patients with sepsis, trauma, and other major illnesses
- In these patient, ACTH and cortisol levels do not correlate to the time of day.

ADH 1-5 pg/ml

- Once the critical care patient has attained neurological stability, ADH levels may be measured to titrate and monitor pharmacological ADH management.

Endocrine Disorders
• *Patient "A"*

- VS
  - B/P – 92/60
  - HR – 110
  - RR – 18
  - O2 sat – 96% on 2L
  - Temp – 37.2 (99F)
- Lab
  - S. Glucose – 140 mg/dL (H)
  - S. Osmo – 310 mOsm/kg (H)
  - S. Sodium – 151 mEq/L (H)
  - U. Osmo – 46 mOsm/kg (L)
- Intake/Output last 4 hrs
  - Intake – 240 ml
  - Output – 2000 ml

- What is Patient "A"s fluid status?

Intravascularly Dry

Volume overload

Euvolemia

Endocrine Disorders
• *Patient "A"*

- VS
  - B/P – 92/60
  - HR – 110
  - RR – 18
  - O2 sat – 96% on 2L
  - Temp – 37.2 (99F)
- Lab
  - S. Glucose – 140 mg/dL (H)
  - S. Osmo – 310 mOsm/kg (H)
  - S. Sodium – 151 mEq/L (H)
  - U. Osmo – 46 mOsm/kg (L)
- Intake/Output last 4 hrs
  - Intake – 240 ml
  - Output – 2000 ml

- What is Patient "A"s fluid status?

Intravascularly Dry

~~Volume overload~~

~~Euvolemia~~

The patient's S&S's are related to massive fluid loss – a result of ↓ADH

Endocrine Disorders
• *Patient "B"*

- VS
  - B/P – 100/82
  - HR – 108; A. fibrillation
  - RR – 24
  - O2 sat – 92% on 2L
  - Temp – 36.5 (97.7F)
- Lab
  - S. Osmo – 268 mOsm/kg (L)
  - S. Sodium – 128 mEq/L (L)
  - U. Osmo – 1230 mOsm/kg (H)
- Additional assessment
  - ↓Urine output
  - Peripheral edema
  - LOC↓, drowsy & confused
  - Disoriented to time, place, & situation

- What is Patient "B"s fluid status?

Euvolemia

Volume overload

Intravascularly Dry

Endocrine Disorders
• *Patient "B"*

- VS
  - B/P – 100/82
  - HR – 108; A. fibrillation
  - RR – 24
  - O2 sat – 92% on 2L
  - Temp – 36.5 (97.7F)
- Lab
  - S. Osmo – 268 mOsm/kg (L)
  - S. Sodium – 128 mEq/L (L)
  - U. Osmo – 1230 mOsm/kg (H)
- Additional assessment
  - ↓Urine output
  - Peripheral edema
  - LOC↓, drowsy & confused
  - Disoriented to time, place, & situation

- What is Patient "B"s fluid status?

~~Euvolemia~~

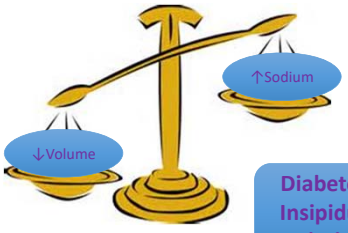
Volume overload

~~Intravascularly Dry~~

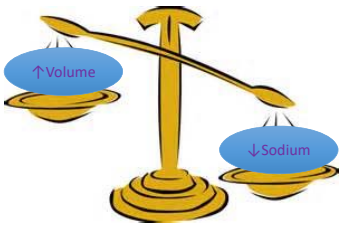
The patient's S&S's are related to fluid overload – resulting in dilutional hyponatremia

Endocrine Disorders
• *Pituitary gland dysfunctions – DI and SIADH*

- Assessment of the pituitary gland's focus is on the effects of too much or too little ADH
  - Observe trends in serum and urine osmolality
  - Fluid and sodium imbalances



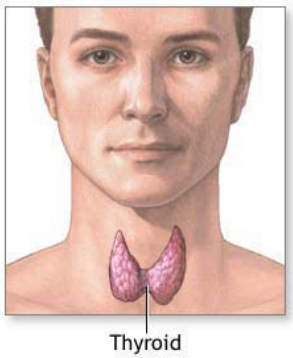
Diabetes Insipidus (DI)



Syndrome of Inappropriate ADH (SIADH)

Endocrine Disorders – Thyroid
• *Thyroid Hormone*

- Thyroid function is not typically assessed in Critical Care
- Unless a patient is exhibiting signs and symptoms suggestive of either hypothyroidism or hyperthyroidism
- The measuring of thyroid stimulating hormone (TSH) will help in the diagnosis and treatment
- TSH is commonly expressed in mIU/L
- Thyroid function assessment will include:
  - Subjective questions
  - Objective examination
  - Diagnostic labs



Endocrine Disorders – Thyroid Gland
• *Thyroid Hormone (TH) – Too Little or too much*

