

# COPD · Chronic Obstructive Pulmonary Disease

Air goes in but cannot come back out.

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## THE STORY IN ONE SENTENCE

Air goes in but cannot come back out.

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

## Normal anatomy — the body's job

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Breathing in takes muscle work. Your diaphragm (the big sheet of muscle under your lungs) pulls down and your chest opens so air rushes in. Breathing out is easy: the diaphragm relaxes, the lungs spring back like a rubber band, and air flows out without any effort. Airways stay open because the stretchy lung tissue around them holds them up like tent stakes.

## BODY'S JOB

The airways are tubes that carry air deep into your lungs, where tiny air sacs (called alveoli) trade oxygen into your blood and pull carbon dioxide waste out. Healthy airways are smooth and open. The lung tissue is stretchy and strong, so it pulls the airways open when you breathe. Breathing out happens by itself because everything relaxes and the lungs shrink back down.

1. You breathe in
2. Oxygen moves into your blood
3. You breathe out
4. Airways stay open

## What breaks

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Years of breathing in cigarette smoke, air pollution, or chemical fumes damage the lungs in two big ways. First, the airways get swollen and irritated on the inside, the walls get thick, and they make extra sticky mucus that clogs them (chronic bronchitis). Second, the stretchy lung

tissue breaks down (that is emphysema), so the airways lose their support and collapse shut when you try to breathe out. Air gets trapped inside. A flare-up (exacerbation) happens when something pushes you over the edge — a cold, the flu, breathing in dust or smoke, or missing your inhaler medicine.

#### CLINICAL TERM

Chronic bronchitis (long-term airway inflammation with mucus hypersecretion) and emphysema (destruction of alveolar walls and loss of elastic recoil) are the two main problems in COPD. Chronic bronchitis causes airway wall thickening, goblet cell hyperplasia (extra mucus-making cells), smooth muscle hypertrophy, and narrowing of the airway lumen. Emphysema destroys the alveolar septa and the elastic fibers that provide radial traction to hold small airways open, so they collapse during expiration (dynamic airway collapse). The result is air trapping and hyperinflation. An acute flare-up (exacerbation) is triggered by a respiratory infection (viral such as rhinovirus or influenza, or bacterial such as *Haemophilus influenzae*, *Streptococcus pneumoniae*, or *Moraxella catarrhalis*), environmental irritants (tobacco smoke, pollution, dust), or non-adherence to controller medications (long-acting beta-agonists, long-acting muscarinic antagonists, and inhaled corticosteroids). The trigger increases airway inflammation mediated by neutrophils and eosinophils, mucus plugging, and bronchospasm (smooth muscle constriction), which worsens air trapping and leads to low oxygen in the blood (hypoxemia) (low oxygen) and hypercapnia (high carbon dioxide).

## How the body compensates

The body fights back to keep oxygen moving and carbon dioxide from building up. You breathe faster and harder. Your heart races. Your body shifts blood flow to vital organs. For a while, these backup plans keep vital signs okay, so you might not look sick yet. But the body is working at maximum effort. When it cannot keep up anymore, things crash fast — carbon dioxide spikes, you get too sleepy to breathe, or a lung collapses. The danger is missing the early warning signs while compensation is still holding.

1. {'name': 'Faster breathing (tachypnea)', 'what': 'Your body senses low oxygen and high carbon dioxide through chemoreceptors (sensors in your neck and brainstem), so it tells you to breathe faster and deeper to try to blow off CO<sub>2</sub> and pull in more oxygen.'}
2. {'name': 'Use of accessory muscles', 'what': 'Normally only your diaphragm does the work. Now your sternocleidomastoid and scalene muscles (in your neck), your trapezius muscles (in your shoulders), and your abdominal muscles all help pull your chest open. You can see someone sitting forward in the tripod position, breathing through pursed lips, using every muscle to move air.'}

