

COPD · Chronic Obstructive Pulmonary Disease

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, breathing in takes muscle work — your diaphragm (the big sheet of muscle under your lungs) pulls down and opens your chest so air rushes in. Breathing out is easy and automatic: the diaphragm relaxes, your lungs spring back like a rubber band, and air flows out without any work. The airways stay open because the stretchy lung tissue around them holds them up like tent stakes. In COPD, years of cigarette smoke or other irritants damage the lungs in two big ways. First, the airways get swollen and thick on the inside, and they make extra sticky mucus that clogs them up. Second, the stretchy lung tissue breaks down (we call that emphysema), so the airways lose their support and collapse shut when you try to breathe out. Now air can get in, but it gets trapped inside. A flare-up happens when something — a cold, the flu, air pollution, or skipping your medicine — makes everything worse all at once. The airways squeeze shut even tighter. Air piles up in the lungs and cannot escape. The diaphragm gets pushed flat and cannot work well anymore. Carbon dioxide (the waste gas you are supposed to breathe out) builds up in the blood, and oxygen (the gas you need) cannot get in fast enough. Your body fights back by breathing faster and harder, and your heart races to pump more blood, but because the air is stuck, none of that helps much. You can see someone working so hard to breathe but barely moving any air. That is the emergency.

What you might feel

- Breathing out slowly through pursed lips (like blowing out a candle)
- Neck and shoulder muscles working hard to breathe
- Barrel chest (the chest looks round and fat, like a barrel)
- Sitting leaning forward with hands on knees (tripod position)
- Chronic cough that brings up mucus (sputum), especially in the morning
- Shortness of breath that gets worse with activity
- Wheezing (a whistling sound when breathing out)
- Unintended weight loss and muscle wasting

- Drowsiness or confusion during a flare-up
- Blue-tinged lips or fingernails (cyanosis)

Call the doctor right away if...

- 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)

What the care team will do

- Start controlled oxygen by nasal cannula to keep oxygen saturation (SpO2) between 88 and 92 percent, not higher.
- 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.
- 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.
- Sit the patient fully upright at 90 degrees or let them lean forward with arms on the bedside table (tripod position).
- Give prednisone 40 mg by mouth once daily or methylprednisolone 125 mg IV if the patient cannot swallow, for 5 days total.
- 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).
- 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.
- Monitor respiratory rate, oxygen saturation, and mental status every 15 to 30 minutes until the patient improves. Set up BiPAP machine at bedside if CO2 is rising or patient is very drowsy.

Medicines you may be given — and why

Albuterol (a short-acting beta-agonist, or SABA)

Albuterol sticks to tiny switches called beta₂ 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.

Ipratropium (a short-acting muscarinic antagonist, or SAMA)

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.

Systemic corticosteroids (prednisone or methylprednisolone)

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.

Antibiotics (azithromycin, amoxicillin-clavulanate, doxycycline, or respiratory fluoroquinolones like levofloxacin or moxifloxacin)

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.

Non-invasive ventilation: BiPAP (bilevel positive airway pressure)

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 (CO₂, 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.

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

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.

Long-acting bronchodilators: LAMA (tiotropium, umeclidinium, glycopyrrolate, aclidinium) and LABA (formoterol, salmeterol, indacaterol, vilanterol, olodaterol)

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 (beta₂ 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.

Inhaled corticosteroids: ICS (fluticasone, budesonide, beclomethasone, mometasone)

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.

Roflumilast

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.

Azithromycin (as chronic prevention (prophylaxis), not for treating an acute infection)

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.

Where this information comes from

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

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