

Acute Metabolic Acidosis

Blood pH is too low—calculate the anion gap to find the source of acid, then fix the upstream cause making or trapping that acid.

THE ONE-LINE TRUTH

Blood pH drops below 7.35 because the body either makes too much acid or loses too much bicarbonate—the main buffer that neutralizes acid.

The chain — how it unfolds

1. Deep, fast breathing called Kussmaul respirations appears — why: The brainstem chemoreceptors—special sensors that detect pH and CO₂—sense the low pH and command the respiratory muscles to hyperventilate. This blows off CO₂, which exists in equilibrium with carbonic acid in blood ($\text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{H}^+ + \text{HCO}_3^-$). Removing CO₂ shifts the equation left and raises pH back toward normal. You hear deep sighing breaths, and the respiratory rate climbs to 25, 30, or even 40 per minute.
2. Nausea, vomiting, and abdominal pain develop — why: Acidemia—low blood pH—directly irritates the lining of the stomach and intestines and stimulates the vomiting center in the brainstem. Many causes of high anion gap too much acid in the blood (acidosis) also damage the gut: DKA causes gastric stasis (stomach stops moving), lactic acidosis (acid from oxygen-starved tissues) from bowel starved blood flow (ischemia) (intestine not getting enough blood) causes severe belly pain, and toxic ingestions directly poison the GI tract. Vomiting worsens volume depletion and can add a metabolic alkalosis (the blood turning too alkaline) on top of the too much acid in the blood.
3. Potassium shifts out of cells and serum potassium rises (hyperkalemia) — why: When blood pH drops, hydrogen ions move into cells to be buffered. To keep the cell electrically neutral, potassium ions move out into the blood in exchange. For every 0.1 unit drop in pH, serum potassium rises by roughly 0.6 mEq/L. This is called transcellular shift. Kidney failure (uremic too much acid in the blood (acidosis)) and insulin deficiency (DKA) also block the kidney's ability to dump potassium into urine, so potassium climbs even higher. Serum potassium may look dangerously high—6.5, 7.0, even 8.0 mEq/L—even though total body potassium is actually low because it has been lost in urine or diarrhea.

4. Blood pressure drops and the patient goes into shock — why: Severe acidemia—pH below 7.1 or 7.2—poisons the heart muscle so it squeezes weakly (suppresses myocardial contractility). It also blocks the receptors on blood vessels that normally respond to epinephrine and norepinephrine, the body's natural adrenaline hormones, so vasopressors stop working. Finally, acidemia dilates peripheral arteries, widening blood vessels and dropping blood pressure further. The heart rate may stay high trying to compensate, but eventually cardiac output falls and organs stop getting enough blood.
5. Confusion, lethargy, or coma appears — why: Low pH crosses the blood-brain barrier and disrupts how neurons transmit electrical signals. Brain cells swell, especially in severe cases. In diabetic ketoacidosis (the acid crisis of missing insulin), if you drop the blood glucose and serum osmolality too fast—by giving too much insulin or too much IV fluid too quickly—water rushes into brain cells and causes cerebral swelling (edema) (brain swelling). This is most dangerous in children. Early signs are headache, vomiting, and confusion; late signs are seizures, unresponsive pupils, posturing, and herniation.
6. Dangerous heart rhythms develop—peaked T waves, wide QRS, sine wave, or cardiac arrest — why: High potassium (Hyperkalemia) from too much acid in the blood (acidosis) makes the electrical charge across the heart muscle cell membrane unstable (depolarizes the membrane). Peaked T waves appear first on the EKG when potassium hits 6.5–7.0 mEq/L. As potassium climbs higher, the P wave flattens, the PR interval lengthens, and the QRS widens into a sine wave pattern. Eventually the heart stops conducting signals—you see a slow heart rate (bradycardia), heart block, ventricular a fast heart rate (tachycardia), ventricular fibrillation, or asystole (flatline). Calcium gluconate stabilizes the heart membrane immediately but does not lower potassium; insulin plus glucose, albuterol, and dialysis actually move potassium out of the blood.
7. Kidney function worsens and urine output drops — why: Low blood pressure from too much acid in the blood (acidosis) means the kidneys do not get enough blood flow to make urine. Severe acidemia also damages the kidney tubules directly. In some cases the too much acid in the blood is caused by kidney failure, so the kidneys cannot recover until you correct the too much acid in the blood and support blood pressure with IV fluids and vasopressors. Watch urine output and creatinine trend closely.
8. None of these findings is random—every symptom is the body trying to survive — why: Hyperventilation corrects pH by blowing off acid as CO₂. Vomiting tries to rid the gut of toxins or relieve gastric distension. The heart races to maintain blood flow to organs despite weak contractions. Potassium shifts out of cells to buffer hydrogen ions moving in. Every lab abnormality and every vital sign change is the body buying time while you find and treat the root cause—lactic acidosis (acid from oxygen-starved tissues) from septic shock needs antibiotics and fluid resuscitation, DKA needs insulin and IV fluids, methanol poisoning needs fomepizole and dialysis, and diarrhea-induced too much acid in the blood (acidosis) needs IV bicarbonate or oral rehydration. Treat the cause, not just the number.