- **Subjective Assessment**
  - Family history of thyroid disease
  - Patient questions –
    - Fatigue
    - Weakness
    - Change in appetite or weight
    - Depression
    - Cold or heat intolerance
    - Change in seating or palpitations
    - Existing thyroid condition
    - Taking thyroid medication

Endocrine Disorders – Thyroid Gland
• *Thyroid Hormone (TH) – Too Little or too much*

- **Hypothyroidism**
  - Neurological status
    - LOC
      - Somnolence
      - Diminished mental acuity
  - Skin condition
    - Pale with yellowish appearance
    - Brittle nails
  - Eyes
    - none
  - Gland
    - Palpate; assess for tenderness or a mass
  - Vital Signs
    - Evaluate all VS
    - HR will be slow

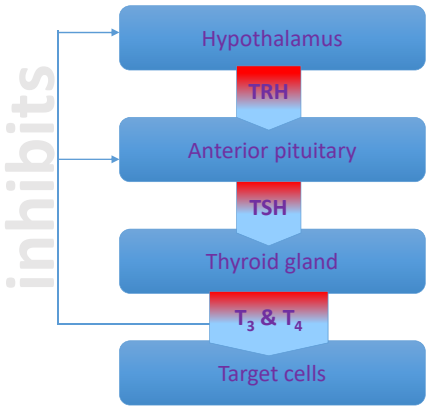
- **Hyperthyroidism**
  - Neurological status
    - LOC
      - Nervousness
      - Confusion
  - Skin condition
    - Hyperpigmentation
    - Pruritis
  - Eyes
    - Exopthalamosis
  - Gland
    - Palpate; assess for tenderness or a mass
  - Vital Signs
    - Evaluate all VS
    - HR will be rapid

Endocrine Disorders – Thyroid Gland

- *Thyroid Stimulating Hormone (TSH) – Too Little or too much*

- Thyroid stimulating hormone (TSH) regulates the amount of thyroid hormone
- The amount of TSH in the blood indicates the integrity of the hypothalamus-pituitary-thyroid (HPT) gland axis

- **Normal TSH levels – 0.5-4.7 mIU/L**
- **Hypothyroidism**
  - ↑TSH with ↓T_4
  - If T_3 and T_4 are too low; the pituitary gland produces additional TSH
- **Hyperthyroidism**
  - ↓TSH and ↑T_3 and T_4
  - If T_3 and T_4 are elevated; the pituitary gland decreases TSH production



Endocrine Disorders

- *Patient "A" – Admitting diagnosis new onset Atrial fibrillation*

- VS
  - B/P – 98/72
  - HR – 160; A fibrillation
  - RR – 20
  - O2 sat – 92% on room air
  - Temp – 37.9 (100.2F)
- Other information
  - Skin hot and dry
  - Can't remember last void
  - Cardizem started in ED to manage A. fibrillation
- Lab
  - Not back yet

Hypothyroidism

Hyperthyroidism

What is the problem?

Endocrine Disorders

- *Patient "A" – Admitting diagnosis new onset Atrial fibrillation*

- VS
  - B/P – 98/72
  - HR – 160; A fibrillation
  - RR – 20
  - O2 sat – 92% on room air
  - Temp – 37.9 (100.2F)
- Other information
  - Skin hot and dry
  - Can't remember last void
  - Cardizem D/C'd and Amiodarone started manage A. fibrillation
- Lab
  - TSH <0.02 mIU/L (L)
  - T_4 – 75 mcg/dL (H)
  - T_3 Uptake 54% (H)

~~Hypothyroidism~~

Hyperthyroidism

With Thyroid Storm

The lab values confirm the diagnosis of Hyperthyroidism with Thyroid Storm

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19

Endocrine Disorders

- *Patient "A" – Admitting diagnosis Hyperthyroidism with Thyroid Storm*

- Following the diagnosis – the doctor orders an Esmolol infusion and discontinues the Amiodarone infusion. Why is the Esmolol a better choice for this patient?

Beta blockers treat the asthma associated with hyperthyroidism

Beta blockers increase the conversion of T_4 to T_3

Beta blockers treat the acute decompensated heart failure (HF)

Beta blockers inhibit beta receptors to control symptoms due to SNS stimulation

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20

Endocrine Disorders

- *Patient "A" – Admitting diagnosis Hyperthyroidism with Thyroid Storm*

- Following the diagnosis – the doctor orders an Esmolol infusion and discontinues the Amiodarone infusion. Why is the Esmolol a better choice for this patient?

- **Beta blockers are the primary treatment for hyperthyroidism with thyroid storm. They inhibit physiological responses to SNS stimulation. Some beta blockers have a minimal effect of decreasing T_3 by inhibiting the conversion of T_4 to T_3**

Beta blockers treat the asthma associated with hyperthyroidism

Beta blockers increase the conversion of T_4 to T_3

Beta blockers treat the acute decompensated heart failure (HF)

Beta blockers inhibit beta receptors to control symptoms due to SNS stimulation

Endocrine Disorders

- *Patient "A" – Admitting diagnosis Hyperthyroidism with Thyroid Storm*

  - Thyroid storm is a relatively uncommon event
  - Nursing care is focused on preventing life-threatening complications
  - Requiring:
    - Frequent and thorough patient assessments
    - Quick and effective interventions when indicated

- 2 hours later after esmolol, acetaminophen for fever and fluids for hydration, Patient A's status is much improved.
  - Thyroid suppressive treatment with Propylthiouracil (PTU) is initiated

- Thyroid storm can be deadly. Positive outcomes require successful management of the symptoms of sympathetic stimulation.

