

Respiratory acidosis

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THE STORY IN ONE SENTENCE

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“Respiratory acidosis (acid building up from trapped carbon dioxide) means CO₂ 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 CO₂ slowly to avoid seizures.”

Normal anatomy — the body's job

The lungs breathe in oxygen and breathe out carbon dioxide (CO₂), keeping the level of CO₂ in your blood steady. The tiny air sacs (alveoli) swap CO₂ from your blood into the air you exhale. Your brainstem (the bottom part of your brain) watches CO₂ levels and tells your diaphragm (the main breathing muscle) how fast and deep to breathe. When this balance works, CO₂ stays low and your blood stays the right pH (not too acidic, not too basic). Keep that picture in your head, because respiratory acidosis (acid building up from trapped carbon dioxide) is what happens when any part of this chain breaks.

BODY'S JOB

Every cell in your body burns fuel (glucose and oxygen) to make energy, and that creates CO₂ as waste. Your blood carries CO₂ to your lungs. In the alveoli (tiny air sacs), CO₂ crosses from blood into air and you exhale it. The medulla (the breathing control center in your brainstem) constantly checks CO₂ levels in your blood and signals your diaphragm and intercostal muscles (muscles between your ribs) to breathe faster or slower to keep CO₂ balanced. Normally, the amount of CO₂ you make equals the amount you breathe out, so your blood pH stays around 7.40.

1. Cells burn fuel → make CO₂ waste
2. Blood picks up CO₂ and carries it to the lungs

3. CO₂ crosses from blood into the alveoli (air sacs)
4. You exhale CO₂ out of your body
5. The medulla (brainstem breathing center) senses CO₂ and adjusts how fast you breathe
6. Balance maintained: CO₂ out = CO₂ made

What breaks

Respiratory acidosis (acid building up from trapped carbon dioxide) happens when your lungs cannot blow off CO₂ fast enough, so it builds up in your blood and makes it acidic. This can happen in three main ways: something blocks your airways (like mucus or swelling in asthma or COPD), your breathing muscles get too weak to move air (from a stroke, spinal injury, or muscle disease), or your brainstem stops telling you to breathe (from opioid overdose, sedatives, or a brain injury). Sometimes the alveoli (air sacs) are damaged or filled with fluid, so CO₂ cannot cross out of your blood. No matter the cause, the result is the same: CO₂ climbs, your blood becomes acidic, and your brain and heart struggle.

CLINICAL TERM

Hypoventilation (not breathing deeply or fast enough) or impaired gas exchange leads to CO₂ retention and a low blood pH (acidemia). Common causes: obstructive lung disease (COPD flare-up (exacerbation), severe asthma, airway foreign body), restrictive lung disease (kyphoscoliosis, obesity hypoventilation), neuromuscular failure (Guillain-Barré, myasthenia gravis, ALS, phrenic nerve injury, spinal cord injury above C3), central respiratory depression (opioid or benzodiazepine overdose, brainstem stroke, severe obstructive sleep pauses in breathing (apnea)), or acute parenchymal lung injury (ARDS, severe pneumonia). You will see somnolence, asterixis (flapping tremor), myoclonus (muscle jerks), swelling of the optic nerve (papilledema) (swollen optic discs from high brain pressure), headache, and in severe cases stupor or coma. Acute rises in CO₂ cause rapid mental status decline and cerebral vasodilation (brain blood vessels open wide, raising brain pressure). Chronic hypercapnia triggers renal compensation (the kidneys hold onto bicarbonate over days to weeks), so the same high CO₂ is tolerated with fewer symptoms.