3. {'name': 'Faster heart rate (tachycardia)', 'what': 'The heart pumps faster to carry whatever oxygen is available to your brain, heart, and kidneys. Your sympathetic nervous system (the fight-or-flight system) drives this.'}
4. {'name': 'Increased sympathetic tone', 'what': 'Your body releases epinephrine and norepinephrine (adrenaline hormones) to open airways a little more through beta-2 receptors in the airway muscles, speed up the heart, and shift blood to vital organs by tightening blood vessels in the skin and gut.'}
5. {'name': 'Pursed-lip breathing', 'what': 'Breathing out through tight lips creates a little back-pressure (positive end-expiratory pressure) that helps hold airways open longer so more air can escape. Many people with COPD learn this naturally.'}
6. {'name': 'The dangerous middle (compensated respiratory failure)', 'what': 'For a while, all this hard work keeps oxygen okay and carbon dioxide from spiking too high. Vital signs look borderline. That is the danger zone, because it hides how much effort the body is spending. You look stable, but you are one step from crashing.'}
7. {'name': 'The crash point (decompensated respiratory failure)', 'what': 'When the body cannot work any harder, carbon dioxide spikes (hypercapnic respiratory failure), the blood gets too acidic (respiratory too much acid in the blood (acidosis) with pH below 7.35), and you get too sleepy or confused to keep breathing (altered mental status from CO2 narcosis). Or a pocket of trapped air bursts through the lung and collapses it (spontaneous pneumothorax). Once you hit this point, the window to recover without a breathing tube (intubation) or ICU care is very narrow.'}
8. {'name': 'Falling respiratory rate is a red flag (agonal respirations)', 'what': 'If someone has been breathing 30 times a minute and suddenly slows to 12 or gasps irregularly, that is not improvement — that is exhaustion. The respiratory muscles (especially the diaphragm) are giving out. This is a pre-arrest warning.'}

## Domino effect

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1. Airways are swollen, clogged with mucus, and collapse shut when you breathe out — 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) — 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) — 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)) — Because air cannot leave, the carbon dioxide you made (the waste gas) stays in your blood instead of being breathed out. CO<sub>2</sub> 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) — Your brain senses the high CO<sub>2</sub> 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) — 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 (CO<sub>2</sub> narcosis and oxygen-starved (hypoxic) encephalopathy) — High carbon dioxide crosses into your brain and makes you sleepy and confused (CO<sub>2</sub> narcosis). Your breathing muscles (especially the diaphragm and intercostals) get exhausted from working so hard. If CO<sub>2</sub> 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) — 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.

## Symptoms · what the patient feels, and why

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- 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.
- Wheezing (a whistling sound when breathing out): Narrowed, inflamed airways make air squeeze through a smaller space, which vibrates and creates a whistling noise—like air rushing through a narrow gap in a door.
- Unintended weight loss and muscle wasting: The body burns extra calories working so hard to breathe. At the same time, inflammation from COPD releases signals (cytokines) that break down muscle. The person may also feel too tired or short of breath to eat enough.
- Drowsiness or confusion during a flare-up: When the lungs fail to clear out carbon dioxide (the waste gas from breathing), it builds up in the blood and acts like a sedative on the brain, making the person sleepy or confused. This is a sign of respiratory failure.
- Blue-tinged lips or fingernails (cyanosis): The blood doesn't have enough oxygen. Oxygen-poor blood looks dark purple-blue instead of bright red, and you can see this color through thin skin on the lips and under the nails.

## Focused exam

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- Barrel chest (round, puffed-out ribcage): Trapped air pushes the ribs out and holds them there, like a balloon that never fully deflates. This shape tells you the lungs cannot empty all the way.

- Pursed-lip breathing (patient breathes out slowly through almost-closed lips): The patient is creating back-pressure to keep weak airways from collapsing. This is the body's own trick to squeeze trapped air out.
- Use of neck and shoulder muscles to breathe: The diaphragm (the main breathing muscle under the lungs) is flattened by trapped air and cannot pump well. The body recruits backup muscles to keep breathing going, which shows the work of breathing is very high right now.
- Tripod position (patient sits leaning forward on their hands): This position lets the shoulder muscles and chest wall push harder. It is the body's way of borrowing extra breathing power when the lungs are tight.
- Decreased or absent breath sounds on listening with a stethoscope: Air is trapped and cannot move in or out. Silent lungs in COPD mean the airways are blocked or the air is stuck behind swollen, thick walls.
- Drowsiness or confusion in a patient who is still breathing: CO<sub>2</sub> (carbon dioxide, the waste gas we breathe out) builds up when air cannot leave the lungs. Rising CO<sub>2</sub> makes the brain sleepy. This is a red flag that COPD is turning into respiratory failure.

