Acid/Base Balances for Medicine B

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Intro

Acid-base balance is a critical homeostatic process in the human body; our proteins can function only within a narrow pH range. Many disease processes contribute to acidifying and alkalizing blood.

Given a venous blood gas (VBG) lab, it is straightforward to determine acidosis or alkalosis. However, how often do we have blood gas available for consultation? This guide draws connections between common disease processes seen in Med. B and the risk for acidosis/alkalosis. By refamiliarizing ourselves with the signs of acid/base imbalance, we can better care for our clients by recognizing the early signs of decompensation and having the knowledge, skill, and judgement to request an order for blood gasses from the provider.

Acidity is the blood's concentration of protons (H^+ ions). The primary sources of H^+ in the body are dissolved CO_2 in the blood (as H_2CO_3, carbonic acid) and exogenous acidic medications and foods. The amount of acid is measured on a logarithmic scale called pH (H is for H^+). acidosis is a process that increases the blood's acidity. Acidemia is a pH below 7.35. Conversely, alkalosis is a process that decreases the acidity of the blood and alkalemia is a pH above 7.45 (Khin, 2020).

Acidemia (Khin, 2020)

Respiratory acidosis is caused by hypoventilation or poor gas exchange in the lungs, causing CO_2 to be retained in the blood as carbonic acid (H_2CO_3). CO_2in the blood acts as a potent vasodilator, especially in the brain.

Metabolic acidosis is caused by ingesting excess acid (or acidic medications) or from a disease process where insufficient bases (H^+ accepting molecules, usually bicarbonate HCO_3^-) are available in the body to buffer the acid. Specific causes include:

- Kidney damage (causing inability to excrete excess H^+)
- Excessive HCO_3^- losses due to diarrhea
- Ketoacidosis (diabetic, alcoholic, or starvation), where the body is producing acidic molecules called ketone bodies.
- Accumulation of lactic acid from excess exercise, anaerobic metabolism (hypoxia), or liver damage.
- Excess ingestion of acidic foods, salicylates, etc.

Signs and Symptoms (p. 329)

- Decreased LoC (confusion, drowsiness)
- Increased respiratory rate and depth
- Headache or feeling of fullness in the head.
- Nausea and Vomiting
- Cold or Clammy Skin

Risks (p. 330)

- Hyperkalemia, dysrhythmias
- Shock: due to vasodilation, reduced preload and cardiac output

Alkalemia (Khin, 2020)

Respiratory alkalosis is exclusively caused by hyperventilation. The increased respiration rate and depth allow more CO_2 to leave the blood, reducing the acidity. This is rarely seen on its own (see page 8 for an example). Still, hyperventilation in the form of rapid, deep breathing called Kussmaul breathing is a sign of a severe acidotic process for which the body is trying to compensate.

Metabolic alkalosis (p. 330):

- loss of acid via vomiting or gastric suction
- excess ingestion of antacids and/or milk (milk-alkali syndrome)
- hypokalemia (causes kidneys to retain K^+ and excrete H^+ and H^+ drawn into cells in exchange for K^+).
- cystic fibrosis (see page 8)

Signs and Symptoms (p. 332)

- Lightheadedness (rt to reduced cerebral blood flow).
- Difficulty concentrating
- Numbness and tingling in fingers and toes (rt hypocalcemia)
- Tinnitus
- Tachycardia
- Decreased respiratory rate and depth (if cause is metabolic)

Risks (p. 330)

- Hypokalemia, dysrhythmias
- Hypocalcemia as more serum Ca^{2+} binds with proteins in alkalotic conditions

Potassium Connection

Acid-base disturbances acutely affect serum potassium levels and vice versa. Potassium (K^+) is an electrically positive ion (cation) like acid (H^+). The body moves one or the other into and out of the cells to maintain homeostasis.