How the body compensates

When CO₂ rises, the body fights to protect the brain and heart. In acute respiratory acidosis (acid building up from trapped carbon dioxide) (minutes to hours), the brain's blood vessels dilate (open wide) to wash out CO₂ faster, but this raises brain pressure and makes you sleepy. The kidneys cannot help fast, so pH drops dangerously. In chronic respiratory acidosis (days to weeks), the kidneys slowly reabsorb bicarbonate (a base) to neutralize the acid, so pH climbs

back toward normal even though CO₂ stays high. This compensation buys time but masks how sick the lungs really are. The danger is missing the slow slide toward respiratory failure because vital signs and labs look 'okay' until the system crashes.

1. {'step': 'Acute phase (minutes to hours): cerebral vasodilation', 'why': 'High CO₂ makes brain blood vessels open wide to flush CO₂ out faster. This raises intracranial pressure (pressure inside the skull), causing headache, swelling of the optic nerve (papilledema) (swollen optic nerve), confusion, and eventually coma. The kidneys have not had time to help, so blood pH falls fast.'}
2. {'step': 'Chronic phase (days to weeks): renal bicarbonate retention', 'why': 'The kidneys sense the low pH and start holding onto bicarbonate (HCO₃⁻, a base) and peeing out more hydrogen ions (acid). This slowly pulls pH back up toward normal, even though CO₂ stays high. On a blood gas, you see high PCO₂ and high bicarbonate with a near-normal or mildly low pH. The patient tolerates high CO₂ much better because the acid is buffered.'}
3. {'step': 'Respiratory drive blunts in chronic hypercapnia', 'why': 'If CO₂ stays high for weeks, the medulla (breathing center) resets its CO₂ set-point higher and stops responding to CO₂ as a signal to breathe. Instead, low oxygen (hypoxia) becomes the main breathing trigger. Giving too much oxygen can wipe out that trigger, drop the respiratory rate, and make CO₂ climb even higher—so use controlled, low-flow oxygen (target SpO₂ 88–92% in COPD patients).'}}
4. {'step': 'Vital signs may look stable while compensation works', 'why': 'Heart rate, blood pressure, and oxygen saturation can stay in normal ranges because the kidneys are holding pH steady and the heart is pumping harder. This is the dangerous middle—teams may miss that the patient is one step from crashing because the numbers look 'fine.' Always trend over time, not just one snapshot.'}
5. {'step': 'Watch for decompensation (the crash point)', 'why': 'Compensation has limits. If CO₂ keeps rising or the kidneys max out, pH will drop sharply, leading to severe acidemia. Signs of decompensation: worsening somnolence, asterixis (flapping tremor when you hold your hands out), myoclonus (muscle jerks), arrhythmias (irregular heartbeat from acidic blood irritating the heart), low blood pressure (hypotension) (low blood pressure), or coma. Once pH falls below ~7.20, the heart and brain fail fast.'}
6. {'step': 'Danger of rapid CO₂ correction in chronic cases', 'why': 'If you ventilate a chronic CO₂ retainer too aggressively and drop CO₂ fast, the kidneys are still holding extra bicarbonate. The blood swings alkaline (high pH), and this can trigger seizures, arrhythmias, or sudden death. Always correct CO₂ slowly in chronic respiratory acidosis (acid building up from trapped carbon dioxide)—aim to drop PCO₂ by no more than 10–15 mm Hg per day.'}

Domino effect

1. Lungs fail to blow off CO₂ — 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. CO₂ production continues normally, but elimination drops, so CO₂ piles up in the blood.
2. PCO₂ rises and pH falls (respiratory too much acid in the blood (acidosis)) — CO₂ dissolves in blood to form carbonic acid (H₂CO₃), which breaks into hydrogen ions (H⁺) and bicarbonate (HCO₃⁻). More CO₂ means more acid, so blood pH drops below 7.35. On a blood gas: high PCO₂ (>45 mm Hg) and low pH in acute cases; high PCO₂, high bicarbonate, and near-normal pH in chronic compensated cases.
3. Brain swells and mental status declines — High CO₂ 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 — 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 — 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) — 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 — 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.

