



(+) **Scott C. Sherman, MD**  
Assistant Professor of Emergency  
Medicine, Rush Medical College;  
Assistant Program Director, Cook County  
Emergency Medicine Residency,  
Chicago, Illinois

### **Acid-Base Made Easy**

The differential diagnoses for acid-base problems can be reduced to a workable few by using a minimal amount of laboratory data. Following well-established principles and formulas, the presenter will help you resolve common acid-base problem cases.

- Identify etiologies of anion gap and non-anion gap acidosis.
- Explain the principle of osmolar gap.
- Differentiate the causes of acid-base disturbance and discuss appropriate management.
- Identify potential life-threatening disorders by working through real ED cases.

WE-226  
Wednesday, October 29, 2008  
4:00 PM - 4:50 PM  
McCormick Place - Lakeside Building

(+) No significant financial relationships to disclose

# Acid-Base Made Easy

Scott C Sherman, MD  
Assistant Residency Director  
Department of Emergency Medicine  
Cook County Hospital (Stroger)  
Assistant Professor of Emergency Medicine  
Rush Medical College

Acid-base analysis strikes fear into the minds of both seasoned clinicians and their junior counterparts. Multiple formulas and rules exist to help guide us through the forest of diagnoses and complex problems. This lecture is set-up to provide a simple, systematic approach to interpreting arterial blood gas (ABG) samples. All that is needed is a little clinical information obtained from a history and physical examination, a few readily available laboratory tests, and the knowledge of five simple steps. Getting in the routine of performing these steps on each patient in which an ABG and electrolytes are performed will help decrease the rate of missed complex acid-base disturbances and hopefully improve patient care.

## Five Steps of Acid-Base Analysis<sup>1-5</sup>

**Step 1:** Acidemia (pH <7.38) or alkalemia (pH >7.42)?

**Step 2:** Primary respiratory or metabolic disturbance?  
(Look at PCO<sub>2</sub> on ABG or HCO<sub>3</sub> on metabolic panel.)

**Step 3:** Is there appropriate compensation for the primary disorder?  
Metabolic acidosis: PCO<sub>2</sub> = [1.5 x (serum HCO<sub>3</sub>)] + 8 (±2)  
Metabolic alkalosis: ↑PCO<sub>2</sub> = 0.6 x ↑HCO<sub>3</sub> (±2)  
Respiratory acidosis: ↑PCO<sub>2</sub> 10, ↑ HCO<sub>3</sub> by 1 (acute) or 4 (chronic)  
Respiratory alkalosis: ↓PCO<sub>2</sub> 10, ↓ HCO<sub>3</sub> by 2 (acute) or 5 (chronic)

**Step 4:** Is there an anion gap metabolic acidosis (AGMA)?  
AG = Na - (HCO<sub>3</sub> + Cl). If > 12, an AGMA is present.

**Step 5:** If metabolic acidosis, is there another concomitant metabolic disturbance?  
If **AGMA**, then calculate ΔGap = ΔAG - Δ HCO<sub>3</sub> = (AG - 12) - (24 - HCO<sub>3</sub>)  
If the ΔGap is > 6, there is a combined AGMA and metabolic alkalosis.  
If the ΔGap is < -6, there is a combined AGMA and NAGMA.  
If **NAGMA**, for every 1 mEq/L ↑Cl, there should be a 1 mEq/L ↓ HCO<sub>3</sub> (±5).  
If HCO<sub>3</sub> decrease is less than predicted, then NAGMA and metabolic alkalosis

# Explanation of the Five Steps

**Step 1.** This step is straightforward. Look at the pH. Is the blood acidemic or alkalemic? This is the primary disorder. Any compensation for a metabolic disturbance by the lungs or vice versa will not bring the pH back to “normal”.

