Clinical Approach to the Patient with Potential Acid-Base Disturbances

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Received: 3/1/07; Accepted: 7/4/07

Introduction

Patient can present with normal acid-base balance, a single disorder, or multiple combinations of two or three disorders. The pH can be estimated in a patient with a single disturbance using the formulas which describe predicted compensation.

Blood gas studies are required for diagnosis and pH estimation when more than one disturbance may be present or when the information is insufficient to allow diagnosis of a single disturbance with confidence (e.g., the patient is unable to give an adequate history).

The diagnostic approach to acid-base disorders utilizes information from the history and physical examination, venous blood laboratory studies, and arterial blood gas studies in a stepwise fashion.

Step 1. Identify potential processes in the history, and recognize the situations that often are associated with mixed disturbances. Potential processes which can be identified in the history include:

- Congestive heart failure = respiratory alkalosis
- Pneumonia = respiratory alkalosis (tachypnea)
- Obstructive airway disease = chronic respiratory acidosis
- Renal failure = metabolic acidosis
- Hepatic failure = respiratory alkalosis
- Vomiting = metabolic alkalosis
- Diarrhea = metabolic acidosis
- Thiazide and loop diuretics = metabolic alkalosis
- Acetazolamide = metabolic acidosis

Situations which are often associated with mixed disturbances include:

- Pulmonary edema or asthma: Respiratory alkalosis followed by respiratory acidosis (as the condition worsens) and metabolic acidosis (lactic acidosis due to hypoxia).
- Sepsis: Respiratory alkalosis and metabolic acidosis (lactic acidosis)
- Hepatorenal syndrome: Respiratory alkalosis (liver failure) and metabolic acidosis (renal failure)
- Cardiopulmonary arrest: Respiratory acidosis (lack of pulmonary function) and metabolic acidosis (lactic acidosis due to tissue hypoperfusion)
- Salicylate poisoning: Respiratory alkalosis (due to stimulation of respiratory system by salicylate) and metabolic acidosis (due to salicylic acid)
- Use of diuretics to treat right sided failure in chronic lung disease: Metabolic alkalosis (diuretics) and chronic respiratory acidosis (chronic lung disease)

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Vomiting in a patient with renal failure: Metabolic alkalosis plus metabolic acidosis

**Step 2.** Review the physical examination for clues to diagnoses. This is usually much less productive than a good history. Clues include:

- Tachypnea = respiratory alkalosis
- Tetany (Chvostek or Trousseau sign) = low ionized Ca concentration. Alkalosis lowers the ionized Ca concentration by increasing Ca binding to protein
- Hypotension = hypoperfusion with metabolic acidosis from lactic acid production.
- Edema can indicate congestive heart failure which is often associated with respiratory alkalosis
- Jaundice, ascites and tremor can indicate impending liver failure which is associated with respiratory alkalosis

**Step 3.** Review the laboratory data. Laboratory data provide information on organ function as well as the electrolytes which provide specific information relative to acid-base. First check the studies that indicate organ function and provide potential diagnoses.

- The BUN and creatinine indicate renal failure
- Liver enzyme tests indicate hepatic function
- Positive blood cultures would confirm the presence of sepsis

Next evaluate the electrolytes as follows:

- **HCO₃ Concentration:** Is the total CO₂ (the bicarbonate concentration) normal, decreased or increased?)
  
  If normal, either the patient has no acid-base disturbances or he has more than one (ie, a mixed or complex disturbance) each of which influences the HCO₃ in a different direction.

  If low, there is either metabolic acidosis or respiratory alkalosis. Determine which fits better with the history and physical examination.

  If high, there is either metabolic alkalosis or chronic respiratory acidosis. Determine which fits better with the history and physical examination.

- **Delta (Anion gap):** Is the AG increased above normal (ie, >12-13 mEq/L)?

  If no, a process which produces H⁺ in association with anions (lactate, Ketoadacidosis, etc) is not present.

  If yes, by definition there is present a process that is producing acid (ie, H⁺ in association with an anion).

  If this AG is increased above normal, there should be concordance between the fall in the HCO₃ concentration and the increase in AG. If there is not concordance, several processes are present.

  As an example, a patient presents with diabetic ketoacidosis and the following set of electrolytes is obtained: Na 140, Cl 102, K 5.1, HCO₃ 10. The AG is 28 and it has increased by 15-16 from a normal value of 12-13. The HCO₃ has fallen by 15 from a normal value of 25. The concordance in the change in the HCO₃ and the AG is consistent with a simple disturbance of metabolic acidosis.