Endocrine Disorders

• Patient "B" – Post-op implanted defibrillator placement

- VS
  - B/P – 90/40
  - HR – 58; Sinus Brady
  - RR – 12
  - O2 sat – 92% on 50% BiPAP
  - Temp – 35.4 (95.8F)
- Other information
  - Altered LOC
  - Drowsy
  - Disoriented to time and place
- PMH
  - Ventricular dysrhythmias controlled with amiodarone

Hypothyroidism

Hyperthyroidism

What is the problem?

Endocrine Disorders

• Patient "B" – Post-op implanted defibrillator placement

- VS
  - B/P – 90/40
  - HR – 58; Sinus Brady
  - RR – 12
  - O2 sat – 92% on 50% BiPAP
  - Temp – 35.4 (95.8F)
- Other information
  - Altered LOC
  - Drowsy
  - Disoriented to time and place
- PMH
  - Ventricular dysrhythmias controlled with amiodarone

Hypothyroidism

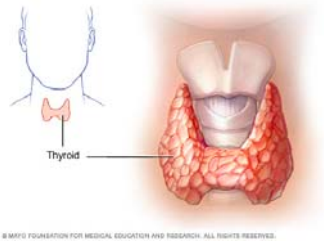
~~Hyperthyroidism~~

With Myxedema Coma

Symptoms related to inadequate or absence of thyroid hormone.

Endocrine Disorders
• *Hypothyroidism*

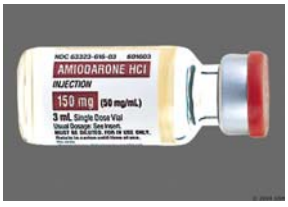
- Thyroid gland damage
  - Inflammation of the thyroid gland causes damage and death to the thyroid tissue
  - Autoimmune thyroiditis is a common form of thyroid inflammation by one's own immune system
    - Hashimoto's Thyroiditis
- Surgical Removal
  - A portion or all of the thyroid tissue is removed. If the remaining tissue cannot produce thyroid hormone – hypothyroidism results



Endocrine Disorders
• *Medications that influence thyroid function*

- Thyroid inhibitors
  - PTU
  - Methimazole (Tapazole)
  - Iodine in large doses

- Other medications
  - Sulfonamides
  - Salicylates
  - Lithium
  - Amiodarone



- Consult with the provider and pharmacy for any medications which might impact thyroid function
- Expect to assess thyroid function

Endocrine Disorders

- *Patient "B" – Post-op implanted defibrillator placement*

- Which of the assessment finding are indicative of hypothyroidism?
- Choose all that apply -

- Altered LOC
- Bradycardia
- Hypothermia
- Diarrhea

Endocrine Disorders

- *Patient "B" – Post-op implanted defibrillator placement*

- Which of the assessment finding are indicative of hypothyroidism?
- Choose all that apply –

- **Hypothyroidism is often missed – the signs and symptoms are similar to those for clinical depression**

- ✓ Altered LOC
- ✓ Bradycardia
- ✓ Hypothermia
- Diarrhea

Endocrine Disorders
• *Hypothyroidism – Myxedema Coma*

- Myxedema Coma is the acute manifestation of hypothyroidism due to diminished or absence of thyroid hormone and the presence of major stressors such as:
  - MI
  - General anesthesia
  - Surgery
  - Systemic Infection
  - Respiratory failure

- Myxedema coma happens most often in women. These patients are managed in critical care

Endocrine Disorders
• *Myxedema Coma*

- Lack of sufficient circulating thyroid hormone
  - Severely depressed metabolic rate
  - Insufficient oxygen delivery to tissue and cells
  - Hypoxia at the cellular level
  - Metabolic acidosis

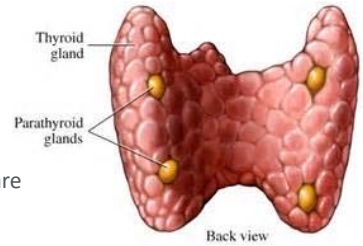
- Systemic effects
  - LOC – decreased LOC – to coma
  - Body temp – hypothermia
  - Hemodynamics – low HR, B/P, and cardiac output
  - Respiratory – resp failure causes resp acidosis may require mechanical ventilation
  - Fluids – decreased urine output, positive for edema, creates fluid and electrolyte imbalances
  - Labs – low T_4 and high TSH. T_3 will not be reduced until severe hypothyroidism is present