**Step 2.** Determine whether the primary disorder is respiratory or metabolic. This is accomplished by looking at the bicarbonate on the chemistry or the pCO<sub>2</sub> on the ABG. In acidemia, low bicarbonate (< 24) and low pCO<sub>2</sub> (< 40) suggests a metabolic acidosis. Alternatively, a high bicarbonate (> 24) and high pCO<sub>2</sub> (> 40) suggests that the primary disorder is a respiratory one. The opposite is true for alkalemia. A patient with an elevated bicarbonate (> 24) and pCO<sub>2</sub> (> 40) supports a metabolic alkalosis, while low bicarbonate (< 24) and low pCO<sub>2</sub> (<40) supports a respiratory alkalosis.

**Step 3.** The next question you would like to answer is whether or not the other body system (kidneys in a primary respiratory disorder or lungs in a primary metabolic disorder) are compensating appropriately.

- **Metabolic acidosis:** Whether compensation is adequate or not is easiest to answer when the primary disorder is a metabolic acidosis. In this case, Winter’s formula is used. Winter’s formula states that the patient’s pCO<sub>2</sub> should be equal to the serum bicarbonate multiplied by 1.5 plus eight<sup>6</sup>. When this number is within two of the pCO<sub>2</sub>, the respiratory system is compensating appropriately.  $PCO_2 = 1.5(HCO_3) + 8 \pm 2$ . If the patient’s pCO<sub>2</sub> is higher than expected, a respiratory acidosis is present in addition to the primary metabolic acidosis. If the patient’s pCO<sub>2</sub> is less than expected, then there is a respiratory alkalosis in addition to the primary metabolic acidosis.
- **Metabolic alkalosis:** The respiratory system compensates for a metabolic alkalosis by increasing the pCO<sub>2</sub> level. However, unlike a metabolic acidosis, the normal respiratory compensation to a metabolic alkalosis is difficult to predict and the pCO<sub>2</sub> level rarely rises above 50 mmHg. However, the increase in the pCO<sub>2</sub> is approximately equal to the increase in HCO<sub>3</sub> multiplied by 0.6<sup>1</sup>.
- **Respiratory acidosis/alkalosis:** Renal compensation for respiratory acid-base abnormalities improves with time. Acute changes (48-72 hours) in respiratory function occur due to titration of bicarbonate by available buffer systems. In chronic situations (> 72 hours), the kidney is able to alter production and resorption of bicarbonate and ultimately affect a larger change. In acute respiratory acidosis, for every pCO<sub>2</sub> increase of 10 mmHg, bicarbonate increases by 1 mEq/L. In chronic respiratory acidosis, a similar change in pCO<sub>2</sub> will result in a bicarbonate change of 4 mEq/L. In patients with acute respiratory alkalosis, a pCO<sub>2</sub> decrease of 10 mmHg produces a drop in bicarbonate of 2 mEq/L, and when chronic, the bicarbonate drops 5 mEq/L. These formulas will give the clinician a rough estimate of whether the respiratory acid-base disorder is being compensated for by the kidneys appropriately and whether the patient is suffering from an **acute, chronic, or acute-on-chronic** respiratory ailment.

**Step 4.** Calculate the anion gap. This step must be carried out on all patients, not just those with a primary metabolic acidosis. The presence of an anion gap, with rare exceptions, means that an anion gap metabolic acidosis is present<sup>7</sup>. The anion gap is calculated by subtracting the sum of the chloride and bicarbonate from the sodium level.  $\text{Na} - (\text{Cl} + \text{HCO}_3)$ . For the purposes of this lecture and the sample calculations within it, the normal anion gap will be assumed to be 12. In reality, it is most likely slightly lower than this (approximately 6-10), but it varies from individual to individual and the most accurate determination would be a baseline anion gap before the patient became ill.

