Electrolytes imbalance and acid-base disorder

Objectives:

1. To be able to identify the type of hyponatremia (euvolemic-hypovolemic-hypervolemic) based on clinical presentation and laboratory finding.
2. Recognize true and pseudo-hyponatremia.
3. To be able to manage a patient with hypokalemia/ hyperkalemia.
4. To be able to Interpret arterial blood gases.
5. To be able calculate respiratory and metabolic compensation for acid/base disturbances.
6. To be able calculate anion gap with correction for serum albumin.

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To be able to identify the type of hyponatremia (euvolemic-hypovolemic-hypervolemic) based on clinical presentation and laboratory finding.

Hyponatremia

Definition

Hyponatraemia (Na <135 mmol/L) is the most common biochemical abnormality in hospitalized patients, with up to 35% of inpatients developing hyponatremia during their stay.
The cause depends on the associated changes in extracellular volume:

A) Hyponatremia with hypovolaemia

- This is due to sodium loss in excess of water loss
- **Causes**:
  - Renal sodium losses: diuretics (especially thiazide), Adrenocortical failure
  - Gastrointestinal sodium losses: Vomiting, Diarrhea
  - Skin sodium losses: burns

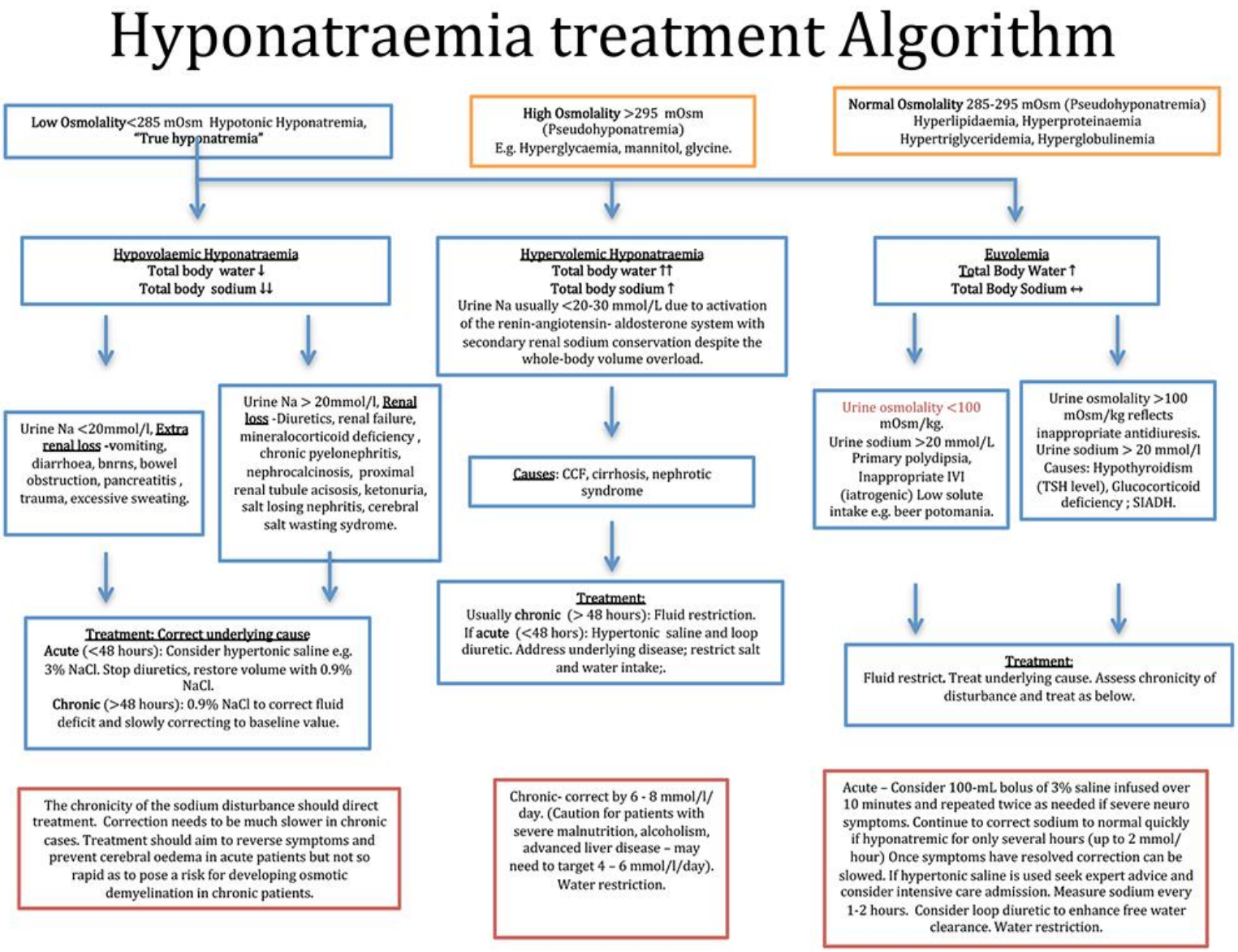
B) Hyponatremia with euvolemia

- This is due to excess body water (no change in total body sodium)
- **Causes**:
  - Primary polydipsia
  - Glucocorticoid deficiency
  - Excessive electrolyte-free water infusion
  - SIADH **(Syndrome of inappropriate ADH)**
  - Hypothyroidism

C) Hyponatraemia with hypervolaemia

- This is due to excess water retention that is associated with sodium retention and volume expansion
- **Causes**:
  - Congestive heart failure
  - Cirrhosis
  - Nephrotic syndrome
  - Chronic kidney disease (during free water intake)

To be able to identify the type of hyponatremia (euvolemic-hypovolemic-hypervolemic) based on clinical presentation and laboratory finding.



Recognize true and pseudo-hyponatremia.

True Hyponatremia (Hypotonic)

Definition:
It is when you have a low serum sodium concentration, associated with low osmolality and low tonicity. If you measure the plasma osmolality it will be low.

Assessment of the Urine Sodium and Urine Osmolality can be used to separate most causes of hyponatremia. However, the clinical scenario often gives the answer. This is especially true on a vignette, where there can't be a mystery to have a single correct answer. If a urine sodium is decreased, the kidney is working and there's poor perfusion to it. If the urine osmoles are concentrated, ADH is activated.

Pseudo-Hyponatremia (Isotonic)

Definition:
Increase in plasma solids lowers the plasma sodium concentration . But the amount of sodium in plasma is normal (hence, pseudohyponatremia). If you measure the plasma osmolality it will be normal.

Causes:

- Any condition that leads to elevated protein ex: multiple myeloma
- Any condition that leads to elevated lipids ex: severe dyslipidemia

Rarely, hyponatremia may be a 'pseudo-hyponatremia'. This occurs in hyperlipidaemia (either high cholesterol or high triglyceride) or hyperproteinemia where there is a spuriously low measured sodium concentration, the sodium being confined to the aqueous phase but having its concentration expressed in terms of the total volume of plasma. In this situation, plasma osmolality is normal and therefore treatment of 'hyponatremia' is unnecessary. Note that artifactual 'hyponatremia', caused by taking blood from the limb into which fluid of low sodium concentration is being infused, should be excluded.