Symptoms · what the patient feels, 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.
- Muscle twitches or hand tremor (asterixis): Severe CO₂ buildup disrupts the normal electrical rhythm of nerves and muscles, causing brief lapses in the signal that keeps muscles tight; this shows up as a flapping tremor of the hands when you hold your arms out.

- An arterial blood gas (ABG) test shows high CO₂ (PaCO₂ above 45 mmHg) with low pH (below 7.35) in sudden respiratory acidosis (acid building up from trapped carbon dioxide), or near-normal pH (7.35–7.40) in long-standing cases where the kidneys have had time to hold onto bicarbonate to buffer the acid: CO₂ dissolves in blood and forms carbonic acid, which splits into hydrogen ions (H⁺) that lower pH; in acute (sudden) cases the kidneys haven't compensated yet so pH drops, but in chronic (long-term) cases the kidneys reabsorb extra bicarbonate (a base) over days to weeks to bring pH back closer to normal even though CO₂ stays high.

Focused exam

- Mental status change — sleepiness, confusion, or trouble waking up: When CO₂ (carbon dioxide, the waste gas your lungs normally blow off) builds up too high, it crosses into the brain and acts like a sleeping pill. The higher it climbs, the more the brain slows down. This is the most dangerous sign because it means the lungs are failing fast and the brain is being poisoned. If the patient cannot wake up, the breathing may stop completely.
- Tremor or muscle twitching (myoclonus): CO₂ trapped in the blood turns into acid (carbonic acid), making the blood too acidic. The acid irritates nerve cells, and they fire in jerky bursts, causing muscles to twitch. This tells you the too much acid in the blood (acidosis) is getting worse and the nervous system is starting to malfunction.
- Slow, shallow breathing or gasping: This is the root problem. The lungs move air in and out to swap oxygen for CO₂. If breathing is slow and shallow, not enough CO₂ escapes with each breath. Count the breaths per minute — if it is under 10 or the chest barely moves, the body cannot blow off CO₂ fast enough and it piles up.
- Swollen optic nerve in the back of the eye (papilledema) on fundoscopy: High CO₂ makes blood vessels in the brain swell and dilate. This raises pressure inside the skull, pushing on the optic nerve where it enters the eye. Seeing a swollen nerve means the too much acid in the blood (acidosis) has been going on long enough to cause brain swelling — a serious red flag.
- Bluish skin (Cyanosis) (blue lips, tongue, or fingernails): The lungs are struggling to blow off CO₂, so they are also struggling to pull in oxygen. When blood does not carry enough oxygen, it looks dark purple-blue instead of bright red. This shows up first in the lips and under the nails — it means the lungs are failing at both jobs.

- Loud wheezing or no air movement at all (silent chest): In respiratory acidosis (acid building up from trapped carbon dioxide) from asthma or COPD (chronic obstructive pulmonary disease, long-term lung damage from smoking or irritants), the breathing tubes are squeezed tight or clogged with mucus. At first you hear wheezing — a high whistle as air squeezes through. But if the tubes close almost completely, no air moves at all and the chest is silent. A silent chest in severe asthma or COPD is a dire emergency because no gas is escaping.
- Flapping tremor of the hands (asterixis): High CO₂ and acid disrupt the brain circuits that hold muscle tone steady. Ask the patient to hold both arms out with wrists bent back — the hands will flap forward and back like a slow wave. This is a sign the brain is being hurt by the acid buildup.

Diagnostics · every test answers a question

- Arterial blood gas (ABG) → Is the pH low? Is the CO₂ high? Is this acute (sudden) or chronic (long-term)?
- Basic metabolic panel (serum electrolytes and bicarbonate) → Is the bicarbonate elevated, suggesting the kidneys are trying to compensate for chronic high CO₂?
- Chest X-ray → What is blocking or breaking the lungs — pneumonia, fluid, collapsed lung, or severe COPD?
- Pulse oximetry (SpO₂) → Is oxygen getting into the blood?
- Urine drug screen or serum toxicology → Did the patient take opioids, benzodiazepines, or alcohol that suppress the brain's breathing center?
- Serum creatine kinase (CK) and thyroid-stimulating hormone (TSH) → Is there muscle breakdown or severe hypothyroidism (very low thyroid) causing weak breathing muscles?