**Step 5.** This step is useful to detect a previously undetected metabolic disorder not discovered on the previous four steps. The premise is that for every increase in positive charges, there must be an equal decrease in negative charges. Therefore, in a patient with an anion gap metabolic acidosis, the elevation of the anion gap over 12 should be accompanied by a similar decrease in the bicarbonate concentration from normal (ie, 24). If the bicarbonate is lower than expected, then an additional metabolic acidosis of the non-anion gap variety exists (this may be a primary NAGMA or compensation for respiratory alkalosis as discovered in step 3). If the bicarbonate concentration is more than expected, then a metabolic alkalosis is present<sup>8</sup>. This calculation is known as the “delta gap” or sometimes the “delta-delta”. To avoid over-diagnosis and to account for the normal range of values for the anion gap and bicarbonate levels, a delta gap of greater than 6 or less than -6 is considered significant<sup>7,9</sup>. This range accounts for two standard deviations around the mean<sup>7</sup>.

In patients with a non-anion gap metabolic acidosis, a previously undetected metabolic alkalosis can be detected by noting that for every 1 mEq/L increase in the chloride (from the normal of 100; hyperchloremic acidosis) there should be a similar 1 mEq/L decrease in the bicarbonate level from 24. If the bicarbonate decrease is less than expected (by five to account for two standard deviations from the mean), then an underlying metabolic alkalosis is also diagnosed<sup>1</sup>.

Once the acid-base disturbances have been determined using the above steps, the finite list of clinical conditions that cause them should be considered based on the patient's medical history. As with any diagnostic test, the results of acid-base determinations cannot be properly interpreted without clinical correlation<sup>10</sup>.

## ● Respiratory Acidosis

Respiratory acidosis is characterized by an elevation in the  $\text{pCO}_2$  and a decrease in blood pH due most commonly to hypoventilation. It results from conditions that decrease the ability of the lungs to excrete carbon dioxide at a rate to keep up with the body's production. A differential diagnosis includes:

- Central nervous system depression (sedatives, CNS disease, sleep apnea)
- Pleural disease (large pneumothorax or pleural effusion)

- Lung disease (ARDS, COPD, pulmonary edema, severe pneumonia)
- Acute airway obstruction (laryngospasm, sleep apnea)
- Neuromuscular disorders (GBS, myasthenia gravis, botulism)
- Thoracic cage injury (flail chest)
- Ventilator dysfunction

The kidney compensates for primary respiratory acidosis by retaining bicarbonate. This compensation occurs over hours to days and is generally at a maximum within four days. The rate of onset of respiratory acidosis can be determined by the degree of renal compensation (increase in  $\text{HCO}_3^-$ ) as listed above in step 3. Alternatively, the chronicity of the respiratory acidosis can be predicted by the change in the pH. In acute respiratory acidosis, the pH decreases by 0.08 units for each increase of 10 mmHg in the  $\text{pCO}_2$  from its baseline of 40 mmHg. Chronic respiratory acidosis is marked by a decrease in the pH of 0.03 units for every increase of 10 mmHg in the  $\text{pCO}_2$ .

Differentiating acute from chronic respiratory conditions can have important clinical implications that may alert the clinician to a patient that is rapidly spiraling downward and might require emergent intubation, from a patient who has chronic disease, but is in less danger of imminent decompensation<sup>5</sup>.

**Example:** A 70 year-old smoker presents with an acute onset of shortness of breath.

ABG: pH 7.30,  $\text{pCO}_2 = 60$  mmHg,  $\text{pO}_2$  60 mmHg

Metabolic panel: Na 135, Cl 100,  $\text{HCO}_3^-$  30

Step 1: Acidosis

Step 2: Respiratory

Step 3: Acute on chronic.  $\text{pCO}_2$  increased by 20, therefore the  $\text{HCO}_3^-$  should increase by 2 if acute and 8 if chronic. Because the  $\text{HCO}_3^-$  increased from 24 to 30 (6), an acute on chronic respiratory acidosis is present.