To be able to manage a patient with hypokalemia/ hyperkalemia.

Hypokalemia <3.4 mmol/L

The most common cause of chronic hypokalemia are diuretic treatment (particularly thiazides)

Management:
Repletion is performed with oral or intravenous potassium. Oral replacement is preferred.
If IV is to be used, the rate must be <10 mEq / hr if by peripheral IV (PIV), or <20 mEq/hr if by central line.

Hyperkalemia >5.5mmol/L

The most common cause of hyperkalemia is acute kidney failure/ chronic kidney disease

Emergency management	Acute management:	Subacute management:
ECG monitor and i.v. access ECG changes 1. Peaking of the T wave 2. widening of the QRS complex 3. prolonged PR interval Protect the myocardium 10 mL of 10% **calcium gluconate** i.v. over 5 min Effect is temporary but dose can be repeated after 15 min	Drive K+ into cells **Insulin 10 units+50 mL of 50% glucose i.v.** over 10–15 min, followed by regular checks of blood glucose and plasma K+. Consider correction of severe acidosis (pH <6.9) **infuse NaHCO3 (Sodium Bicarbonate)** Stop all potassium supplements, and drugs reducing urinary excretion of potassium	Deplete body K+ (to decrease plasma K+ over the next 24 h) Novel potassium binders (**Patiromer** or sodium zirconium cyclosilicate) – May have faster action of onset/may be better tolerated Older potassium binders –Polystyrene sulfonate resins (e.g. calcium resonium) **Haemodialysis** or peritoneal dialysis if the above failed

Doctor's slides

Components of Acid - Base physiology

Acid

- **Exogenous**
  - Physiological: Diet
  - Pathological: toxins (Methanol, Ethylene Glycol)
- **Endogenous**
- Physiological: metabolism (volatile & non-volatile acids)
- Pathological: Ketoacids and lactate

Base

- HCO_3 is the kidney favorite's player
- Liver produces HCO_3 from some precursors (Lactate, Citrate)

Protective mechanisms that keep us alive

1- Blood Buffers:

Bicarbonate -Carbonic acid system	53%
Hemoglobin	35%
Albumin	7%
Phosphate	5%

$H_2O + CO_2 = H_2CO_3 = H + HCO_3$

2- Respiratory mechanism:

- Very quick reaction
- PCO_2 and H have a potent stimulatory effect
- on the respiratory centre

3- Renal mechanisms:

- Increase of HCO_3: Absorption, Generation
- H acid secretion: NH_3 synthesis

Doctor's slides

Response to Acid load

If 10 mmol/l of Acid is added to the blood
pH = 6.1 + log (Bicarb/carbonic acid)
pH = 6.1 + log (26-10)/(1.3+10)
= 6.1 + 0.15

pH = 6.25 (if no protective mechanism exists)

How things can go wrong

- Impaired respiratory response
- Impaired renal response

Acid base interpretation

Major tools:

pH
[H]
[HCO3]
PCO2
Clinical data

Supplementary tools:

The GAPs !
Anion Gap
Delta Gap
Plasma osmolar Gap
Urine anion Gap
Urine osmolar Gap

To be able calculate anion gap with correction for serum albumin.

Anion gap calculation:

- AG= Unmeasured anions - Unmeasured cations
- AG= measured Cations – measured anions
- AG = Na - (Cl + HCO3)
- Elevated Gap indicates excess acids in the blood = metabolic acidosis
- Watch out for hypoalbuminemia!
- For each 10 point drop in albumin, add 2.5 to the calculated AG

Delta Gap mystery

- In metabolic acidosis, the drop in HCO3 should match the elevation in AG
- **Delta gap=** $\Delta AG/\Delta HCO3 = 1$
- Delta gap < 1 = the drop in HCO3 is more than expected= 2 metabolic acidotic processes !
- Delta gap > 1 = the drop in HCO3 is less than expected= additional metabolic alkalotic process is present !

To be able to Interpret arterial blood gases.

ABG interpretation

Steps:

- Describe the pH
- Identify the primary drive for pH
- Predict the compensatory response
- Assess the actual compensatory response
- Calculate the Anion gap (AG)
- Correct the AG for albumin

Normal values:

- **pH**= 7.4
- **HCO3** = 24 mmol/l
- **PCO2** = 40 mmHg
- **Anion Gap** = 12
- **Albumin** = 40 g/l

Examples:

	pH (7.4)	PCO2 mmHg (40)	HCO3 mmol/L (24)
A	7.32	28	14
B	7.47	20	20
C	7.51	49	38
D	7.08	49	14

A- Metabolic Acidosis
B- Respiratory Alkalosis
C- Metabolic Alkalosis
D- Respiratory Acidosis + Metabolic acidosis (decompensated) NOT SURE

Steps in Acid-Base Analysis

Step 1 | History & physical examination

look for clues that may lead to the abnormalities in pH

- Vomiting: causes loss of acid and gastric contents, which suggests development of alkalosis
- Diarrhea
- Hypoventilation
- Respiratory disease
- Medications (laxatives, diuretics, etc)
- Diabetes

Step 2 | Look at the pH

Determine if it is

- Normal 7.35 – 7.45 (No abnormality or presence of mixed acidosis and alkalosis)
- Low <7.35 (acidemic)
- High >7.45 (alkalemic)

Step 3a | Determine the primary abnormality that is causing the abnormal pH

- If the pH is acidemic (<7.35), then look for Low HCO3 (Metabolic) or High PCO2 (Respiratory)
- If the pH is alkalemic (>7.45), then look for High HCO3 (Metabolic) or Low PCO2 (Respiratory)

Note: Compensation will not return the pH to the normal range, it's just a mechanism which the body trying to reduce the impact

Step 3b | If pH is normal, that doesn't rule out mixed acidosis and alkalosis (Determine what is being mixed[1])

- Look for high or low PCO2= Low PCO2 suggests respiratory alkalosis/High PCO2 suggests respiratory acidosis
- Look for high or low HCO3= Low HCO3 suggests metabolic acidosis/High HCO3 suggests metabolic alkalosis

How to determine Is the respiratory disturbance acute or chronic?

- Acute respiratory acidosis: HCO3 increase by 1 mEq/l for every 10 mmHg increase in PaCO2.
- Chronic respiratory acidosis: HCO3 increase by 3-3.5 mEq/l for every 10 mmHg increase in PaCO2.
- Acute respiratory alkalosis: HCO3 decrease by 2 mEq/l for every 10 mmHg decrease in PaCO2.
- Chronic respiratory alkalosis: HCO3 decrease by 4-5 mEq/l for every 10 mmHg decrease in PaCO2.

