

COPD Exacerbation

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THE STORY IN ONE SENTENCE

The patient's airways are already narrow from long-term damage, and now something (usually an infection) has made them even tighter and filled with mucus.

“COPD flare-up (exacerbation) — an infection or other trigger suddenly worsens the already-narrowed, damaged airways in chronic obstructive pulmonary disease, trapping air and raising CO₂.”

Normal anatomy — the body's job

In healthy lungs, air flows easily in and out through open, stretchy airways. Tiny air sacs (alveoli) at the end of each airway grab oxygen from the air you breathe in and dump carbon dioxide (CO₂) into the air you breathe out. The diaphragm (the big dome-shaped muscle under your lungs) pulls down to let air in, then relaxes so the lungs spring back and push air out. The airways have elastic walls that keep them open, and a thin layer of mucus traps any dust or germs so tiny hairs (cilia) can sweep them out. Your brain watches the CO₂ level in your blood and tells you to breathe faster when CO₂ goes up. In COPD, this system is already broken before any flare-up (exacerbation) happens.

BODY'S JOB

The lungs' job is to trade gases: pull oxygen into the blood and push carbon dioxide out. The airways must stay open and elastic so air can flow in and out smoothly. The diaphragm and chest muscles do the work of breathing, and the brain fine-tunes how fast and deep you breathe based on CO₂ and oxygen levels. In COPD, years of smoking or other lung irritants have destroyed the elastic walls of the airways and air sacs, leaving floppy, narrow tubes and fewer working gas-exchange surfaces. The patient's baseline is already high CO₂, low oxygen, and a breathing pattern driven by oxygen sensors (because CO₂ sensors have reset). Any worsening from this fragile baseline tips the person into crisis.

1. Air flows in through the nose or mouth, down the windpipe (trachea), and into branching airways (bronchi and bronchioles) that spread through both lungs like an upside-down tree.

2. At the tips of the smallest airways sit clusters of tiny air sacs (alveoli), wrapped in blood vessels so thin that oxygen can jump from air into blood and CO₂ can jump from blood into air.
3. The diaphragm contracts (pulls down) to make the chest bigger and suck air in; then it relaxes and the elastic lungs recoil (spring back) to push air out—breathing out is normally passive, not hard work.
4. Mucus and cilia line the airways to catch and sweep out dust and germs, keeping the air passages clean.
5. The brain's breathing center in the brainstem watches CO₂ in the blood; when CO₂ rises, it signals you to breathe faster and deeper to blow it off.
6. In COPD, the airways are permanently narrowed by scarring and inflammation, the elastic tissue is destroyed (so lungs do not recoil well), and many alveoli are gone (less surface for gas exchange). The baseline is already high CO₂ (chronic respiratory acidosis (acid building up from trapped carbon dioxide)) with kidneys holding extra bicarbonate to keep pH near normal (compensated). The brain now relies mostly on low oxygen to trigger breathing, because the CO₂ level has been high for so long that the brainstem has adapted to it.

What breaks

In a COPD flare-up (exacerbation), something irritates or infects the already-damaged airways, making them swell, fill with thick mucus, and squeeze tight. Now the patient cannot blow air out, so stale air with CO₂ gets trapped inside and oxygen cannot get in. The most common triggers are respiratory infections (viruses like rhinovirus, influenza, or RSV; bacteria like *Haemophilus influenzae*, *Streptococcus pneumoniae*, or *Moraxella catarrhalis*). Other triggers include breathing in air pollution, cigarette smoke, cold air, or having a blood clot travel to the lung (pulmonary a traveling clot lodging in a vessel (embolism)). Sometimes no clear trigger is found. The key break is that the baseline narrow, floppy airways—already barely able to let air out—now become even more obstructed, and the patient's breathing muscles cannot keep up with the extra work.

CLINICAL TERM

Acute flare-up (exacerbation) of COPD: an acute worsening of respiratory symptoms (increased trouble breathing (dyspnea), cough, coughed-up mucus (sputum) volume or purulence) beyond normal day-to-day variation, requiring a change in medication. The pathophysiology is increased airway inflammation, mucus hypersecretion, bronchospasm, and dynamic hyperinflation superimposed on fixed airflow obstruction. Triggers include viral or bacterial respiratory infection (most common), environmental irritants, pulmonary embolism (a clot lodging in a lung artery), pneumothorax, heart failure, or medication nonadherence. The result is worsening ventilation-blood flow (perfusion) mismatch, low oxygen in the blood (hypoxemia), hypercapnia, and increased work of breathing that can lead to respiratory muscle fatigue and acute-on-chronic respiratory failure.