## Diagnostics · every test answers a question

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- Arterial blood gas (ABG) → Is the patient holding on to too much CO<sub>2</sub>, and is the body still able to balance the acid in the blood?
- Chest X-ray → Is there something new on top of COPD that is making the patient worse, like pneumonia, a collapsed lung, or fluid in the lungs?
- Spirometry (breathing test with forced expiration) → How bad is the airway obstruction, and is it reversible with medicine?
- Oxygen saturation (pulse oximetry or ABG) → Is the blood carrying enough oxygen to the tissues?
- B-type natriuretic peptide (BNP) or NT-proBNP → Is shortness of breath from COPD alone, or is the heart failing too?
- Coughed-up mucus (Sputum) culture (if coughed-up mucus is thick, colored, or patient is very sick) → Is there a bacterial infection triggering the COPD flare, and which antibiotic will work?

## Treatment logic

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- 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 CO<sub>2</sub> can escape.

- 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.
- 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.
- Bilevel positive airway pressure (BiPAP or noninvasive ventilation) if CO<sub>2</sub> 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 CO<sub>2</sub> fast without needing a breathing tube down the throat.
- 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.
- Remove the trigger (stop smoking, restart home inhalers, treat infection, move away from dust or fumes) → Removes whatever pushed the patient into crisis, so the body calms down and the breathing system settles back toward baseline instead of spiraling worse.
- Pulmonary rehabilitation and vaccination (influenza yearly, pneumococcal, COVID-19) after acute crisis → Strengthens breathing muscles and teaches energy-saving techniques so the patient can do more with less effort. Vaccines prevent infections that trigger future flares.

## Medications

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### **Albuterol (a short-acting beta-agonist, or SABA) · Short-acting beta<sub>2</sub>-agonist bronchodilator**

Albuterol sticks to tiny switches called beta<sub>2</sub> receptors on the muscle wrapped around your airways. When it flips those switches, the muscle relaxes and the airways spring open wider—like unclenching a fist around a straw—so air can flow in and out more easily.

1. You breathe in the albuterol mist through an inhaler or nebulizer machine.
2. The drug lands on beta<sub>2</sub> receptors sitting on the surface of airway muscle cells.
3. Flipping the receptor wakes up a helper molecule inside the cell called cyclic AMP (a chemical messenger) that tells the muscle to let go.
4. The muscle loosens, the airway opens, and air flows freely again.
5. Relief starts in 5 to 15 minutes and lasts about 4 to 6 hours.

**SAFETY WATCH**

Can make your heart race or cause shaky hands because the drug can reach beta receptors in the heart and muscles. · May lower potassium in the blood if used heavily; important for people already on water pills (diuretics). · Needing rescue albuterol more than twice a week for symptoms is a red flag that COPD is not well controlled and the treatment plan needs to be stepped up.

**Ipratropium (a short-acting muscarinic antagonist, or SAMA) · Short-acting anticholinergic bronchodilator**

Ipratropium blocks acetylcholine (a nerve signal that tells airway muscles to squeeze tight). By blocking that signal, the muscles stay relaxed and the airways stay open—like cutting the pull-cord on a drawstring bag so it cannot cinch closed.

1. You breathe in the ipratropium mist through an inhaler or nebulizer.
2. The drug covers muscarinic receptors on airway muscle cells, blocking the docking spots where acetylcholine normally attaches.
3. Acetylcholine (the squeeze signal from your nerves) cannot attach and cannot trigger muscle contraction.
4. The muscle stays loose, the airway stays open, and breathing gets easier.
5. Peak effect comes in 1 to 2 hours and lasts about 6 to 8 hours.

**SAFETY WATCH**

Can cause dry mouth and a bitter taste. · Rarely, if mist gets into your eyes, it can blur vision or trigger a dangerous eye-pressure spike (narrow-angle glaucoma) in people at risk—keep the mist away from your face and eyes. · May worsen trouble emptying the bladder (urinary retention) in older men with an enlarged prostate.

**Systemic corticosteroids (prednisone or methylprednisolone) · Glucocorticoid (anti-inflammatory steroid)**

Steroids turn down the volume on your immune system's alarm bells. They slip into immune cells—like neutrophils, eosinophils, and lymphocytes (types of white blood cells)—and stop them from releasing the swelling chemicals (cytokines, histamine, leukotrienes) that make airways red, puffy, and clogged with mucus.