1: Sometimes you may have mixed diseases (Metabolic Alkalosis AND Respiratory Acidosis)

Step 4 | check for compensation

Compensation is the mechanism by which the body adapts to either acidosis or alkalosis, it will not fully correct the abnormality
example:

- A patient has diabetic ketoacidosis, pH is 7.29, HCO3 is 15 (hence, it is metabolic acidosis)
- Use the metabolic acidosis formula: Expected PCO2 by using Winter's formula PCO2 = 1.5 x HCO3 + 8 (±2[1]) = 1.5 x 15 + 8 = 30.5
- So: you expect the PCO2 in this patient to be in the range of 28.5– 32.5[3]
- Now, determine whether there is a compensation or an additional disorder:
  - If the PCO2 in this patient is higher than 32.5 → consider additional[2] respiratory acidosis.
  - If the PCO2 in the patient is lower than 28.5 → consider additional respiratory alkalosis.

Compensation calculation

Primary disorder		Expected compensation
Metabolic acidosis		• $PaCO_2$= 1.5 x HCO_3 + 8 ± 2 • ↓$PaCO_2$= 1.2 x ΔHCO_3 • $PaCO_2$~ last two digits of pH
Metabolic alkalosis		• ↑$PaCO_2$= 0.7 x ΔHCO_3
Respiratory acidosis	Acute	• ↑HCO_3= 0.1 x Δ$PaCO_2$
	Chronic	• ↑HCO_3= 0.35 x Δ$PaCO_2$ • ↓pH = 0.003 x Δ$PaCO_2$
Respiratory alkalosis	Acute	• ↓HCO_3= 0.2 x Δ$PaCO_2$
	Chronic	•↓HCO_3= 0.4 x Δ$PaCO_2$

Step 5 | Calculate the anion gap

anion gap (AG): AG = Na – (Cl + HCO3)

- Normal anion gap = 6-12[4]
- Albumin is the main unmeasured anion. To overcome the effects of hypoalbuminemia on the AG, the corrected AG can be used which is AG + (0.25 X (40-albumin)) expressed in g/L.
- An increase in anion gap that means there's additional acids like lactic acid and keto acid.
- Get back to pages to check for high AG metabolic acidosis vs normal AG metabolic acidosis

1: gives you a range
2: Please make sure that you differentiate between additional and compensated.
3: Memorize one compensation equation for each acid base abnormality. Example:
-If the PCO2 of this patient was 35, then the patient's acid-base status will be : Metabolic Acidosis AND Respiratory Acidosis.
-If the PCO2of this patient was 30, then the patient's acid-base status will be : Metabolic Acidosis Compensated by Respiratory Alkalosis. .
4: The normal range is up to 14. It is Especially important in Metabolic Acidosis, crucial for the differential diagnosis.

To be able calculate respiratory and metabolic compensation for acid/base disturbances.

Compensatory mechanisms

Acid base defect	Primary defect	pH	Compensation
Met acidosis	Low Bicarb	Low	Low PCO2
Met alkalosis	High Bicarb	High	High PCO2
Resp alkalosis	Low PCO2	High	Low Bicarb
Respiratory acidosis	High PCO2	Low	High Bicarb

Acid base disorder	Primary defect	Compensation
Metabolic acidosis	⬇ HCO3	1.2 drop in PCO2 for each 1 mmol decrease in HCO3
Metabolic alkalosis	⬆ HCO3	0.7 rise in PCO2 for every 1 mmol rise in HCO3
Acute resp acidosis	⬆ PCO2	1 mmol rise in HCO3 for every 10 point increase in PCO2
Chronic resp acidosis	⬆ PCO2	3.5 mmol rise in HCO3 for every 10 point increase in PCO2
Acute resp alkalosis	⬇ PCO2	2 mmol drop in HCO3 for every 10 point fall in PCO2
Chronic resp alkalosis	⬇ PCO2	4 mmol drop in HCO3 for every 10 point fal in PCO2

Interactive Cases - 1

❖ **15 year old boy with abdominal pain**

	Case	Normal
pH	**7.1**	7.40
$PaCO_2$	**17**	40 mmHg
HCO_3	**5**	24 mmol/L
Na^+	130	136-145 mmol/L
Cl^-	105	96 - 106 mmol/L

A. What is the acid base disorder? Metabolic Acidosis, adequately compensated

B. What else do we need to know ? Anion gap= 20
130 - (105+5) = 20
C. What is the clinical diagnosis? High anion gap metabolic acidosis (DKA)

Interactive Cases - 2

❖ **23 year old man with a 3 day history of diarrhea.**

	Case	Normal
pH	**7.28**	7.40
$PaCO_2$	**26**	40 mmHg
HCO_3	**12**	24 mmol/L
Na^+	135	136-145 mmol/L
Cl^-	110	96 - 106 mmol/L
K^+	3.2	3.6-5.2 mmol/L

What is the acid base disorder? Metabolic Acidosis (normal anion gap), adequately compensated

Interactive Cases - 3

❖ **55 yo man k/c of BA. In ER with SOB and cough for 2 days**

	Case	Normal
pH	**7.32**	7.40
$PaCO_2$	**50**	40 mmHg
HCO_3	**25**	24 mmol/L
Na^+	134	136-145 mmol/L
Cl^-	100	96 - 106 mmol/L
K^+	4.5	3.6-5.2 mmol/L

What is the acid base disorder? Acute Respiratory Acidosis (adequately compensated)

Interactive Cases - 4

❖ **55 yo man with COPD. Admitted for elective hernia repair. Pre operative ABG showed :**

	Case	Normal
pH	**7.37**	7.40
$PaCO_2$	**55**	40 mmHg
HCO_3	**31**	24 mmol/L
Na^+	136	136-145 mmol/L
Cl^-	96	96 - 106 mmol/L
K^+	3.5	3.6-5.2 mmol/L

What is the acid base disorder? Chronic respiratory Acidosis (adequately compensated)

Interactive Cases - 5

- **40 yo woman with repeated vomiting for 1 day.**

	Case	Normal
pH	**7.49**	7.40
$PaCO_2$	**48**	40 mmHg
HCO_3	**35**	24 mmol/L
Na^+	130	136-145 mmol/L
Cl^-	85	96 - 106 mmol/L
K^+	2.8	3.6-5.2 mmol/L

What is the acid base disorder? Metabolic Alkalosis (adequately compensated)

Interactive Cases - 6

- **28 yo man with abdominal pain and diarrhea. He is clinically volume depleted (low BP, tachycardia..)**

	Case	Normal
pH	**7.29**	7.40
$PaCO_2$	**21**	40 mmHg
HCO_3	**8**	24 mmol/L
Na^+	133	136-145 mmol/L
Cl^-	105	96 - 106 mmol/L

What is the acid base disorder? Combined anion gap and non-gap metabolic acidosis (Diarrhea induced HCO3 loss and lactic acidosis), adequately compensated

Introduction

Disorders of sodium are really disorders of water balance. Normally, there are two compartments - the blood and the brain. These compartments are in equilibrium. If there's a disturbance in how much "stuff" is in the blood the water will shift. When there's too much "stuff" in the blood, water will move out of the cells and into the blood to balance it (**Hypernatremia**), **dehydrating** the cells. If there's too much water (less "stuff") in the blood (**hyponatremia**), the water will move out of the blood and into the cells to balance it, causing them to **swell**. Either way, that's bad news for the cells. It's the dehydrating and swelling that leads to symptoms.