Step 4:  $\text{AG} = 135 - (100 + 26) = 9$ . No anion gap metabolic acidosis

Step 5: XX

**Answer:** Acute on chronic respiratory acidosis due to COPD exacerbation

## ● Respiratory Alkalosis

In contrast to respiratory acidosis, respiratory alkalosis is characterized by a decrease in the  $\text{pCO}_2$  and an elevation in the blood pH. The  $\text{pO}_2$  can be used to distinguish between disease of the lungs and other causes of hyperpnea (eg, fever)<sup>11</sup>. The causes of a primary respiratory alkalosis include:

- CNS disease (CVA)
- Toxins (Salicylates)
- High altitude

- Severe anemia
- Pregnancy
- Lung disease/hypoxia (asthma, pneumonia, PE, pulmonary edema, pulmonary fibrosis)
- Anxiety
- Cirrhosis of the liver
- Fever (Sepsis)
- Ventilator dysfunction

**Example:** A 22 year-old woman presents with 4 hours of numbness in both hands typical of previous episodes of anxiety.

ABG: pH 7.48, pCO<sub>2</sub> 30 mmHg, pO<sub>2</sub> 86 mmHg

Metabolic panel: Na 140, Cl 110, HCO<sub>3</sub> 22

Step 1: Alkalosis

Step 2: Respiratory

Step 3: Acute. Drop in the pCO<sub>2</sub> by 10 corresponds to a drop in the HCO<sub>3</sub> by 2 if acute and 5 if chronic. 24-22 = 2 and therefore, as would be expected by the clinical history, an acute disorder is diagnosed. Note also that the pH went up by 0.08 as would be expected in an acute condition.

Step 4: AG 140-(110+22) = 8

Step 5: XX

**Answer:** Acute respiratory alkalosis secondary to a panic attack

## ● Metabolic Acidosis

In the presence of a pH < 7.38, metabolic acidosis is diagnosed as a primary condition when the pCO<sub>2</sub> is < 40 mmHg or the bicarbonate is < 24 mEq/L. Metabolic acidosis can be further classified based on the presence of an anion gap. The anion gap reflects the balance between positively and negatively charged particles in the blood. Sodium is the only significant positively charge particle that is measured, while the measured anions are chloride and bicarbonate. Therefore, the anion gap is calculated by the formula: **Na – (Cl + HCO<sub>3</sub>)**.

A “normal” anion gap is 6-12, although based on newer techniques for measurement using ion selective electrodes, the normal anion gap is approximately 6 (range 3-11)<sup>12</sup>. Unmeasured anions are negatively charged proteins (15 mEq/L), phosphates (2 mEq/L), sulfates (1 mEq/L), and organic acids (5 mEq/L); for a total of 23 mEq/L. Unmeasured cations include potassium (4.5 mEq/L), calcium (5mEq/L), and magnesium (1.5 mEq/L); for a total of 11 mEq/L.

One potential pitfall in the measurement of the anion gap is patients with low albumin. Albumin has several negative charges on it and therefore, in a patient with a low albumin

level, their “normal” anion gap might be much lower than 12. For every 1 gram drop in serum albumin level, the anion gap decreases by 2.5<sup>1</sup>. A patient with a calculated anion gap of 10 and a 2 gram drop in their albumin may actually have an anion gap metabolic acidosis (recalculated AG 15).

## **Anion Gap Metabolic Acidosis (AGMA)**

Detection of an AGMA is important because only a few conditions commonly cause it<sup>13</sup>. In addition, in mixed acid-base disorders, an elevation in the anion gap may be the only signal that a metabolic acidosis is present. The causes of an AGMA are divided into four main categories: renal failure, ketoacidosis, toxins, and lactic acidosis. Causes of lactic acidosis include conditions with inadequate tissue perfusion (hypoxemia, low cardiac output, peripheral shunting, sepsis).

### **A CAT MUDPILES**

- Analgesics (massive NSAID, acetaminophen)
- Cyanide, Carbon monoxide
- Arsenic, Alcoholic ketoacidosis
- Toluene
- Methanol, Metformin
- Uremia
- Diabetic ketoacidosis
- Paraldehyde, Phenformin
- Iron, Isoniazid
- Lactic acidosis
- Ethylene glycol
- Salicylates

An anion gap of 20 or more suggests that metabolic acidosis is present regardless of the pH or serum bicarbonate level. This statement is accurate because the body does not compensate for alkalosis by generating a large anion gap. The degree of rise in the anion gap also suggests how likely the cause will be diagnosed. In one study, when the anion gap was between 12-20, the specific cause was found in < 30% of patients. In patients with an anion gap > 20, the cause was identified in 77%, and when the gap was > 30, 100% of patients were diagnosed<sup>14</sup>.