Endocrine Disorders
• *Myxedema Coma*

- Patient with myxedema coma will experience multi-system failure
- Focus of care will be to support these systems.
  - Medications – thyroxine or liothyronine; glucocorticoids may help the body respond to the thyroid hormone
  - Respiratory support – may require non-invasive or mechanical ventilation
  - Cardiac support – bradycardias and blocks; may require atropine or temporary pacing; pressors if needed to maintain cardiac output
  - Temperature control – warm blankets and other measures to slowly increase the patients temperature
  - Skin care – measures in place to increase mobility and reduce risk for pressure ulcers
  - Education – patient education regarding hypothyroidism and the prevention of future episodes of myxedema coma

Endocrine Disorders
• *Parathyroid Gland*

- Overactivity of the gland or underactivity of the gland
- Parathyroid glands produce parathyroid hormone (PTH)
- Necessary for calcium and phosphorus regulation
- In critical care, parathyroid dysfunction usually presents as hypocalcemia or hypercalcemia



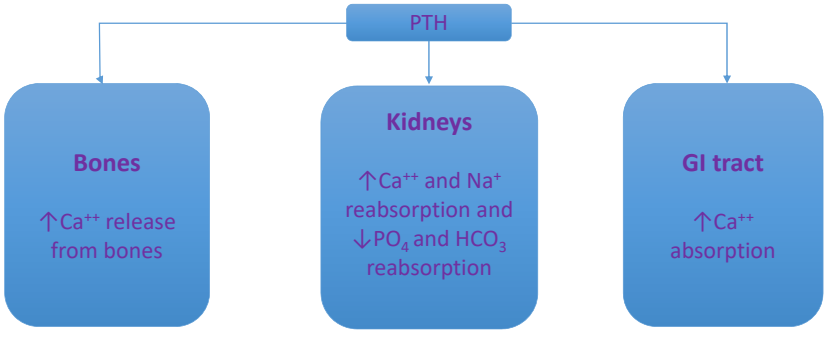
- Normal PTH – 10-55 pg/ml
- Levels are not routinely assessed in critical care

Endocrine Disorders
• *Parathyroid Hormone level imbalances*

- **Hypoparathyroidism symptoms**
  - Low Ca^{++} levels
  - Muscle cramps
  - Muscle aches
  - Muscle spasms especially around the mouth
  - Tingling and numbness lips and fingertips
  - Patchy hair loss
  - Brittle nails
  - Dry skin
- **Hyperparathyroidism symptoms**
  - High Ca^{++} levels
  - Osteopenia and kidney stones
  - c/o's of recent nausea and vomiting
  - Muscle weakness
  - Quickness to tire

Endocrine Disorders
• *Hyperparathyroidism*

- Result of parathyroid glands secreting too much PTH.
  - Primary hyperparathyroidism results in hypercalcemia



Endocrine Disorders - Hyperparathyroidism
• *Signs and Symptoms*

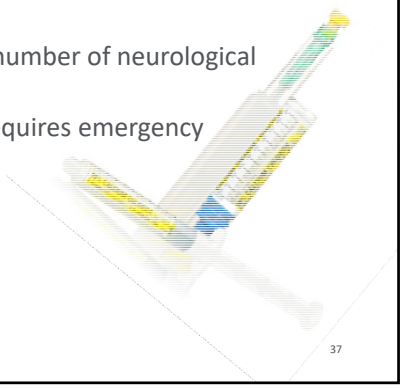
- The length of time and severity of the hyperparathyroidism will determine the degree of symptom severity
  - Osteoporosis
  - Kidney stones
  - Bone and joint pain
  - Depression
  - Forgetfulness
  - Nausea and/or loss of appetite
- Elevated calcium levels may cause
  - Smooth muscle relaxation
  - Increased cardiac contractility
  - Cardiac dysrhythmias – bradycardia and block

Endocrine Disorders - Hyperparathyroidism
• *Interventions and evaluations*

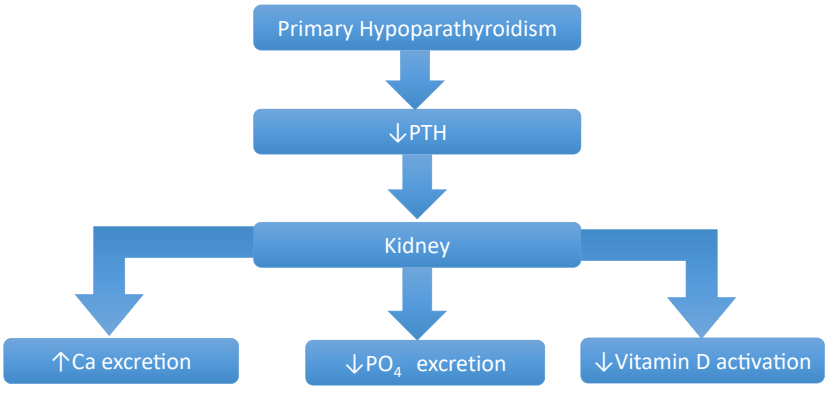
- Fluid and diuretic therapy
  - Increase renal excretion of calcium with large volumes of isotonic solution and diuretics
- Monitor patient response
  - Accurate I & O
  - Assess lung sounds
  - Cardiac dysrhythmias – be prepared to treat
- Surgical gland removal
  - Indicated for patient experiencing severe signs and symptoms due to hyperparathyroidism

