ACID-BASE BALANCE

Epidemiology/Pathophysiology

Understanding the physiology of acid-base homeostasis is important to the surgeon. The two acid-base buffer systems in the human body are the metabolic system (kidneys) and the respiratory system (lungs). The simultaneous equilibrium reactions that take place to maintain normal acid-base balance are:

$$H^{+}\ aq\ +\ HCO^{-}_{3}\ aq\ \leftrightarrow H_2CO_3\ aq\ \leftrightarrow H_2O\ l\ +\ CO_2\ (g)$$

To classify the type of disturbance, a blood gas (preferably arterial) and basic metabolic panel must be obtained. A basic understanding of normal acid-base buffer physiology is required to understand alterations in these labs. The normal pH of human blood is 7.40 (7.35-7.45). This number is tightly regulated by the two buffer systems mentioned above. The lungs contain carbonic anhydrase which is capable of converting carbonic acid to water and CO_2. The respiratory response results in an alteration to ventilation which allows acid to be retained or expelled as CO_2. Therefore, bradypnea will result in respiratory acidosis while tachypnea will result in respiratory alkalosis. The respiratory buffer system is fast acting, resulting in respiratory compensation within 30 minutes and taking approximately 12 to 24 hours to reach equilibrium. The renal metabolic response results in alterations in bicarbonate excretion. This system is more time consuming and can typically takes at least three to five days to reach equilibrium.

Five primary classifications of acid-base imbalance:

- Metabolic acidosis
- Metabolic alkalosis
- Respiratory acidosis
- Respiratory alkalosis
- Mixed acid-base disturbance

It is important to remember that more than one of the above processes can be present in a patient at any given time.

Primary Respiratory:

A primary respiratory acid-base imbalance is a result of over- or under-ventilation. As described above, if minute-ventilation is decreased there will be an increase in CO_2 and acid in the blood, resulting in respiratory acidosis. This is a common problem in the surgical patient as a result of opioids, anxiolytics and anesthetics which cause sedation and decrease the respiratory drive. Conversely, post- operative pain or anxiety can result in increased minute-ventilation, resulting in decreased CO_2, and subsequent respiratory alkalosis. Patients who are mechanically ventilated can develop disturbances from inappropriate sedation or ventilator settings. The ventilator can be used to compensate for metabolic disturbances (*see Management*).

Epidemiology/Pathophysiology (continued)

Primary Metabolic:

A primary metabolic acid-base imbalance is a result of changes in bicarbonate excretion by the kidneys. CO_2 levels will be normal or compensatory. In the surgical patient, the most common is metabolic acidosis which has a broad differential diagnosis and is further sub-divided by the presence or absence of an abnormal anion gap. Anion gap is the difference between major plasma cations and anions and is calculated by the following formula:

$$\mathrm{AG} = \mathrm{Na}^{"} - (\mathrm{HCO}^{*} + \mathrm{Cl}^{*})$$

Normal values are 8-12 mmol/l. The differential diagnosis for anion-gap metabolic acidosis (AGMA) is easily remembered with the mnemonic “MUDPILES”:

- **M** Methanol toxicity
- **U** Uremia
- **D** Diabetic ketoacidosis
- **P** Paraldehyde
- **I** Isoniazid or Iron toxicity
- **L** Lactic acidosis
- **E** Ethylene glycol toxicity
- **S** Salicylate toxicity

Lactic acidosis is the most common cause of AGMA in the surgical patient and is a result of inadequate end-organ perfusion and resultant anaerobic metabolism producing lactate and other anions as byproducts.

The causes of non-anion gap metabolic acidosis (NAGMA) can be remembered with the mnemonic “HARDUP”:

- **H** Hyperalimentation
- **A** Acetazolamide
- **R** Renal tubular acidosis
- **D** Diarrhea
- **U** Ureterosigmoid fistula
- **P** Pancreatic fistula

Metabolic alkalosis is another disturbance commonly seen by the surgeon. While the differential diagnosis is broad, the most common causes in the surgical patient are gastrointestinal loss of hydrochloric acid (vomiting) and volume contraction (dehydration, excessive diuretic use).

Epidemiology/Pathophysiology (continued)

Mixed acid-base disturbance

As stated, multiple disturbances can occur simultaneously. This increases the diagnostic challenge of determining the primary disturbance. Further, the body will attempt to compensate with either the metabolic or respiratory system if the other is out of balance.

