**Lesson Plan: Basic Lab Values**

**West Michigan Regional Medical Consortium**

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| **Topic:** | Basic Lab Values |
| **Presenter:** | Nicholas McManus, DO FAAEM |
| **Location:** | https://wmrmcc.thinkific.com/courses/basic-lab-values |
| **Credit Category:** | Preparatory |
| **License Level:** | PARAMEDIC |
| **Credits:** | 1 |
| **Format:** | 1 hour lecture |

**Objectives:** Upon completion of this CE, the participants will be able to:

Cognitive

1. Define basic metabolic panel.
2. Identify normal lab values.
3. Discuss reason lab values may vary.
4. Identify treatments for outside of normal lab value findings.

**Outline for Presentation:**

**A. Basic Metabolic Panel**

a. Chemistry panel, also known as the Basic Metabolic Panel which typically contains 8 values.

b. Also known as a Chem-7, Chem-8 or Chem-10 (depending on the number of components of the test).

c. The Comprehensive Metabolic Panel is the same as a Basic Metabolic Panel, but also includes liver function studies.

d. In clinical practice, the Chemistry panel is written as short hand using what is known as a **fish bone.**

i. The numeric readings for each value sits in the designated spot on the fishbone, which is quicker and easier than writing them all out with the associated word.

ii. Universal understanding of the components of the fishbone allows clinicians to efficiently communicate multiple lab values in written documentation.

**B. iSTAT**

a. An iSTAT is a handheld bedside blood analyzer that allows rapid analysis of many lab values including lactic acid, hematology values such as hemoglobin and hematocrit,

Chemistry panel, Cardiac enzymes, Coagulation labs such as the PT/INR, arterial or venous blood gases and a blood pregnancy test.

b. For the purpose of this lecture, most of the lab values we will discuss are pertinent to the management of critical and emergency medical management of patients and we will focus on the most common lab values that will give you the broadest and most complete understanding of these measurements.

**C. Biomedical Pathways**

a. This lecture will include many pathways, which may seem overwhelming depending on your current level of understanding.

b. For the purpose of this lecture, it is important to understand the overall purpose of each pathway, which should give you a better understanding on a particular physiological process, but don't get caught up in the details. At least not until you grasp the overall concepts.

**c. The Renin Angiotensin aldosterone system (also known as RAAS for short):**

i. A system of different hormones that regulates blood pressure and fluid balance.

ii. Steps:

1. A drop in blood pressure decreases the amount of blood that flows to the kidney. This drop in blood pressure is sensed by specialized cells in the kidney known as the juxtaglomerular cells.
2. The kidney then activates and secretes a hormone known as Renin into circulation.
3. The liver secretes a hormone known as angiotensinogen, which circulates in the blood waiting for the signal from Renin to become activated.
4. Renin then converts Angiotensinogen to Angiotensin I.
5. The lungs contain an Enzyme known as Angiotensin-Converting Enzyme (or ACE for short). This enzyme is the target of the anti-hypertensive medications ACE-inhibitors such as Lisinopril. ACE converts Angiotensin I into Angiotensin II.
6. Angiotensin II is the main player in this pathway and has many functions:
    a. First, it directly increases sympathetic activity. The fight or flight mechanism of the body.
    b. Secondly, Angiotensin II acts directly on the renal tubules to increase the reabsorption of Sodium, Chloride and Water and at the same time, excretes potassium to maintain electrolyte balance.
    c. This mechanism is also managed a second way. Angiotensin II stimulates the adrenal gland, that sits on top of the kidney, to secrete aldosterone, which has the same effect in electrolyte balance.

d. Angiotensin II also acts directly at the smooth muscle cells of blood vessels, causing vasoconstriction, which increased blood pressure.
e. Finally, Angiotensin II acts in the pituitary gland to secrete a hormone known as Anti-Diuretic Hormone (or ADH), which works in the kidney as well, but this time at the collecting ducts and reabsorbs water. So, it is true to its name in that it is the opposite of a diuretic, or an antidiuretic. Another name for ADH, is vasopressin, which I am sure you are all familiar with in its use as a medication given to help increase blood pressure.

7. As blood pressure increases, the increase in renal blood flow is again sensed by the juxtaglomerular cells in the kidney, and renin is no longer produced.