1. You swallow a prednisone pill (or receive methylprednisolone through an IV in the hospital during a severe flare-up).
2. The steroid travels through the bloodstream to reach immune cells everywhere, including the lungs.

3. Inside each cell, the steroid binds to a glucocorticoid receptor and moves into the nucleus (the cell's control room).
4. It shuts off genes that make swelling chemicals and turns on genes that calm inflammation.
5. Swelling drops, mucus production slows, and airway walls shrink back toward normal—usually within 24 to 48 hours.
6. Current guidelines (GOLD 2024) recommend a 5-day course (40 mg of prednisone daily for adults) for COPD flare-ups; longer courses add side effects without extra benefit.

**SAFETY WATCH**

Even short 5-day courses can raise blood sugar (a problem for people with diabetes), raise blood pressure, cause trouble sleeping, and trigger mood swings or jitteriness. · Longer or repeated courses increase the risk of infections (steroids suppress your immune defenses), bone thinning (osteoporosis), stomach ulcers, and cataracts (clouding of the eye lens). · Never stop a long steroid course suddenly—your adrenal glands (small glands above the kidneys that make natural steroids like cortisol) need time to wake back up or you can crash into dangerous low blood pressure and shock.

**Antibiotics (azithromycin, amoxicillin-clavulanate, doxycycline, or respiratory fluoroquinolones like levofloxacin or moxifloxacin) · Antibiotic (varies by drug: macrolide, beta-lactam, tetracycline, or fluoroquinolone)**

Antibiotics kill or stop the growth of bacteria that have infected the lungs during a COPD flare-up. Each class attacks bacteria in a different way: beta-lactams (like amoxicillin-clavulanate) punch holes in the bacteria's outer wall so it bursts; macrolides (like azithromycin) jam the bacteria's protein-making machinery (the ribosome); tetracyclines (like doxycycline) also block protein assembly; and fluoroquinolones (like levofloxacin) cut and tangle the bacteria's DNA so it cannot copy itself.

1. You swallow the antibiotic pill (or receive it by IV in the hospital if you are very sick).
2. The drug enters the bloodstream and reaches the infected lung tissue.
3. It attacks the bacteria using its specific mechanism: macrolides block the ribosome; beta-lactams break down the cell wall; tetracyclines jam protein assembly; fluoroquinolones cut and tangle bacterial DNA (using enzymes called topoisomerase and gyrase).
4. Bacteria die or stop multiplying, your immune system clears the debris, and the infection resolves over 5 to 7 days.

**SAFETY WATCH**

Common side effects: nausea, diarrhea (antibiotics also kill helpful bacteria in your gut). · Azithromycin can stretch out the heart's electrical recovery time (QT interval on an EKG) and cause dangerous irregular heartbeats (arrhythmias) in people with heart disease or low potassium or magnesium; get an EKG first if the patient is at risk. · Fluoroquinolones carry FDA black-box warnings for tendon rupture (especially the Achilles tendon in the heel), permanent nerve damage (peripheral neuropathy), and worsening of myasthenia gravis (a muscle-weakness disease); reserve them for severe cases when other antibiotics will not work. · Overuse of antibiotics drives antibiotic resistance—bacteria learn to survive the drugs—so only use them when truly needed based on clinical signs of bacterial infection.

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

BiPAP is a machine that pushes air into your lungs through a snug mask over your nose or nose and mouth. It gives a higher pressure when you breathe in (inspiratory positive airway pressure, or IPAP) to inflate stiff, tired lungs, and a lower pressure when you breathe out (expiratory positive airway pressure, or EPAP) to keep airways from collapsing and to help blow off trapped carbon dioxide (CO<sub>2</sub>, the waste gas your body must get rid of). Think of it as a mechanical helper that takes over part of the work your exhausted breathing muscles—the diaphragm and intercostal muscles (the muscles between your ribs)—cannot do anymore.

