Objectives
Upon completion of the lecture the participant will be able to:

• 1. List 2 non-excretory functions of the kidney
• 2. Describe the action of the antidiuresis hormone
• 3. Define hypochloremic metabolic alkalosis
• 4. Describe 4 methods of reducing serum potassium in hyperkalemia
Kidney Erythropoietin Prostaglandins

- Na/H2O retention
- Plasma volume
- Venous load (preload)

Kidney

- Aldosterone

Prostaglandins

- Renin
  - Angiotension I-II
  - Vasoconstriction

Blood Pressure

- Red cell mass
- Cardiac output
- Peripheral resistance
Function of the Kidney

• Nonexcretory functions
  ▪ Renin
  ▪ Erythopoietsin
  ▪ Metabolizes Vit D
  ▪ Degrades insulin
  ▪ Produces prostaglandins
Endogenous pathway
Skin UV exposure

Vit D

Liver
(25, OH D)

Kidney
(1,25, OH D)

Diet, GI track
Bile acids
Arachidonic Acid Pathways

- Phospholipids
  - Lipooxygenase pathway
    - Leukotrienes
  - Cyclooxygenase pathway
    - Prostaglandins
      - Cyclooxygenase inhibitors
      - Prostaglandin synthase inhibitors
        - Indomethacin
        - Neoprofen
Functions of the Kidney

• Excretory functions
  ▪ Maintains plasma osmolarity
  ▪ Maintains electrolyte balance
  ▪ Maintains water balance
  ▪ Excretes nitrogenous end products
1. Note relationship to vascular lines
2. Gross anatomy of the GU system
3. Adrenal glands
1. Gross anatomy of the kidney
   Cortex
   Medulla
   Renal sinus/pelvis
   Ureter
   Bladder
   Urethra
2. Urine is produced by 9-10 weeks gestation
1. Low renal blood flow due to high vascular resistance
   4-6% CO in first 12 hours
   8-10% first week of life
   20-24% CO - adult values

2. Low renal blood flow = low glomerular filtration rate

3. Nephrogenesis complete at 34 weeks gestation

4. Nephron - Filtration Reabsorption Secretion
Concentration of Urine

- Afferent to Efferent
- Enters via glomerulus and is filtered into Bowman’s capsule
- Proximal tubule
  Na, H₂O, AA, glucose
  NaHCO₃ - reabsorbed

Concentration of Urine

- Filtrate flows through the descending and ascending Loops of Henle
- Reabsorption of solutes and water
- Na, H₂O, glucose, AA returned to circulation
Distal tubule and collecting duct

ADH

Aldosterone

Concentrated Urine

Renin Angiotensin Aldosterone Loop

Antidiuretic Hormone

[Diagram showing the mechanism of Antidiuretic Hormone (ADH) with labeled parts: Osmoreceptors, Hypothalamus, Baroreceptors, Aortic arch, Carotid sinus, Posterior pituitary, Kidney, Circulation, ADH or vasopressin, Osmotic pressure, Hypothalamus, Baroreceptors, Aortic arch, Carotid sinus, I(blood pressure), ADH or vasopressin, Posterior pituitary, Kidney, Circulation, (Increased reabsorption of water), (I'blood volume and blood pressure), (I'blood volume and blood pressure).]
Acid Base Balance

- Lungs - Kidneys - Blood buffers
- Renal response to acidosis
- Renal response to alkalosis

- Chronic lung disease and the use of diuretics
  - Hypochloremic metabolic alkalosis
Atrial Natriuretic Peptide

- Synthesized by the walls of the right atrium
- Secreted in response to elevated pressure in the right atrium
- Increases urine production
  - Inhibits Na reabsorption
  - Inhibits secretion of ADH
- Felt to contribute to initial diuresis in newborns
Lasix
- Loop diuretic
- Blocks reabsorption of Cl
- Increases renal blood flow
- Impairs Ca^{++} and Mg^{+} reabsorption
- Prolonged 1/2 life in neonate
- Onset of action 1 hour
- Duration of action 6 hours
Common Medications & Renal function

- Thiazides
  - Acts of distal tubules
  - Less potent
  - Augments $K^+$ wasting
  - Stimulates $Ca^{++}$ reabsorption
  - Onset of action 2 hours
  - Peak action 3-6 hours
Cl⁻ channel blocker, Cl⁻ stays in filtrate

Na⁺ is reabsorbed and K⁺ is given in exchange

Na & H₂O follow the Cl⁻

Glucose
Common Medications & Renal function

- Aldactone
  - K\(^+\) sparing
  - Competitive inhibition of aldosterone
  - Increased excretion of Na\(^+\)
- Theophylline/Caffeine
  - Increases UOP
  - Inhibits Na\(^+\) reabsorption in proximal and distal tubules
Dopamine

- Dilates renal arteries (dopaminergic effect)
- Increases GFR by inhibition of angiotension II
- Specific renal dose (1-6 mcg/kg/min)
- High doses causes vasoconstriction
Common Medications & Renal function

- **Indocin**
  - Prostaglandin synthetase inhibitor
    - Inhibits cyclooxygenase pathway and prevents PGE synthesis from arachidonic acid
  - Prostaglandins vasodilate kidneys
  - Decreases GFR
  - Increases ADH secretion - may lead to SIADH
Acute Renal Failure