Endocrine Disorders
• *Hypoparathyroidism*

- An uncommon condition associated with inadequate levels of circulating PTH causing hypocalcemia
- Usually iatrogenic – unintended removal of parathyroid or impaired blood flow to the gland
- May be an autoimmune issue related to low magnesium levels
- Vitamin D may also be low.
- Rate calcium level drops correlates to the number of neurological symptoms present
- Hypoparathyroidism may be so severe it requires emergency intervention to treat the hypocalcemia



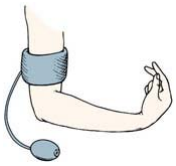
Endocrine Disorders
• *Hypoparathyroidism*



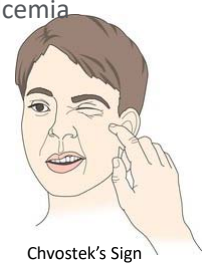
Endocrine Disorders - Hypoparathyroidism
• *Hypocalcemia*

- The signs and symptoms are based on the levels of calcium in the blood.
- Mild hypocalcemia – patient may not be aware of any symptoms
- Moderate hypocalcemia – numbness around the mouth, fatigue and weakness
- Severe symptoms may include tetany, laryngospasm or seizures.

- Patients with severe Hypoparathyroidism will exhibit positive Trousseau's and Chvostek's signs.
- Trousseau's may become positive before any other symptoms of hypocalcemia are exhibited



Trousseau's Sign



Chvostek's Sign

Endocrine Disorders - Hypoparathyroidism
• *Lab Values*

Endocrine Disorders - Hypoparathyroidism
• *Patient Management*

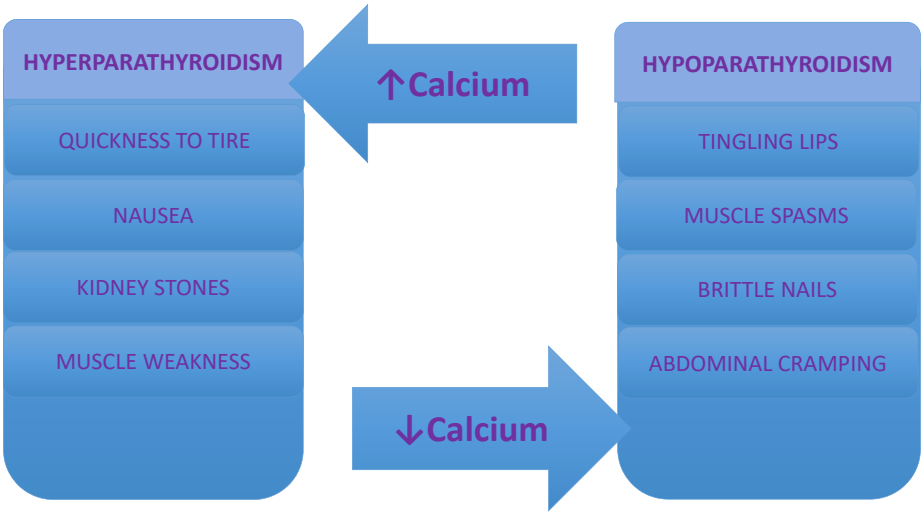
- Calcium
  - IV Calcium Chloride, Calcium Gluconate or Calcium Gluceptate
  - Check calcium levels frequently to avoid hypercalcemia
- Vitamin D
  - Be cautious with Vitamin D administration
  - Overdosage of Vitamin D can cause irreversible kidney damage
- Airway
  - Must be monitored and secured as necessary
- Seizure precautions
  - Seizure precautions should be initiated
- Monitor
  - Cardiac dysrhythmias
  - Intake and output
  - Assess Ca^{++}, PO_4^{3-}, K^+, and Mg^{++} levels
  - Monitor for Chvostek's and Trousseau's sign

Hyperparathyroidism and Hyperparathyroidism
• *Assessments unique to each diagnosis*

HYPERPARATHYROIDISM		HYPOPARATHYROIDISM
QUICKNESS TO TIRE	MUSCLE SPASMS	TINGLING LIPS
	NAUSEA	
	KIDNEY STONES	
	BRITTLE NAILS	
	ABDOMINAL CRAMPING	
	MUSCLE WEAKNESS	

Hyperparathyroidism and Hyperparathyroidism

- *Assessments unique to each diagnosis*



Endocrine Disorders

- *Parathyroid Hormone level imbalances*

- Patient A – recently had a thyroidectomy for cancer
- C/O's:
  - Frequent muscle spasms
  - Cramping
  - Brittle nails
  - Headaches
  - Ca^{++} level 5.4 mg/dl
- Recently reports issues with memory problems since surgery

Hypoparathyroidism

Hyperparathyroidism

What is the problem?