  However, if the values were Na 140, Cl 108, K 5.1, HCO₃ 10, there is not concordance. The AG is 22, an increase of 10-11 above the normal while the HCO₃ fell by 15. Therefore, some process in addition to increased metabolic acid production is contributing to the fall in HCO₃ without affecting the AG. This could be respiratory alkalosis or it could be non-AG metabolic acidosis (eg, from diarrhea) both conditions which lower the HCO₃ concentration but do not increase the AG.

**Step 4.** Assemble a tentative acid-base diagnosis using the information obtained with the history, physical examination and laboratory data.

- **Simple disturbance:** If all the data sources are consistence with the presence of a single disturbance, estimate the pH from the
bicarbonate concentration using the modified Henderson Hasselbach equation as follows: 
\[ H^+ = 24 \, pCO_2 \cdot HCO_3^- \]
To convert H into pH use the equation listed below:

\[ \Delta 10 \, nEq/L \, \text{increment in } H^+ = 0.10 \, \text{fall in } pH \]

(every 0.1 fall in pH is equivalent to a 10 nEq/L rise in plasma H\(^+\) concentration):

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<tr>
<th>pH</th>
<th>H(^+) (nEq/L)</th>
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<tr>
<td>7.40</td>
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Blood gases are not necessary unless there is likely to be a marked change in pH.

- **Complex disturbance**: If the data suggest that there is more than one disturbance present, the pH cannot be estimated. It is necessary to measure the pH to corroborate the diagnoses present and to determine treatment for the abnormal pH.

Mistakes can be made in the laboratory. Always ensure the blood gas data are correct (i.e., fit the Henderson Hasselbach equation before using the information). If the data are correct, are they consistent with normal compensation of a simple disturbance? If yes, the process is complete. If no, why? When you are happy with the validity of the pH and the diagnosis, complete a treatment plan.

**Normal Compensation**

The following equations are used to evaluate the appropriateness of compensation for a single acid-base disorder.

- **Acute respiratory acidosis**: \( \Delta HCO_3^- = 0.1 \, \Delta pCO_2 \)
  The plasma HCO\(_3^-\) concentration rises by 1 mEq/L for every 10 mmHg elevation in the pCO\(_2\).

- **Acute respiratory Alkalosis**: \( \Delta HCO_3^- = 0.2 \, \Delta pCO_2 \)
  The plasma HCO\(_3^-\) concentration falls by 2 mEq/L for every 10 mmHg decline in the pCO\(_2\).

- **Chronic respiratory acidosis**: \( \Delta HCO_3^- = 0.3 \, \Delta pCO_2 \)
  The plasma HCO\(_3^-\) concentration rises by 3 mEq/L for every 10 mmHg elevation in the pCO\(_2\).

- **Chronic respiratory alkalosis**: \( \Delta HCO_3^- = 0.4 \, \Delta pCO_2 \)
  The plasma HCO\(_3^-\) concentration falls by 4 mEq/L for every 10 mmHg decline in the pCO\(_2\).

- **Metabolic acidosis**: \( \Delta pCO_2 = 1.2 \Delta HCO_3^- \)
  The respiratory compensation results in a 1.2 mmHg fall in the pCO\(_2\) for every 1 mEq/L reduction in serum HCO\(_3^-\) concentration.

- **Metabolic alkalosis**: \( \Delta pCO_2 = 0.6 \Delta HCO_3^- \)
  The respiratory compensation results in an increase in pCO\(_2\) of 0.6 mmHg for every 1 mEq/L elevation in serum HCO\(_3^-\) concentration.

**ACID-BASE DISTURBANCES**

**CLINICAL QUIZ**

1. A 12-year old female is brought to the Emergency Room because of increasing weakness. She had been having low grade fever and severe diarrhea for four days. Laboratory studies reveal sodium 140 mEq/L, potassium 2.4 mEq/L, chloride 115 mEq/L, bicarbonate 15 mEq/L, BUN 21 mg/dL, creatinine 1.5 mg/dL, glucose 88 mg/dL, calcium 10.0 mg/dL, phosphate 3.5 mg/dL, magnesium 1.8 mg/dL, and plasma osmolality 284 mosmol/kg.