Treatment logic

- 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.
- 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).

- 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.
- 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.
- 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.
- Avoid sedatives, opioids, and high-dose oxygen unless carefully monitored → Prevents further suppression of the brainstem breathing center or worsening CO₂ retention. Protects the patient from harm caused by medical care (iatrogenic) (doctor-caused) harm.

Medications

Naloxone (Narcan) · Opioid antagonist (opioid blocker)

Kicks opioid drugs off the receptors in the brain's breathing center, so the brain wakes up and starts telling the lungs to breathe again. Works in 1–2 minutes.

1. An opioid drug (morphine, fentanyl, oxycodone) locks onto mu opioid receptors in the medulla and tells the brain to breathe slowly or stop.
2. Carbon dioxide builds up because the brain is not ordering the lungs to clear it out.
3. Naloxone bumps the opioid off the receptor and blocks the spot.
4. The medulla wakes up, sends normal breathing signals to the diaphragm, and the lungs start moving air again.
5. CO₂ drops back toward normal as ventilation picks up.

SAFETY WATCH

Can trigger sudden opioid withdrawal (shaking, sweating, agitation, pain) in someone who uses opioids regularly. · Wears off in 30–90 minutes; opioid can outlast naloxone, so patient needs close monitoring and repeat doses or an IV drip. · Do NOT give to newborns of opioid-dependent mothers without neonatal resuscitation readiness—withdrawal can be severe.

Albuterol (ProAir, Ventolin) · Short-acting beta-2 agonist (SABA) bronchodilator

Relaxes the tight muscle bands wrapped around airways in the lungs, opening the tubes so air (and CO₂) can move in and out more easily. Works in 5–15 minutes.

1. In asthma or COPD flare, airway smooth muscle squeezes tight, narrowing the tubes and trapping air and CO₂.
2. Albuterol lands on beta-2 receptors on the muscle cells.
3. This triggers a chemical chain (cAMP rises) that tells the muscle to relax.
4. Airways widen, air flows out, trapped CO₂ escapes, and respiratory acidosis (acid building up from trapped carbon dioxide) improves.
5. Effect lasts 4–6 hours, then may need another dose.

SAFETY WATCH

Can speed heart rate (palpitations) and cause hand tremor—usually mild. · Overuse (more than every 4 hours for days) is a red flag that asthma or COPD is not controlled; patient needs a controller medication and urgent provider check. · Rarely lowers potassium if used in very high doses; monitor in hospital setting.

Ipratropium (Atrovent) · Short-acting muscarinic antagonist (SAMA) anticholinergic bronchodilator

Blocks the nerve signal (acetylcholine) that tells airways to squeeze, so the tubes stay open. Often combined with albuterol for a stronger effect. Works in 15–30 minutes.

1. The vagus nerve releases acetylcholine, which lands on M3 receptors and tells airway muscle to contract.
2. Ipratropium blocks those receptors so acetylcholine cannot dock.
3. Airway muscle relaxes, tubes widen, airflow improves, CO₂ clears better.
4. Peak effect at 1–2 hours; lasts 4–6 hours.

SAFETY WATCH

Dry mouth is common. · Do NOT spray into the eyes—can trigger acute narrow-angle glaucoma (sudden eye pain, blurred vision). · Use with caution in men with large prostate (can worsen urinary retention, though uncommon with inhaled form).

Prednisone (oral) or Methylprednisolone (IV) · Corticosteroid (anti-inflammatory steroid)

Shuts down the inflammatory cascade inside airways, shrinking swelling and mucus production so air can move and CO₂ can escape. Takes 4–6 hours to start working; full effect by 24–48 hours.