How the body compensates

The body tries to keep oxygen delivery and pH stable even as the lungs fail. The patient breathes faster, uses neck and shoulder muscles to help, and the kidneys hold onto bicarbonate to buffer the rising CO₂. Blood vessels in under-oxygenated parts of the lung squeeze down to shunt blood toward better-ventilated areas. The right heart pumps harder to push blood through those squeezed vessels. For a while, vital signs and mental status can look almost normal—this is the dangerous window where the patient is working hard but not yet crashing. Spotting this compensation (pursed-lip breathing, using accessory muscles, mild confusion, rising CO₂ on blood gas) is the key to preventing full respiratory failure.

1. The patient breathes faster (tachypnea) to try to move more air and blow off CO₂, even though each breath is shallow because the diaphragm is weak and the lungs are over-inflated.
2. The patient uses accessory muscles (neck, shoulders, abdominal muscles) and leans forward with hands on knees (tripod position) to help the tired diaphragm push air out.
3. The patient purses their lips when breathing out (pursed-lip breathing) to keep a little back-pressure in the airways, preventing them from collapsing and trapping even more air.
4. The kidneys retain bicarbonate over days to weeks to keep blood pH from dropping too low (metabolic compensation for chronic respiratory acidosis (acid building up from trapped carbon dioxide)). In an acute flare-up (exacerbation), CO₂ rises faster than the kidneys can compensate, so pH starts to fall.
5. Blood vessels in poorly ventilated parts of the lung clamp down (hypoxic pulmonary vasoconstriction) to redirect blood toward areas still getting oxygen, but this raises pressure in the lung circulation (pulmonary high blood pressure (hypertension)).

6. The right side of the heart works harder to pump blood through the high-pressure lung vessels. Over time this can cause right heart strain or failure (cor pulmonale), seen as swollen ankles, a large liver, and bulging neck veins.
7. The body tolerates low oxygen by increasing heart rate and red-blood-cell production (polycythemia), but there is a limit—severe low oxygen in the blood (hypoxemia) causes confusion, arrhythmias, and organ damage.
8. Warning: compensation can hide how sick the patient really is. A normal or near-normal oxygen saturation (especially on home oxygen), normal blood pressure, and mild confusion can all be present while the patient is minutes to hours from respiratory arrest. The crash points to watch for are: (1) rising CO₂ with falling pH (below 7.25–7.30) despite noninvasive ventilation (BiPAP), or worsening drowsiness (CO₂ narcosis); (2) sudden calm after agitation, with a slow respiratory rate and falling oxygen—this is pre-arrest exhaustion, not improvement. Both mean the patient needs intubation and mechanical ventilation immediately.

Domino effect

1. A trigger (infection, pollution, or pulmonary embolism (a clot lodging in a lung artery)) hits already-damaged airways. — The most common trigger is a viral or bacterial respiratory infection that inflames the airway lining, but any irritant or added lung stress can tip a fragile system into crisis.
2. Airways swell, fill with thick mucus, and squeeze tight (bronchospasm). — Inflammation brings more fluid and immune cells into the airway walls (making them thicker), mucus glands pour out thick secretions (clogging the passage), and smooth muscle contracts (narrowing the tube even more).
3. Air gets trapped in the lungs because breathing out through narrow airways is too slow. — It takes longer to push air out through a narrow, floppy tube, so the patient does not finish exhaling before the next breath in—leftover air stacks up breath after breath (dynamic hyperinflation).
4. The chest barrel-shapes and the diaphragm flattens. — Trapped air over-inflates the lungs like a balloon that never fully deflates, pushing the ribs out and the diaphragm down into a flat, weak position instead of its normal dome.
5. The diaphragm and other breathing muscles tire out from working at a bad angle and against high resistance. — A flattened diaphragm cannot contract efficiently, and pushing air through swollen, mucus-filled airways is like breathing through a narrow straw—both exhaust the muscles quickly.

