Electrolytes: A Review and Refresher

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Composition of Body Fluids

- **Total Body Water (~ 40 liters)**
  - Approximately 60% of body weight (males)
  - Approximately 50% of body weight (females)
  - Approximately 75% in newborns (less body fat)
  - Obese/elderly decrease above by 10%

To calculate TBW (L) = Current wt (kg) x 60%
Composition of Body Fluids

- **Intracellular Fluids**
  - inside the cell

- **Extracellular Fluids**
  - Intravascular- plasma
  - Interstitial-surround all cells
    - Some interstitial fluid is transcellular or under the influence of metabolic activity (ie fluid from the respiratory and GI tract, CSF, lymph, pleural, pericardial, peritoneal, synovial, and gland secretions)
A 70 kg male has 40-42 liters of fluid in the following distribution:

- **Intracellular fluid** = 28 L

- **Extracellular fluid** = 14 L
  - Interstitial = 11 L
  - Intravascular = 3 L

  - Total blood volume = 5 liter (2 liters consist of RBCs)
Daily Intake & Output

Intake 2,500 ml
- Liquids 1,500 ml
- Water in food 700 ml
- Water of oxidation 250 ml

Output 1,400-2,300 ml
- Respiration & Perspiration (insensible) 600-900 ml
- Urine 800-1500 ml
- Stool 250 ml
Fluid Movement

Fluid movement is constant and is influenced by:

- membrane permeability
- colloid osmotic pressure (plasma proteins)
- hydrostatic pressure
Fluid Movement

Membrane Permeability

- Transport of substances across the cell membrane depends on the substance to be transported
  - **Passive** transport does not require energy and is accomplished by osmosis, diffusion or the force of hydrostatic pressure
  - **Active** transport requires the expenditure of metabolic energy by the cell. (Larger and electrically charged particles ie Na/K pump)
Fluid Movement

Starling’s Law

- pressure difference in the arterial and venous ends of the capillaries influence the direction and rate of water and solute movement

Forces favoring filtration
- Forces opposing filtration

Net Filtration
Fluid Movement

Hydrostatic Pressure (capillary bed pressure)

- is the mechanical force of water pushing against a membrane. In the intravascular space, it is the pumping action of the heart that generates this force.
- at the arterial end of the capillary, hydrostatic pressure forces water, sodium and glucose across the membrane into the interstitial space
Fluid Movement

Osmotic Pressure

- movement of fluid between the ICF and ECF is primarily a function of osmotic forces- plasma proteins pull water back into the vascular space at the venous end of the capillary bed
- measurement of solute concentrations in body fluids: osmolality mOsm/kg water
  osmolarity mOsm/liter (exogenous)
Renal Regulation

Kidneys are the most important regulators of volume and composition of body fluids.

Hormonal Control

- ADH
- Renin-angiotensin-aldosterone system
- Natriuretic factors
Serum Osmolarity
(Concentration)

- Normal 275 - 295
  - $>295 = \text{concentrated (dehydrated)}$
  - $<275 = \text{dilute (fluid overloaded)}$

- Serum Osmo = $2(Na) + \text{glucose} + \text{BUN}$
  - 18 2.8

- Serum Osmo = sodium x 2 (quick reference)
Serum Laboratory Findings
(Normal)

- Serum sodium: 135-145 mEq/L
- Serum osmolarity: 275-295 mOsm/L
- BUN: creatinine: 10:1 ratio
- Hematocrit (males 40-52% ~ females 37-46%)
- Total protein: 6.2-8.2 g/dL
Types of Acute Renal Failure

Figure 39.1. Causes of acute oliguria based on anatomic location of the problem.
So, why did Valerie spend all this time talking about fluid movement?

