

Respiratory acidosis

Respiratory acidosis (acid building up from trapped carbon dioxide) means CO_2 builds up because the lungs cannot breathe it out fast enough. Support breathing with oxygen, non-invasive ventilation (a tight mask that pushes air in), or a breathing tube if needed. Fix the underlying cause—weak breathing muscles, airway blockage, or a suppressed breathing drive. In chronic cases, drop CO_2 slowly to avoid seizures.

THE ONE-LINE TRUTH

The lungs cannot blow off enough carbon dioxide, so it piles up in the blood and makes the body too acidic.

The chain — how it unfolds

1. Lungs fail to blow off CO_2 — why: The root problem: blocked airways (asthma, COPD), weak breathing muscles (Guillain-Barré, spinal injury), suppressed breathing drive (opioid overdose, brainstem stroke), or damaged alveoli (ARDS, pneumonia) mean air does not move in and out fast enough. CO_2 production continues normally, but elimination drops, so CO_2 piles up in the blood.
2. PCO_2 rises and pH falls (respiratory too much acid in the blood (acidosis)) — why: CO_2 dissolves in blood to form carbonic acid (H_2CO_3), which breaks into hydrogen ions (H^+) and bicarbonate (HCO_3^-). More CO_2 means more acid, so blood pH drops below 7.35. On a blood gas: high PCO_2 (>45 mm Hg) and low pH in acute cases; high PCO_2 , high bicarbonate, and near-normal pH in chronic compensated cases.
3. Brain swells and mental status declines — why: High CO_2 crosses into the brain and dilates (opens) cerebral blood vessels, increasing blood flow and raising intracranial pressure. You see headache, confusion, somnolence (hard to wake), asterixis (flapping hand tremor), myoclonus (sudden muscle jerks), and swelling of the optic nerve (papilledema) (swollen optic disc seen on eye exam). In severe cases, brainstem compression leads to stupor or coma. Acute rises are much more dangerous than chronic because the brain has no time to adapt.

4. Heart rhythm and blood pressure become unstable — why: Acidic blood irritates the heart muscle and makes it prone to arrhythmias (irregular beats). Severe acidemia (pH <7.20) reduces the heart's ability to respond to adrenaline and other stress signals, leading to low blood pressure (hypotension) (low blood pressure) and shock. High CO₂ also causes pulmonary vasoconstriction (lung blood vessels squeeze tight), raising pressure in the right side of the heart.
5. Oxygen delivery to tissues falls — why: Acidic blood shifts the oxygen-hemoglobin curve to the right (the Bohr effect), which helps oxygen unload to tissues—but if the lungs are also failing to bring in oxygen (common in the same diseases that cause CO₂ retention), low oxygen in the blood (hypoxemia) (low blood oxygen) worsens. Organs start to fail from combined too much acid in the blood (acidosis) and low oxygen.
6. Kidneys try to compensate (in chronic cases) — why: Over days to weeks, the kidneys reabsorb bicarbonate and excrete hydrogen ions to pull pH back toward normal. You see high bicarbonate (HCO₃⁻ >30 mEq/L) on the blood gas. This buys time and makes the patient feel better despite high CO₂, but it also means the body has reset to a new, fragile equilibrium. Any new stress (infection, sedative, surgery) can tip them into acute-on-chronic failure.
7. Without treatment, coma and death — why: If CO₂ keeps climbing, severe acidemia shuts down the brain (coma), the heart (arrest), and the lungs (apnea—breathing stops completely). The only fix is to support breathing (non-invasive ventilation or intubation) and treat the root cause (bronchodilators for asthma, reverse opioid overdose with naloxone, drain fluid from the lungs, fix the broken nerve or muscle signal). Every symptom—sleepiness, tremor, swollen optic nerve, irregular heartbeat—is the body's alarm that CO₂ is dangerously high. Trace backwards from the symptom to the broken lung or breathing drive, fix that one upstream break, and the dominoes stop falling.

Spot it — what you see, and why

- Shortness of breath or feeling like you can't catch your breath: When CO₂ builds up in the blood because the lungs aren't blowing it off fast enough, your body senses the gas piling up and triggers faster, harder breathing to try to fix it — but if the lungs are struggling, the breathing still feels hard and never feels like enough.
- Confusion, trouble thinking clearly, or feeling sleepy when you shouldn't be: High CO₂ in the blood crosses into the brain and makes the fluid around brain cells more acidic, which slows down the electrical signals neurons use to talk to each other — this dulls thinking, memory, and alertness.
- Headache: Extra CO₂ in the blood causes the blood vessels in the brain to widen (dilate), which stretches pain sensors in the vessel walls and creates a dull, throbbing headache.