1. In asthma or COPD flare-up (exacerbation), immune cells flood the airway walls, releasing chemicals that cause swelling, mucus, and muscle spasm.
2. Steroid enters the cell nucleus and turns off genes that make inflammatory proteins (cytokines, leukotrienes).
3. Swelling shrinks, mucus production drops, airways open wider.
4. Ventilation improves, CO₂ clears, pH rises back toward normal.
5. Taper dose over 5–14 days to avoid rebound inflammation.

SAFETY WATCH

Short courses (5–7 days) are generally safe; longer use raises blood sugar, blood pressure, and infection risk. · Can cause mood swings, trouble sleeping, stomach upset. · Do NOT stop suddenly after weeks of use—must taper to let the adrenal glands wake back up. · Check blood sugar in diabetics; steroid can spike glucose.

Supplemental oxygen (O₂) · Gas therapy (not a drug, but a key intervention)

Raises the oxygen level in blood to keep tissues fed while the team works to fix the CO₂ problem. In COPD, use the lowest flow that keeps O₂ saturation 88–92 % to avoid blunting the drive to breathe.

1. Oxygen flows through nasal prongs or mask into the lungs.
2. Alveolar oxygen pressure rises.
3. More oxygen dissolves in blood and binds to hemoglobin.
4. Tissues get the oxygen they need, preventing organ damage while ventilation is being restored.

SAFETY WATCH

In chronic CO₂ retainers (advanced COPD), aim for 88–92 % O₂ saturation, NOT 95–100 %. Higher levels can suppress breathing and worsen too much acid in the blood (acidosis). · Monitor with pulse oximeter and arterial blood gas. · Nasal prongs dry out nasal passages; humidify if flow > 4 L/min.

Non-invasive positive-pressure ventilation: BiPAP (bilevel positive airway pressure) · Mechanical ventilation support (device, not a drug)

A tight-fitting face mask pushes air into the lungs at two pressures—higher when you breathe in (to inflate lungs and overcome airway resistance) and lower when you breathe out (to help CO₂ escape). Buys time for medications to work and avoids the need for a breathing tube.

1. Mask seals over nose and mouth.
2. Machine delivers inspiratory pressure (IPAP, often 10–20 cm H₂O) to push air in, opening collapsed airways and filling alveoli.
3. Expiratory pressure (EPAP, often 4–8 cm H₂O) keeps airways open during exhale so CO₂ can escape instead of being trapped.
4. Ventilation improves, CO₂ drops, pH climbs back toward 7.35–7.45.
5. Patient must be awake enough to cooperate and protect their airway (no vomiting or choking risk).

SAFETY WATCH

Requires an awake, cooperative patient. · Risk of air swallowing (gastric distension), mask pressure sores on bridge of nose. · If patient tires out, becomes confused, or too much acid in the blood (acidosis) worsens despite BiPAP, need to intubate (insert breathing tube) and use full mechanical ventilation. · Monitor vital signs, oxygen saturation, and repeat blood gas in 1–2 hours.

Endotracheal intubation and mechanical ventilation · Invasive airway and ventilation support (procedure + device)

A breathing tube is placed through the mouth into the trachea, and a ventilator machine takes over breathing. The machine controls how much air goes in, how fast, and at what pressure, clearing CO₂ and resting exhausted breathing muscles.

1. Patient is sedated (and often paralyzed briefly) so a tube can be passed between the vocal cords into the trachea.
2. Tube is connected to a ventilator.
3. Ventilator delivers breaths at a set rate and volume, inflating the lungs and clearing CO₂.

4. Arterial blood gas is checked every few hours; vent settings are adjusted to bring pH and CO₂ back to safe range.
5. Once the cause is treated (e.g. pneumonia cleared, COPD flare controlled), patient is weaned off the ventilator and tube is removed.