Solutions (liquid solvents) containing dissolved substances (solute) are classified according to their concentration (or tonicity):

- Hypertonic
- Isotonic
- Hypotonic
Intravenous Fluids

- Crystalloids
  - Hypertonic
  - Isotonic
  - Hypotonic

- Colloids
  - Albumin
  - Hetastarch
  - Dextrans
Crystalloids

- Isotonic
  - osmolarity equals serum
  - stays in the intravascular space thus expanding the volume
  - good choice for hydration
Crystalloids

Hypertonic

- solute concentration higher than the solute concentration of the serum
- infusion causes an increase in the solute concentration of the serum, pulling fluid from the interstitial space to the vascular space through osmosis
Crystalloids

- Hypotonic
  - lower solute concentration than the serum
  - infusion decreases the solute concentration in the vascular space into the intracellular and interstitial spaces where the solute concentration is higher
    - fluid moves into the cells
### IV Fluids - Crystalloids

<table>
<thead>
<tr>
<th>Isotonic</th>
<th>Hypertonic</th>
<th>Hypotonic</th>
</tr>
</thead>
<tbody>
<tr>
<td>LR (275)</td>
<td>D5½NSS (408)</td>
<td>½ NSS (154)</td>
</tr>
<tr>
<td>Ringer’s (275)</td>
<td>D5NSS (560)</td>
<td>.33%NaCl (103)</td>
</tr>
<tr>
<td>NSS (308)</td>
<td>D5LR (575)</td>
<td>D2.5%W (126)</td>
</tr>
<tr>
<td>D5W (260)</td>
<td>3% NSS (1,025)</td>
<td></td>
</tr>
<tr>
<td>Plasma-lyte (295)</td>
<td>7.5% NaCl (2,400)</td>
<td></td>
</tr>
</tbody>
</table>

(All numbers are mOsmo/l)
Volume Expansion

Increase in Plasma Volume

Increase in Interstitial Volume

1000  500  0  500  1000

D_5W (1L)

0.9% NaCl (1L)

5% Albumin (1L)

7.5% NaCl (250 mL)

(mL)
Colloids

- Synthetic, commercially prepared volume expanders containing polysaccharide molecules (sugar & starch)
- More effective for volume resuscitation in moderate to severe blood loss
  - Albumin (5% and 25%)
  - 10% Dextran 40 and 6% Dextran 70
  - Hetastarch (hespan)
  - 10% Pentastarch (hetastarch derivative)
Perioperative Fluid Therapy

-General considerations:
  - NPO status
  - Tendency for Na$^+$ and H$_2$O retention
  - Evaporative losses
  - Third spacing
Objectives for Fluid & Electrolyte Management

- Beware of treating a lab value!
- Fluid needs are not static - always re-assess
- Multiple fluid, electrolyte and acid/base abnormalities should be corrected in the following sequence:
  - Fluid and perfusion deficits
  - pH
  - K, Ca, and Mg abnormalities
  - Na and Cl abnormalities
# Blood and Body Fluid Volumes

<table>
<thead>
<tr>
<th>Body Fluid</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Body</td>
<td>600 ml/kg</td>
<td>500 ml/kg</td>
</tr>
<tr>
<td>Whole Blood</td>
<td>66 ml/kg</td>
<td>60 ml/kg</td>
</tr>
<tr>
<td>Plasma</td>
<td>40 ml/kg</td>
<td>36 ml/kg</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>26 ml/kg</td>
<td>24 ml/kg</td>
</tr>
</tbody>
</table>
Maintenance Guidelines

- Urine output and daily weights are useful in fluid and perfusion status.
- Ca, Mg, Phos, protein and MVI may be necessary after 5-7 days of IV fluids.
- Insensible losses increase 100-150 ml per 24 hours for each degree >37 C.
Electrolytes

- **Major intracellular electrolytes**
  - Potassium (cation)
  - Phosphorus (anion)

- **Major extracellular electrolytes**
  - Sodium (cation)
  - Chloride (anion)

  - Sodium is the determinant of osmolality (tonicity) since it is the major ECF cation
Daily Electrolyte Needs

- NaCl 80-120 meq
- K 50-100 meq
- Ca 1-3 gm
- Mg 20 meq
Hypernatremia

Either from a fluid deficit (water loss exceeds Na loss) or inadequate water intake

**Etiology:** impaired thirst or access to water, excessive renal or extrarenal losses of water without Na⁺ loss

**Symptoms:** extreme thirst, dry mucous membranes, dry flushed skin, tachycardia, low grade fever, hyperactive DTR, weakness/lethargy, oliguria or polyuria

- D5W fluid of choice to replace H2O deficits
Hypernatremia IVF Infusion

Rate

- 6 liter deficit: give D$_5$W (or NSS)
- Also need to give maintenance fluid requirements (replace renal and extra-renal losses).

