

COPD · Chronic Obstructive Pulmonary Disease

COPD flare-up (exacerbation) means the airways are so squeezed and clogged that air gets trapped inside the lungs and cannot leave.

THE ONE-LINE TRUTH

Air goes in but cannot come back out.

The chain — how it unfolds

1. Airways are swollen, clogged with mucus, and collapse shut when you breathe out — why: Years of smoke or irritants make the airway walls thick and inflamed (chronic bronchitis). The airways make extra sticky mucus from too many goblet cells. The lung tissue that used to hold the airways open (elastic fibers providing radial traction) breaks down (emphysema), so the airways collapse like a wet noodle when you try to push air out.
2. Air gets trapped inside the lungs (air trapping and hyperinflation) — why: Air can get in (because breathing in opens the airways), but it cannot get out (because breathing out squeezes them shut and they have no support to stay open). Each breath adds a little more air, and the lungs blow up like a balloon that cannot deflate. This is called hyperinflation and air trapping.
3. The diaphragm gets pushed flat and cannot work (diaphragmatic dysfunction) — why: Normally the diaphragm is dome-shaped and can pull down hard to open the chest. Trapped air pushes the diaphragm flat, so it has no leverage. It is like trying to pull on a rope when you are standing right under the pulley — you cannot generate any force. The diaphragm moves into a mechanically disadvantaged position.
4. Carbon dioxide builds up in the blood and oxygen drops (hypercapnia and low oxygen in the blood (hypoxemia)) — why: Because air cannot leave, the carbon dioxide you made (the waste gas) stays in your blood instead of being breathed out. CO₂ mixes with water in your blood to make carbonic acid (through the enzyme carbonic anhydrase), so your blood gets acidic (respiratory too much acid in the blood (acidosis) with low pH). At the same time, fresh oxygen cannot get in fast enough, so oxygen levels fall (hypoxemia).

5. You breathe faster and harder, but it does not help much (increased work of breathing with poor gas exchange) — why: Your brain senses the high CO₂ and low oxygen through chemoreceptors in the carotid bodies (in your neck) and the medulla (in your brainstem) and screams at you to breathe. You work every muscle you have — sternocleidomastoid, scalenes, trapezius, abdominal muscles — to try to move air. But because the airways are collapsed and clogged, all that effort barely moves any air. You look like you are breathing hard, but not much air is actually flowing (high minute ventilation with poor alveolar ventilation).
6. Your heart races and blood pressure may rise (sympathetic activation) — why: Your body releases epinephrine and norepinephrine (adrenaline hormones) from the adrenal glands to try to save you. Your heart beats faster to pump more blood. Blood vessels tighten (vasoconstriction) to shift blood to your brain and heart. This is the sympathetic nervous system (the fight-or-flight system) trying to buy time.
7. You get tired, confused, or sleepy (CO₂ narcosis and oxygen-starved (hypoxic) encephalopathy) — why: High carbon dioxide crosses into your brain and makes you sleepy and confused (CO₂ narcosis). Your breathing muscles (especially the diaphragm and intercostals) get exhausted from working so hard. If CO₂ keeps climbing and oxygen keeps falling, your brain does not get enough oxygen (hypoxic encephalopathy), and you can lose consciousness or stop breathing (respiratory arrest).
8. Risk of pneumothorax (collapsed lung) — why: Trapped air stretches the lung tissue so tight that a weak spot in an over-inflated air sac (a bleb or bulla) can pop like an over-inflated balloon. Air leaks into the pleural space (the space between the lung and the chest wall) and collapses the lung. This is a life-threatening emergency that needs immediate treatment with a chest tube.

Spot it — what you see, and why

- Breathing out slowly through pursed lips (like blowing out a candle): The small air tubes in the lungs collapse too early when the person breathes out. Pursing the lips creates a little back-pressure—like putting your thumb partly over a garden hose—which props the tubes open long enough to empty more air out of the lungs.
- Neck and shoulder muscles working hard to breathe: The main breathing muscle (the diaphragm) sits flat and weak because the lungs are over-inflated and trap air. The body recruits helper muscles in the neck and shoulders to lift the ribs and pull in air.
- Barrel chest (the chest looks round and fat, like a barrel): Damaged lungs lose their elastic snap-back, so old air gets trapped inside. Over time, the chest wall stays puffed out in the inhaled position, giving it a round, barrel shape.