Spot it — what you see, and why

- Kussmaul respirations (deep, rapid breathing): Your brain has tiny sensors that watch your blood's acid level. When they see too much acid, they tell your lungs to work harder—blow out more CO₂ gas—because getting rid of CO₂ makes blood less acidic, like opening a window to let out smoke.
- Nausea and vomiting: Acid irritates the stomach lining and also tickles a special alarm center in your brainstem, making you feel sick and throw up. This is especially common when acid builds up from diabetes complications, not enough blood reaching your gut, or poisons.
- Belly pain: Acid hurts the lining of your intestines. Many things that cause acid buildup—like uncontrolled diabetes, not enough blood to the gut, or antifreeze poisoning—also damage organs inside your belly, so pain is a common warning.
- Confusion, sleepiness, or trouble thinking: Acid particles (hydrogen ions) slip into brain tissue and mess up the tiny electrical signals that brain cells use to talk. If acid gets very high or you fix it too fast, the brain can swell with water, which is dangerous.
- Low blood pressure or shock: When blood becomes too acidic, your heart muscle squeezes less hard, and your blood vessels stop responding well to your body's own adrenaline hormones (epinephrine and norepinephrine). Blood vessels also widen, so pressure drops and blood can't get to organs.
- Weakness and feeling exhausted: Acid stops muscle cells from making energy properly—like a battery running low. Acid also pushes potassium out of cells, and that change makes muscles weaker and harder to use.

Red flags — act now

- Blood pH below 7.1 or bicarbonate below 10 mEq/L (heart can stop or shock may not respond to treatment)
- Potassium above 6.5 mEq/L or EKG showing peaked T waves, wide QRS, or sine wave (heart rhythm about to become lethal)
- Confusion, unresponsiveness, or coma (brain swelling risk in diabetic ketoacidosis (the acid crisis of missing insulin) or severe acid affecting brain function)
- Blood pressure staying low despite IV fluids or strong medicines (acidic blood blocks adrenaline from working on heart and vessels)
- Lactic acid above 4 mmol/L (signals shock, sepsis, or organs not getting oxygen—find and fix the cause now)

Treat it — in this order

1. Identify and treat the root cause (shock causing lactic acidosis (acid from oxygen-starved tissues), insulin deficiency causing ketoacidosis, kidney failure causing uremic too much acid in the blood (acidosis), diarrhea causing bicarbonate loss, or toxin ingestion such as methanol, ethylene glycol, or aspirin) → Stops the body from making more acid or losing more bicarbonate. Once the source is controlled, the lungs can slow down their emergency breathing and the heart can pump more effectively. The kidneys can begin clearing the acid that has built up.
2. Give intravenous fluids (0.9% normal saline, typically 10–20 mL/kg bolus over 1 hour, then ongoing replacement at 250–500 mL/hour, adjusted to patient size and response) → Increases blood volume and blood pressure. Restores blood flow to the kidneys so they can filter and excrete acid and waste. Improves blood flow to tissues so cells switch back from making lactic acid (anaerobic metabolism, energy without oxygen) to normal energy production. Reduces strain on the heart.
3. Give intravenous regular insulin (0.1 units/kg/hour continuous infusion; some protocols use an initial 0.1 units/kg bolus) with dextrose-containing fluids (add 5% dextrose to IV fluids) once glucose drops below 200–250 mg/dL (for diabetic ketoacidosis (the acid crisis of missing insulin) only) → Insulin stops the body from breaking down fat and making ketones. It allows glucose to enter cells so they can make energy normally instead of burning fat. Dextrose prevents blood sugar from dropping too low (hypoglycemia, below 70 mg/dL) once insulin starts working.
4. Treat dangerous high potassium (above 6.5 mEq/L or with ECG changes: tall peaked T waves, wide QRS, or sine wave) with IV calcium gluconate (1 g over 2–3 minutes) or calcium chloride to protect the heart; insulin (10 units IV) with dextrose (25 g, or 50 mL of 50% dextrose) and/or inhaled albuterol (10–20 mg nebulized) to move potassium into cells; and dialysis if kidneys are not working → Calcium stabilizes heart muscle membranes so the heart does not develop a fatal arrhythmia (abnormal rhythm). Insulin (with dextrose to prevent low blood sugar) and albuterol push potassium from the blood back into cells, temporarily lowering blood potassium. Dialysis physically removes potassium from the body when kidneys cannot.
5. Give intravenous sodium bicarbonate (50–100 mEq mixed in 0.45% saline or dextrose solution, infused over 30–60 minutes) ONLY if pH is below 7.0–7.1 AND the patient has severe shock, life-threatening high potassium not responding to other treatments, or specific toxin ingestion (aspirin, tricyclic antidepressant, or toxic alcohol like methanol or ethylene glycol) → Bicarbonate directly buffers (neutralizes) hydrogen ions, temporarily raising blood pH. It also helps move potassium back into cells.

Your top priorities at the bedside

1. Obtain venous or arterial blood gas, basic metabolic panel with sodium, potassium, chloride, bicarbonate, glucose, and creatinine within 15 minutes of patient arrival. — why: The pH confirms too much acid in the blood (acidosis) severity, bicarbonate level guides replacement needs, the anion gap (a calculated number showing unmeasured acids) identifies the too much acid in the blood type, potassium reveals life-threatening high potassium (hyperkalemia) risk, and glucose plus creatinine point to DKA or kidney failure as the cause.
2. Place the patient on continuous cardiac monitoring and obtain a 12-lead EKG immediately, watching for peaked T waves, widened QRS complexes, or sine-wave pattern. — why: Too much acid in the blood (Acidosis) causes potassium to leave cells and enter the bloodstream; high potassium (hyperkalemia) changes how the heart's electrical system works, and these EKG changes appear right before lethal arrhythmias like ventricular fibrillation (chaotic quivering that stops effective pumping) or asystole (flatline).
3. Start a large-bore IV (18-gauge or larger) and infuse 0.9% normal saline at 10–20 mL per kg over the first hour, then continue at 250–500 mL per hour based on blood pressure and urine output. — why: Volume expansion improves kidney blood flow so kidneys can start removing acid and regenerating bicarbonate; in lactic acidosis (acid from oxygen-starved tissues) and DKA, fluids alone often reverse too much acid in the blood (acidosis) by restoring tissue oxygen delivery and shutting off anaerobic acid production.