**d. Fluid and Solute Shifts**

**i. Osmosis**

1. The spontaneous net movement of solvent molecules, such as water, through a semi-permeable membrane into a region of higher solute concentration.
2. If the body, water shifts in and out of cellular structures based on the concentration of different solutes on each side of that membrane (Solutes include electrolytes such as sodium, or different proteins such as Albumin).
3. Keep in mind, with osmosis, the water molecules are small enough to pass through the cellular membranes, but the different solutes cannot.
4. With osmosis, water will always follow solute, until the concentration of solute is equal on both sides.

**ii. Diffusion**

1. The movement of molecules from a region of high concentration to an area of low concentration.
2. The gaps in the cellular membrane are large enough for the solute to pass, and therefore, they will move down their concentration gradient until an equal concentration is achieved on each side of the membrane.

**D. Basic Metabolic Panel - FISH BONE**

**a. SODIUM**

i. Sodium is the primary extracellular cation in the body.
ii. It regulates osmotic forces, it is involved in membrane transport systems, it helps regulate many chemical reactions as well as aid in acid-base balance.

**iii. Na/K pump**

1. The picture shown here is of a cell membrane. The Green box in the center is the Na/K pump. This is an enzyme that acts as a pump to pump

Sodium out of cells while pumping potassium into cells. Both of these ions are moving AGAINST their concentration gradient, and this movement requires energy which is formed by cleaving a phosphate from the molecule ATP.

2. The movement of these ions gives energy for other transport systems that move amino acids, glucose and other nutrients into cells, it controls signaling within many cells, controls cell volume and controls neuron activity.
3. This small electrolyte shift is important to so many mechanisms in the body, that you can hopefully appreciate the importance that Sodium, Potassium and even phosphorus play in cellular function.

iv. Normal Range of Sodium

1. The normal range of Sodium is typically 138-146 mmol/L.
2. Values outside this range can have detrimental effects on many cellular functions throughout the body.

**v. Hypernatremia (sodium that is too high)**

1. Causes:
    a. Dehydration (the concentration of sodium is elevated in relation to the amount of water in the body).
        i. In the setting of dehydration, a decrease is blood pressure is sensed by the kidneys and the RAAS system is activated, increasing the absorption of water in the kidneys.
        ii. Further, the chemoreceptors in the brain sense a higher sodium concentration, and the pituitary will secrete ADH, causing more free water to be reabsorbed. It also activates the thirst centers, causing the patient to feel thirsty.
    b. Excess saline administration.
    c. Renal loss of water, such as aggressive diuretic use or Diabetes Insipidus.
2. Treatment:
    a. As clinicians, we can help this process by giving the patient free water. What that means is, we are giving water in the pure form, without sodium.
    b. If the patient can tolerate oral fluids, we can give them water to drink.
    c. It is not uncommon the amount of free water that they are deficient is quite large, in the 7-10-liter range.

d. This deficit is a calculated value based on a patients gender, age, weight and sodium level.
e. If they need more free water than they can drink, or they are unable to tolerate PO, we can give them free water through the IV. However, it is not safe to infuse free water into an IV. So IV free water is given in the form of Dextrose and water, or D5W.

**vi. Hyponatremia (sodium concentration that is too low)**

1. Causes:
   a. Excessive vomiting or diarrhea, where sodium is expelled from the body before it can be absorbed, or reabsorbed by the GI tract).
   b. Certain diuretics
   c. Inadequate salt intake
   d. Drinking too much free water (as seen in diabetes insipidus)
2. Symptoms:
   a. Headaches, confusion, lethargy
   b. If the levels drop below 120 mEq/L, the patient is at risk for seizures, coma and death.
3. Treatment:
   a. Gradually increase in the sodium concentration by slow infusion of 0.9% normal saline.
   b. If sodium is corrected too rapidly, a devastating phemonenon called Central Pontine Myolinolysis can occur, where the myelin sheath on neuronal cells in the pons of the brainstem degenerates, and results in irreversible paralysis, difficulty swallowing, difficulty speaking and other neurological symptoms.
   c. A 1 liter bag of normal saline will increase sodium by about 1 mEq/L.
   d. Our goal in treatment is an increase in serum sodium by 1-2 mEq/L/hr in acute hyponatremia, and no more than 0.5 mEq/L/hr in patients with chronic hyponatremia to avoid central Pontine myolinolysis.
   e. If a patients sodium drops too low, and they start to have significant neurological changes and certainly if they develop seizures, the treatment is to give a higher concentration of sodium. This is done with 3% hypertonic saline given at 25-100 mL/hr with very close monitoring of rate correction.
   f. If seizures occur, treatment of the seizure with benzodiazepines should be attempted, but unlikely to respond until the sodium is brought up to a safer level, typically > 120 mEq/L