What do you estimate her arterial pH to be?

A. 7.20-7.24  
B. 7.25-7.29  
C. 7.30-7.34  
D. 7.40-7.44  
E. 7.45-7.49

The correct answer is **C**. The acid-base diagnosis is uncomplicated hyperchloremic acidosis due to severe diarrhea. This would allow you to estimate her pCO\(_2\) from Winter’s formula which applies only when simple
(uncomplicated) metabolic acidosis is present: \( \Delta p\text{CO}_2 = 1.2 \times \Delta \text{HCO}_3 \) or 12 mmHg. Thus, the predicted pCO\(_2\) compensation would be 28 mmHg, the difference between normal pCO\(_2\) and the expected fall in pCO\(_2\) [normal pCO\(_2\) (40)] – [\( \Delta p\text{CO}_2 \) (12)]. The pH can then be calculated with the modified Henderson-Hasselbach equation: \( H^+ = 24 \times p\text{CO}_2 : \text{HCO}_3 \). The value obtained is 45 nEq/L, which is equivalent to a pH of 35.0 (every 0.1 fall in pH is equivalent to a 10 nEq/L rise in plasma H\(^+\) concentration):

\[
\Delta 10 \text{ nEq/L increment in } H^+ = \Delta 0.10 \text{ fall in pH}
\]

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The point of this exercise is to remind you that the principal indication for a blood gas is the inability to estimate pH when there are multiple acid-base abnormalities present or when you cannot be sure that there is only one abnormality present.

2. An 18-year old boy is brought to the ER in a coma. There is no other history available. Physical examination revealed a thin, nonicteric Caucasian female in coma. The patient is comatose without focal neurological signs, Temp is 37ºC; BP 136/88 mmHg; pulse 98; respiratory rate 24/min; weight 70 kg; length 170 cm. The chest is clear without rales or rhonchi. The heart rate is regular; there are no extra sounds or murmurs. The abdomen is soft. Mild hepatosplenomegaly is present without jaundice or ascites. No other masses are palpable. Bowel sounds are present. Mild kyphosis is noted. Extremities are free of rashes or edema. Laboratory studies reveal sodium 140 mEq/L, potassium 5.5 mEq/L, chloride 106 mEq/L, HCO\(_3\)- 6.6 mEq/L, BUN 30 mg/dL, creatinine 0.8 mg/dL, glucose 95 mg/dL, calcium 9.0 mg/dL, phosphate 2.9 mg/dL, magnesium 1.7 mg/dL, plasma osmolality 340 mosmo/kg, and serum ketones 1+.

Would you like to draw a blood gas?
A. Yes, I believe one is indicated
B. No, I do not believe it is necessary

The correct answer is A. A blood gas is indicated in this case. This is because you cannot be sure that there is only one acid-base disturbance present when there is no history available and thus you cannot predict the pH.

3. An arterial blood gas was obtained which revealed: pH 7.20; pCO\(_2\) 16 mmHg; HCO\(_3\)- 15 mEq/L; pO\(_2\) 110 mmHg.

What is the acid-base diagnosis?
A. Metabolic acidosis
B. Metabolic acidosis plus metabolic alkalosis
C. Metabolic acidosis plus respiratory acidosis
D. Metabolic acidosis plus respiratory alkalosis
E. Metabolic acidosis plus metabolic alkalosis plus respiratory acidosis

The correct answer is D. This is anion gap (AG) metabolic acidosis in a patient without signs suggestive of hepatic coma. Laboratory studies on venous blood reveal an increased anion AG and a reduced HCO\(_3\)- compatible with AG metabolic acidosis. The fall in the bicarbonate concentration (−18) is close to the change in the AG (+17) suggesting that another acid-base process is not present. The blood gas data are compatible with metabolic acidosis and inappropriate respiratory compensation as determined by Winter’s formula discussed earlier.

4. What is the likely cause of the anion gap metabolic acidosis in this patient?
A. Alcoholic ketoacidosis
B. Idiopathic or primary lactic acidosis
C. Toxic ingestion (methanol, ethylene glycol)
D. Renal failure
E. Starvation ketoacidosis

The correct answer is C. The potential causes of AG metabolic acidosis include: starvation ketoacidosis, alcoholic ketoacidosis, methanol intoxication, ethylene glycol intoxication, lactic acidosis, inborn error of metabolism, and
renal failure. Blood ketones are only trace positive and the serum creatinine is normal. However, the measured serum osmolality is considerably higher than the calculated osmolality (340 vs. 296 mosmol/kg).