1. A tight-fitting mask covers your nose or your nose and mouth; a good seal is essential.
2. The BiPAP machine senses when you start to breathe in and immediately delivers a higher pressure (IPAP, typically 10–20 cm H<sub>2</sub>O) to push air deep into your lungs.
3. When you exhale, the machine drops to a lower pressure (EPAP, typically 4–8 cm H<sub>2</sub>O) so you can push air out more easily, but the pressure stays high enough to keep airways from pinching shut and trapping air.
4. Carbon dioxide blows out more efficiently, oxygen gets in, and your tired muscles rest and recover.
5. Blood oxygen rises, carbon dioxide falls, breathing rate slows, and the risk of needing a breathing tube inserted down your throat (endotracheal intubation) drops by about 50 percent in patients with acute COPD flare-ups complicated by respiratory acidosis (acid building up from trapped carbon dioxide) (high CO<sub>2</sub> making the blood acidic).

**SAFETY WATCH**

The tight mask can rub and cause painful skin sores on the bridge of your nose if worn for many hours; protective dressings and mask adjustments help. · Some people feel trapped or panicked (claustrophobic) with the mask on; coaching, reassurance, and short practice sessions can help you get used to it. · If you vomit while wearing the mask, you can breathe in (aspirate) stomach contents into your lungs, causing a dangerous chemical pneumonia or choking—BiPAP is only safe if you are awake, alert, and can protect your throat and cough effectively. · Air pushed under pressure can also enter the stomach, causing bloating, belching, or—very rarely—stomach rupture; using the lowest effective pressures reduces this risk. · BiPAP is contraindicated (should not be used) in patients with facial trauma, recent facial or airway surgery, uncontrolled vomiting, altered mental status, or inability to clear secretions.

**Controlled oxygen therapy (delivered by nasal cannula or Venturi mask) · Supplemental oxygen therapy**

In COPD, damaged lungs struggle to load oxygen into the blood. Giving extra oxygen raises the oxygen level in the air you breathe—from the normal 21 percent up to 24–28 percent or higher—so even poorly functioning lungs can grab enough oxygen to keep your brain, heart, kidneys, and other vital organs alive. The key word is *\*controlled\**: the dose of oxygen must be carefully adjusted, because too much oxygen can be dangerous in COPD.

1. Oxygen flows through plastic tubing into your nose (nasal cannula, typically 1–6 liters per minute) or through a special mask that mixes precise oxygen percentages with room air (Venturi mask, delivering 24, 28, 31, 35, or 40 percent oxygen).
2. You inhale the oxygen-enriched air into your lungs.
3. In the alveoli, the higher oxygen concentration creates a steeper pressure gradient that pushes more oxygen molecules across the thin alveolar membrane and onto hemoglobin in red blood cells.
4. Oxygen-rich blood travels from the lungs to the left side of the heart, which pumps it out to the brain, kidneys, heart muscle, and every other organ.
5. The goal in acute COPD exacerbations is to keep oxygen saturation ( $\text{SpO}_2$ , measured by a finger clip called a pulse oximeter) between 88 and 92 percent—enough to protect organs but not so high that it shuts off your breathing drive.

**SAFETY WATCH**

In severe COPD, the brain's normal breathing trigger (a rise in CO<sub>2</sub> sensed by chemoreceptors in the brainstem) is blunted because CO<sub>2</sub> has been high for so long. The body switches to a backup alarm: low oxygen (hypoxemia) becomes the main signal that tells the brain to breathe. If you give too much oxygen and push SpO<sub>2</sub> above 92–94 percent, that backup alarm shuts off, breathing slows or stops, and carbon dioxide climbs to dangerous levels, causing confusion, sleepiness, and even coma (this is called oxygen-induced hypercapnia or CO<sub>2</sub> retention). · Always titrate (carefully adjust) supplemental oxygen to a target SpO<sub>2</sub> of 88–92 percent in patients with COPD and risk of CO<sub>2</sub> retention, unless there is another life-threatening reason (like a heart attack or massive bleeding) that demands higher oxygen levels. · Oxygen supports combustion (burning)—keep oxygen equipment away from open flames, cigarettes, candles, and sparks to avoid serious fires. · Dry oxygen flowing for hours can irritate and dry out the inside of your nose and throat; humidifying the oxygen flow helps if you need high flow rates or long-term use. · Venturi masks are preferred over nasal cannulas in acute COPD exacerbations because they deliver a precise, controlled oxygen percentage regardless of how fast or slow the patient breathes.