4. Correction of Na for Hyperglycemia
    a. Whenever hyponatremia is seen on laboratory evaluation, look at the glucose.
    b. Significant elevations in glucose will cause sodium concentrations to appear low, and this is actually a falsely low reading even though the actual sodium concentration may be normal.
    c. If you notice a significantly elevated glucose level (like in the hundreds) and the sodium is low, you must correct the sodium level with the following equation.

$$\text{Corrected serum sodium} = \underset{(\text{mEq per L})}{\text{sodium}} + \frac{1.65 \times (\text{glucose [mg per dL]} - 100)}{100}$$

    d. *Alternate Method:*
        i. Typically, sodium will falsely read low by 1.6 mEq/L for every 100 mg/dL of glucose AFTER 100.
        ii. Ex: if the patients' Sodium level is 129 and blood glucose is 300:
            1. Add 1.6 for every 100 mg/dL of glucose AFTER 100.
            2. So in this case, we use a glucose level of 200.
            3. So we will have to add 1.6 twice (so, 3.2) to the patients measured sodium to get the corrected, and accurate sodium level.
            4. Corrected Sodium for a glucose of 300 = 132.2 (round it to 132).
    e. *Even Easier Method*
        i. Download the app MDCalc, and you can just plug the numbers in.

**b. POTASSIUM**

i. The major INTRACELLULAR cation in the body.
ii. Along with sodium, it regulates intracellular volume, plays an important role in not only nerve conduction, but also contraction of skeletal and cardiac muscle cells and plays an important role in many metabolic processes.
iii. The normal range is 3.5-4.9 mEq/L.
**iv. Hyperkalemia (concentration of potassium is too high)**
    1. Causes:
        a. Renal failure (potassium is no longer filtered or excreted through the kidneys)
        b. Excessive potassium replacement.

c. Trauma - *remember that potassium likes to be inside of cells, so trauma causing massive tissue damage, such as a crush injury that causes large amounts of cellular damage can cause dangerously high levels of potassium to spill into systemic circulation.*
    i. Reperfusion syndrome - patients extremities are crushed and trapped by a large object, such as a fallen building or the dash of a car for a prolonged period of time, and when the object is removed and the blood that was trapped in the pinned extremity is released into systemic circulation, a toxic level of potassium reaches the heart resulting in cardiac arrest.

2. Signs and Symptoms
    a. Cardiac dysrhythmias
        i. Typically not seen until the potassium level reaches a level of about 6.5.
    b. EKG Changes
        i. Potassium level of 6.5-7.5
            1. Prolonging of the PR interval
            2. Shortening of the QT segment
            3. Peaking of the T waves.
        ii. Potassium level of 7.5-8.0
            1. P-waves begin to flatten
            2. QRS begins to widen.
        iii. Potassium levels greater than 8.0
            1. QRS segment begins to widen so much that it is difficult to appreciate a normal QRS segment, and it starts to become more of a sine wave appearance
            2. Rapidly degrades into v-fib and then asystole.
3. Treatment
    a. Get an EKG!
        *i. If there are no significant EKG changes, and the potassium level is significantly elevated, the blood specimen should be redrawn prior to treatment as many factors can lead to a false reading. A prolonged tourniquet time while searching for a vein to start an IV, for example can cause a falsely elevated reading as cells leak potassium into the hypoxic arm, causing a higher*