Endocrine Disorders

- *Parathyroid Hormone level imbalances*

- Patient A – recently had a thyroidectomy for cancer
- C/O's:
  - Frequent muscle spasms
  - Cramping
  - Brittle nails
  - Headaches
  - Ca^{++} level 5.4 mg/dl
- Recently reports issues with memory problems since surgery

Hypoparathyroidism may require long-term calcium supplementation

Hypoparathyroidism

~~**Hyperparathyroidism**~~

Management of this patient is focused on correcting and maintaining the calcium levels. Emergent treatment of hypocalcemia requires IV calcium replacement.

Endocrine Disorders

- *Parathyroid Hormone level imbalances*

- Patient B – recently has had repeated trips to ED for kidney stones
- C/O's
  - Frequent kidney stones
  - Ca^{++} level 15.4 mg/dl
- Referred to endocrinologist for workup

Hypoparathyroidism

Hyperparathyroidism

What is the problem?

Endocrine Disorders
• *Parathyroid Hormone level imbalances*

- Patient B – recently has had repeated trips to ED for kidney stones
- C/O's
  - Frequent kidney stones
  - Ca^{++} level 15.4 mg/dl
- Referred to endocrinologist for workup for hyperparathyroidism

In severe cases of hyperparathyroidism – surgical removal of the parathyroid gland is necessary

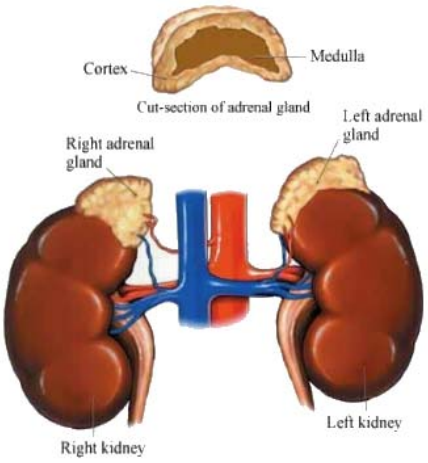
~~Hypoparathyroidism~~

Hyperparathyroidism

Emergency management of this patient is focused on administration of isotonic fluids and diuretics.

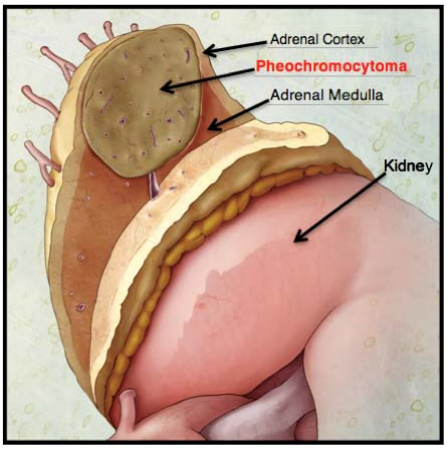
Endocrine Disorders
• *Adrenal Gland*

- Located on top of each kidney
  - Composed of two separate organs
    - Cortex
    - Medulla
  - Each organ
    - Secretes separate hormones
    - Regulated by different mechanisms
- Cortex – 80% of gland
  - Produces mineralcorticoids, glucocorticoids and gonadocoricoids
- Medulla – 20% of gland
  - Produces epinephrine and norepinephrine



Endocrine Disorders – Adrenal Gland
• *Pheochromocytoma*

- Tumor in the medulla of the adrenal gland
  - Usually benign
  - Large amounts of epinephrine and norepinephrine released
    - Increased metabolic rate
    - Hyperglycemia
    - Constant state of fight or flight
- **Left untreated -**
  - Severe HTN
    - Hypertensive encephalopathy
    - Cardiomyopathy
    - Kidney failure
    - Death



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Endocrine Disorders – Adrenal Gland
• *Pheochromocytoma*

- Patient "A" is admitted with:
  - Hypertensive crisis
  - PMH
    - Headaches
    - HTN
    - Bilateral rentinal hemorrhages
  - Tx HTN for 4 months
    - ARB
    - Loop diuretic
    - Calcium channel blocker
- Has yet to achieve B/P control with medications

- **In addition to HTN and headaches what other symptoms would you expect to see?**

  - Tremors and anxiety
  - Hypoglycemia
  - Unexplained abdominal and/or chest pain
  - Profound sweating
  - Tachycardia with palpitations

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Endocrine Disorders – Adrenal Gland
• *Pheochromocytoma*

- Patient "A" is admitted with:
  - Hypertensive crisis
  - PMH
    - Headaches
    - HTN
    - Bilateral rentinal hemorrhages
  - Tx HTN for 4 months
    - ARB
    - Loop diuretic
    - Calcium channel blocker
- Has yet to achieve B/P control with medications

- **In addition to HTN and headaches what other symptoms would you expect to see?**

✓ Tremors and anxiety
• Hypoglycemia
✓ Unexplained abdominal and/or chest pain
✓ Profound sweating
✓ Tachycardia with palpitations

Signs and symptoms are the result of hyperactive sympathetic response. Other signs might be hyperglycemia, flushing or pallor

Endocrine Disorders – Adrenal Gland
• *Pheochromocytoma*

- Diagnostic tests –
  - ~95% will have elevated serum catecholamine levels
  - 24 hour urine collection is positive if the catecholamine levels are more than double the normal level
  - CT scan may be helpful in localizing the tumor