- 161 mEq to 140 mEq Na$^+$ = 21 mEq
- 21 mEq / 0.5 mEq per hour = 42º (to correct)
  
  6 liters / 42º = 142 cc/hr

(also need to give maintenance fluid)
Hyponatremia

(Found in 10-15% of hospitalized patients)

- Decrease in measured serum Na concentration below 135 mEq/L
  - Mild 125-134 mEq/L
  - Moderate 110-124 mEq/L
  - Severe 100-109 mEq/L
- Evaluate serum osmolarity
- If osmolarity is hypotonic, evaluate volume status:
  - Hypervolemia
  - Euvolemia
  - Hypovolemia
Sodium Concentration in Body fluids

<table>
<thead>
<tr>
<th>Fluids Commonly Lost</th>
<th>Sodium Concentration (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine*</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>40</td>
</tr>
<tr>
<td>Gastric secretions</td>
<td>55</td>
</tr>
<tr>
<td>Sweat</td>
<td>80</td>
</tr>
<tr>
<td>Furosemide diuresis</td>
<td>75</td>
</tr>
<tr>
<td>Pancreatic secretions</td>
<td>145</td>
</tr>
<tr>
<td>Small bowel secretions</td>
<td>145</td>
</tr>
</tbody>
</table>

* Urinary sodium concentration varies according to daily sodium intake.
Hyponatremia - Serum Osmo

- Normal (275-295)
  - Isotonic Hyponatremia (Inc. Lipids, Inc. Proteins)
- Low (<275)
- High (>295)
  - Hypertonic Hyponatremia (Hyperglycemia, Mannitol)

Volume Status?

- Hypovolemic
  - Extrarenal Salt Loss
  - Renal Salt Loss
- Euvolemic
  - SIADH
    - Post op
    - Hypothyroidism
    - Water intoxication
- Hypervolemic
  - Edematous States
    - CHF
    - Liver disease
    - Renal failure
True Hyponatremia

- Hyponatremia is **ALWAYS** due to ingestion of water in excess of the amount the kidneys can excrete.
- **This occurs in 3 situations:**
  - Ingestion of water that overwhelms the kidney
  - Ingestion of normal amounts of water in kidney failure
  - Ingestion of normal amounts of water in the presence of ADH
Hyponatremia: Pathophysiology Overview

- Maximum daily urine volume depends on the daily solute load and minimum urinary concentration.
- Normal kidneys can dilute urine (to as low as 30-50mmol/L) and can excrete large amounts of water.
  - Can be determined directly by measuring the urine osmolality.
Hyponatremia

**ROS** - headache, muscle cramping, anorexia/nausea, nervousness

**Physical Exam** weight loss, altered mental status, dec. temperature, ST/hypotension, diminished DTR, pupillary abnormalities if impending herniation, Cheyne-Stokes respirations, tremors/ seizure/ coma

**Management**

\[
\text{Na deficit} = \text{nl TBW} \times (130 - \text{serum Na})
\]

**ACUTE**: replace 1-2 mEq/L/hr with NSS or 3\% NSS - SLOWLY!!! No > than 10 meq/24 hours

**CHRONIC**: replace 0.5mEq/L/hr with NSS
Summary of Management

Hypovolemic hyponatremia is treated by restoring volume with 0.9% saline. Patients are usually symptomatic from volume loss, not hyponatremia.

Hypervolemic hyponatremia is treated by identifying and treating the underlying disorder. Fluid restriction is also used to specifically correct the sodium concentration.
Potassium

Total Body K (mEq)

3400 mEq

Intracellular Potassium

70 mEq

Extracellular Potassium
Hyperkalemia

**Etiology:** excess intake K, renal insufficiency, tubular unresponsiveness to aldosterone, metabolic acidosis, MI widespread injuries (burns, trauma, crush, extensive surgery, infection), drugs such as ACE inhibitors, NSAIDS, heparin, K-sparing diuretic, digitalis toxicity, betablockers, insulin deficient

**Clinical symptoms:** malaise/ weakness, parasthesias/ numbness, hyperreflexia, bradycardia, dysrhythmias (cardiac arrest), hypotension

**ECG changes:** flattened or absent P waves, prolonged PR, wide QRS, peaked T waves followed by ST segment
Hyperkalemia Treatment