*local concentration that does not represent the true systemic concentration.*

b. Calcium (either gluconate or chloride)
    i. Always given first
    ii. Stabilizes the cardiac membrane
    iii. Does nothing to actually decrease potassium levels.
c. Insulin
    i. Will drive potassium into cells, and this hides it.
    ii. This is only a temporizing measure, as it doesn't decrease the actual whole body level of potassium, only temporarily decreases the amount of potassium in systemic circulation.
    iii. Giving 10 units of insulin to a patient who is not hyperglycemic could be a big problem, so we always give glucose with the insulin when it is used for hyperkalemia.
d. Sodium Bicarbonate
    i. Also helps to hide potassium in cells, providing a temporary fix.
e. Inhaled albuterol
    i. Also helps to hide potassium in cells, providing a temporary fix.
f. Lasix
    i. Sometimes used
    ii. Increases the amount of potassium excreted at the kidneys.
g. Kayexalate
    i. Binds potassium in the GI tract, preventing its re-absorption.
    ii. This process is very slow
    iii. Literature is mixed on its actually clinical efficacy.
h. Dialysis
    i. Indicated for significant hyperkalemia and will actually rapidly remove potassium from the body.

**v. Hypokalemia (serum potassium levels less than 3.5).**

1. Causes:
    a. Over-diuresis
    b. Inadequate dietary intake of potassium
    c. GI wasting with diarrhea and vomiting
    d. Other metabolic conditions.
2. Signs/Symptoms

a. Metabolic alkalosis
    i. potassium is lost as a compensatory mechanism in the kidney in exchange for sodium, which we will talk more about later. Remember that potassium is vital for cell depolarization.
b. Confusion
c. Drowsiness
d. Weakness
e. Fatigue
f. More prone to arrhythmias.
g. Ileus
h. Nausea and Vomiting

3. Treatment
    a. Replacement of Potassium.
    b. Potassium is significantly more efficiently absorbed through the GI tract, than through IV administration.
    c. So, if the patient can take oral medications, we prefer this route.
    d. However, it may be beneficial to give potassium both PO as well as IV.
    e. IV potassium must be given over a long period of time to help minimize risks associated with the IV infusion and is typically given at a rate of 10 mEq per hour at a maximum of 40-50 mEq per treatment.
    f. There are dextrose containing potassium formulations, however, this should really just be avoided as it can cause an anaphylactic reaction in patients who have an allergy to corn products.
    g. For each 10 mEq dose of potassium given IV, will increase the serum concentration of potassium by 0.1 mEq.
        i. So if a patients potassium level is 3.1, we can estimate that giving them 40 mEq of IV potassium will increase their serum concentration back to 3.5.
        ii. Oral potassium replacement is much less predictable, so we can't use this rule for estimating enteral potassium replacement

**c. BICARBONATE (HCO3/CO2)**

i. The normal range of Bicarbonate is 23-28 mmol/L.
ii. In the majority of the time, you will actually not find Bicarbonate listed on the chemistry panel, but it is there. That is because it is actually listed as CO2.
    1. *In the body, most CO2 is actually being carried around in a molecule known as Bicarbonate, or HCO3*

2. *Therefore, a CO2 blood test is really a measurement of your blood bicarbonate level.*
3. *CO2 and HCO3 are used interchangeably when referring to the basic metabolic or chemistry panel.*
4. *It is important NOT TO CONFUSE this with the pCO2, which is the partial pressure of CO2 in the blood and important in blood gas and pH analysis. We will discuss that more later.*

*iii.* Blood pH is maintained by the bicarbonate buffer system.

1. The lungs and the kidneys are constantly involved in a tug-of-war to help maintain acid-base homeostasis.
2. CO2 reacts with water to form carbonic acid, which readily dissociates into bicarbonate and a hydrogen ion.
3. An increase in the formation of the molecules on any one side of this equation will result in a compensatory increase in the formation of molecules on the opposite side of the equation to keep blood pH at the physiological level of 7.35-7.45.
4. CO2 is actually safely carried by red blood cells in the form of bicarbonate and transported throughout the body and eventually to the lungs to participate in gas exchange.
5. **Response to acidosis**
    a. The body will compensate and do what it can to shift the equation to the right, in favor of bicarbonate formation which will buffer the blood and increase the pH.
    b. This is done by generation of new bicarbonate in the kidney and increased absorption of bicarbonate in the kidney.
    c. The equation can readily move from right to left and left to right, so the kidney will also excrete hydrogen ions to prevent further formation of carbonic acid.
    d. Further, the lungs help out by increasing the respiratory rate and blowing off more CO2.
6. **Response to alkalosis**
    a. The body will compensate and do what it can to shift the equation to the left, in favor of carbonic acid, and decrease the pH.
    b. This is done by renal wasting of bicarbonate and decreased bicarbonate production by the kidney.
    c. Hydrogen ions are saved at the kidney and the lungs decrease the respiratory rate to increase the amount of CO2 that is now present to allow for more carbonic acid production.