- Sitting leaning forward with hands on knees (tripod position): Leaning forward helps anchor the shoulder and arm muscles so they can pull harder on the ribs to help inflate the lungs. It's the body's way of turning every available muscle into a breathing helper.
- Chronic cough that brings up mucus (sputum), especially in the morning: Damaged airways make too much thick mucus and lose the tiny hairlike sweepers (cilia) that normally clear it out. Mucus pools overnight, so the person coughs it up when they wake and start moving.
- Shortness of breath that gets worse with activity: The lungs can't move air in and out fast enough because the airways are narrowed and the air sacs are destroyed. When the body needs more oxygen during exercise, the damaged lungs can't keep up.

Red flags — act now

- New confusion, drowsiness, or difficulty staying awake (sign of carbon dioxide building up and respiratory failure)
- Blue or gray lips, tongue, or fingernails (sign of dangerously low oxygen)
- Chest pain or coughing up blood (may mean infection has spread, a clot in the lung, or lung tissue tearing)
- Sudden worsening shortness of breath at rest or inability to speak in full sentences (sign of severe flare-up or collapsed lung)
- Swelling in the ankles and legs with a bloated belly (sign the right side of the heart is failing from lung strain)

Treat it — in this order

1. Short-acting bronchodilators (albuterol nebulized plus ipratropium) → Relaxes the squeezed smooth muscle around the airways so trapped air can actually leave the lungs. This lets the diaphragm flatten back out and CO₂ can escape.
2. Systemic corticosteroids (prednisone 40 mg by mouth daily or methylprednisolone IV) for 5 days → Reduces swelling inside the airway walls so the tube gets wider and less mucus sticks around. Wider tube means air moves easier and the breathing muscles do not have to work as hard.
3. Controlled supplemental oxygen to keep oxygen saturation at 88 to 92 percent → Fixes low blood oxygen without flooding the lungs with too much oxygen, which would turn off the breathing drive in someone whose brain already relies on low oxygen to trigger breaths.
4. Bilevel positive airway pressure (BiPAP or noninvasive ventilation) if CO₂ rises above 50 mmHg and drowsiness starts → Pushes air down into the lungs on inhale and keeps airways propped open on exhale so trapped air can escape. Wakes up the brain and drops CO₂ fast without needing a breathing tube down the throat.

5. Antibiotics (azithromycin, amoxicillin-clavulanate, or doxycycline) if coughed-up mucus (sputum) is pus-filled (purulent) or patient is very sick → Kills bacteria that thicken mucus and trigger inflammation, which unplugs airways so air can move and reduces the swelling signal.

Your top priorities at the bedside

1. Start controlled oxygen by nasal cannula to keep oxygen saturation (SpO₂) between 88 and 92 percent, not higher. — why: COPD patients breathe because of low oxygen, not high CO₂ like healthy people. Too much oxygen (above 92 percent) shuts off their breathing drive, CO₂ builds up, and they become drowsy and stop breathing. This is called hypercapnic respiratory failure.
2. Give albuterol 2.5 mg plus ipratropium 0.5 mg together by nebulizer (breathing treatment) right away, then every 20 minutes for the first hour if very short of breath. — why: Albuterol is a beta-agonist that relaxes smooth muscle around airways in seconds to minutes. Ipratropium is an anticholinergic that blocks signals that squeeze airways shut. Together they open airways faster and better than either alone.
3. Watch for signs of high CO₂ and respiratory failure: confusion, extreme sleepiness, or very slow breathing. Check ABG (arterial blood gas) if patient becomes drowsy. — why: When lungs cannot blow out CO₂, it dissolves in blood and makes acid (respiratory too much acid in the blood (acidosis)). High CO₂ also acts like a sedative on the brain. If CO₂ goes above 50 mmHg with drowsiness, the patient needs BiPAP or a breathing tube to survive.