

COPD Exacerbation

A plain-English guide for you and your family

WHAT IS HAPPENING

People with COPD have lungs that are permanently damaged. The small airways are scarred and floppy, and many of the tiny air sacs (where oxygen goes into the blood) are destroyed. Because of this damage, their lungs cannot push air out well, so stale air with carbon dioxide (CO₂) gets trapped inside. Over months and years, their body adjusts to living with high CO₂ by raising bicarbonate (a chemical that keeps blood from getting too acidic). Their brain also switches to using low oxygen—not high CO₂—as the main signal to breathe. This is their new normal. But when something triggers a flare-up (most often a cold, flu, or lung infection, sometimes air pollution or a blood clot in the lung), the already-narrow airways swell even more, fill with thick mucus, and squeeze tight. Now air cannot get out at all. CO₂ climbs higher, oxygen drops lower, and the patient has to work much harder to breathe. The chest puffs up with trapped air, the breathing muscle under the lungs (the diaphragm) flattens and tires out, and the patient takes fast, shallow breaths that move very little air. Blood vessels in the lungs clamp down because of low oxygen, forcing the right side of the heart to pump harder. If this goes on too long without treatment, the patient can become exhausted, confused from high CO₂, and even stop breathing. Every strange finding you see—barrel chest, wheezing, using neck muscles to breathe, swollen ankles—points back to this one problem: air trapped in damaged lungs.

What you might feel

- Breathing gets harder or faster than usual
- More coughing than usual
- Coughed-up mucus (Sputum) (the mucus you cough up) changes color to yellow, green, or brown, or you cough up more of it
- Chest feels tight
- Wheezing (a whistling sound when you breathe)
- Using neck and shoulder muscles to breathe, or belly moving in and out a lot
- Breathing out takes much longer than usual, or you purse your lips (make them tight) when you breathe out

- Feeling more tired than usual, or confused

Call the doctor right away if...

- New confusion or can't stay awake
- Lips or fingernails turn blue or gray
- Chest pain
- Breathing is so hard you can't speak a full sentence
- Using neck and shoulder muscles to breathe, or sitting and leaning forward to breathe

What the care team will do

- Position the patient sitting upright at 60 to 90 degrees, leaning slightly forward with arms resting on the overbed table.
- Start controlled oxygen therapy with a nasal cannula or Venturi mask, targeting oxygen saturation (SpO₂) of 88 to 92 percent, and monitor closely for decreased respiratory rate or increased sleepiness.
- Administer nebulized albuterol (a short-acting beta-agonist that opens airways) and ipratropium (a short-acting anticholinergic that also opens airways) together, usually every 4 to 6 hours or more often if ordered.
- Give systemic corticosteroids (prednisone 40 mg by mouth daily for 5 days, or IV methylprednisolone if the patient cannot swallow) as ordered.
- Monitor respiratory rate, oxygen saturation, heart rate, and level of consciousness every 1 to 2 hours, and notify the provider immediately if the patient becomes confused, very sleepy, or SpO₂ drops below 88 percent despite oxygen.
- Encourage the patient to cough and deep breathe every 1 to 2 hours, and help them perform controlled coughing (huff cough technique: take a deep breath, hold it, then make a 'huff' sound to push air out).
- Ensure the patient stays hydrated by encouraging oral fluids (at least 1500 to 2000 mL per day if not fluid-restricted) or give IV fluids as ordered.
- Administer antibiotics (azithromycin, doxycycline, or amoxicillin-clavulanate) as ordered if the patient has at least 2 of these: worse shortness of breath, more coughed-up mucus (sputum), or pus-filled (purulent) (yellow-green) coughed-up mucus.

Medicines you may be given — and why

Albuterol (short-acting beta-2 agonist, SABA)

Albuterol attaches to tiny switches (called beta-2 receptors) on the muscle wrapped around the breathing tubes in your lungs. When it flips these switches, the muscle relaxes and the tubes open wider, making it easier to breathe.

Ipratropium (short-acting muscarinic antagonist, SAMA)

Ipratropium blocks a natural signal (called acetylcholine) that normally tells the airway muscles to squeeze tight. By blocking that signal, the muscles stay relaxed and the breathing tubes stay open.

Prednisone or methylprednisolone (systemic corticosteroid)

Corticosteroids are man-made versions of a natural hormone your body uses to calm down inflammation. They turn down the volume on the immune cells that are causing swelling, mucus, and irritation in the airways, which helps the flare-up settle faster.

Azithromycin, amoxicillin-clavulanate, or doxycycline (antibiotic)

Antibiotics kill or stop the growth of bacteria that may have triggered the COPD flare-up. They work by breaking down the bacteria's cell wall, blocking its ability to make proteins, or stopping it from copying its DNA – different drugs use different tactics.

Supplemental oxygen

Extra oxygen is given to raise the oxygen level in your blood when your damaged lungs can't pull in enough on their own. The goal is a safe range (88–92% oxygen saturation), NOT 100%, because too much oxygen can be harmful in COPD.

Bilevel positive airway pressure (BiPAP or NIPPV)

BiPAP is a mask that fits over your nose or face and pushes air into your lungs with gentle pressure. It helps you breathe in (with higher pressure) and breathe out (with lower pressure), making it easier to move air and blow off excess carbon dioxide when your lungs are too tired or blocked to do it on their own.

Where this information comes from

- GOLD 2024 Report
- AARC Clinical Practice Guideline on Noninvasive Ventilation
- American Thoracic Society
- NEJM COPD Review

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