**d. CHLORIDE**

i. An abundant anion in the extra-cellular fluid.
ii. Mechanisms
    1. Moves in and out of cells with sodium
    2. Involved in many cellular transport systems
    3. Helps transport CO2 to RBC's

**e. BLOOD UREA NITROGEN (BUN)**

i. Ammonia is a waste product of amino acid catabolism and is quite a toxic substance to the body.
ii. Our body converts the toxic ammonia, to a less toxic substance such as urea nitrogen (Urea Cycle).
iii. Urea nitrogen that is produced is released into the blood where it eventually travels to the kidneys and is excreted in urine.
iv. We can measure the amount of urea nitrogen in the blood as an indirect measurement of how well the kidneys are functioning.
v. If the kidneys are not filtering the way they should, the serum blood urea nitrogen level will be increased.
vi. Azotemia (An elevation of BUN)
    1. However, this really not a specific lab value to completely assess renal function, as other things can make this value elevate such as heart failure, dehydration, and a high protein.
    2. UPPER GI Bleed
        a. Consider if you see levels greater than 30 mg/dL
        b. As blood enters the stomach, the protein components of blood, including hemoglobin and immunoglobulins are digested and just like any other protein, their amino acids are utilized with the same waste product of Ammonia, and eventually Urea Nitrogen.
        c. It is important to note that this is only seen with upper GI bleeds, and not lower GI bleeds as proteins are broken down to amino acids in this portion of the digestive tract.
vii. Clinically, BUN is used in conjunction with creatinine to assess fluid status.
    1. As intravascular volume decreases, so does the oncotic pressures that filter substances through the kidney.
viii. Mechanisms of Kidney Injury
    1. Prerenal (an insult occurring before the kidney)
        a. Etiologies: Sudden drop in blood pressure, or dehydration will cause a decrease in the blood flow to the kidney, and will cause an elevation in BUN that typically is elevated significantly higher than the other 2 mechanisms of insult.
        b. Clinically, this is likely the most often utilized benefit of analyzing BUN.

c. If the kidney injury is due to a prerenal cause the radio on BUN/Creatinine will typically be > 20.
d. Treatment: IV fluids

2. Renal (direct insult to the kidney)
   a. Etiologies: Direct infectious process, an autoimmune disorder or toxic medication effect.
   b. The BUN will also increase, but not to the level that it is seen with a prerenal etiology.
   c. The ratio of BUN/Creatinine is typically less than 20.
3. Post-Renal (insult offering after the level of the kidney)
   a. Etiologies: Impedance of urine flow from a kidney stone blocking the urinary outflow.
   b. Again, BUN will elevate, but not to the level of that seen in a prerenal etiology.

**f. CREATININE**

i. Creatinine is a much more specific indicator of how the kidney is doing.
ii. Creatinine is a chemical waste molecule generated from muscle metabolism.
iii. It is transported through the bloodstream to the kidneys and most of this waste product is filtered out through the kidneys and into the urine.
iv. It is more specifically elevated in renal failure, or during a pathologic process that causes injury to the kidney such as profound dehydration and shock, which decreases blood flow to the kidney resulting in injury.
v. The normal value of Creatinine is patient specific, as patients may have chronic renal failure and their “normal” value may be outside of the range listed here.
vi. Clinically, it is the change in creatinine that we use to assess changes in renal function. However, in an otherwise healthy patient with no history of renal disease, their value is typically 0.6-1.3 mg/dL.
vii. Low creatinine levels aren’t typically of clinical concern, as the goal of the body is to get rid of this waste product anyways, but can be seen in pregnancy, starvation states and in patients after dialysis.
viii. AKIN Criteria
   1. Stage 1 AKI - Creatinine increases by more than 1.5 from the patients baseline or an elevation by more than 0.3 mg/dl.
   2. So, if you don’t remember anything else from this chart just remember that if the patients baseline
ix. Example
   1. Cr is 0.7, and today it is 1.1, the patient is in an acute kidney injury.
   2. Now lets tie in BUN. If the patients BUN is elevated, we look at the ratio of BUN elevation in relation to Cr elevation.