**Long-acting bronchodilators: LAMA (tiotropium, umeclidinium, glycopyrrrolate, aclidinium) and LABA (formoterol, salmeterol, indacaterol, vilanterol, olodaterol) · Long-acting muscarinic antagonist (LAMA) or long-acting beta<sub>2</sub>-agonist (LABA)**

These inhaled medicines are the backbone of daily COPD treatment for stable disease (not for sudden flare-ups). LAMAs block the squeeze signal (acetylcholine on muscarinic receptors) on airway muscles all day long, and LABAs flip the relax switches (beta<sub>2</sub> receptors) on airway muscles all day long. Together they keep airways open, reduce flare-ups, improve how far you can walk and how much you can do, and improve your quality of life.

1. You inhale the medicine once or twice daily through a dry-powder inhaler or soft-mist inhaler (timing depends on which specific drug you use).
2. The drug binds tightly to its target receptor on airway muscle cells and stays attached for 12 to 24 hours.
3. Airway muscles remain relaxed throughout the day and night, keeping the breathing tubes open even when you sleep.
4. Symptoms—shortness of breath (dyspnea), wheezing, chronic cough—drop, exercise capacity and endurance rise, and the rate of flare-ups (exacerbations) falls by about 15–25 percent compared to no treatment.

**SAFETY WATCH**

LAMAs: dry mouth, trouble starting urination or fully emptying the bladder (urinary retention, especially in older men with enlarged prostates), and—rarely—narrow-angle glaucoma (a painful eye-pressure spike) if mist accidentally gets in the eyes. · LABAs: hand tremor, fast or pounding heartbeat (tachycardia), and low potassium in the blood (hypokalemia)—the same side effects as albuterol but usually milder because inhaled LABAs reach lower levels in the bloodstream. · NEVER use a LABA alone (without an inhaled steroid) in asthma because it can mask worsening inflammation and increase the risk of death, but in COPD using a LABA alone or combined with a LAMA is safe and follows guideline recommendations (GOLD 2024). · If you still need rescue albuterol more than twice a week for symptoms despite taking a LAMA or LABA daily, your COPD is not well controlled—talk to your doctor about stepping up therapy (adding the other class of long-acting bronchodilator or adding an inhaled steroid if you have frequent flare-ups).

**Inhaled corticosteroids: ICS (fluticasone, budesonide, beclomethasone, mometasone) · Inhaled glucocorticoid (anti-inflammatory steroid)**

Inhaled steroids land directly on the lining of your airways and calm the chronic inflammation—swelling, extra mucus, and thickened walls—that drives COPD. Because the drug goes straight to the lungs and very little enters the bloodstream, side effects are far fewer than with steroid pills.

1. You inhale the steroid mist or powder daily (once or twice a day, depending on the medicine).
2. The drug coats the airway walls and slips into cells.
3. Inside each cell, the steroid binds to a glucocorticoid receptor and moves into the nucleus (the control center).
4. It dials down genes that make swelling chemicals—like interleukin-8, tumor tissue death (necrosis) factor, and leukotrienes—and turns up genes that calm inflammation.
5. Over days to weeks, airway walls become less swollen, mucus-making cells (goblet cells) shrink back, mucus production drops, and the rate of flare-ups falls.

**SAFETY WATCH**

Oral thrush (candidiasis)—a white, patchy yeast infection in the mouth and throat that causes soreness. Rinse your mouth with water and spit it out after every dose to wash the steroid off your tongue and gums; using a spacer device with your inhaler also helps reduce mouth deposition. · Hoarse voice or sore throat (dysphonia)—the steroid irritates the vocal cords. Rinsing and using a spacer reduce the risk. · Higher risk of pneumonia (lung infection) in COPD patients on inhaled steroids—the steroid weakens local immune defenses in the lungs, making it easier for bacteria to take hold. The risk is real but small: about 1 extra case of pneumonia per 50 patients treated for one year. Fluticasone seems to carry the highest risk; budesonide may be safer. · Long-term use of high-dose inhaled steroids can thin bones (osteoporosis), raise the risk of cataracts and glaucoma, and—in very high doses—cause some of the same side effects as oral steroids (high blood sugar, easy bruising, thin skin), though far less than pills. · Do NOT reflexively start every COPD patient on an inhaled steroid—use only when guideline criteria (frequent exacerbations or high eosinophils) are met. If a patient has been stable without flare-ups for a year on triple therapy (LAMA + LABA + ICS), consider stepping down by removing the ICS to reduce pneumonia risk.