Patients with alcoholic ketoacidosis will have an AGMA with ketones in their blood and urine. Because alcoholics can present with a multitude of problems (DT's, infection, vomiting, dehydration, pancreatitis) their acid-base abnormalities may be quite complex and require careful consideration. Although the pH is most frequently low, it can also be elevated or normal. The pathogenesis is related to both the effects of ethanol and acute starvation from lack of any other caloric intake. The metabolism of ethanol provides substrate for ketone production and decreases gluconeogenesis<sup>15</sup>.

In any patient with an AGMA, calculate an osmol gap. Osmol gaps are a clue to a potentially life-threatening toxic alcohol ingestion (ie. ethylene glycol and methanol).

The osmol gap is determined by subtracting the calculated osmolality from the measured osmolality. Calculated osmolality =  $2(\text{Na}) + \text{Glc}/18 + \text{BUN}/2.4 + \text{ETOH}/4.6$ . When the measured osmolality is more than the calculated osmolality by 10 mOsm/kg H<sub>2</sub>O, then the presence of an unmeasured substance should be considered<sup>16</sup>. The mnemonic for remembering the clinically important substances that cause an osmol gap is ME LIE (methanol, ethylene glycol, lactate, isopropyl alcohol, and ethanol)<sup>17</sup>. Unfortunately, a normal osmol gap does not exclude the possibility of a toxic alcohol ingestion<sup>18</sup>.

**Example:** 32 year old man with depression and alcohol abuse presents with altered mental status.

ABG: pH 6.9, pCO<sub>2</sub> 29, pO<sub>2</sub> 100

Metabolic panel: Na 140, Cl 101, HCO<sub>3</sub> 5

Step 1: Acidosis

Step 2: Metabolic

Step 3:  $\text{pCO}_2 = 1.5(\text{HCO}_3) + 8 = 15$ , but the patient's pCO<sub>2</sub> is higher than 15. Therefore, a respiratory acidosis is also present, possibly secondary to CNS depression.

Step 4:  $\text{AG} = 140 - (101 + 5) = 34$

Step 5:  $\text{Delta gap} = (34 - 12) - (24 - 5) = 3$ . No additional metabolic disorders other than AGMA.

**Answer:** Anion gap metabolic acidosis and respiratory acidosis. The patient had an osmol gap of 174 and a methanol level of 510 mg/dL.

## Non-Anion Gap Metabolic Acidosis (NAGMA)

A NAGMA is due to either GI or renal losses of bicarbonate. If desired, GI mediated and renally mediated losses can be distinguished by obtaining urine electrolytes (ie. Na, K, and Cl) and calculating the urine anion gap. The urine anion gap is the difference between the spot urine positive ions and spot urine negative ions. If an excess of negatively charged ions is present, the acidemia is due to the kidney<sup>1</sup>. Examples of NAGMAs include<sup>11</sup>:

- GI bicarbonate losses (diarrhea).
- Renal tubular acidosis.
- Carbonic anhydrase inhibitors.
- Ureteral diversions.
- Rehydration.
- K sparing diuretics
- NSAIDs
- Addison's disease

**Example:** A 68 year old man who recently took antibiotics for a skin infection presents with 10 episodes of watery diarrhea per day for the last 5 days.

ABG: pH 7.34, pCO<sub>2</sub> 34, pO<sub>2</sub> 80

Metabolic panel: Na 135, Cl 108, HCO<sub>3</sub> 18

Step 1: Acidosis

Step 2: Metabolic

Step 3:  $p\text{CO}_2 = 1.5(\text{HCO}_3) + 8 = 35$

Step 4:  $\text{AG} = 135 - (108 + 18) = 9$

Step 5:  $\text{Cl} \uparrow$  by 8 and  $\text{HCO}_3 \downarrow$  by 6; therefore there is no metabolic alkalosis.