- Membrane stabilization
  - Calcium (Gluconate vs. chloride)
  - Immediate onset
- Transcellular shift
  - Insulin-dextrose
  - Sodium bicarbonate
- Enhanced clearance
  - Kayexalate
  - Loop diuretics
  - Hemodialysis
Hyperkalemia Management

1.) 1 amp Ca gluconate (cardioprotective)
2.) Ca gluconate (10%) IV infusion over 2-5 minutes to counteract the myocardial depressant effects of hyperkalemia- MUST be on a monitor/Needs repeat Rx
3.) D50W 1 amp and regular IV insulin 10-20 units (to facilitate K movement into the cells
4.) Bicarbonate 1 amp - drives K into the cells
5.) Aerosolized albuterol - good for uremic patients
6.) Kayexelate- a cation exchange resin (K moves into bowel space and Na moves into the bowel cells
7.) Dialysis if renal failure
Hypokalemia

**Etiology** - excessive excretion or inadequate intake of K Beta agonist bronchodilators with thiazide/loop diuretics, GI losses (NGT, diarrhea), alkalosis, magnesium depletion, insulin excess, licorace

**Clinical symptoms** - lethargy, N/V, leg cramps/muscle weakness, hyporeflexia, decreased bowel motility (ileus), cardiac dysrhythmias (PAC, PVC)

**ECG changes** - ST segment depression, flattened T waves, U waves, prolonged QT interval, AV Block, ventricular ectopy or ST
Hypokalemia Management

- Reverse underlying cause (diuretics)/ alkalosis
- Control extra renal losses
- Treat associated electrolyte disorders (low Mg)
- Standard 20 meq/100 ml NSS over 1 hour
  - 40 mEq/ 100 ml NSS may be used if K < 1.5 meq or serious arrhythmias
- Otherwise, give KCL 20-40 mEq PO q 2 - 24 hours. Check serum levels after each dose.
Potassium

Ekg

Decreasing $S_k$
- Flat T wave
- Prominent U wave
- Depressed S-T segment

Increasing $S_k$
- Normal
- Peaked T wave
- Prolonged P-R interval
- Absent P wave
- Ventricular fibrillation

TU
Calcium

8.5-10.5mg/dL
Ionized: 4.65-5.28mg/dL

- Most abundant electrolyte; 99% in bone
- Processes
  - Neuromuscular transmission
  - Smooth muscle contraction
  - Blood coagulation
- Protein binding
  - Unbound ionized fraction is physiologically active-
    amount depends on pH (eg. acidosis increases ionized
    CA and decreases bound CA)
  - Correct for albumin
    - Add 0.8 mg/dl for every 1 g/dl serum albumin below 4
    - Measure ionized calcium
Hypocalcemia

- Neuromuscular excitability
  - Hyperreflexia
  - Tetany
  - Seizures

- Cardiovascular effects
  - Ventricular ectopy
  - Hypotension
  - Decreased cardiac output
Hypocalcemia

<table>
<thead>
<tr>
<th>Table 43.2. Causes of Ionized Hypocalcemia in the ICU</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alkalosis</strong></td>
</tr>
<tr>
<td><strong>Blood transfusions (15%)</strong></td>
</tr>
<tr>
<td><strong>Cardiopulmonary bypass</strong></td>
</tr>
<tr>
<td><strong>Drugs:</strong></td>
</tr>
<tr>
<td>Aminoglycosides (40%)</td>
</tr>
<tr>
<td>Cimetidine (30%)</td>
</tr>
<tr>
<td>Heparin (10%)</td>
</tr>
<tr>
<td>Theophylline (30%)</td>
</tr>
</tbody>
</table>

Numbers in parentheses show the frequency of ionized hypocalcemia reported in each condition.
Hypocalcemia  Total Ca < 8.5/dL  
Ionized Ca <1.0 mmol/L

**Etiology** - Mg depletion, alkalosis (more Ca will bind to protein), blood transfusions, renal failure, Vitamin D deficiency, malabsorption syndromes/diarrhea, bypass surgery, drugs/diuretics, pancreatitis, hypoparathyroidism

**Clinical Symptoms** - hypotension, SB, heart failure/arrhythmia, digitalis insensitivity, QT prolongation, weakness/muscle spasms/laryngospasms/ hyperreflexia/ tetany/paresthesias/seizures

