

Acute Metabolic Acidosis

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

Three systems work together every second to keep blood pH between 7.35 and 7.45: bicarbonate buffer (instant response), lungs blowing off CO₂ (acts within minutes), and kidneys dumping acid into urine (takes hours to days). Bicarbonate is the largest and fastest-acting buffer in the blood.

BODY'S JOB

Think of pH as the voltage in your body's electrical system. Just like your phone battery needs to stay between 20% and 100% to work properly, blood pH must stay between 7.35 and 7.45 or proteins stop working, enzymes fail, and chemical reactions slow down or speed up dangerously. Bicarbonate is like a sponge that soaks up extra acid the instant it appears. Your lungs act like an exhaust fan—when acid builds up, the brainstem senses it and makes you breathe faster to blow off CO₂, which turns into acid if it stays in the blood. Your kidneys are the cleanup crew—they work slowly over hours and days to dump acid into urine and make fresh bicarbonate to refill the sponge.

What breaks

Metabolic acidosis (acid building up in the blood) happens when the body either makes or absorbs too much acid, or loses too much bicarbonate. To figure out which, you calculate the anion gap—a simple formula that sorts the causes into two buckets.

CLINICAL TERM

High anion gap (>12 mEq/L) means acid is accumulating: lactic acidosis (acid from oxygen-starved tissues) (from shock, sepsis, seizure, or tissue not getting enough oxygen), ketoacidosis (diabetic, alcoholic, or starvation—when the body burns fat for fuel and makes ketones, which are acids), severe waste buildup in the blood (uremia) (kidney failure with GFR below 15 mL/min/1.73 m², so toxins pile up), or toxic ingestions (methanol, ethylene glycol, aspirin, metformin, propylene glycol). Normal anion gap (8–12 mEq/L) means bicarbonate was lost: GI loss from diarrhea, ileostomy, or pancreatic fistula (bicarbonate-rich fluid pouring out of the intestine), renal tubular too much acid in the blood (acidosis) (kidney tubules fail to reclaim bicarbonate or dump acid properly—types 1, 2, or 4), or rapid IV saline infusion (dilutes bicarbonate in the blood). The break does not stay local—the downstream cascade starts with deep, fast breathing, and every other clinical finding flows from that physiologic starting point.

How the body compensates

The body compensates for low pH by hyperventilating to blow off CO₂, but this only buys time. Vital signs may look stable while the patient is silently heading toward cardiovascular collapse.

1. {'compensationMechanism': 'Brainstem chemoreceptors sense low pH and drive respiratory rate up', 'howItWorks': 'Special sensors in the brainstem and carotid arteries detect the drop in pH and the rise in CO₂. They send signals to the respiratory muscles to breathe deeper and faster. For every 1 mEq/L drop in bicarbonate, PaCO₂ drops by about 1.2 mmHg. You can predict the expected PaCO₂ with the Winter formula: $\text{PaCO}_2 = 1.5 \times \text{HCO}_3 + 8 \pm 2$. If the actual PaCO₂ matches this formula, compensation is appropriate. If PaCO₂ is higher than predicted, the patient also has respiratory acidosis (acid building up from trapped carbon dioxide) (lungs are not keeping up, maybe from fatigue or narcotic overdose). If PaCO₂ is lower than predicted, suspect a second process causing respiratory alkalosis (blowing off too much carbon dioxide), such as sepsis, pain, or anxiety.'}
2. {'compensationMechanism': 'Hyperventilation keeps pH closer to normal for hours', 'howItWorks': 'This compensation can hold pH in the low 7.2s even when bicarbonate is very low. This is the dangerous middle zone—the patient looks tired but not crashing, so teams may underestimate severity. But the respiratory muscles can only hyperventilate for so long before they fatigue. Once they tire, PaCO₂ rises, pH drops further, and the patient decompensates rapidly.'}