- Prerenal - a relative state of hypoperfusion in an otherwise normal kidney
  - Hypotension
  - Hypovolemia
  - Hypoxemia

Etiology

- RDS
- CHF
- Asphyxia
- Septic shock
- Hemorrhage
- Surgery
Acute Renal Failure

• Intrinsisic Renal Failure
  ▪ Cellular damage to the glomerulus, tubules and/or collecting system
  ▪ Conditions causing ARF
    • Conditions resulting in ARF
      ▪ ATN (acute tubular necrosis)
      ▪ Infection/Inflammation
      ▪ Congenital anomalies of the kidneys
      ▪ Vascular conditions
Acute Tubular Necrosis

- Ischemia/injury to kidney
- Cellular damage
- Tubule epithelial cells slough into the tubule lumen
- Cr and other products re-enter circulation

ARF vs SIADH

- Little to no urine output
- Increasing BUN/Cr
- Electrolye disturbances

- Little to no urine output
- Normal BUN/Cr
- Hyponatremia with a normal potassium
- Low serum osmolality
- High urine osmolality
Fluids & Electrolytes

- Body water distribution
  - ICF
  - ECF - at term about 75% of weight is water

- Adjustments after birth
  - Physiologic contraction of ECF volume resulting in a diuresis and a drop in post natal weight
    - Term 5-10%
    - Premie 20%
Regulation of fluid balance

- Renal mechanisms - nephrons not mature or fully present until 34 weeks
- Increased renal vascular resistance
- May have delayed response to fluid boluses
- Reabsorption of Na⁺, NaHCO₃, and glucose is limited
- Antenatal steroids will decrease ISWL
Fluids & Electrolytes

• Normal urine output 1-4cc/kg/hr, with higher rates during ECF contraction
• Factors affecting ISWL
  - Extreme prematurity
  - <1 week of age
  - Hyperthermia
  - High ambient temp
  - Low humidity
  - Radiant warmer
  - Phototherapy
  - Convection loss
Fluids & Electrolytes

- Fluid therapy
  - Initial total fluids
  - Growth and development
  - Serum Na^+
- Type of IV fluid
  - Glucose (GIR)
  - Electrolytes
Fluids & Electrolytes

Day 1

- Term
  - 60-80 cc/kg/d
  - No lytes
  - D10W
  - GIR - vol x % = grm/d, divide by weight and 1.44 to obtain the mg/kg/min

- Preterm
  - 80-100 cc/kg/d
  - No lytes, Ca++
  - D5W - D10W
Fluids & Electrolytes

- Day 2
  - Term
  - Preterm
  - +20cc/kg/day
  - Start TPN
  - Add Na⁺/Ca²⁺
  - +/- K⁺ depending on UO
  - Feeds vs TPN
Fluids & Electrolytes

- Day 3
  - Term
    - +20cc/kg/day
  - Preterm
    - Full electrolytes as needed, goal is 150cc/kg/day for IVF and 180cc/kg/day for enteral feeds
  - Goal GIR 11-14 grm/kg/day
Fluids & Electrolytes

- Assessment of fluid balance
  - Body weight
  - Urine volume
  - Specific gravity
  - Physical assessment
  - Laboratory information
    - Serum Na$^+$ osmolality
    - BUN/Cr Hematocrit

THIRD SPACING
Sodium

Hyponatremia - Serum Na⁺ <130
Prematurity
Dilutional hyponatremia
Renal losses
Inadequate intake during periods of rapid growth
Hypernatremia

Serum Na$^+$ > 150

Excessive ISWL

Hypovolemic/dehydration
Potassium

Hypokalemia - assessed indirectly
  Urine losses  Inadequate intake
  GI losses     Metabolic alkalosis
  Medications (glucose, bicarb, diuretics)

Clinical presentation - EKG, ileus

Correction
  Potassium infusions
  Potassium sparing medications

Complications - too rapid infusion
Fluids & Electrolytes

Hyperkalemia
- Extreme prematurity
- Metabolic acidosis
- Adrenal insufficiency

Clinical presentation - EKG

Correction - Check IVF
- CaGluconate
- Glucose/Insulin

- Bicarb/Tham
- kayexalate
Calcium

**PTH** increases Ca by mobilizing Ca from the bone, increases absorption from GI and decreases urinary losses. Released by low serum Ca and Mg levels.

**Vit D** acts with PTH to increase Ca by increasing absorption of Ca and Phos from the GI tract and bone.

**High Phos** level will inhibit the absorption of Ca
Metabolic bone disease - osteopenia of prematurity (Rickets)
Prematurity
Unsupplemented Breast milk in a premie
Prolonged use of diuretics
Prolonged use of TPN/lack of enteral feedings
Acid Base Balance

- pH
- Buffering system
- Lung regulation
- Kidney regulation
- Compensation
Metabolic Acidosis

• Why is my baby acidotic
  ▪ Are we giving too much acid
  ▪ Is he/she losing base
  ▪ Is the baby making acid

• 3 Types of acid
  ▪ Lactic acid
  ▪ Ketoacids
  ▪ Organic acids
Metabolic Alkalosis

• Why is my baby alkalotic
  ▪ Loss of acid
  ▪ Too much substrate
• Hypochloremic metabolic alkalosis