6. The patient shifts to rapid, shallow breathing and uses neck and shoulder muscles to help.
— Shallow breaths take less work per breath than deep ones when the diaphragm is weak, and recruiting accessory muscles buys a little more time—but shallow breathing moves air mostly in the dead space (mouth, throat, large airways) and very little reaches the alveoli for gas exchange.
7. Oxygen drops (hypoxemia) and carbon dioxide rises (hypercapnia). — Shallow breathing and mucus-plugged airways mean fresh air does not reach the alveoli to load oxygen, and stale air full of CO₂ cannot be pushed out, so both gases go the wrong way in the blood.
8. Blood pH falls (worsening too much acid in the blood (acidosis)) because CO₂ is an acid when dissolved in blood. — The kidneys have been holding extra bicarbonate for years to buffer chronic high CO₂, but in an acute flare-up (exacerbation) CO₂ climbs faster than the kidneys can keep up, so pH drops—acidic blood impairs heart rhythm, brain function, and muscle strength.
9. Low oxygen tells lung blood vessels to squeeze down (hypoxic pulmonary vasoconstriction), raising pressure in the lung circulation. — This reflex normally redirects blood to better-oxygenated lung areas, but in COPD flare-up (exacerbation) large areas are poorly oxygenated, so widespread vasoconstriction raises the pressure the right heart must pump against (pulmonary high blood pressure (hypertension)).
10. The right side of the heart strains and can fail (acute cor pulmonale). — The right ventricle is a thin-walled pump built for low resistance; pumping against high lung pressure makes it swell, work inefficiently, and eventually back up blood into the body (causing swollen legs, liver, and neck veins).
11. The patient becomes confused, drowsy, or combative from high CO₂ (hypercapnic encephalopathy or CO₂ narcosis). — CO₂ crosses into the brain and acts like a sedative at high levels, slowing thinking and reflexes—this is a medical emergency because it means the brain's drive to breathe is failing.
12. If untreated, the patient stops fighting, breathing slows or stops, and cardiac arrest follows (respiratory arrest). — Exhausted muscles, severe too much acid in the blood (acidosis), and lack of oxygen to the heart and brain shut down the body's ability to keep breathing and pumping blood—without immediate intubation and ventilation, the patient will die.

Symptoms · what the patient feels, and why

- Breathing gets harder or faster than usual: The airways are squeezed tighter and filled with more mucus than normal, so air can't flow in and out easily. Your body tries to breathe faster to get enough oxygen.
- More coughing than usual: The airways are irritated and filled with extra mucus. Your lungs try to clear it out by coughing.

- Coughed-up mucus (Sputum) (the mucus you cough up) changes color to yellow, green, or brown, or you cough up more of it: When bacteria grow in the mucus, white blood cells rush in to fight them. Those cells and dead bacteria turn the mucus yellow or green.
- Chest feels tight: The muscles around your airways squeeze down, and the airways themselves swell. This makes your chest feel like a band is wrapped around it.
- Wheezing (a whistling sound when you breathe): Air is being pushed through narrowed, swollen airways. The narrow space makes the air vibrate and whistle.
- Using neck and shoulder muscles to breathe, or belly moving in and out a lot: Your main breathing muscle (the diaphragm) can't keep up alone, so extra muscles in your neck, shoulders, and belly join in to help move air.
- Breathing out takes much longer than usual, or you purse your lips (make them tight) when you breathe out: Air gets trapped in your lungs because the airways collapse or close too early. Pursing your lips keeps a little pressure in the airways so they stay open longer and you can push the air out.
- Feeling more tired than usual, or confused: Your blood isn't carrying enough oxygen to your brain and muscles, or carbon dioxide is building up in your blood because you can't breathe it out well. Both make you feel foggy and weak.

Focused exam

- Pursed-lip breathing (lips puckered tight when breathing out): The patient is trying to keep airways propped open longer so trapped air can escape. This tells you the airways are collapsing and badly clogged right now.
- Using neck and belly muscles to breathe (accessory muscle use): The diaphragm is weak and pushed flat because air is trapped in the lungs. When the body recruits helper muscles, it means breathing is extremely hard work and the patient is tiring out.
- Barrel chest (chest puffed up and round like a barrel): Air stays stuck in the lungs because damaged airways collapse when the patient tries to push it out. The chest stays inflated like a balloon that will not deflate. This is chronic hyperinflation (too much air trapped) from long-standing COPD.
- Coughed-up mucus (Sputum) that is thick, yellow, or green instead of clear: Bacteria or a virus infected the already-damaged airways and triggered this flare-up. Colored coughed-up mucus (sputum) means white blood cells are fighting the infection and died, turning the mucus yellow or green.
- Low oxygen saturation (SpO₂ below 88 percent on room air): The airways are so clogged that oxygen cannot get into the blood and carbon dioxide cannot get out. This tells you the lungs are failing to do their job of gas exchange right now.