**Management** - (IV Rx only with symptoms or ionized Ca < 0.65 mmol/L) 10% CaCl or CaGluconate 20ml mixed with NSS IV over 10 min (2ml/min), followed by 30-40ml/500ml NSS over 4 hours. (NO BICARB)
Hypercalcemia: Total Ca > 11mg/dl
Ionized Ca >1.3 mmol/L

**Etiology** - Hyperparathyroidism, Ca$^+$ release from bone (osteo/tumors), malignancy, alkalosis, Vit D excess, prolonged immobility, CRF, thiazide diuretics

**Clinical Symptoms** - hypotension, conduction abnormalities, SB, dehydration/polyuria/polydipsia, weakness, leg pain/path fractures, decreased LOC, seizures/coma, pancreatitis, anorexia/N/V, dig toxicity flank pain/renal stones

**Management** -
Hydration and diuretics (ie 4-6 liters of NSS with lasix q 1-2 hours + K$^+$ 60 meq/day + Mg 60 mmol/day)
Hypercalcemia Rx (continued)

**Pamidronate** 15-90 mg/1000cc IV over 24 hours- delayed response of 4-5 days but more potent than calcitonin- mainstay of Rx with cause related to malignancy

**Mithramycin** 25 mcg/kg IV over 4 hours x1

**Calcitonin** is a naturally occurring hormone that prevents bone resorption
- give 4U/kg IM or SC q 12 hours- onset is within a few hours (but drop in level is mild- 0.5 mmol/L is max drop)

**Hydrocortisone** (adjunct to calcitonin) 200 mg IV TID

**Plicamycin** 25 mcg/kg over 4 hours; can repeat every 24 hours (potential toxicity)
Phosphorus 4.5-5.5mg/dL

Effects of Phosphate Depletion

1. Low Cardiac Output
2. Anemia
3. Impaired HbO₂ Dissociation
4. Impaired ATP Production
Hyperphosphatemia

**Etiology** - renal insufficiency, or PO4 release from cell due to widespread cell necrosis (rhabdomyolysis or tumor), hypocalcemia, increase intake of alkali (baking soda), Addison’s disease, Vitamin D excess

**Clinical Symptoms** - not well documented except for formation of insoluble calcium-phosphate complexes that are deposited in soft tissues and promote tissue damage

**Management** - Promote phosphate binding in upper GI tract with sucralfate or aluminum-containing antacids. If concurrent, hypocalcemia, give PhosLo tabs 2 TID ~ in CRF- hemodialysis
Hypophosphatemia

**Etiology** - resp. alkalosis, beta agonist bronchodilators, sepsis, phosphorus binding agents (carafate/amphogel), diabetic ketoacidosis, malabsorption

**Clinical Symptoms** - often clinically silent - impaired myocardial contractility and decreased CO, hemolytic anemia, shift in the oxyhemoglobin dissociation curve to the left, muscle weakness

**Management** - IV Rx if P₀₄ is <1.0 mg/dL, once > 2, give oral supplements 0.6-0.9 mg/kg NaP₀₄ or KP₀₄ per hour, check levels every 6 hours
Magnesium 1.5-2.5 meq/L

- Second most abundant intracellular cation
- Cofactor for enzyme reactions involving ATP
- Membrane pump for electrical gradient across cell membranes
  - Activity of electrically excitable tissues
  - Regulates calcium flux in smooth muscle cells
- Plasma levels poorly reflect body stores
Hypermagnesemia

- **Etiology**: CRF, inc. intake (laxatives/antacids), Addison’s disease, aspiration on salt water, hyperparathyroidism, hypothyroidism, dehydration

- **Clinical Symptoms**: confusion, feeling of warmth/sweat followed by severe depression, hypotension, SB, heart block, muscle weakness, flaccid paralysis, respiratory muscle paralysis, hypoactive DTR

- **Management**: 10-20 ml of 10% Ca Gluconate IV over 10 minutes HD with renal failure
Hypomagnesemia

- **Etiology** - excessive diuretics, starvation, alcoholism, antibiotics, drugs, malabsorption, GI losses, diabetes, acute MI, cisplatin Rx, acute/chronic pancreatitis, hypokalemia hypocalcemia