3. {'compensationMechanism': 'Kidneys try to dump more acid and make more bicarbonate, but this takes days', 'howItWorks': 'The kidneys upregulate hydrogen ion secretion in the collecting duct and increase ammonia production to buffer acid in the urine. They also reclaim every bit of filtered bicarbonate. But this response takes 3–5 days to reach full capacity. In acute metabolic acidosis (acid building up in the blood) (shock, DKA, poisoning), the kidneys cannot keep up, so compensation depends almost entirely on the lungs.'}
4. {'compensationMechanism': 'Watch the trend, not just a single snapshot', 'howItWorks': 'Track serial labs: rising lactate, falling bicarbonate, widening anion gap, climbing potassium, or dropping pH. A patient with pH 7.25 and stable labs is very different from one with pH 7.25 and lactate climbing from 4 to 8 mmol/L over two hours. The second patient is heading toward arrest. Recheck labs every 2–4 hours in severe too much acid in the blood (acidosis).'} }
5. {'compensationMechanism': 'Anticipate the crash points where compensation fails', 'howItWorks': 'pH below 7.0–7.1: vasopressors (norepinephrine, epinephrine) stop working because acidemia blocks the receptors on blood vessels. Blood pressure drops despite maximal doses. High potassium (Hyperkalemia) from too much acid in the blood (acidosis): potassium above 6.5–7.0 mEq/L can cause a slow heart rate (bradycardia), heart block, wide QRS, sine wave on EKG, or cardiac arrest. Cerebral swelling (edema) during DKA treatment: happens when you drop glucose and serum osmolality too fast, especially in children; brain cells swell, causing headache, confusion, vomiting, then herniation. Mixed acid-base disorders: a patient with DKA (metabolic too much acid in the blood) who also vomits (metabolic too little acid in the blood (alkalosis)) may have a near-normal pH but both problems are present and must be treated.'}

Domino effect

1. Deep, fast breathing called Kussmaul respirations appears — 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 — 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) — 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 — 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 — 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 — 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 — 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 — 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.

Symptoms · what the patient feels, 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.
- Heart racing or skipping beats: Acid pushes potassium out of cells into the blood. High blood potassium changes the electrical charge in heart muscle cells, making the heart's rhythm unstable and dangerous—like a guitar string tuned too tight, it snaps.
- Peeing a lot and feeling very thirsty (in diabetic ketoacidosis (the acid crisis of missing insulin)): When blood sugar is very high, the kidneys can't reabsorb it all, so sugar spills into urine and drags water along with it. You lose so much water you get dehydrated, and your brain tells you to drink more.
- Sweet or fruity breath smell (in ketoacidosis): Your body breaks down fat for fuel and makes ketones. One ketone, called acetone, evaporates easily and you breathe it out through your lungs, giving your breath a fruity or nail-polish smell.
- Muscle cramps or twitching: Acid shifts calcium and potassium in and out of cells, and those minerals control how muscles contract. When their balance is off, muscles can cramp, twitch, or feel weak—like an engine misfiring.

Focused exam

- Deep, fast breathing (Kussmaul respirations): When blood has too much acid (pH below 7.35), the body tries to blow off carbon dioxide (CO₂, which turns into acid in the blood) by breathing faster and deeper. This emergency breathing pattern tells you the blood is dangerously acidic and the body is fighting hard to fix it.
- Fruity breath smell: A sweet or nail-polish-remover smell on the breath usually means ketones (acids the body makes when it burns fat for energy instead of sugar) are building up. This points to diabetic ketoacidosis (the acid crisis of missing insulin) (DKA, a life-threatening complication when the body has too little insulin). One ketone, acetone, evaporates out through the lungs and causes the smell.
- Confusion or altered mental status: When blood becomes too acidic, brain cells cannot work properly. Confusion or sleepiness means the pH is dropping fast and the brain is struggling. This signals the body's defenses are failing.

- High potassium on blood test (hyperkalemia): When blood pH drops, acid (hydrogen ions) moves into cells and potassium moves out into the blood in exchange. If potassium rises above 6.5 mEq/L, the heart's electrical system can malfunction and the heart can stop. Continuous heart monitoring is essential.
- Weak pulse and low blood pressure: Acidic blood weakens the heart's squeeze and causes blood vessels to widen. The heart cannot pump blood effectively and tissues do not get enough oxygen and nutrients. This leads to shock (circulation failure).
- Cool, clammy skin: The body shuts down blood flow to the skin and limbs to save blood for the brain, heart, and kidneys. Cool and sweaty skin means the body is in survival mode, redirecting resources to keep vital organs alive.