**Roflumilast · Phosphodiesterase-4 (PDE4) inhibitor (oral anti-inflammatory pill)**

Roflumilast is a pill that blocks an enzyme called phosphodiesterase-4 (PDE4) inside immune cells—especially neutrophils and macrophages (types of white blood cells). Blocking PDE4 raises the level of a helper molecule called cyclic AMP inside those cells, and more cyclic AMP tells the cells to quiet down and stop releasing swelling chemicals (like tumor tissue death (necrosis) factor, interleukin-8, and leukotriene B4). It reduces inflammation from the inside out, especially in patients with chronic bronchitis (daily cough and thick coughed-up mucus (sputum)) and repeated flare-ups.

1. You swallow one 500-microgram tablet once daily (usually in the morning).
2. The drug enters the bloodstream and spreads throughout the body, reaching immune cells in the lungs.
3. It blocks the PDE4 enzyme, cyclic AMP levels rise inside immune cells, and inflammatory signaling drops.
4. Over weeks to months, the frequency of flare-ups (exacerbations) falls by about 15–20 percent in patients with severe COPD (FEV<sub>1</sub> less than 50 percent predicted), chronic bronchitis, and a history of frequent exacerbations despite being on maximal inhaled therapy.

**SAFETY WATCH**

Diarrhea, nausea, stomach pain, and weight loss (often 5–10 pounds) are very common, especially in the first month. To reduce side effects, start at half-dose (250 µg daily) for the first four weeks, then increase to the full 500 µg dose if you tolerate it. · Can worsen depression, anxiety, insomnia, and—rarely—trigger suicidal thoughts or behavior. Do NOT use roflumilast in patients with a history of severe depression, anxiety disorder, or suicidal ideation. Monitor mood closely, especially in the first weeks; stop the drug immediately if mood worsens. · Headache is common (reported by about 1 in 5 patients). · Roflumilast interacts with strong cytochrome P450 enzyme inducers (like rifampin, phenobarbital, carbamazepine, phenytoin) and inhibitors (like erythromycin, ketoconazole, fluvoxamine); adjust the dose or avoid the combination. · Because of the side-effect profile, roflumilast is reserved for severe COPD patients (FEV<sub>1</sub> <50 percent predicted) with chronic bronchitic symptoms (daily productive cough) and repeated exacerbations (≥2 per year) who are already on optimized inhaled therapy (LAMA + LABA ± ICS) and still flaring up.

**Azithromycin (as chronic prevention (prophylaxis), not for treating an acute infection) · Macrolide antibiotic (used here for its anti-inflammatory and immune-modulating effects, not primarily to kill bacteria)**

When taken long-term at a low dose (250 mg three times a week or 500 mg once daily), azithromycin reduces the frequency of COPD flare-ups by calming airway inflammation, thinning mucus so it is easier to cough up, and changing the mix of bacteria living in the lungs (the airway microbiome) in ways that seem to protect against infections. The exact mechanism is still being studied, but it is NOT just about killing germs—azithromycin seems to turn down the volume on the immune system's overreaction and make the airway environment less hospitable to harmful bacteria.

1. You take a low-dose azithromycin pill three times a week—a common schedule is 250 mg every Monday, Wednesday, and Friday, or 500 mg once daily.
2. The drug enters the bloodstream and accumulates in lung tissue over weeks, reaching concentrations far higher in the lungs than in the blood.
3. Azithromycin reduces the release of inflammatory chemicals (like interleukin-8 and neutrophil elastase), decreases the stickiness and thickness of mucus by interfering with mucin production, and alters bacterial populations in the airways.
4. Over months, inflammation markers drop, mucus becomes thinner and easier to clear, harmful bacteria are suppressed, and the rate of flare-ups falls by about 25 percent in carefully selected patients.