The presence of an osmol gap is consistent with toxic ingestion with a substance such as alcohol, methanol or ethylene glycol which produce acidosis by interfering with metabolism and in part by producing lactic acidosis.

5. A 19-year old boy presents to the ER with severe vomiting and the recent onset of chest pain. The patient has a history of peptic ulcer disease. Vomiting began 48 hours ago and continued until the present time without improvement. Fifteen minutes before arriving in the ER, the patient developed the sudden onset of left sided pleuric chest pain, shortness of breath and hemptysis. Physical exam revealed a tachypneic male in acute distress with a respiratory rate of 30/min and complaining of chest pain. The remainder of the examination was normal. Lab data revealed, sodium 140 mEq/l, potassium 3.0 mEq/L, chloride 92 mEq/L, CO₂ 36 mEq/L, BUN 30 mg/dL, creatinine 1.3 mg/dL, calcium 10.0 mg/dL, phosphate 3.5 mg/dL, blood ketones negative, glucose 90 mg/dL and plasma osmolaity 280 mosoml/kg. The chest x-ray showed marked pleural effusion on the left pleural chest with the left lower lobe infiltrate.

Would you like to draw a blood gas?
A. No
B. Yes

The correct answer is B. Yes, this is because the history suggests that more than one acid-base disturbance may be present and you cannot therefore predict the arterial pH.

6. The blood gas revealed a pH of 7.69 with a pCO₂ of 30 mmHg, a HCO₃ of 35 mEq/l and a pO₂ of 75 mmHg.

What is the acid-base diagnosis?
A. Simple metabolic alkalosis
B. Respiratory alkalosis
C. Respiratory acidosis
D. Mixed metabolic and respiratory alkalosis
E. Mixed metabolic alkalosis and acidosis

The correct answer is D. This is an example of primary metabolic alkalosis plus primary respiratory acidosis. The history of vomiting suggests the presence of metabolic alkalosis. The pulmonary embolus suggests that there may be superimposed acute respiratory alkalosis. Laboratory studies reveal an increased BUN to creatinine ratio consistent with volume contraction. The bicarbonate is increased and the AG is normal consistent with metabolic alkalosis without metabolic acidosis. Blood gas values show a markedly increased pH with an increased HCO₃ and a low pCO₂ confirming the presence of both metabolic alkalosis and respiratory alkalosis.

7. A 16-year old women is seen for a routine examination. The history is basically non-revealing and she has no current complains. She denies taking any medications now but does admit to using diet pills in the past. She also relates that her potassium has been low on several occasions in the past. Physical exam revealed a thin Caucasian female in no acute or chronic distress. Tem 37ºC; BP 116/80 mmHg; pulse 78 beats/min; respiratory rate16/min; weight 49 kg, Ht 162 cm. The heart is regular; there are no extra sounds or murmurs. The lungs are clear without rales or rhonchi, abdomen is soft without palpable masses. Bowel sounds are present. There is no edema. Neurological exam is intact. Laboratory data revealed sodium 136 mEq/L, chloride 95 mEq/L, potassium 3.0 mEq/L, CO₂ 36 mEq/L, BUN 15 mg/dL, creatinine 1.1 mg/dL, calcium 10.0 mg/dL, phosphate 3.6 mg/dL, magnesium 1.7 mg/dL and albumin 4.6 g/dL.

Would you like to draw a blood gas?
A. Yes
B. No

The correct answer is B. A blood gas is not necessary in this case. There is sufficient information in the history and laboratory data to make a diagnosis of a single acid-base disturbance. This allows prediction of the arterial pH from Winter’s formula for that disturbance and a blood gas is not necessary.
8. What is the acid-base diagnosis and which studies would help with the differential diagnosis of this condition? (Select all that apply)
   A. Respiratory alkalosis
   B. Metabolic acidosis
   C. Metabolic alkalosis
   D. Respiratory acidosis
   E. Measurement of urinary aldosterone
   F. Urinary diuretic screen
   G. Measurement of urinary chloride excretion

The correct answers are C, F and G. This is a case of metabolic alkalosis. The history reveals that hypokalemia might be present. Diet pills are often diuretics which may cause a metabolic alkalosis.