Diagnostics · every test answers a question

- Arterial blood gas (ABG) → Is the blood too acidic, and is the body compensating?
- Basic metabolic panel (BMP) with anion gap calculation → What is causing the too much acid in the blood (acidosis)—unmeasured acids or bicarbonate loss?
- Serum lactate → Is lactic acid the culprit?
- Serum and urine ketones → Are ketones causing the too much acid in the blood (acidosis)?
- Serum glucose → Is this diabetic ketoacidosis (the acid crisis of missing insulin)?
- Serum osmolality and osmolal gap → Did the patient ingest a toxic alcohol?
- Urinalysis with urine pH → Are the kidneys handling acid properly?
- Serum creatinine and blood urea nitrogen (BUN) → Are the kidneys failing and unable to excrete acid?

Treatment logic

- 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.

- 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.
- 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.
- 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.
- 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.
- Support breathing with supplemental oxygen (nasal cannula or mask to keep oxygen saturation $\geq 94\%$) and monitor for fluid in the lungs; prepare for mechanical ventilation if the patient's breathing muscles tire or too much acid in the blood (acidosis) is so severe the lungs cannot compensate → Maintains adequate oxygen levels while the lungs work hard to compensate. Prevents respiratory failure (lungs cannot maintain gas exchange) from muscle exhaustion.

- Initiate urgent hemodialysis for refractory too much acid in the blood (acidosis) (pH remains below 7.1 despite treatment), severe severe waste buildup in the blood (uremia) (advanced kidney failure with BUN above 100 mg/dL or symptoms of uremic encephalopathy), refractory high potassium (hyperkalemia) (potassium above 6.5 mEq/L not responding to medical therapy), fluid overload with pulmonary edema (fluid flooding the lungs), or confirmed toxic alcohol ingestion (methanol, ethylene glycol) with end-organ damage → Dialysis physically removes acid, potassium, and toxins from the blood. It bypasses failed kidneys and provides immediate metabolic correction when medical therapy cannot keep up.

Medications

Insulin (regular, IV) · Hormone replacement

Insulin is the key that unlocks your cells so sugar can go inside and be burned for energy instead of building up in your blood. When you have diabetic ketoacidosis (the acid crisis of missing insulin) (DKA—a dangerous state where your body burns fat for fuel and makes acids called ketones because there is no insulin to help sugar get into cells), insulin stops your liver from making ketones, moves sugar out of your blood and into muscle and fat cells, and also pushes potassium (a mineral that keeps your heart beating normally) back into cells where it belongs.

1. You give insulin through an IV at 0.1 units per kilogram of body weight every hour (a slow, steady drip)
2. Insulin binds to receptors on the surface of muscle and fat cells, like a key fitting into a lock
3. Gates inside the cell membrane open, and glucose rushes in from the blood; the sugar level in your blood starts to fall
4. At the same time, potassium (which had leaked out of cells during too much acid in the blood (acidosis)) gets pulled back inside
5. In the liver, insulin turns off the enzyme that breaks fat into ketones, so ketone production stops within hours
6. As ketones disappear, the acid level in your blood drops and pH starts climbing back toward normal
7. When blood sugar falls below 200–250 mg/dL, you add dextrose (sugar water) to the IV so the patient does not crash into low blood sugar (hypoglycemia) (dangerously low blood sugar) while insulin is still running

SAFETY WATCH

Low blood sugar (Hypoglycemia) (blood sugar dropping below 70 mg/dL): causes confusion, sweating, shaking, and can lead to seizures or coma if severe; check glucose every hour · Low potassium (Hypokalemia) (potassium falling below 3.5 mEq/L): as too much acid in the blood (acidosis) improves, potassium rushes back into cells and blood levels can plummet, triggering dangerous heart rhythms; replace potassium when level is below 5.2 mEq/L and the patient is making urine · Cerebral swelling (edema) (brain swelling): rare but life-threatening, especially in children with DKA; avoid pushing fluids or insulin too fast · Do NOT start insulin if potassium is below 3.3 mEq/L—replace potassium first or the heart may stop

Sodium bicarbonate (IV) · Alkalinizing agent (base)

Sodium bicarbonate is baking soda given through an IV. It is a base (the opposite of acid) that soaks up extra hydrogen ions (the particles that make your blood acidic) like a sponge. When bicarbonate meets hydrogen in your blood, they combine and form water and carbon dioxide (which you breathe out), raising your blood pH and making it less acidic. It is used only in very severe too much acid in the blood (acidosis) (pH below 7.1) when the heart is struggling, potassium is dangerously high, or the patient is in shock and needs medications to keep blood pressure up.