**Answer:** NAGMA due to diarrhea

## • Metabolic Alkalosis

Metabolic alkalosis is characterized by an increase in the serum bicarbonate concentration. The most common cause of metabolic alkalosis is volume contraction. This may come in the form of obvious losses such as vomiting, but also occurs with significant third spacing of fluids. The mechanism of contraction alkalosis is related to increased mineralocorticoid secretion in response to extracellular volume depletion. Renal sodium resorption is enhanced in exchange for increased potassium and hydrogen ion excretion. Hypokalemia is a frequent finding in patients with metabolic alkalosis due to vomiting or diuretic use, but its presence also promotes metabolic alkalosis. The mechanism involves a transcellular shift of potassium out of the cell. Hydrogen ion enters the cell and this effect in the renal tubular cells results in increased hydrogen ion secretion and increased plasma bicarbonate. The causes of metabolic alkalosis are listed below.

- Volume contraction (vomiting, NG suction, loop or thiazide diuretics).
- Excess glucocorticoids or mineralocorticoids (eg, Cushing's syndrome).
- Hypokalemia
- Bartter's syndrome.
- Alkali ingestion/infusion.
- Post-hypercapnic alkalosis

**Example:** A 20 year old student presents with excessive vomiting after binge drinking.

ABG: pH 7.50,  $p\text{CO}_2$  44,  $p\text{O}_2$  100

Metabolic panel: Na 138, Cl 100,  $\text{HCO}_3$  30

Step 1: Alkalosis

Step 2: Metabolic

Step 3: Increase in  $p\text{CO}_2$  should equal 0.6 multiplied by the elevation of the  $\text{HCO}_3 \pm 2$ .

The increase of the  $p\text{CO}_2$  of 4 is within two of 6(0.6) or 3.6; therefore there is appropriate compensation.

Step 4:  $\text{AG} = 138 - (100 + 30) = 8$

Step 5: XX

**Answer:** Metabolic alkalosis secondary to vomiting.

## Practice Cases<sup>2,7</sup>

### CASE 1

A diabetic presents with diarrhea and cough. CXR reveals an infiltrate.

pH 7.31; pCO<sub>2</sub> 10

Na 123; Cl 99; HCO<sub>3</sub> 5

Step 1: Acidemic or Alkalemic

Step 2: Primary respiratory or metabolic

Step 3: Compensation appropriate?

YES or NO

If met acidosis, then  $1.5(\text{HCO}_3) + 8 = \text{_____} \pm 2 = \text{pCO}_2?$

If NO, respiratory acidosis or respiratory alkalosis

Step 4:  $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{_____}$  AGMA?

YES or NO

Step 5: If metabolic acidosis, other metabolic disturbances?

If AGMA, calculate delta gap.  $(\text{AG}-12) - (24- \text{HCO}_3) = \text{_____}$

> 6, then AGMA and metabolic alkalosis

< -6, then AGMA and NAGMA

### CASE 2

An alcoholic presents with vomiting.

pH 7.20; pCO<sub>2</sub> 25

Na 130; Cl 80; HCO<sub>3</sub> 10

Step 1: Acidemic or Alkalemic

Step 2: Primary respiratory or metabolic

Step 3: Compensation appropriate?

YES or NO

If met acidosis, then  $1.5(\text{HCO}_3) + 8 = \text{_____} \pm 2 = \text{pCO}_2?$

If NO, respiratory acidosis or respiratory alkalosis

Step 4:  $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{_____}$  AGMA?

YES or NO

Step 5: If metabolic acidosis, other metabolic disturbances?