Laboratory data reveal an increased bicarbonate, a normal AG, and hypokalemia associated with renal K wasting and metabolic alkalosis due to diuretics or upper GI losses (vomiting). Rarely the etiology is one of the inherited salt-wasting nephropathies (Bartter’s syndrome or Gitelman’s syndrome). Urinary Cl excretion helps to distinguish between diuretics and vomiting as causes of metabolic alkalosis. Vomiting is associated with ECF volume depletion, chloride depletion and a urinary Cl excretion less than 15 mEq/L. In contrast, urinary Cl excretion is usually greater than 40 mEq/L in conditions associated with Bartter’s syndrome, Gitelman’s syndrome, hyperaldosteronism or use of diuretics. Suggestive diuretic abuse can be confirmed with a urinary diuretic screen.

9. You are called to see a 19-year old male found to be stuporous at home. According to his father who found him this morning, he had apparently been complaining of continuous tension type headaches for the past several months. He was otherwise asymptomatic in good health with only a history of mild hypertension for which he had been treated with a beta-blocker. On examination he appeared well-developed, stuporous. Temp was 37°C; BP 141/88 mmHg; Pulse 88 beats/min; respiratory rate 34/min, weight 59 kg, height160 cm. The heart rate is regular, there are no extra sounds or murmurs. The chest is clear without rales or rhonchi. The abdomen is soft. No palpable masses. Bowel sounds are present. There is no edema. Several ecchymoses are apparent over his trunk and limbs. Laboratory data revealed serum sodium 140 mEq/L, chloride 108 mEq/L, potassium 3.8 mEq/L, HCO₃ 13 mEq/L, BUN 14 mg/dL, creatinine 1.2 mg/dL, glucose 96 mg/dL; blood ketones 2+; calcium 10.0 mg/dL, phosphate 3.5 mg/dL, magnesium 1.8 mg/dL and albumin 4.0 g/dL and plasma osmolality 284 mosmol/kg. Would you like a blood gas?
   A. Yes
   B. No

The correct answer is A. The blood gas determination is indicated in this case. The presence or absence of more than one disturbance cannot be determined in the absence of an adequate history. In addition, there is a discrepancy between the increase in the AG and the fall in the serum bicarbonate level.

10. A blood gas was obtained and revealed the following: pH 7.4, pCO₂ 20; HCO₃ 12; pO₂ 105

What is the acid-base diagnosis? (Select all that apply)
   A. Metabolic acidosis
   B. Metabolic alkalosis
   C. Respiratory acidosis
   D. Respiratory alkalosis

The correct answers are A and D. This is a mixed disturbance of metabolic acidosis and chronic respiratory alkalosis. There are no specific clues to an acid-base disturbance in the history. Headaches might suggest the use of salicylates or a CNS lesion both of which might be associated with respiratory alkalosis. The physical examination suggests that hyperventilation is present which could indicate severe acidosis or primary alkalosis. Ecchymoses are consistent with the use of salicylates. Laboratory studies reveal a low HCO₃ and an increased AG consistent with the presence of AG metabolic acidosis. However, the fall in the HCO₃ concentration (-11) is greater than the increase in the AG (+7)
suggesting the presence of another process which lowers the HCO₃⁻; either chronic respiratory alkalosis or hyperchloremic acidosis. Blood gas values reveal a low HCO₃, low pCO₂ and a normal pH indicative of a mixed disturbance with metabolic acidosis and primary respiratory alkalosis. The combination of AG metabolic acidosis and primary respiratory alkalosis occurs in several situations including sepsis, salicylate intoxication, and lactic acidosis in a patient with hepatic failure. Salicylate intoxication is the likely diagnosis in this patient in view of the history of severe headaches, stupor and the presence of ecchymoses.

11. A 16-year old boy with a recent history of tuberculosis, diagnosed 2 months ago, returns for follow-up examination. He has been complaining of shortness of breath on exertion as well as chronic cough with sputum production in the morning. He was placed on furosemide several weeks ago. On examination: Temp 37°C, BP 148/82 mmHg; Pulse 84 beat/min; respiratory rate 22/min; weight 63 kg; height 157 cm. The heart rate is regular, there are no extra sounds or murmurs. Respiratory excursions are shallow and there is occasional wheezing. The abdomen is soft without palpable masses. Bowel sounds are present. There is 2+ edema on lower extremities. Lab data revealed: Hemoglobin 12.0g/dL; WBC 6600 cells/μL; sodium 140 mEq/L; potassium 3.4 mEq/L; HCO₃⁻ 42 mEq/L; calcium 10.0 mg/dL; phosphate 3.5 mg/dL; magnesium 1.8 mg/dL; albumin 3.4 g/dL; blood ketones 0; glucose 94 mg/dL and plasma osmolality 284 mosmol/kg.