1. You give 50–100 milliequivalents (mEq) of sodium bicarbonate through an IV over 30–60 minutes
2. Bicarbonate ions spread through the bloodstream and bind to free hydrogen ions, neutralizing them
3. pH rises within minutes, and the heart becomes more stable; blood vessels respond better to medications that raise blood pressure
4. In salicylate (aspirin) overdose, bicarbonate is also used to make the urine alkaline (pH 7.5–8.0), which traps ionized aspirin molecules in the urine so they cannot sneak back into the blood; the kidneys then flush the drug out faster
5. Bicarbonate does NOT fix the root problem—if your body is still making lactic acid from poor oxygen delivery, or ketones from uncontrolled diabetes, or if your kidneys cannot remove acid, the pH will drop again as soon as the bicarbonate wears off

SAFETY WATCH

High sodium (Hypernatremia) (sodium rising above 145 mEq/L): bicarbonate is a sodium salt, so it loads your blood with sodium; too much can cause confusion, seizures, and muscle twitching · Volume overload (fluid backing up into lungs and legs): each dose adds fluid to your bloodstream; in patients with weak hearts or kidney failure, this can cause pulmonary edema (fluid flooding the lungs) (fluid in the lungs) and shortness of breath · Overshoot too little acid in the blood (alkalosis) (pH climbing above 7.45): giving too much bicarbonate flips too much acid in the blood (acidosis) into too little acid in the blood, which shifts the oxygen-release curve and makes it harder for tissues to grab oxygen from red blood cells · Paradoxical CNS too much acid in the blood (acidosis) (brain pH dropping even though blood pH rises): carbon dioxide (CO₂) crosses from blood into the brain much faster than bicarbonate; when bicarbonate neutralizes acid in blood, CO₂ rises, sneaks into the brain, and makes brain fluid more acidic · Low potassium (Hypokalemia) (potassium falling below 3.5 mEq/L): raising pH drives potassium into cells, unmasking a dangerous shortage in the blood; always replace potassium before or during bicarbonate therapy · Do NOT use bicarbonate in DKA unless pH is below 6.9 AND the patient has severe high potassium (hyperkalemia) or shock—it does not improve outcomes and may worsen brain swelling in children · In salicylate toxicity, bicarbonate can worsen low potassium (hypokalemia); give potassium aggressively before alkalinizing the urine

Fomepizole (IV) · Alcohol dehydrogenase inhibitor (antidote)

Fomepizole blocks an enzyme in your liver called alcohol dehydrogenase—the chemical scissors that normally cut apart alcohol molecules. When someone drinks methanol (wood alcohol found in antifreeze or fuel) or ethylene glycol (sweet-tasting chemical in antifreeze), the liver breaks these alcohols down into extremely poisonous acids: formic acid (from methanol) burns the optic nerve and causes blindness, and glycolic and oxalic acids (from ethylene glycol) destroy the kidneys. Fomepizole jams the scissors so the toxic alcohols stay whole and harmless in the blood long enough to be filtered out by the kidneys or removed by dialysis.

1. You give a loading dose of 15 milligrams per kilogram of body weight through an IV over 30 minutes
2. Fomepizole travels to the liver and locks onto alcohol dehydrogenase enzymes, shutting them down
3. Methanol or ethylene glycol stays in its original, less harmful form instead of being converted into formic acid or glycolic acid
4. The unchanged alcohol is slowly filtered out by the kidneys in urine, or removed by hemodialysis (a machine that cleans the blood)

5. You give maintenance doses of 10 mg/kg IV every 12 hours to keep the enzyme blocked until the toxic alcohol level drops below 20 mg/dL
6. If the patient is on dialysis, you increase the dose to 15 mg/kg every 12 hours because dialysis removes fomepizole from the blood