3. So if the BUN is 24, we divide that by 1.1, and we get a value of 21.8. This is greater than 20.
4. So if the clinical appearance of the patient fits intervascular volume depletion, the treatment for this patient will likely be to initiate IV fluids while other possible causes of renal injury are ruled out.

**g. GLUCOSE**

i. Glucose levels will change based on fasting vs fed states.
ii. Fasting blood glucose level is 70-105 mg/dL in a nondiabetic patient.
iii. Serves as nearly the only energy source for the brain.
iv. ***As blood glucose drops*** → pancreas promotes glucagon release → stimulates glycogen breakdown into glucose in the liver.
v. ***As blood glucose rises*** → pancreas secretes insulin which stimulates the uptake of glucose into tissue and cells and stimulates he storage of excess glucose as glycogen by the liver.
vi. **Hyperglycemia**

1. Symptoms
   a. Thirst
   b. Urinary output increases – due to increased water secretion
   c. Blurred Vision – due to fluid shifts in the eye
2. Treatment
   a. Normal saline – correct body fluid depletion
   b. Insulin – once hydrated
   c. Potassium – Insulin shifts potassium into cells, so much watch closely.

**3. Diabetic Ketoacidosis**

a. Increased release of glucose by the liver, but also leads to release of free fatty acids and from adipose tissue which are converted to ketones to try to use as an energy source.
b. Without insulin, the body cannot uptake glucose into cells.
c. Ketones cause pH to decrease → metabolic acidosis.
d. Bicarbonate system rapidly overwhelmed.
e. Typically occurs in Type 1 diabetics, as they have a complete lack of insulin.
f. Causes include infection, after exogenous steroid use or following a stroke or MI.

**4. Hyperglycemic Hyperosmolar Syndrome**

a. Seen with a relative insulin deficiency that leads to an increase in serum glucose.
b. Typically seen in Type 2 diabetics that still have some insulin secretion.

c. As insulin is still present in these patients, ketones are not typically produced as glucose can still be used by cells as a source of energy.
d. However, the glucose levels increase to a level that increases the overall serum osmolarity.
e. Typically, glucose levels are significantly higher than those seen in DKA, and not uncommonly greater than 900.
f. Ketones are not produced → no metabolic acidosis
g. Bicarbonate buffer system is not depleted
h. Causes: Infection, or other stress such as exogenous steroid use, stroke and MI.
i. The fluid deficit is usually much more profound than in DKA
j. Mortality is much higher than in DKA

**vii. Hypoglycemia**

1. Causes
   a. excess in insulin levels
   b. adverse effect of oral hypoglycemic
   c. Infection
   d. Liver failure (as glucose cannot be utilized from glycogen stores)
   e. Alcoholics (inadequate diet)
2. Symptoms
   a. Headache
   b. Dizziness
   c. Extreme fatigue
   d. Seizures
   e. Coma.
   f. Hunger.
   g. Shakiness, Tachycardia, Sweating (autonomic nervous system activated)
      i. Patients on beta blocker may not show these symptoms!
3. Treatment
   a. Glucose replacement with juice or oral glucose gel, paste or tabs if the patient can tolerate PO
   b. IV dextrose
   c. IM glucagon

**h. ANION GAP**

i. Helps identify causes of metabolic acidosis.
ii. Calculation: **$Na^+ – (Cl^- – HCO_3^-)$**
iii. Normal value = 12
**iv. Causes**

1. M – methanol
2. U – uremia
3. D – diabetic ketoacidosis/alcoholic ketoacidosis
4. P – propylene glycol
5. I – isoniazide/iron
6. L – lactic acidosis
7. E – ethylene glycol
8. S – salicylates (aspirin overdose)

**v. Symptoms**

1. Weakness, confusion, loss of consciousness and coma
2. Shortness of breath
3. Tachycardia, arrhythmias
4. Nausea, vomiting and diarrhea