If AGMA, calculate delta gap.  $(\text{AG}-12) - (24- \text{HCO}_3) = \text{_____}$

> 6, then AGMA and metabolic alkalosis

< -6, then AGMA and NAGMA

### **CASE 3**

A man with arthritis presents with confusion, shortness of breath, and diaphoresis.

pH 7.30; pCO<sub>2</sub> 18

Na 147; Cl 108; HCO<sub>3</sub> 16

Step 1: Acidemic or Alkalemic

Step 2: Primary respiratory or metabolic

Step 3: Compensation appropriate?

YES or NO

If met acidosis, then  $1.5(\text{HCO}_3) + 8 = \text{_____} \pm 2 = \text{pCO}_2?$

If NO, respiratory acidosis or respiratory alkalosis

Step 4:  $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{_____}$  AGMA?

YES or NO

Step 5: If metabolic acidosis, other metabolic disturbances?

If AGMA, calculate delta gap.  $(\text{AG}-12) - (24 - \text{HCO}_3) = \text{_____}$

> 6, then AGMA and metabolic alkalosis

< -6, then AGMA and NAGMA

### **CASE 4**

A patient with COPD presents with shortness of breath.

pH 7.18; pCO<sub>2</sub> 80

Na 135; Cl 93; HCO<sub>3</sub> 30

Step 1: Acidemic or Alkalemic

Step 2: Primary respiratory or metabolic

Step 3: Compensation appropriate?

↑PCO<sub>2</sub> 10, ↑ HCO<sub>3</sub> by 1 (acute) or 4 (chronic)

Acute or Chronic or Both

Step 4:  $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{_____}$  AGMA?

YES or NO

Step 5: If metabolic acidosis, other metabolic disturbances?

xxx

### **CASE 5**

A woman with Crohn's disease presents with fever, vomiting, and diarrhea.

pH 7.36; pCO<sub>2</sub> 22

Na 147; Cl 121; HCO<sub>3</sub> 14

Step 1: Acidemic or Alkalemic

Step 2: Primary respiratory or metabolic

Step 3: Compensation appropriate?

YES or NO

If met acidosis, then  $1.5(\text{HCO}_3) + 8 = \text{_____} \pm 2 = \text{pCO}_2?$

If NO, respiratory acidosis or respiratory alkalosis

Step 4:  $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{_____}$  AGMA?

YES or NO

Step 5: If metabolic acidosis, other metabolic disturbances?

If NAGMA, increase in Cl should equal decrease in HCO<sub>3</sub>

If HCO<sub>3</sub> decrease is less than predicted, then metabolic alkalosis

### **CASE 6**

A noncompliant patient with diabetes and cirrhosis presents with vomiting.

pH 7.46; pCO<sub>2</sub> 17

Na 133; Cl 84; HCO<sub>3</sub> 15

Step 1: Acidemic or Alkalemic

Step 2: Primary respiratory or metabolic

Step 3: Compensation appropriate?

↓PCO<sub>2</sub> 10, ↓HCO<sub>3</sub> by 2 (acute) or 5 (chronic)

Acute or Chronic or Both

Step 4:  $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{_____}$  AGMA?

YES or NO

Step 5: If metabolic acidosis, other metabolic disturbances?

If AGMA, calculate delta gap.  $(\text{AG}-12) - (24 - \text{HCO}_3) = \text{_____}$

> 6, then AGMA and metabolic alkalosis

< -6, then AGMA and NAGMA

**Answers:**

- Case 1: Primary AGMA (DKA), respiratory alkalosis (pneumonia), NAGMA (diarrhea)  
Case 2: Primary AGMA (alcoholic ketoacidosis), metabolic alkalosis (vomiting)  
Case 3: Primary AGMA and respiratory alkalosis (Salicylate toxicity—107 mg/dl)  
Case 4: Primary respiratory acidosis—acute-on-chronic (COPD exacerbation)  
Case 5: Primary NAGMA (diarrhea), respiratory alkalosis (fever), metabolic alkalosis (vomiting)  
Case 6: Primary chronic respiratory alkalosis (cirrhosis), AGMA (DKA), metabolic alkalosis (vomiting)

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