Acidemia $\rightleftharpoons$ Hyperkalemia

As H^+ increases (acidosis), the body's cells absorb the excess acid to maintain homeostasis. With the acid comes excess positive electrical charge. The cells expel K^+ into the bloodstream to maintain electrical neutrality. This release of K^+ can increase serum levels above 5 mmol/L, leading to hyperkalemia. Similarly, in cases of hyperkalemia, K^+ in the blood will be drawn into cells, expelling H^+ and increasing acidosis.

Signs and Symptoms of hyperkalemia (Khin, 2020, p.316):

- Muscular weakness escalating to quadriplegia
- ECG changes: T waves narrow and peaked, ST depression, Shortened QT interval

Alkalemia $\rightleftharpoons$ Hypokalemia

As H^+ decreases (alkalosis), the body's cells release the acid (H^+) to maintain homeostasis. To maintain electrical neutrality in the cells, the cells must absorb K^+ from the bloodstream. This can decrease serum K^+ below 3.5 mmol/L, leading to hypokalemia. Similarly, hypokalemia will cause cells to pull in H^+ (acid) and expel K^+, resulting in alkalosis.

Signs and Symptoms of hypokalemia (Khin, 2020, p. 314):

- nausea and vomiting
- fatigue, muscle weakness and cramps
- glucose intolerance
- dysrhythmias

Nephrology Considerations

Chronic Kidney Disease (CKD) — Acidotic

A crucial acid-base homeostasis method is excreting excess H^+ in urine. As the glomerular filtration rate (GFR) decreases, the kidneys produce less urine and H^+ is retained in the blood. Since the change can be gradual, the body compensates. However, the overall acid load increases, heightening the risk of acidosis from other sources. In the most severe case, end-stage renal disease (ESRD), the clients produce little to no urine (anuria), leading to chronic acidemia (Power-Kean, 2020, p. 1284).

Secondarily, the kidneys are less able to excrete K^+ in urine as GFR declines, potentially leading to hyperkalemia, which increases acidosis (see page 5).

Call the provider for a VBG when the client has:

- Decreased LoC, drowsiness
- Increased respiration rate

Acute Kidney Injury (AKI) — Acidotic

In the oliguric phase of AKI, the client is at risk for acidemia from the same causes as in CKD, retention of H^+ and K^+ due to reduced kidney function. In the diuresis phase of AKI, urine output may increase above baseline. This can lead to electrolyte imbalances, like hypokalemia and alkalosis, but this is less common.

Respirology Considerations

Chronic Obstructive Pulmonary Disease (COPD) — Acidotic

COPD can result in progressively impaired gas exchange. In many cases, the increase in dead space (lung surface area not involved in respiration) and reduced elasticity result in retention of CO_2 (hypercapnia) and acidosis (Power-Kean, 2020). This process is slow, and clients can compensate. However, the high baseline acid load can make further compensation for other acidotic disease processes difficult.

Watch for:

- Signs of respiratory acidosis, cerebral vasodilation (headache)
- Decreased LoC, drowsiness

Asthma (Power-Kean, 2020) — Acidotic or Alkalotic

In the early stages of an asthma exacerbation, inflammation promotes hyperventilation which can cause respiratory alkalosis. However, if the ventilation is severely compromised, hypercapnia from respiratory collapse causes acidosis. Asthma can exacerbate alkalotic and acidotic processes even when the exacerbation itself is sub-acute.

Respirology Considerations (cont.)

Pulmonary Embolism (PE) — Alkalotic

During PE, inflammatory cytokines build up, stimulating respiratory drive and hyperventilation. This leads to hypocapnia and respiratory alkalosis (Vyas et al., 2025).

Watch for:

- Signs of alkalemia (page 4)
- Numbness and tingling in fingers and toes

Cystic Fibrosis (CF) — Alkalotic

In patients with CF, the kidney has impaired HCO_3^- excretion capability, leading to metabolic alkalosis (Berg et al., 2022). With the kidneys unable to correct the alkalosis, hypoventilation is triggered to create compensating respiratory acidosis. Hypoventilation can interact negatively with their compromised respiratory function. Clients with CF are expected to have baseline alkalosis, which impairs adjustment to other alkalotic insults such as vomiting, dehydration, and hypokalemia (see page 5).