To fix **Hypernatremia** (which is always a deficiency in water) the task is easy. **PO Water** is always the best way to replace a water deficit. However, there may also be the need for **Hypotonic Solutions** such as D5W or 1/2NS. Before replacing the free water deficit, replace volume with Normal Saline.

To fix **Hyponatremia** the task is a bit more challenging. If the patient is in a **severe state** (regardless of the diagnosis), use **Hypertonic Saline** (**3%**). If not severe, then the management is based on the underlying diagnosis. For example, SIADH is treated with volume restriction, volume overload with diuresis, and volume depletion with volume resuscitation. To determine which course of action to take, further investigation is required.

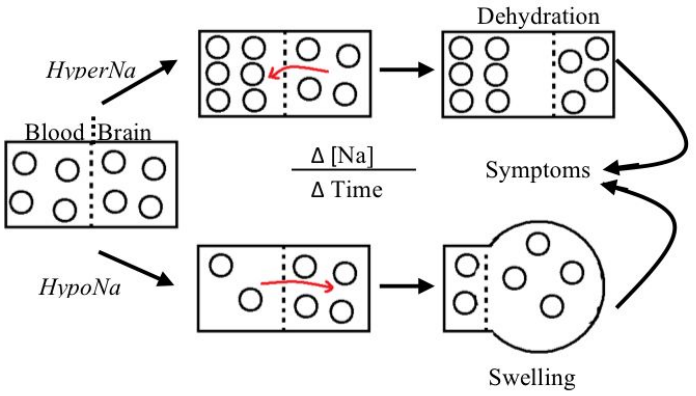
Sodium correction should occur **no faster than 0.25mmol/hr** unless severe. Correction by 4-6 is all that is required to eliminate symptoms in most cases. If sodium is corrected to quickly, it may result in **osmotic demyelination syndrome** (formerly called central pontine myelinolysis), leaving the patient a spastic quadriplegic. Thus, a **regular assessment of the Na** is required.

Isotonic Hyponatremia = Pseudohyponatremia

Pseudohyponatremia is a product of laboratory artifact. The **calculated osmoles** will be elevated, but **measured** will be normal. This is a result of fats and proteins. Newer labs rarely encounter this issue.

Hypertonic Hyponatremia

There can be other "stuff" in the blood that accounts for osmotic activity other than sodium. While the measured sodium is low, the **measured osmoles are elevated.** This is usually a product of **glucose**, **BUN**, or sugar alcohols. For every **100mg/dL** of glucose above 100 adjust for the Na by 1.6. If the corrected sodium is normal, correct the osmotic compound alone.



Onset	*Symptoms*	*Treatment*
Mild	Asymptomatic	HypoNa: Dz-Specific HyperNa: Po Water
Moderate	Nausea, Vomiting, Headache (all non-specific)	Hypo Na: IV NS HyperNa: IV NS NOT Hypertonic
Acute or Severe (Na<110, Hours)	Coma, Seizures, Death	HypoNa: IV Hypertonic HyperNa: IV D5W

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Hypotonic Hyponatremia = True Hyponatremia

If the **measured osmoles** are low, then the original assessment of the water status was accurate. Now it's up to you to determine the underlying etiology and correct it to correct the sodium.

Assessment of the **Urine Sodium** and **Urine Osmolality** can be used to separate most causes of hyponatremia. However, the clinical scenario often gives the answer. This is especially true on a vignette, where there can't be a mystery to have a single correct answer. If a urine sodium is decreased, the kidney is working and there's poor perfusion to it. If the urine osmoles are concentrated, ADH is activated. The appropriateness of this is discussed in the Posterior Pituitary lecture.

Hypervolemic Hyponatremia

If the patient is **wet** (i.e. JVD, edema, CHF, Anasarca), they're **overloaded**. The fluid is in the third space and needs to be mobilized. Treat with **diuretics**.

Hypovolemic Hyponatremia

If the patient is **volume down** (dry mucous membranes, burns, fevers, tachypnea, hypotension), then all the patient needs is **Volume resuscitation**. The sodium should correct with IVF.

Euvolemic Hyponatremia

If the patient is **euvolemic,** we're left with **RATS**. Rule out each disease one at a time. Renal Tubular Acidosis is assessed with a urinalysis, Addison's disease with cortisol, and Thyroid disease with a TSH.

SIADH is a diagnosis of exclusion. It's treated with **volume restriction** and **gentle diuresis**. Refractory cases can be treated with **demeclocycline**. Check out the endocrine topics for details.

Vaptans

Vaptans are absolutely contraindicated in hypovolemic hyponatremia. They're **never the right answer**.

Fluids

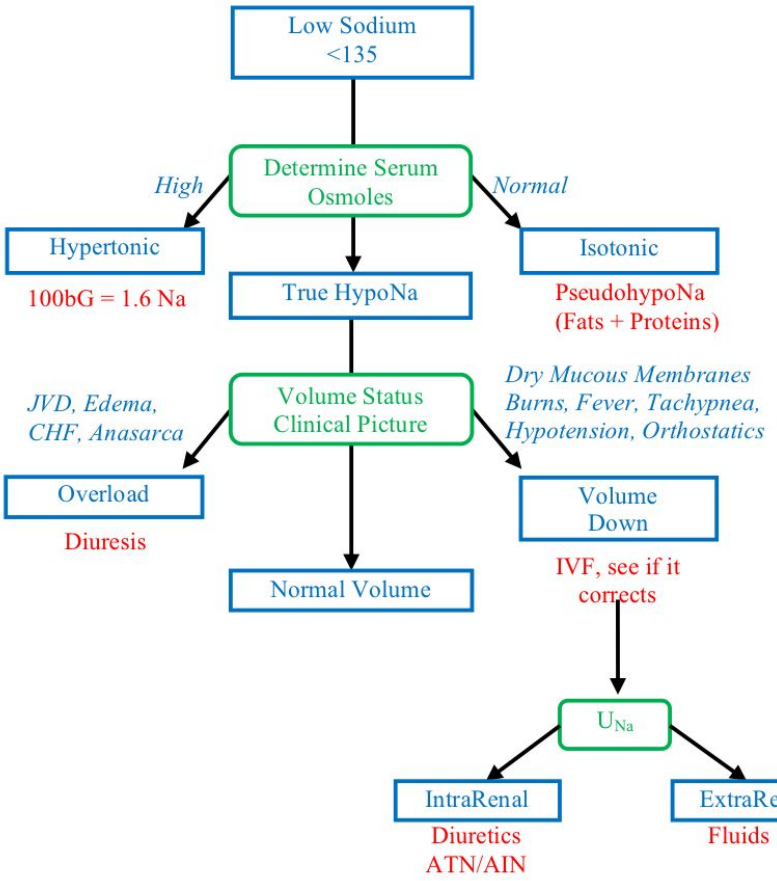
Fluids are discussed in greater detail in the Intern content.