SAFETY WATCH

Headache, nausea, dizziness: common but usually mild · Rare allergic reactions: rash, itching, or in very rare cases anaphylaxis (severe allergic reaction with low blood pressure and trouble breathing) · Increase dose during hemodialysis: the dialysis machine removes fomepizole, so you must give 15 mg/kg every 12 hours while dialysis is running to keep the enzyme blocked · Safe in pregnancy when the alternative is death or blindness from methanol, or kidney failure from ethylene glycol

Potassium chloride (IV or oral) · Electrolyte replacement

Potassium is a charged mineral (electrolyte) that lives mostly inside your cells and keeps your heart beating in a steady rhythm and your muscles working. During too much acid in the blood (acidosis), hydrogen ions (acid particles) flood into cells and kick potassium out into the blood, so blood potassium looks normal or even high. When you fix the too much acid in the blood with insulin or bicarbonate, hydrogen ions leave the cells, potassium rushes back inside, and the blood level can crash—triggering dangerous heart rhythms or even cardiac arrest. Giving potassium refills the tank so there is enough for both blood and cells.

1. Check the potassium level in blood; if it is below 5.2 mEq/L and the patient is making urine, start replacement
2. Give potassium chloride through an IV (usually 10–20 mEq per hour in a peripheral line, up to 40 mEq/hour through a central line if critically low) or by mouth if the patient can swallow
3. Potassium enters the bloodstream and is carried to cells throughout the body
4. Pumps in the cell membrane (especially the sodium-potassium pump) actively move potassium into cells in exchange for sodium
5. Inside the cell, potassium stabilizes the electrical charge across the membrane, which is critical for the heart to beat normally and muscles to contract
6. Recheck potassium every 2–4 hours during too much acid in the blood (acidosis) correction because levels can drop fast as pH rises

SAFETY WATCH

High potassium (Hyperkalemia) (potassium above 5.5 mEq/L): causes dangerous heart rhythms, muscle weakness, and can lead to cardiac arrest; do NOT give potassium if the level is already above 5.2 mEq/L, the patient has no urine output, or severe kidney failure is present · IV potassium can burn veins and cause phlebitis (vein inflammation) if given too fast through a small peripheral IV; dilute well and infuse slowly · EKG changes with high potassium: tall peaked T-waves, wide QRS complex, and eventually a sine-wave pattern that signals imminent cardiac arrest; stop potassium immediately and treat high potassium (hyperkalemia) · Always confirm the patient is making urine before giving potassium—if the kidneys are not working, potassium will pile up in the blood

Hemodialysis · Extracorporeal blood purification (not a drug, but a procedure)

Hemodialysis is a machine that does the work of failed kidneys by cleaning your blood outside your body. Blood is pumped out through a tube into a filter (dialyzer) that has thousands of tiny hollow fibers; waste products, toxins (like methanol, ethylene glycol, or salicylates), extra potassium, and acid pass through the fiber walls into a cleansing fluid (dialysate) and are thrown away, while cleaned blood flows back into your body. It removes small molecules that your kidneys cannot clear when they are shut down or overwhelmed, and it is the fastest way to pull out life-threatening poisons or correct severe too much acid in the blood (acidosis) from kidney failure (uremia).

1. A large IV catheter is placed in a central vein (neck, chest, or groin), or blood is accessed through an arteriovenous fistula (a surgically created connection between an artery and vein in the arm)
2. Blood is pumped out of the body at 200–400 milliliters per minute into the dialyzer
3. Inside the dialyzer, blood flows past dialysate fluid on the other side of a thin membrane; toxins, urea, creatinine, potassium, and acid (hydrogen ions) diffuse across the membrane into the dialysate and are discarded
4. Cleaned blood is pumped back into the patient
5. A typical session lasts 3–4 hours for routine kidney failure, but may run longer or continuously (continuous renal replacement therapy, CRRT) in ICU patients with unstable blood pressure
6. In toxic alcohol poisoning, dialysis is started urgently if the methanol or ethylene glycol level is above 50 mg/dL, pH is below 7.25, or there is kidney injury or vision changes
7. In severe salicylate (aspirin) toxicity, dialysis is started if the level is above 100 mg/dL, the patient has severe too much acid in the blood (acidosis), altered mental status, or pulmonary edema (fluid flooding the lungs)