Watch for:

- Signs of alkalemia (page 4, hypocalcemia
- Diarrhea or dehydration (will exacerbate alkalosis)

ETOH / Opioid / Benzos / Barbituates — Acidotic

CNS depressants cause respiratory depression. Respiratory depression leads to CO_2 retention and acidosis. This can increase acidosis by adding a respiratory acidosis component to a patient with an existing respiratory or metabolic acidotic process.

Watch for:

- Decreased respiration rate (<12/min)
- Decreased LoC, drowsiness

Diarrhea — Acidotic

When stomach contents enter the small intestine, the acidic contents are neutralized to a basic pH with HCO_3^- from pancreatic juices. The intestinal contents remain basic until HCO_3^- is reabsorbed in the colon. In diarrhea, the contents are expelled before the colon reabsorbs HCO_3^- and water. This HCO_3^- is replenished by removing it from the blood, which becomes more acidic in its absence.

Dehydration is often the primary concern, but watch for:

- Increased respiration rate
- Decreased LoC, drowsiness

Vomitting/Gastric Suction — Alkalotic

The stomach is full of strong acid. When this acid is lost due to vomiting, the body must remove and concentrate H^+ from the blood to replenish the lost supply. Because the acid is so strong, large amounts of H^+ are consumed, leading to alkalosis.

General Medicine (cont.)

Shock / Heart Failure — Acidotic

Shock and heart failure can result in hypoperfusion of the kidneys, leading to acidosis via AKI (see page 6). When body tissues have insufficient oxygen (hypoxia), they generate energy without oxygen (anaerobic). Lactic acid is a by-product of anaerobic metabolism, which can cause rapid acidosis.

Shock is a medical emergency. In less acute cases (CHF), watch for:

- Signs of acidemia (page 3)
- Cool, clammy skin
- Tachypnea, reduced blood pressure.
- Oliguria (indicating AKI)

Liver disease (Scheiner et al., 2017) — Acidotic or Alkalotic

The body is constantly producing lactate / lactic acid as part of energy metabolism. The liver is the main consumer of lactate, using between 30-70%. In disease processes resulting in poor liver function, lactic acid can build up in the blood (lactic acidosis).

However, liver disease can also produce alkalosis. Albumin is synthesized exclusively by the liver, is the most abundant protein in the blood, and a weak acid (McAuliffe et al., 1986). Reduced albumin levels can increase the pH of the blood (metabolic alkalosis).

Watch for signs of acidemia (page 3) and other lactic acid-producing processes:

- Signs of infection/sepsis
- Medications (metformin overdose, propofol, NSAIDs, drugs which reduce cardiac output)
- Signs of shock

General Medicine (cont.)

Diabetes Mellitus — Acidotic

In a hyperglycemic state, the client will become thirsty (polydipsia) and urinate in larger volumes (polyuria), which can deplete potassium and lead to acidosis (see page 5). The breakdown of fat for energy due to insufficient insulin results in the production of acidic ketone bodies. Extreme states such as hyperosmolar hyperglycemic syndrome and diabetic ketoacidosis are medical emergencies. However, less severe hyperglycemic states may contribute to acidemia in the presence of other disease processes.

Watch for:

- Signs of hyperglycemia (polydipsia, polyuria, blurred vision)
- Signs of acidosis (page 3), e.g. decreased LoC, drowsiness

Obesity (Hodgson et al., 2015) — Acidotic

Excess mass can compress the rib cage and abdomen when a client is supine, limiting diaphragm movement and fatiguing respiratory muscles. This can lead to a shallow breathing pattern, reduced lung volume and compliance. Bariatric clients can be retainers of /cotowo like clients with COPD, for similar reasons. Semi-Fowler's position (called beach chair positioning in surgical literature) effectively increases lung expansion.