Test	Typical Findings
Blood Test	• Elevated catecholamines
Urinalysis	• Elevated fractionated metanephrines (catecholamine metabolites) • Elevated creatinine

Endocrine Disorders – Adrenal Gland
• *Pheochromocytoma – Medical Management*

- Most pheochromocytomas require surgical removal
- Prior to surgery – the focus of medical management is on the control of HTN and observing for S/S of severe sympathetic stimulation
- Treatment includes
  - Alpha blockers for vasodilation – but there are few true alpha blockers
  - Beta blockers for negative chronotrope and inotrope action – reduce HR and contractility, also help manage cardiac dysrhythmias
  - Combo alpha/beta blockers – for vasodilation and some reduction in HR and contractility
  - Calcium channel blockers – for those with ineffective response to alpha or beta blockers
  - Metrosine – another antihypertensive that may be used

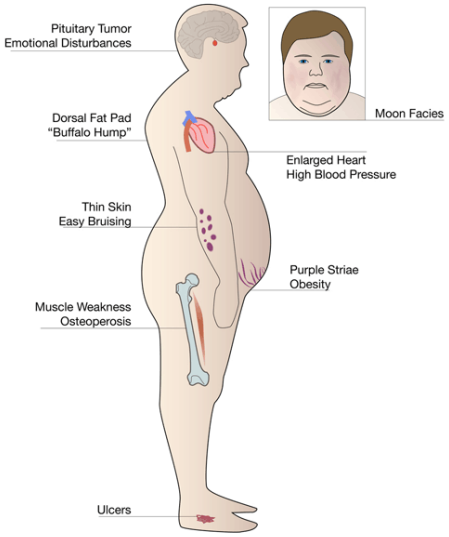
Endocrine Disorders – Adrenal Gland
• *Pheochromocytoma Removal – Post-operative care*

- Post surgical removal of the tumor
  - Typically admitted to Critical Care
    - Blood pressure monitoring
    - Fluid volume management
  - Other post-operative management includes
    - Pain control
    - VTE prophylaxis
    - Early mobilization
    - Atelectasis prevention
- Be prepared to treat hypotension
- Patient "A":
  - After surgery
    - After a 3 day stay was discharged to home
    - Annual follow-up
      - assess recurrence of tumor and monitor plasma metanephrines levels
- Self-management of care includes
  - Monitoring B/P routinely

Endocrine Disorders – Adrenal Gland
• *Cushing's Syndrome*

- Relatively uncommon condition that occurs when the body has prolonged exposure to high levels of cortisol

- Causes –
  - Excessive amount of cortisol-like medications
  - A tumor either –
    - Produces cortisol
    - Makes the adrenal gland increase cortisol production



Endocrine Disorders – Adrenal Gland
• *Cushing's Syndrome*

Any condition that leads to excess production of cortisol can cause Cushing's syndrome

- Primary
  - Damage to the adrenal cortex has occurred
    - Hypercortisolism results

- Secondary
  - Hypersecretion of ACTH from the anterior pituitary gland
  - Non-pituitary tumor capable of producing ACTH

- Most common cause in Critical Care the administration of exogenous steroids

Endocrine Disorders – Adrenal Gland

- *Cushing's Syndrome – treatment depends on the source or cause of excess cortisol*

Source/Cause of Cortisol Excess	Treatment
ACTH-producing tumor of the pituitary	Medication, surgery or radiation
Adrenal gland tumor	Removal of the tumor; other remaining adrenal gland will produce sufficient cortisol
Exogenous steroids	Slow steroid taper to prevent adrenal insufficiency

Endocrine Disorders – Adrenal Gland

- *Cushing's Syndrome*

- Patient "B" admitted for urosepsis.
  - On steroids for the last 6 months
  - Gained 35 pounds
  - Extremely fatigued
  - VS
    - B/P 102/34
    - HR 109 S. Tacycardia
    - RR 28 labored
    - SPO_2 92% on 4L/min NC
    - Temp 35.4 (95.8 F)
    - Glucose 244 mg/dL
    - WBC 14,000 mm^3
- DX – Cushing's syndrome
- Orders to initiate insulin gtt if next glucose is >180 mg/dL

- Of the choices below what further orders do you expect?

- **Administer IV steroids**
- **Administer 2 units of PRBC's**
- **Administer diuretics and hypertonic fluid resuscitation**
- **Remove both adrenal glands**

Endocrine Disorders – Adrenal Gland
• *Cushing's Syndrome*

- Patient "B" admitted for urosepsis.
  - On steroids for the last 6 months
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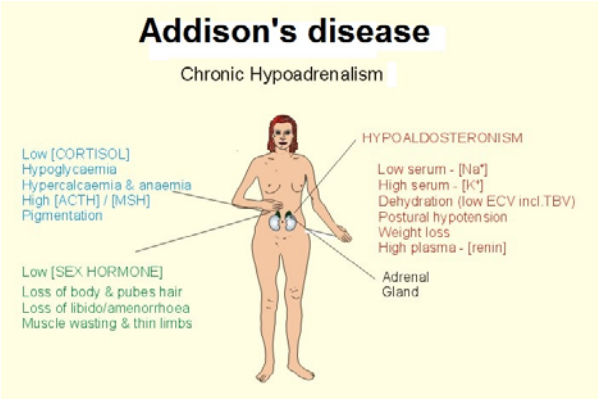
- next glucose is >180 mg/dL

- Of the choices below what further orders do you expect?