**i. CALCIUM**

i. 80% of all calcium is bound to albumin in circulation.
ii. Ionized (free) calcium is the biologically active form
iii. Calcium plays many important biological functions, including building strong bones and teeth, clotting of blood, Sending and receiving nerve signals, muscle contraction and relaxation, cardiac contraction and in the release of many hormones from both the anterior and posterior pituitary in the brain.
iv. Normal level is around 10 mg/dL.
v. As blood calcium levels fall → Parathyroid gland PTH
    1. Kidney → directly stimulate Calcium uptake in the kidney
    2. Kidney → activates Vitamin D → increased calcium uptake by the intestines
    3. Bones → stimulates calcium reabsorption from calcium stores in bones

**vi. Hypercalcemia**

1. Causes
    a. Hyperparathyroidism (excess PTH)
    b. Excessive Vitamin D intake
    c. Thiazide diuretic use (calcium sparing in the kidney)
    d. Adrenal insufficiency
    e. Certain types of cancers (multiple myeloma)
    f. Milk-alkali syndrome (tums ingestion)
2. Symptoms
    a. “stones, bones, groans and psychiatric overtones.”
    b. Kidney stones and kidney disease
    c. Bone loss – from Ca reabsorption and bone remodeling
    d. Psychiatric disturbances and abdominal pain
3. Treatment

a. IV fluids to flush calcium through the kidneys
b. IV Lasix - a loop diuretic causing calcium wasting at the kidney

**vii. Hypocalcemia**

1. Ionized calcium levels below 4.6 mg/dL.
2. Causes
   a. Underactive parathyroid gland
   b. Vitamin D deficiency
   c. Renal failure
   d. Any disease process that causes calcium sequestration such as pancreatitis.
3. Symptoms
   a. Muscle spasms
   b. Convulsions
   c. Tetany
   d. Cardiac arrhythmias.
   e. EKG changes: **prolongation of the QT interval**.
4. Treatment
   a. Replace calcium.
   b. Calcium gluconate or calcium chloride IV.
   *c. Caution should be given to patients on Digoxin receiving IV calcium, specifically calcium gluconate, as there is a theoretical risk of causing calcium influx into myocardial cells resulting in a phenomenon known as "stone heart" resulting in malignant dysrhythmias.*

**E. PT/INR**

**a. Prothrombin Time (PT)**

i. Evaluates the coagulation factors **1, 2, 5, 7 and 10.**
ii. ***Prolonged in:*** liver disease, vitamin K deficiency or a coagulation factor deficiency.
iii. ***Decreased in:*** patients with high vitamin K intake, oral contraceptive use and in patients on hormone replacement therapy.

**b. International Normalized Ratio (INR)**

i. A calculated value based on the Prothrombin time
ii. We use this calculated value clinically to measure the therapeutic effects of the anticoagulant medication Warfarin (Coumadin).
iii. < 1.2: Normal in a patient not on Warfarin
iv. 2-3: Therapeutic range for patients with nonvalvular a-fib on Warfarin
v. 2.5-3.5: Therapeutic range for patients with mechanical heart valves on Warfarin
vi. Elevated INR

1. A patient with a supratherapeutic INR is a concern, as they are at a significantly increased risk for major bleeding event.
2. INR < 10 and there is no signs of bleeding, treatment typically includes holding the patients Coumadin and restarting it when the INR falls to therapeutic levels.
3. INR > 10 regardless of bleeding, the Coumadin should be reversed with oral Vitamin K, which serves as a cofactor for the coagulation factors previously listed.
4. Any sign of major bleeding should be treated more aggressively with IV Vitamin K PLUS either fresh frozen plasma or Prothrombin Complex Concentrate.

**F. Cardiac Injury Profile (Troponin I, CK-MB, and other CK isoforms)**

a. The enzymes are released into circulation as a result of injury to cardiac myocytes.

**b. CK and CK-MB**

i. Are not specific to cardiac muscles, but also found in skeletal muscle.
ii. Therefore, an elevation of these do not necessarily mean the cardiac myocytes are injured.
iii. Blood levels tend to peak earlier than Troponin in cardiac injury, but also normalize rather rapidly.
iv. Therefore, a patient who has a repeat coronary event with elevated CK isoforms should help with diagnosis

**c. Troponin I**

i. Is specific to cardiac cells.
ii. Typically takes up to 6 hours to show up in the blood after cellular injury, which is why these labs are often repeated several times in the emergency department.
iii. Troponin goes up after several hours but stays elevated for days

**Evaluation of Presentation:** Continuing Education Program Sponsor Evaluation Form will be filled out by all participants.