Volume resuscitation is done with Normal Saline or Lactated ringers; it's provided as a bolus.

Free Water is replaced with **hypotonic solutions**, given either as PO free water or D5W. ½ NS is a hypotonic solution and can be used to administer free water, but I want you to learn it as maintenance fluid.

Maintenance fluid is administered as any combination of ½ NS, ¼ NS, with or without D5.

Nutrition is provided as PPN or TPN. D5 containing solutions do not count.



$$\text{Serum Osmoles} = (2xNa) + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8}$$

Volume	*Maintenance*	*Free Water*	*Nutrition*
NS	½ NS	PO Water	TPN
LR	½ NS D5	D5W	PPN
	¼ NS		
	¼ NS D5		

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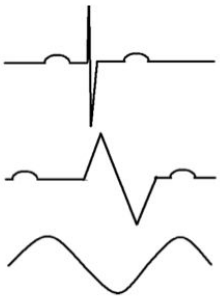
Hyperkalemia

Extracellular potassium is tightly regulated. It doesn't take that much extra potassium in a syringe to kill someone (**death penalty**). The range is typically **3.5 – 5.5** (>4.0 is normal in cardiac patients). There are many causes of hyperkalemia - some rare, some common. They all lead to the same symptoms: **areflexia**, **flaccid paralysis**, **paresthesia** (aka decrease motor and sensation) and **ECG Changes**. Whenever there's an abnormal potassium level, the first thing to do is **repeat the lab** (the sample could be busted or the cells could have hemolysed). Yet, the crucial evaluation is the **ECG 12-Lead**. Remember that "everything gets bigger…" as the **K goes up** the **PR prolongs**, the **QRS widens**, and the **T waves peak**. There are 3 phases of treatment. They're dependent on severity and ECG changes.

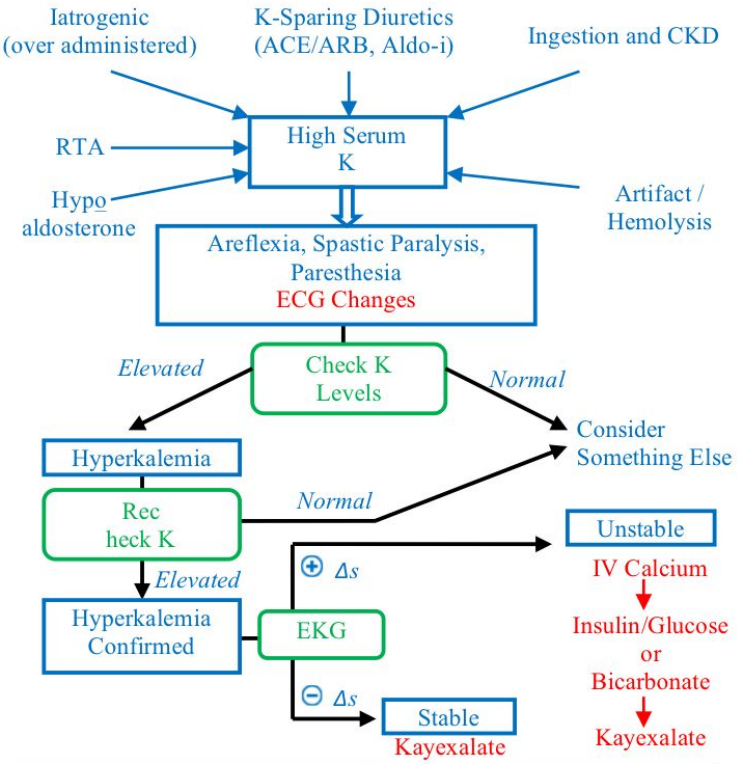
Phase I is to stabilize the myocardium with **IV calcium gluconate**. Phase II is to **decrease serum K** by sequestering it - hiding it - in the cells. Do that with **Insulin and glucose** (the insulin shifts the K, the glucose prevents hypoglycemia) or with **Bicarbonate**. Phase III is to actually **decrease total body K** with either K-wasting **diuretics** or more commonly with **Kayexalate**. If in Renal Failure or the K is extreme, use **Dialysis**.



Hypokalemia

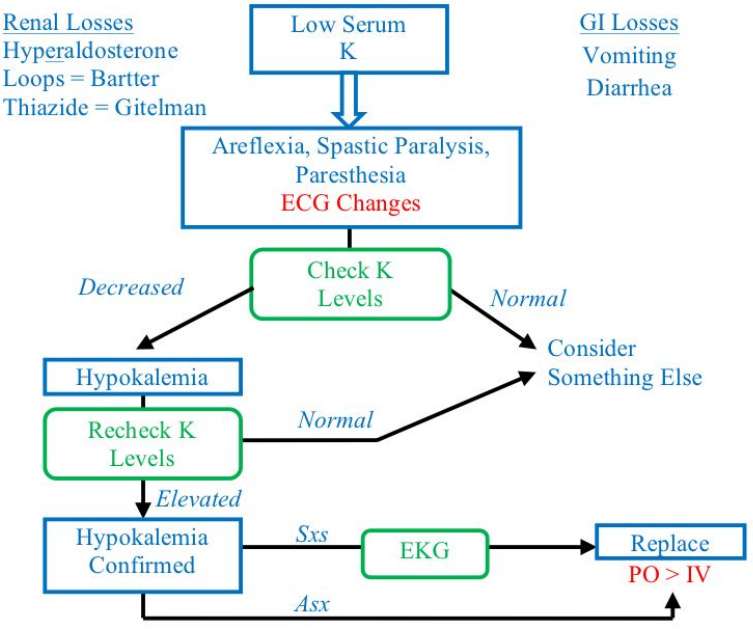
Less exciting than hyperkalemia but just as deadly, a **low potassium** has multiple potential causes. It's usually going to be through either **GI losses** (diarrhea, laxatives, vomiting) or **Renal Losses** (hyperaldo states, loop diuretics or thiazide diuretics).

While **rechecking the K** and **checking an EKG** could be done, mostly the K is simply repleted. Repletion is performed with oral or intravenous potassium. **Oral replacement is preferred**. If IV is to be used, the rate must be **<10mEq / hr** if by peripheral IV (PIV), or **<20mEq/hr** if by central line.