✓ **Administer IV steroids**
- Administer 2 units of PRBC's
- Administer diuretics and hypertonic fluid resuscitation
- Remove both adrenal glands

Patients on long term steroids must be maintained to prevent the effects of abrupt withdrawal. Once recovered from the illness steroids can then be tapered off

Endocrine Disorders – Adrenal Gland
• *Addison's Disease*



- A disorder of hyposecretion of the adrenal gland
  - Addisonian Crisis is rare
    - Inadequate production of
      - Cortisol
      - Aldosterone

Endocrine Disorders – Adrenal Gland
• *Addison's Disease – 3 different patient presentations*

- Adrenal Hyposecretion
  - Stress event
  - Underproduction of cortisol
  - Supplemental steroids until acute episode of adrenal insufficiency resolves
- Addison's Disease
  - Treated with long-term steroids in past – weaned off
  - Crisis event
    - Adrenal gland unable to produce cortisol
    - Steroids required to stabilize condition
- Addisonian Crisis
  - Disease treatment requires continuous steroids indefinitely
  - Stops taking steroids
  - Now in acute crisis requiring steroids

Endocrine Disorders – Adrenal Gland
• *Addison's Disease*

- Primary
  - Rare
  - Adrenal cortex is damaged
  - Result
    - Hyposecretion of cortisol
    - Sometimes hyposecretion of aldosterone as well
- Secondary
  - Prolonged use of glucocorticoid containing medications
    - Suppresses the glands ability to produce hormone
    - Abrupt cessation results in deficiency
  - Pituitary gland damage
    - Damage leads to reduced ACTH production
      - Reduced cortisol production

Endocrine Disorders – Adrenal Gland

- *Addison's Disease*

- Patient has presented after abruptly stopping her steroids.

- **What symptoms do you expect to see?**

- **Hypoglycemia**
- **Hypotension**
- **Hyperkalemia**
- **hypervolemia**

Endocrine Disorders – Adrenal Gland

- *Addison's Disease*

- Patient has presented after abruptly stopping her steroids.

- **What symptoms do you expect to see?**

Hypoglycemia, hypotension and hyperkalemia are the triad of symptoms seen with Addison's disease. This is because of the lack of cortisol and aldosterone.

- ✓ **Hypoglycemia**
  - ✓ **Loss of cortisol**
- ✓ **Hypotension**
  - ✓ **Loss of cortisol and aldosterone**
- ✓ **Hyperkalemia**
  - ✓ **Loss of aldosterone**
- **hypervolemia**

Endocrine Disorders – Adrenal Gland
• *Addison's Disease*

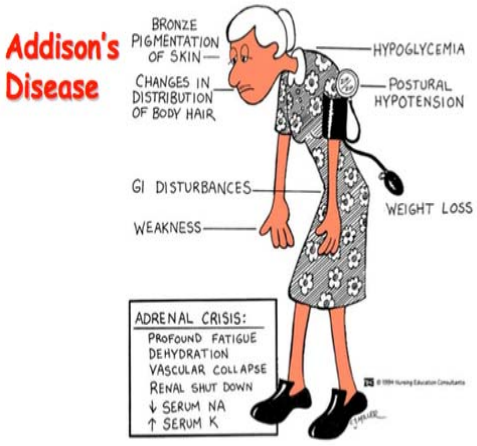
Addison's Disease
- Weakness
- Anorexia
- Fatigue
- Hyperpigmentation of skin and mucosa
- Irritability and depression

Acute Addison's Disease
- Hyponatremia due to sodium loss
- Decreased extracellular fluid volume
- Hyperkalemia]
- Hypovolemia leading to cardiac shock

Addisonian Crisis
- Hypertension
- Hypoglycemia
- Severe pain in back, legs, abdomen
- Severe nausea, vomiting and diarrhea
- Dehydration and hypotension that progresses to shock

Endocrine Disorders – Adrenal Gland
• *Addison's Disease - treatment*

- Fluid resuscitation
  - D5NS
  - Vasopressors for hypotension
  - Treat electrolyte imbalances
    - Hyponatremia
    - Hyperkalemia
    - Hypercalcemia
  - D50W for hypoglycemia
  - Administer steroids
  - Assess/intervene
    - Monitor V/S, labs and I/O closely



Endocrine Disorders – Adrenal Gland
• *Addison's Disease - treatment*

- Patient now
  - B/P 80/40
  - HR 114
  - Glucose 84
  - Sodium 124
  - Potassium 6.4

  - **What do our next interventions focus on?**

- Serial CXR
- Administration of steroids
- B/P management
- Electrolyte management
- Glucose administration
- Nutritional support

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Endocrine Disorders – Adrenal Gland
• *Addison's Disease - treatment*

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