Suggested method to approach diagnosis of acid-base pathology:

1. "emia" - Check the arterial pH
   a. pH < 7.4 = Acidemia
   b. pH > 7.4 = Alkalemia

2. "osis" - Look at the pattern in PCO_2 & $[HCO_3]$
   a. If both PCO_2 & $[HCO_3]$ are low
      i. Suggests presence of metabolic acidosis or respiratory alkalosis
   b. If both PCO_2 & $[HCO_3]$ are high
      i. Suggests presents of metabolic alkalosis or respiratory acidosis
   c. If PCO_2 & $[HCO_3]$ move in opposite directions
      i. Mixed disturbance
   d. If high anion gap is present
      i. Strongly suggests metabolic acidosis
   e. If there is a base deficit**
      i. Metabolic acidosis
      ii. Can also be a result of compensation
   f. If there is a base excess **See Relevant Diagnostic Studies for more information.*
      i. Metabolic alkalosis

3. Is there compensation? Is it appropriate?
   a. Both the bicarbonate level or PCO2 can be abnormal as a result of the primary process or as a compensatory response. There are a few rules for compensation that are important to remember.
      i. Compensation will rarely restore pH to normal
      ii. This table shows appropriate compensation based on the primary disorder:

Primary Disorder	Primary Abnormality	Compensatory Change	
Metabolic acidosis	$\downarrow HCO_3^*$		$\downarrow pCO_2 = 1.2\times\Delta HCO_3^*$
Metabolic alkalosis	$\uparrow HCO_3^*$		$\uparrow pCO_2 = 0.7\times\Delta HCO_3^*$
Respiratory acidosis	$\uparrow pCO_2$	Acute	$\uparrow HCO_3^* = 0.1\times\Delta pCO_2$
			$\uparrow [H^+] = 0.8\times\Delta pCO_2$

Epidemiology/Pathophysiology (continued)

Primary Disorder	Primary Abnormality	Compensatory Change	
Respiratory acidosis		Chronic	$\uparrow HCO^* = 0.35 \times \Delta pCO$
			$\uparrow [H^+] = 0.3 \times \Delta pCO$
Respiratory alkalosis	$\downarrow pCO$	Acute	$\downarrow HCO^* = 0.2 \times \Delta pCO$
			$\downarrow [H^+] = 0.8 \times \Delta pCO$
		Chronic	$\downarrow HCO^* = 0.6 \times \Delta pCO$
			$\downarrow [H^+] = 0.2 \times \Delta pCO$

4. Is there a GAP?
   a. If so, primary disorder is very likely to be metabolic acidosis

5. Delta/Delta (ΔΔ) *(applicable if abnormal anion GAP present)*
   a. Calculating a ΔΔ is a method of asking the following question: *What would the bicarbonate be, if the anion gap acidosis wasn't there?*
   b. To calculate ΔΔ
      i. First calculate the ΔGAP: $GAP_{patient} - GAP_{normal}$
      ii. Next calculate the ΔΔ: Add ΔGAP + HCO_3
   c. Compare ΔΔ to normal bicarbonate level
      i. ΔΔ ≈ Normal HCO_3 = No further disturbance present
      ii. ΔΔ > Normal HCO_3 = Additional presence of a metabolic alkalosis
      iii. ΔΔ < Normal HCO_3 = Presence of a non-gap metabolic acidosis

Signs and Symptoms

Signs and symptoms of a major acid-base disturbance can range from asymptomatic to total cardiovascular collapse. There are no specific symptoms to help differentiate between the types of disturbance but there are a few clinical scenarios that may suggest the presence of a specific disorder. The first is in the critically ill surgical patient who has an increasing vasopressor requirement with progressively worsening hemodynamic collapse. The relative binding affinities of inotropes and vasopressors to adrenergic receptors can be altered by acidosis which decreases their effect. Therefore, this clinical scenario is highly suggestive of primary metabolic acidemia. Another common surgical scenario is the post-operative patient who is somnolent and/or bradypneic. As mentioned previously, the medications used for pain, anxiety and anesthesia cause powerful sedation which can decrease the respiratory drive. This can result in hypercarbia which can cause encephalopathy and somnolence. This scenario is highly suggestive of primary respiratory acidosis.