K comes back high on a lab draw	Repeat the draw
K comes back confirmed high but the patient is asx, Ø EKG Δ	Kayexalate, Stop K-Sparing drugs
Symptoms or EKG Δs With or without elevated K	IV Calcium, Insulin/Glucose, Kayexalate, Consider Dialysis
Renal Failure and any of the above	Dialysis

IV Calcium	Stabilize	Stabilizes Myocardium
Insulin + Glucose	Temporize	Shifts K into cells
Bicarbonate	Temporize	Shifts K into cells
Kayexalate	↓ Total K	↓ Total Body K
Dialysis	↓ Total K	↓ Total Body K



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You'll be asked to do two things: interpret a blood gas (which comes later) and decide what to do next. We first handle "what to do next," the potential diagnoses that might be encountered and how to spot them on a vignette.

The first step is to determine what the primary disturbance is. It's discussed in greater detail in gas interpretation, but basically <7.4 is acidic while >7.4 is basic. Then use the CO2 (with a cutoff of 40) to separate into respiratory or metabolic.

Respiratory Acidosis

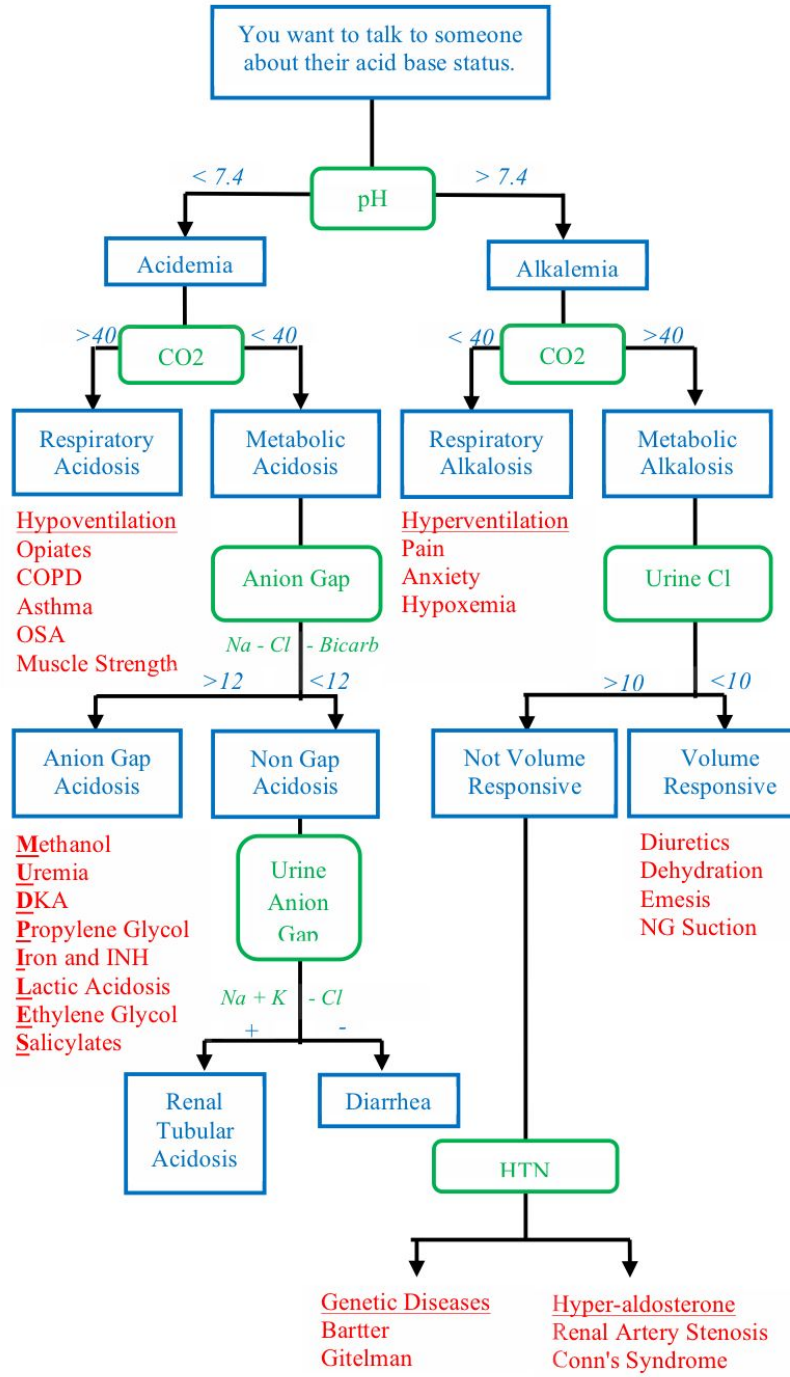
This is a product of hypoventilation. The less ventilation the more CO2 will accumulate. Whether it's a low tidal volume (COPD) or a low respiratory rate (opiate overdose), if either falls the CO2 rises. Look for things like wheezing (Obstructive Lung Disease), obesity (OSA), cyanosis and pinpoint pupils (opiates), or signs of muscle weakness (like paralysis from Guillain–Barré)

Respiratory Alkalosis

Conversely, respiratory alkalosis is from hyperventilation. Very few things will do that as a primary disturbance. It'll either be pain, anxiety or hypoxemia. Lots of things cause hypoxemia (pneumonia, PE, ARDS) so the patient can get complex, but in terms of acid-base respiratory alkalosis means hyperventilation.

Metabolic Alkalosis

The only thing that causes this is a high aldosterone. The decision is if the person is volume responsive - that is, will giving him/her volume improve their alkalosis? This is done in one of two ways: using the history to say he/she is volume down and give fluids, then recheck the bicarb OR by checking the **urine chloride**. The test loves the urine chloride. If it's low (**<10**) the patient is salt-sensitive, or volume responsive, and giving him/her volume will improve his/her condition. Look for the use of diuretics, emesis or NG suction, or another reason for them to be dehydrated (looking for insensible water losses like sepsis, fever, tachypnea, or tachycardia).



Test	*When To Use It*
pH	Start here
pCO2	After pH to get primary disturbance
Anion Gap	Metabolic Acidosis
Urine Anion Gap	Non-Gap Acidosis
Urine Chloride	Metabolic Alkalosis

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If the Urine Chloride is high (**>10**) it's a condition that has nothing to do with volume. It's then time to assess for the presence of **hypertension**.

If there is **+ HTN**, consider diseases of too much aldosterone; inappropriate elevations in aldosterone levels. It's most likely to be renal artery stenosis or Conn's syndrome (primary hyperaldosteronism). Keep in mind that the aldosterone was up in volume depletion to keep the pressure up. In this case it's up inappropriately, so it causes a rise in blood pressure.

If the patient is **- HTN**, think of Bartter and Gitelman syndromes - genetic, always present forms of HCTZ and Furosemide, respectively.

Metabolic Acidosis

Metabolic Acidosis is the hardest to handle; it's the most complex by far in gas interpretation. But it's pretty easy to get the answer right when trying to make a diagnosis based on the clinical scenario.