Watch for:

- Signs of acidosis (page 3), e.g. decreased LoC, drowsiness
- Moderate to severe use of accessory muscles for respiration.
- Shallow respirations, hypoventilation

Blood Gas Review

After you ask for a blood gas, what are we looking for? Values on a blood gas:

Value	*Normal Range*		Units
	ABG	VBG	
pH	7.35–7.45	7.35–7.45	
$paCO_2/pCO_2$	35–45	42–50	mmHg
HCO_3^-	22–26	22–26	mmol/L
paO_2/pO_2	95–100	$\approx$ 40 mmHg	mmHg

pH: $<$7.35 is acidemia, $>$7.45 is alkalemia. If pH is within the normal range, but pCO_2 and/or HCO_3^- is out of range (see page 13).

pCO_2: *Respiratory* component of VBG. Increases in pCO_2 drive pH in the *opposite* direction (downward).

HCO_3^-: *Metabolic* component of VBG. Increases in HCO_3^- drive pH in the same (*equal*) direction.

ROME - Respiratory Opposite, Metabolic Equal

ROME is an algorithm for reading ABG/VBG. First, look at pH value, noting acidosis if the pH is $<$7.35 or alkalosis if $>$7.45. Next, consider the respiratory component, pCO_2: if it is out of range in the *opposite* direction (elevated when pH is low or depressed when pH is high), then the cause is respiratory. Next, if HCO_3^- is out of range in the same (*equal*) direction (high when pH is high, low when pH is low), then the cause is *metabolic*.

If all values are normal, the client has neither acidosis nor alkalosis.

Blood Gas Review (cont.)

Uncompensated acidosis/alkalosis

Example:

pH	7.33
pCO_2	55 mmHg
HCO_3^-	24 mmol/L

The body is not trying to increase HCO_3^- to increase pH, as evidenced by HCO_3^- within the normal range. We call this uncompensated respiratory acidosis.

Partially compensated acidosis/alkalosis

Example:

pH	7.33
pCO_2	55 mmHg
HCO_3^-	30 mmol/L

In this case, the increased HCO_3^-increases pH against the work of pCO_2lowering the overall pH). The HCO_3^-pushback cannot bring the pH into the normal range, so we call this partially compensated respiratory acidosis.

Compensated acidosis/alkalosis

Example:

pH	7.35
pCO_2	55 mmHg
HCO_3^-	30 mmol/L

In this case, the pH is closer to acidic (low). pCO_2 (*respiratory* component) is deviated in the *opposite* direction (high). However, increased HCO_3^- is keeping pH within normal range. In cases like this, we say the acidosis is fully compensated.

Putting it all Together

Given the list of conditions and associated acid/base imbalances. How can we use this to improve our practice?

Adding up the Risks

We cannot see the future, but we can predict likely complications based on patient history and current disease processes.

For example, with a patient with COPD (+1 acidosis risk), BMI >30 (+1 acidosis), moderate CKD (+1 acidosis), admitted for UTI (acid/base risk unknown). We can expect that the client's baseline acid load is high. If we see a VBG, we'll know that a "critical low" pH maybe normal for them. We can also be on the lookout for symptomatic acidosis and notify the team

Cole's Notes

Acidemia looks like decreased LoC, drowsiness, confusion, and headache.

Alkalemia looks like decreased respiratory rate, lightheadedness, difficulty concentrating, numbness in fingers/toes, and tachycardia.

When you see these symptoms, think of the patient-specific risk factors. If the client has 1-2 disease processes pushing in the same direction, call the provider for an order for blood gases, using the patient's history and new symptoms as rationale.

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About the Author

Shawn Nock is a Fanshawe College RPN student completing consolidation in Medicine B. He can be a little "try-hard", but he hopes it's somewhat endearing, rather than cloying. You can see more of his work at his nursing portfolio website: https://nurse.win/.