- **Clinical Symptoms** - confusion, seizures, coma, arrhythmias (PVC, Vfib, torsades de pointes), hyperreflexia

- **Management (Mild)** - 1 gm MgSO₄ = 8 mEq
  - 50% MgSO₄ 500 mg/ml (4 mEq/L) or
  - 20% MgSO₄ 200 mg/ml (1.6 mEq/L)
  
  Replace with 1mEq/kg for 24 hours, then 0.5 mEq/kg for 3-5 days
# Hypomagnesemia

## Table 42.3. Markers of Possible Magnesium Depletion

<table>
<thead>
<tr>
<th>Predisposing Conditions</th>
<th>Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug therapy*:</td>
<td>Electrolyte abnormalities*:</td>
</tr>
<tr>
<td>Furosemide (50%)</td>
<td>Hypokalemia (40%)</td>
</tr>
<tr>
<td>Aminoglycosides (30%)</td>
<td>Hypophosphatemia (30%)</td>
</tr>
<tr>
<td>Amphotericin, pentamidine</td>
<td>Hyponatremia (27%)</td>
</tr>
<tr>
<td>Digitalis (20%)</td>
<td>Hypocalcemia (22%)</td>
</tr>
<tr>
<td>Cisplatin, cyclosporine</td>
<td>Cardiac manifestations:</td>
</tr>
<tr>
<td>Diarrhea (secretory)</td>
<td>Ischemia</td>
</tr>
<tr>
<td>Alcohol abuse (chronic)</td>
<td>Arrhythmias (refractory)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>Digitalis toxicity</td>
</tr>
<tr>
<td>Acute MI</td>
<td>Hyperactive CNS Syndrome</td>
</tr>
</tbody>
</table>

* Numbers in parentheses indicate incidence of associated hypomagnesemia.
Anion Gap (AG)

- Anion gap is an acid base parameter used to help evaluate the cause of metabolic acidosis.
  - an increase in $\text{H}^+$ ions (lactic acidosis)
  - loss of bicarbonate (diarrhea)
- Law of electroneutrality says that plasma must have an equal number of +/- charges

**Anions** = $\text{HCO}_3^-$, $\text{Cl}^-$, proteins, organic acids, phosphates, sulfates

**Cations** = $\text{Na}^+$, $\text{K}^+$, $\text{Mg}^+$, $\text{Ca}^+$
Measured cation = \( \text{Na}^+ \)
Measured anions = \( \text{HCO}_3^- , \text{Cl}^- \)

<table>
<thead>
<tr>
<th>Unmeasured Anions</th>
<th>Unmeasured Cations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proteins</td>
<td>Calcium</td>
</tr>
<tr>
<td>Organic acids</td>
<td>Potassium</td>
</tr>
<tr>
<td>Phosphates</td>
<td>Mg</td>
</tr>
<tr>
<td>Sulfates</td>
<td>Mg</td>
</tr>
<tr>
<td>1 meq/L</td>
<td></td>
</tr>
<tr>
<td>23 meq/L</td>
<td></td>
</tr>
<tr>
<td>23-11 = 12 “anion gap”</td>
<td>(Normal AG = 8-12)</td>
</tr>
<tr>
<td>(relative abundance of unmeasured anions)</td>
<td></td>
</tr>
</tbody>
</table>
Anion Gap
Anion Gap
Anion Gap
High Anion Gap
Metabolic Acidosis

High anion gap (increased H\(^+\) in ECF)
- H\(^+\) combines with bicarb = carbonic acid
- Decreased bicarb in ECF increases the AG
  - Organic acid accumulation: alcoholic and diabetic ketoacidosis, renal failure, lactic acidosis, rhabdomyolysis, toxins (methanol, ethylene glycol, paraldehyde, salicylates)
Normal Anion Gap
Metabolic Acidosis

- Normal anion gap (loss of bicarb)
  - increase in chloride ions to maintain electrical neutrality, therefore AG remains unchanged
  - seen in early renal failure/bicarb wasting, GI bicarb loss (diarrhea, ureteral diversions), and with carbonic anhydrase inhibitors (amphetamine, ephedrine, salicylates, phenytoin, quinidine, methenamine)
Thanks to:

- Valerie Sabol, RN, MSN, ACNP
- Co-Director ACNP Program University of Maryland School of Nursing