First, calculate the **anion gap** (Na - Cl - Bicarb). A normal gap is 12, or Albumin x 3. If greater, there's an anion gap acidosis, which can be reminded by a number of mnemonics. We've chosen MUDPILES in this section (just don't forget about Toluene). In an **anion gap metabolic acidosis** the diagnosis is made by the rest of clinical picture. Highlights of the ones you must know are to the right.

For **non-gap acidosis** the next step is the **urine anion gap**. The urine anion gap is calculated from similar but not the same electrolytes as the regular anion gap (frustrating), so be careful. If **positive** the answer is renal tubular acidosis. If **negative** the answer is diarrhea.

UCl < 10 = Volume Responsive

UCl > 10 = Not Volume Responsive

UCl > 10 and HTN = Inappropriate Aldosterone

UCl > 10 and no HTN = genetic diseases

ANION GAP: Na - Cl - Bicarb
(NO POTASSIUM)

Highlights to MUDPILES Diagnoses

DKA	Diabetic who is acidotic. Look for ketones. Treat with insulin, fluids, and replete potassium
Methanol	Homemade liquor (moonshine), causes blindness, no cure
Ethylene Glycol	Crystals in the urine, urine turns color under Wood's Lamp. Give either ethanol or fomepizole
Lactic Acidosis	Either Metformin + Acute Kidney Injury or... Patient in shock (fix the shock)

URINE ANION GAP: Na + K - Cl
(No Bicarb)

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Gas interpretation of acid-base disturbances is difficult. There will be one on your shelf. You're guaranteed at least one on the Step 2 as well. Unfortunately, being able to appropriately interpret a blood gas doesn't always prove incredibly useful in actual practice. But being able to master acid base disturbances can lead to an impressive evaluation (and can impress all your friends since they won't be able to do it). But in reality, if this stuff just takes too long and you still don't get it, take the hit on the test and move on. Better to randomly guess and get it wrong than spend 15 minutes on a question you may not get right (thereby wasting precious minutes that could have been used on other questions). With that in mind, let's get started.

Follow the Steps

Step 1: Acidemia or Alkalemia. Use 7.4
- Is the pH < 7.4 (acidemia)
- Is the pH > 7.4 (alkalemia)

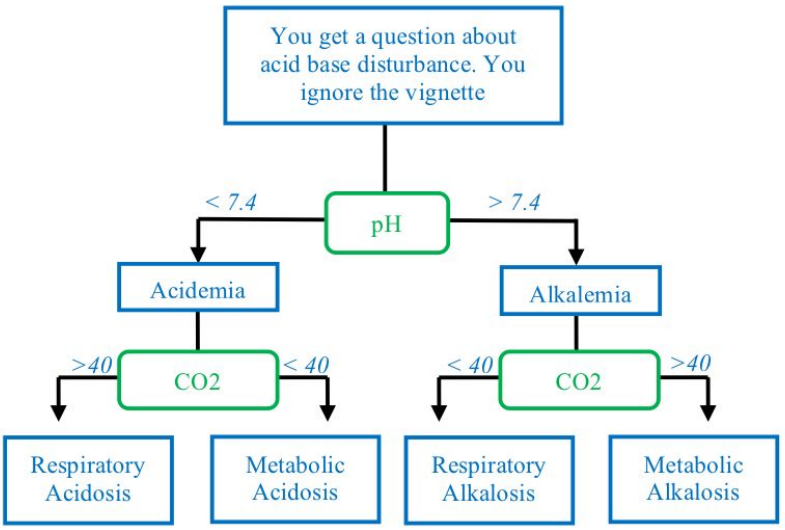
Step 2: Respiratory or Metabolic
See CO2 as respiratory and acid. CO2 is the respiratory acid. If you get rid of CO2 you get rid of respiratory acid; this should create an alkalotic environment. If you retain CO2 you hold onto more respiratory acid; it should create an acidotic environment.

After deciding if there's an Acidemia or Alkalemia ask, "What do I expect the CO2 to be - high or low?"

If there's a pH < 7.4, expect the CO2 to be higher than normal - that is >40. If it is, the acidemia is caused by a respiratory acidosis. If it isn't, the acidemia is caused by a metabolic acidosis.

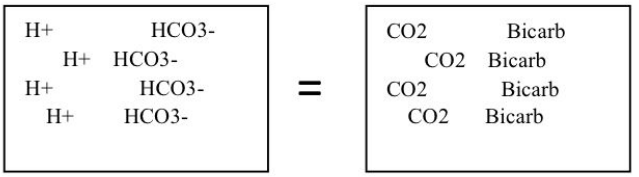
If there's a pH >7.4, expect the CO2 to be lower than normal (loss of respiratory acid). If it is, the alkalemia is caused by a respiratory alkalosis. If it isn't, the alkalemia is caused by a metabolic alkalosis.

This step is SUPER important because it decides what Step 3 is going to be. Once the primary disturbance is determined you then go through that disturbance start to finish.



Determining the Primary Disturbance

Step 1: Acidemia or Alkalemia
Step 2: Respiratory or Metabolic
....
Step 3: is there something else wrong?



H+ = "Respiratory Acid" = pCO2
HCO3- = "Metabolic Base" = Bicarb

More H+ = More pCO2 = Low pH
Less H+ = Less pCO2 = High pH

More HCO3- = More Bicarb = High pH
Less HCO2- = Less Bicarb = Low pH

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Step 3a: Check the anion gap.

Always check the anion gap. It's normally 12. It's actually about 3* Albumin, normal albumin being 4, so this may change in real life. When handling acid-base problems, view them w/ the assumption of a normal anion gap = 12. The reason to always check the anion gap is because if present (regardless of other findings), there must also be an anion gap metabolic acidosis. That's true even if it isn't the primary disturbance.

Anion Gap = Na - Cl - Bicarb

Normal Anion Gap = 12... or Albumin x 3

If the calculated anion gap (Na-Cl-Bicarb) is greater than the normal anion gap there is an anion gap metabolic acidosis

REGARDLESS of whatever else is going on

Respiratory Acidosis

Step 3b: Acute or Chronic

If the respiratory acidosis is acute then for every dime change (every 10 points) of CO2 the pH should change by 0.08. If the respiratory acidosis is chronic, then for every dime change of CO2 the pH should change by 0.04. Step 3b is to find out which it is: acute or chronic.

To do that, find out how many dimes from normal the CO2 is. Multiply that by 0.08 and subtract from the normal pH of 7.4. Do it again multiplying by 0.04 and subtracting that from the normal pH of 7.4. Compare both scores to whatever the pH actually is. Whichever is closer determines the chronicity.