SAFETY WATCH

Requires ICU care and sedation. · Risks: ventilator-associated pneumonia, vocal cord injury, tracheal damage, barotrauma (lung rupture from high pressure). · Prolonged intubation (> 7–10 days) may need tracheostomy (surgical breathing tube through the neck). · Must monitor for auto-PEEP (air trapping) in COPD; adjust settings to allow longer exhale time.

Acetazolamide (Diamox) · Carbonic anhydrase inhibitor diuretic

Makes the kidneys dump bicarbonate (the blood's buffer) into urine, creating a mild metabolic acidosis (acid building up in the blood) that nudges the brain's breathing center to breathe faster and blow off CO₂. Used mainly in chronic stable hypercapnia (e.g. obesity hypoventilation, high-altitude periodic breathing), NOT in acute respiratory acidosis (acid building up from trapped carbon dioxide).

1. Acetazolamide blocks carbonic anhydrase, the enzyme that recycles bicarbonate in kidney cells.
2. Bicarbonate spills into urine, blood bicarbonate drops.
3. Blood pH nudges downward (mild metabolic acidosis (acid building up in the blood)).
4. The medulla senses the pH drop and cranks up breathing drive.
5. Patient breathes deeper and faster, CO₂ falls, respiratory acidosis (acid building up from trapped carbon dioxide) improves over days to weeks.

SAFETY WATCH

Can cause tingling fingers and toes (paresthesias), common and harmless. · Makes urine alkaline; raises risk of calcium kidney stones. · Do NOT use in severe liver disease (can trigger hepatic encephalopathy) or severe kidney disease (won't work). · Monitor potassium—can cause low potassium (hypokalemia).

Red flags

- 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)

Nursing priorities

1. Place patient on continuous pulse oximetry and capnography (CO₂ monitor) and assess mental status every 15 minutes — 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 — 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 — Bronchospasm (tight airways) traps CO₂ in the lungs; albuterol relaxes smooth muscle around airways so air flows out and CO₂ can escape with exhalation
4. Prepare and assist with BiPAP setup if patient is alert and cooperative, or gather intubation equipment if patient is abnormally sleepy (somnolent) or has myoclonus — BiPAP pushes air into lungs with positive pressure to inflate alveoli and blow off CO₂ without needing a tube; but if the brain is too sedated by high CO₂ the patient cannot protect their airway from aspiration so intubation with mechanical ventilation is safer
5. Hold or reduce any sedating medications and notify provider immediately; prepare naloxone 0.4 mg IV if opioid overdose is suspected — Opioids and benzodiazepines suppress the medullary respiratory center (breathing control area in brainstem) so the brain forgets to signal the lungs to breathe; naloxone reverses opioid receptor binding and restarts normal breathing drive within 2 minutes
6. Start IV access and draw arterial blood gas (ABG) as ordered to confirm pH and PCO₂ levels — ABG is the only test that directly measures pH and PCO₂ to diagnose respiratory acidosis (acid building up from trapped carbon dioxide) and tell us if it is acute (pH below 7.35) or chronic with compensation (pH near normal with high bicarbonate); this guides how aggressively we escalate respiratory support

7. Suction oral secretions and encourage coughing every 2 hours if patient has weak cough or thick mucus — Mucus plugs and secretions block airways so CO₂ cannot leave the lungs; clearing the airway restores ventilation (air movement) and prevents atelectasis (collapsed alveoli) which worsens gas trapping
8. Monitor for signs of respiratory muscle fatigue: use of neck muscles to breathe, paradoxical abdominal breathing, or respiratory rate above 30 or below 8 — When the diaphragm and intercostal muscles (rib muscles) tire from working too hard they can no longer move enough air; paradoxical breathing (belly sucks in during inhale instead of pushing out) and extreme rates mean muscles are failing and patient will need mechanical ventilation within minutes

Sources

- American Thoracic Society (ATS)
- GOLD COPD 2024 guideline
- UpToDate clinical reference
- American Association for Respiratory Care (AARC)

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