SAFETY WATCH

Low blood pressure (Hypotension) (blood pressure dropping): removing fluid too quickly can cause dizziness, fainting, or shock; ICU patients may need continuous slow dialysis (CRRT) instead of rapid intermittent sessions · Bleeding: dialysis requires blood thinners (usually heparin) to prevent clotting in the machine; risk of bleeding at the catheter site or elsewhere in the body · Infection: the large IV catheter is a direct highway for bacteria into the bloodstream; strict sterile technique and close monitoring are essential · Electrolyte shifts: potassium, calcium, and phosphorus can swing rapidly during dialysis; monitor labs closely · Air a traveling clot lodging in a vessel (embolism) (air bubble entering the bloodstream): rare but life-threatening; modern machines have air detectors and automatic shut-offs · Dialysis removes some medications (including fomepizole), so doses must be adjusted during treatment

Red flags

- 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)
- Suspected poison ingestion—methanol, antifreeze, or aspirin—with vision changes, kidney failure, or ringing in ears

Nursing priorities

1. Obtain venous or arterial blood gas, basic metabolic panel with sodium, potassium, chloride, bicarbonate, glucose, and creatinine within 15 minutes of patient arrival. — 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. — 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. — 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.
4. Assess and support airway and breathing; apply oxygen to keep saturation above 94%, and notify the provider immediately if respiratory rate exceeds 30 or the patient becomes too tired or confused to breathe deeply. — The lungs compensate for metabolic acidosis (acid building up in the blood) by blowing off carbon dioxide (another acid) through deep Kussmaul breathing; if the patient tires out or becomes too altered to hyperventilate, carbon dioxide will rise, pH will drop dangerously fast, and emergency intubation (breathing tube placement) may be needed.
5. If potassium is above 6.0 mEq/L or EKG shows high potassium (hyperkalemia) changes, immediately give calcium gluconate 1–2 grams IV over 2–3 minutes to stabilize the heart membrane, then prepare insulin plus dextrose and albuterol nebulizer to shift potassium back into cells. — Calcium does not lower potassium but protects the heart muscle from dangerous electrical instability for 30–60 minutes; insulin with sugar and inhaled albuterol (a bronchodilator that also moves potassium) drive potassium from blood into cells within 15–30 minutes, buying time to fix the too much acid in the blood (acidosis) and prevent cardiac arrest.
6. Identify and treat the underlying cause: give IV insulin for DKA (regular insulin 0.1 units/kg/hour after initial bolus), start antibiotics within 1 hour for sepsis, consult nephrology (kidney specialist) or toxicology for dialysis if methanol, ethylene glycol, severe severe waste buildup in the blood (uremia), or salicylate poisoning is suspected. — Too much acid in the blood (Acidosis) will not resolve until you stop the source—insulin stops ketone production in DKA, antibiotics and source control reverse septic lactic acidosis (acid from oxygen-starved tissues), and dialysis physically removes toxic alcohols and their acidic metabolites (breakdown products) while also clearing acid the kidneys cannot handle.

7. Monitor blood glucose every 1–2 hours in DKA patients; once glucose drops below 250 mg/dL, add dextrose 5% to the IV fluids while continuing insulin to clear ketones and close the anion gap. — In DKA, glucose falls faster than ketone acids disappear; stopping insulin too early leaves too much acid in the blood (acidosis) unresolved, but letting glucose drop without adding sugar back causes dangerous low blood sugar (hypoglycemia) (low blood sugar) that can trigger seizures or loss of consciousness.
8. Recheck blood gas, electrolytes, and anion gap every 2–4 hours; watch for signs of overcorrection such as pH rising above 7.45, tetany (muscle spasms), or confusion worsening despite improving lab values. — Too-rapid correction of chronic too much acid in the blood (acidosis) can cause rebound too little acid in the blood (alkalosis) (blood becoming too basic), shift ionized calcium and cause seizures, or trigger cerebral swelling (edema) in DKA patients; serial monitoring lets you adjust treatment speed and catch complications like persistent high potassium (hyperkalemia) or new organ failure early.

Sources

- NEJM: Berend et al. Physiological Approach to Acid-Base Disturbances, 2014
- NEJM: Seifter. Integration of Acid-Base and Electrolyte Disorders, 2014
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