- Feeling anxious or restless: The brainstem's respiratory centers (the parts that control breathing) sense rising CO₂ and fire alarm signals to wake you up and push you to breathe harder, which triggers a feeling of inner panic or discomfort even if you don't know why.
- Blue or gray tint to lips, fingernails, or skin (cyanosis): When the lungs aren't moving air well, oxygen levels in the blood often drop at the same time CO₂ rises; hemoglobin (the oxygen-carrier in red blood cells) without enough oxygen looks dark blue-red instead of bright red, and that color shows through the skin.
- Fast heart rate (tachycardia): High CO₂ and low oxygen both trigger the sympathetic nervous system (the body's fight-or-flight wiring), which releases adrenaline to speed up the heart and try to deliver more blood — and therefore more oxygen — to tissues that need it.

Red flags — act now

- Severe confusion, hard to wake up, or unconscious (CO₂ narcosis — the brain is so slowed by acid that breathing drive shuts down)
- Gasping for air, unable to speak full sentences, or breathing has become very shallow and slow (respiratory failure is imminent)
- Blue lips, tongue, or fingertips that don't improve with extra oxygen (sign of critically low oxygen)
- New chest pain or very fast or irregular heartbeat (the heart may be struggling under low oxygen and high acid)

Treat it — in this order

1. Treat the root cause: bronchodilators (albuterol, ipratropium) and steroids for COPD or asthma flare-up (exacerbation); naloxone for opioid overdose; antibiotics for pneumonia → Fixes the broken step so CO₂ can escape again. Bronchodilators open squeezed airways. Naloxone wakes up the brainstem breathing center. Antibiotics clear infection clogging the air sacs. Once the blockage or suppression is gone, the lungs can blow off CO₂.
2. Start non-invasive positive-pressure ventilation (BiPAP or CPAP mask) if the patient is alert enough to protect their airway and cooperate → The mask pushes air into the lungs with pressure, forcing the chest to expand more and driving CO₂ out faster. It recruits collapsed or weak parts of the lung and reduces the work of breathing. This can prevent the need for a breathing tube (intubation).
3. Intubate (place breathing tube) and start mechanical ventilation if pH < 7.25, patient is unresponsive or abnormally sleepy (somnolent), has severe muscle twitching, or BiPAP fails → The ventilator breathes for the patient, forcing a set amount of air in and out every minute to blow off CO₂. This stabilizes pH and protects the airway from aspiration (breathing in vomit or secretions). Buys time for treatments to work.

4. In chronic COPD with baseline high CO₂ (50–60 mmHg), target the patient's usual CO₂ level, not normal (35–45 mmHg), and use controlled low-flow oxygen (1–2 L/min nasal cannula, target SpO₂ 88–92%) → Prevents oxygen-induced hypercapnia (too much oxygen shuts down the breathing drive in patients whose brain relies on low oxygen as the trigger to breathe). Keeps oxygen just high enough to protect organs without suppressing ventilation.
5. Treat chronic causes: CPAP or BiPAP at night for obstructive sleep pauses in breathing (apnea); respiratory physical therapy or long-term non-invasive ventilation for neuromuscular weakness (ALS, muscular dystrophy); weight loss for obesity hypoventilation syndrome → Prevents repeated nightly CO₂ buildup (sleep pauses in breathing (apnea)) or progressive weakening of breathing muscles. Keeps baseline CO₂ stable and prevents acute-on-chronic respiratory failure.

Your top priorities at the bedside

1. Place patient on continuous pulse oximetry and capnography (CO₂ monitor) and assess mental status every 15 minutes — why: High CO₂ sedates the brain causing somnolence and confusion; oxygen saturation may look normal but rising CO₂ means respiratory failure is progressing so we must catch declining consciousness before the patient cannot protect their airway
2. Position patient upright at 45 degrees or higher and encourage deep breathing every hour if awake — why: Sitting upright lets gravity pull the diaphragm (main breathing muscle) down so lungs expand more easily; this lowers the work of breathing and helps the patient blow off CO₂ with each breath
3. Administer prescribed bronchodilators (albuterol nebulizer) immediately if patient has wheezing or known COPD or asthma — why: Bronchospasm (tight airways) traps CO₂ in the lungs; albuterol relaxes smooth muscle around airways so air flows out and CO₂ can escape with exhalation