**SAFETY WATCH**

Hearing loss (ototoxicity)—usually reversible if caught early, but can be permanent. Get a baseline hearing test (audiogram) before starting chronic azithromycin and repeat it every 6–12 months while on therapy. Stop the drug immediately if hearing worsens or ringing in the ears (tinnitus) develops. · QT prolongation—azithromycin can stretch out the heart's electrical recovery time (the QT interval on an EKG), which can trigger life-threatening irregular heartbeats (torsades de pointes) and sudden cardiac death. Get a baseline EKG before starting; avoid azithromycin in patients with a long QT interval, low potassium or magnesium, or who take other QT-prolonging drugs (like amiodarone, sotalol, certain antipsychotics, or methadone). · Diarrhea, nausea, and stomach upset are common because macrolides also kill helpful bacteria in the gut. · Antibiotic resistance—long-term azithromycin can select for resistant bacteria, especially macrolide-resistant *Streptococcus pneumoniae* (a common pneumonia germ) and nontuberculous mycobacteria (NTM, slow-growing bacteria related to TB that cause chronic lung infections). Before starting chronic azithromycin, test for NTM infection if there is any suspicion based on symptoms (chronic cough, weight loss, night sweats) or chest imaging (nodules, cavities, or bronchiectasis—damaged, dilated airways). Do NOT use azithromycin prevention (prophylaxis) if NTM is present, because it will breed resistance and make NTM nearly impossible to treat. · Liver enzyme elevations (usually mild and reversible). · Current guidelines (GOLD 2024, ATS/ERS) reserve chronic azithromycin for FORMER smokers (not current smokers) with optimized inhaled therapy (LAMA + LABA ± ICS) who still have at least one flare-up (exacerbation) per year requiring oral steroids or antibiotics. Duration of therapy is usually at least one year; reassess benefit, side effects, and resistance risk every 6–12 months and consider stopping if exacerbations have been controlled for a year.

**Red flags**

- 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)

## Nursing priorities

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1. Start controlled oxygen by nasal cannula to keep oxygen saturation (SpO<sub>2</sub>) between 88 and 92 percent, not higher. — COPD patients breathe because of low oxygen, not high CO<sub>2</sub> like healthy people. Too much oxygen (above 92 percent) shuts off their breathing drive, CO<sub>2</sub> 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. — 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 CO<sub>2</sub> and respiratory failure: confusion, extreme sleepiness, or very slow breathing. Check ABG (arterial blood gas) if patient becomes drowsy. — When lungs cannot blow out CO<sub>2</sub>, it dissolves in blood and makes acid (respiratory too much acid in the blood (acidosis)). High CO<sub>2</sub> also acts like a sedative on the brain. If CO<sub>2</sub> goes above 50 mmHg with drowsiness, the patient needs BiPAP or a breathing tube to survive.
4. Sit the patient fully upright at 90 degrees or let them lean forward with arms on the bedside table (tripod position). — Sitting upright lets the diaphragm (main breathing muscle) drop down and pull in more air. Leaning forward braces the shoulder and neck muscles so they can help lift the ribcage. Lying flat makes the belly push up on the diaphragm and worsens shortness of breath.
5. Give prednisone 40 mg by mouth once daily or methylprednisolone 125 mg IV if the patient cannot swallow, for 5 days total. — Corticosteroids (steroids) stop inflammation (swelling and mucus) inside the airways. Inflammation makes airways narrow and fills them with sticky mucus. Steroids shrink this swelling in hours and cut hospital stay and treatment failure in half.
6. Encourage the patient to cough and spit out mucus into a tissue or cup. If mucus is thick and stuck, give nebulized saline or help with chest percussion (gentle tapping on the back). — Mucus plugs block airways and trap air, making it even harder to breathe out. Clearing mucus opens airways and lowers the work of breathing. Thick mucus also grows bacteria and causes infection.
7. Start antibiotics (azithromycin 500 mg day one, then 250 mg daily, or amoxicillin-clavulanate 875 mg twice daily) if mucus is green, yellow, or brown, or if patient has fever. — Bacterial infection is present in about half of COPD flare-ups. Bacteria release toxins that increase inflammation and mucus production. Antibiotics shorten symptoms by 1 to 2 days and prevent treatment failure when infection is present.

8. Monitor respiratory rate, oxygen saturation, and mental status every 15 to 30 minutes until the patient improves. Set up BiPAP machine at bedside if CO<sub>2</sub> is rising or patient is very drowsy. — COPD patients can tire out fast because breathing takes huge energy. If breathing rate suddenly slows or the patient gets quiet and sleepy, muscles are failing and respiratory arrest (breathing stops) is minutes away. BiPAP pushes air in and supports tired muscles.

## Sources

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- GOLD 2024 Global Initiative for Chronic Obstructive Lung Disease
- ATS/ERS COPD Guidelines
- CDC Chronic Obstructive Pulmonary Disease
- Cochrane Airways Group

Last reviewed: 2026-07-07