Relevant Diagnostic Studies

1. Arterial blood gas (ABG)
   a. pH, pCO_2, pO_2
   b. Base deficit/excess
      i. A *calculated value* using the pH and the [HCO_3^-]
      ii. Some labs report this value, some do not.
      iii. Basically tells you if there is an excess or deficit in the amount of HCO_3^- (base) in circulation.
         a) BD < -2 = metabolic acidosis
         b) BE > 2 = metabolic alkalosis
2. Chemistry panel
   a. HCO_3^- (some labs report this value with the ABG)
   b. Values necessary to calculate AG
      i. $Na^{"}$
      ii. $HCO_{)}^{*}$
      iii. Cl^{*}

Management

The most important principle in the management of a major acid-base disturbance is to treat the cause (not the effect). Sometimes it is difficult to determine the cause, and once the source has been identified, it can be difficult to treat. There are supportive measures that can employed to temporarily treat acid-base disturbances which are effectively used to “buy time”. If a patient has severe acidemia and is hemodynamically unstable, alkaline medications such as sodium bicarbonate may be infused intravenously to temporarily increase the pH in order to improve the function of the vasoactive medications and improve hemodynamics. This practice is controversial and should be implemented cautiously in very specific clinical scenarios as it has been shown to increase mortality. In the mechanically ventilated patient, settings on the ventilator can be altered to provide additional compensation to assist in the correction of a disturbance. In the rare scenario where a patient has severe metabolic alkalosis, intravenous hydrochloric acid can be given to decrease the pH.

Questions

1. A 47-year-old female is 4 hours status-post cholecystectomy. You are performing a postoperative check and find her to be unresponsive. Her respiratory rate is 5 breaths/min. All other vital signs are normal. Her pupils are miotic. What is the expected acid-base disturbance in this patient?

   a. Metabolic acidosis
   b. Respiratory acidosis
   c. Metabolic alkalosis
   d. Respiratory alkalosis
   e. Mixed acid-base disturbance

2. A 39-year-old male presents to the ED with multiple orthopedic injures after a fall from an intentional ingestion of an unknown substances. The patient is agitated and confused. An ABG is obtained which demonstrates pH 7.11, pCO2 30, pO2 94. The results of his basic metabolic panel are: Na 141, K 4.4, Cl 95, HCO3- 17, BUN 10, Cr 0.86. Which acid-base disturbance is present in this patient? (You may choose more than one answer if appropriate.)

   a. Anion gap metabolic acidosis
   b. Non anion gap metabolic acidosis
   c. Respiratory acidosis
   d. Respiratory alkalosis
   e. Mixed acid-base disturbance

3. A 74-year-old male is intubated and in the SICU with intra-abdominal sepsis due to perforated diverticulitis. His ABG is: pH 7.21, pCO2 35, pO2 150mmHg, HCO3 18. Anion gap is 18. Which acid-base disorder is present?

   a. Metabolic acidosis with respiratory acidosis
   b. Respiratory acidosis with compensatory metabolic compensation
   c. Metabolic acidosis with compensatory respiratory compensation
   d. Metabolic alkalosis with respiratory acidosis
   e. Respiratory alkalosis with compensatory metabolic compensation

4. You are called to the bedside of a patient in the surgical ICU who is being prepared for extubation. The patient is somnolent but following commands. On pressure-support ventilation, the most recent ABG is: pH 7.22, pCO2 58, pO2 127. The nurse is concerned about the pH and asks you to order 2 ampules of sodium bicarbonate for the patient. You write an order for 2 amps of sodium bicarbonate and extubate the patient. Thirty minutes later, you are called back to the bedside because the patient is non-responsive. You draw another ABG (pH 7.06, pCO2 106, pO2 90). The patient requires emergent re-intubation. How could this complication have been avoided?

   a. Giving the patient 4 amps of sodium bicarbonate
   b. Not giving the patient sodium bicarbonate
   c. Increasing the FIO2 after extubation
   d. Using inhaled bronchodilators
   e. Extubating from lower PEEP

Questions (continued)

5. You are taking care of a patient who is status-post thoracotomy and pulmonary resection. The patient had severe pulmonary edema post-operatively and has been on aggressive diuretic therapy for the past four days. He seems to be less dyspneic but you want to be sure he is oxygenating well, so you perform an ABG which reveals a pH of 7.54, pCO2 of 49, pO2 106, HCO3 36. Which acid-base disorder is present?

   a. Combined respiratory alkalosis and metabolic alkalosis
   b. Respiratory alkalosis with compensatory respiratory acidosis
   c. Metabolic alkalosis with compensatory metabolic alkalosis
   d. Metabolic alkalosis with compensatory respiratory acidosis

Answers

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