Would you like to order a blood gas?
A. Yes
B. No

The correct answer is A. The history suggests the possibility of more than one disturbance and thus the pH cannot be predicted with certainty.

12. An arterial blood gas was obtained and revealed the following: pH 7.45; pCO₂ 60; HCO₃⁻ 40, pO₂ 55.

What is the acid-base diagnosis? (Select all that apply)
A. Metabolic alkalosis
B. Respiratory acidosis
C. Respiratory alkalosis
D. Metabolic acidosis

The correct answers are A and B. This is a mixed disturbance with chronic respiratory acidosis and metabolic alkalosis. The history and physical exam indicate that the patient has chronic bronchitis and COPD which can produce chronic respiratory acidosis. He was placed on loop diuretics for apparent right sided congestive heart failure. These may produce metabolic alkalosis. Laboratory data show an elevated HCO₃⁻ and a normal AG which could represent either chronic respiratory acidosis or metabolic alkalosis or both. There is also mild hypokalemia consistent with the use of diuretics and metabolic alkalosis.

The pH is only minimally elevated above the normal range with a markedly elevated pCO₂ and HCO₃⁻. These data are not compatible with either metabolic alkalosis alone or chronic respiratory acidosis with normal compensation alone. Therefore both metabolic alkalosis and chronic respiratory acidosis are present.

13. A 16-year old female decided to attempt a starvation diet. To speed up initial weight loss, she also took furosemide. After one week she had lost 10 pounds but felt terrible and decided to see her physician. As she approached the office, she became acutely anxious and felt weak. Physical examination in the office revealed a BP of 90/60 mmHg and respiratory rate 20/min. There was a positive Trousseau’s sign. The remainder of the examination was normal. Laboratory data showed: Hemoglobin 13.5 g dL; BUN 40 mg/dL; creatinine 1.5 mg/dL; sodium 140 mEq/L; chloride 98 mEq/L; potassium 3.0 mEq/L; HCO₃⁻ 18 mEq/L; calcium 10.0 mg/dL; phosphate 3.5
mg/dL; magnesium 1.8 mg/dL; albumin 4.0 g/dL; blood ketones 0; glucose 99 mg/dL; and plasma osmolality 283 mosmol/kg.

**Would you like to obtain an arterial blood gas?**

A. Yes  
B. No

The correct answer is A. The history suggests the possibility of more than one disturbance and thus the pH cannot be predicted with certainty.

14. An arterial blood gas was obtained and revealed the following: pH 7.53; pCO₂ 20; HCO₃ 16; pO₂ 105.

What is the acid-base diagnosis? (Select all that apply)

A. Metabolic alkalosis  
B. Respiratory alkalosis  
C. Metabolic acidosis  
D. Respiratory acidosis

The correct answers are A, B, and C. This is a triple disturbance with metabolic alkalosis, metabolic acidosis, and acute respiratory alkalosis. The history and physical examination suggest that three acid-base processes may be present.

1. Starvation is associated with ketoacidosis  
2. Loop diuretics with metabolic alkalosis  
3. Acute anxiety with hyperventilation and acute respiratory alkalosis

Hypotension suggests volume contraction which can maintain the metabolic alkalosis. Trousseau’s sign is indicative of a reduction in ionized calcium concentration which can be caused by the sudden increase in pH due to respiratory alkalosis. The change in pH leads to increased binding of ionized calcium to protein, principally albumin. Laboratory studies reveal an increased BUN to creatinine ratio consistent with volume contraction. The HCO₃ is reduced and the AG is increased consistent with AG metabolic acidosis. However, the increase in the AG (+12) is significantly greater than the decrease in the HCO₃ (-7) suggesting that metabolic alkalosis is also present. Blood gas studies show a significantly increased pH with a low pCO₂ confirming the presence of respiratory alkalosis.

Thus, the data from the history and physical examination, laboratory studies, and blood gas studies together indicate the presence of a triple acid-base disturbance.

**References**