For Every "Dime" Change in CO2

Δ pH		*Δ Bicarb*	
If Acute	0.08	If Acute	1
If Chronic	0.04	If Chronic	3

Formula for memorizers:

7.4 - (Dimes * 0.08) = pH if acute
7.4 - (Dimes * 0.04) = pH if chronic

Pick the one closest to the actual pH

Step 3c: Is there a Metabolic Derangement

For respiratory acidosis the bicarbonate should change as well. For every dime change in CO2 the bicarb should change by 1 point if acute or 3 points if chronic. Bicarb should change to compensate for the CO2; in a respiratory acidosis the bicarb should go up.

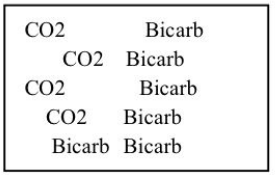
Multiply the number of dime change of CO2 by 1 (if acute) and by 3 (if chronic). Add that to a normal bicarb of 24. Compare to the bicarb you have. If there are more bicarbs than expected, there's also a metabolic alkalosis. If there are too few bicarbs, however, it's an additional metabolic acidosis.

Note that in the example the CO2s don't change. When exploring Step3c the only care is the bicarb number (too few, enough, too many). The CO2 doesn't matter except to the extent that we use it to determine how much the bicarb should have changed.

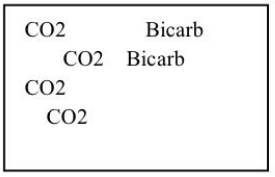
Formula for memorizers:

24 + (dimes * 1) = Expected bicarb if acute
24 + (dimes * 3) = Expected bicarb if chronic

If actual bicarb > expected bicarb: too many bicarbs = Metabolic Alkalosis



If actual bicarb < expected bicarb: not enough bicarbs = Metabolic Acidosis



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Respiratory Alkalosis

It's literally the same for respiratory acidosis, except that the bicarb changes by 2 (if acute) or 4 (if chronic) for every dime change. Let's spell it out here.

Step 3b: Acute or Chronic

If the respiratory alkalosis is acute then for every dime change (every 10 points) of CO2 the pH should change by 0.08. If the respiratory acidosis is chronic, then for every dime change of CO2 the pH should change by 0.04. Step 3b is to find out which it is: acute or chronic.

To do that, find out how many dimes from normal the CO2 is. Multiply that by 0.08 and subtract from the normal pH of 7.4. Do it again multiplying by 0.04 and subtracting that from the normal pH of 7.4. Compare both scores to whatever the pH actually is. Whichever is closer determines the chronicity.

Step 3c: Is there a Metabolic Derangement

For respiratory alkalosis the bicarbonate should change as well. For every dime change in CO2 the bicarb should change by 2 point if acute or 4 points if chronic. Bicarb should change to compensate for the CO2; in a respiratory acidosis the bicarb should go up.

Multiply the number of dime change of CO2 by 2 (if acute) and by 4 (if chronic). Add that to a normal bicarb of 24. Compare to the bicarb you have. If there are more bicarbs than expected, there's also a metabolic alkalosis. If there are too few bicarbs, however, it's an additional metabolic acidosis.

Metabolic Alkalosis

The only way this will happen is if the aldosterone is up. Don't care about the gas interpretation, but instead whether it's "salt sensitive," which always means, "volume responsive," which also asks, "are they volume deplete?" To figure that out simply give the patient volume.

The way Metabolic Alkalosis will appear on an acid-base interpretation question is as a secondary disturbance to a respiratory problem or on its own. That's it.

For Every "Dime" Change in CO2

Δ pH		*Δ Bicarb*	
If Acute	0.08	If Acute	2
If Chronic	0.04	If Chronic	4

Formula for memorizers:

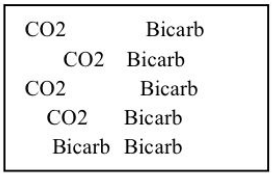
7.4 + (Dimes * 0.08) = pH if acute
7.4 + (Dimes * 0.04) = pH if chronic

Pick the one closest to the actual pH

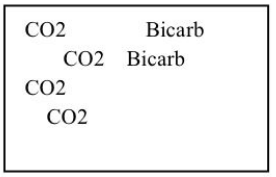
Formula for memorizers:

24 - (dimes * 2) = Expected bicarb if acute
24 - (dimes * 4) = Expected bicarb if chronic

If actual bicarb > expected bicarb:
too many bicarbs = Metabolic Alkalosis



If actual bicarb < expected bicarb:
not enough bicarbs = Metabolic Acidosis



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Respiratory Alkalosis

It's literally the same for respiratory acidosis, except that the bicarb changes by 2 (if acute) or 4 (if chronic) for every dime change. Let's spell it out here.

Step 3b: Acute or Chronic

If the respiratory alkalosis is acute then for every dime change (every 10 points) of CO2 the pH should change by 0.08. If the respiratory acidosis is chronic, then for every dime change of CO2 the pH should change by 0.04. Step 3b is to find out which it is: acute or chronic.

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For Every "Dime" Change in CO2

Δ pH		*Δ Bicarb*	
If Acute	0.08	If Acute	2
If Chronic	0.04	If Chronic	4

Formula for memorizers:

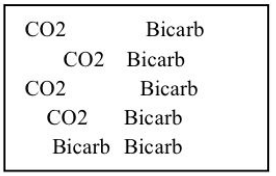
7.4 + (Dimes * 0.08) = pH if acute
7.4 + (Dimes * 0.04) = pH if chronic

Pick the one closest to the actual pH

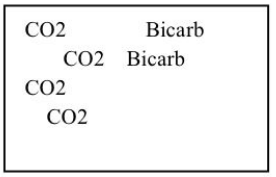
Formula for memorizers:

24 - (dimes * 2) = Expected bicarb if acute
24 - (dimes * 4) = Expected bicarb if chronic

If actual bicarb > expected bicarb:
too many bicarbs = Metabolic Alkalosis



If actual bicarb < expected bicarb:
not enough bicarbs = Metabolic Acidosis



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Lecture Quiz

1:C / 2:A / 3:B

Q1: A 32-year-old builder presents in accident and emergency in a distressed state. He reports suffering from chest pain for the last 2 weeks, the pain is sharp and only occurs when he moves heavy objects. He has a family history of cardiovascular disease and is worried about a heart attack. His blood gas findings are as follows: pH = 7.47; PCO2 = 3.3; PO2 = 15.3; bicarbonate = 17.53. The most likely diagnosis is:

A. Respiratory acidosis with metabolic compensation
B. Respiratory alkalosis with metabolic compensation
C. Acute respiratory alkalosis
D. Metabolic Alkalosis

Q2: A 47 y/o male presented to the ER with 2 days history of diarrhea. His vitals are BP=75/45, HR=113 , RR=23. How would you manage this patient?

A. Normal saline
B. Half normal saline
C. Quarter normal saline
D. 5% Dextrose

Q3: Calculate the anion gap, Na=132 , HCO3=16 , Cl=96

A. 24
B. 20
C. 12
D. 15