- Prolonged expiration (breathing out takes much longer than breathing in): The airways collapse when the patient tries to push air out. A long, slow exhale is the body's attempt to keep airways open long enough to empty the lungs. The longer the exhale, the worse the obstruction.
- Drowsiness, confusion, or altered mental status: Carbon dioxide is rising so high in the blood that it is acidifying the brain and making it work poorly. This is a crash warning that the patient may stop breathing soon and need a breathing machine.

Diagnostics · every test answers a question

- Arterial blood gas (ABG) → Is the patient building up too much carbon dioxide and acid in the blood because breathing is failing? Is this a new problem or an old problem getting worse?
- Chest X-ray → Is there pneumonia, a collapsed lung (pneumothorax), fluid around the lungs (pleural a fluid collection (effusion)), or heart failure triggering this flare-up (exacerbation)?
- Electrocardiogram (ECG or EKG) → Is the right side of the heart being strained because the lungs are so sick? Is the patient also having a heart attack (myocardial tissue death from blocked blood flow (infarction)) at the same time?
- Complete blood count (CBC) → Is there a bacterial infection driving this flare-up (exacerbation)? Is the patient's blood thick from chronic low oxygen?
- Procalcitonin → Does this patient need antibiotics, or is the trigger a virus or pollution instead of bacteria?
- Basic metabolic panel (BMP or Chem-7) → Are the kidneys trying to fix the acid problem? Is the patient dehydrated from working so hard to breathe?
- Brain natriuretic peptide (BNP) if heart failure is suspected → Is shortness of breath from COPD flare-up (exacerbation), heart failure, or both together?

Treatment logic

- Nebulized short-acting beta-agonist (albuterol) plus short-acting anticholinergic (ipratropium) → Relaxes the smooth muscle squeezing the airways so air can move in and out again. Opens the tubes within minutes and gets oxygen up and carbon dioxide down faster. This is the fastest way to relieve airway obstruction.
- Systemic corticosteroids (prednisone 40 mg by mouth daily for 5 days, or equivalent IV dose) → Calms down the inflammation and swelling inside the airway walls and reduces mucus production. Keeps the airways more open for days so the patient can breathe easier and recovers faster. Cuts hospital stay by about 1 day and lowers the chance of treatment failure.

- Antibiotics (azithromycin, doxycycline, or amoxicillin-clavulanate) if at least 2 of these are present: worse trouble breathing (dyspnea), more coughed-up mucus (sputum), or pus-filled (purulent) (thick yellow or green) coughed-up mucus → Kills the bacteria causing the infection so the inflammation and mucus production stop getting worse. Shortens recovery time and lowers the chance the patient will come back to the hospital.
- Controlled oxygen therapy to target SpO₂ 88 to 92 percent (NOT higher) → Raises oxygen in the blood just enough so organs get what they need and the body works less hard. Keeps a little bit of low oxygen so the brain's breathing center stays awake and reminds the patient to keep breathing.
- Noninvasive positive-pressure ventilation (BiPAP or CPAP) if pH is below 7.35, PaCO₂ is above 45 mmHg, and respiratory rate is above 25 despite initial treatment → Pushes air into the lungs so the patient does not have to work so hard and carbon dioxide escapes more easily. Lowers the need for intubation (breathing tube) by about half and cuts mortality. Buys time while medications start working.
- Smoking cessation counseling and nicotine replacement (patch, gum, or lozenge) → Stops the daily damage to airways so COPD slows down and future exacerbations become less frequent. This is the single most effective intervention to change the course of COPD long-term.
- Pulmonary rehabilitation referral before discharge → Teaches breathing techniques and builds strength so the patient can do more with less shortness of breath. Lowers hospital readmissions and improves quality of life over months.

Medications

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

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.

1. You breathe in the albuterol mist through a nebulizer or inhaler.
2. The drug lands on beta-2 receptor switches sitting on the surface of the airway muscle cells.
3. When albuterol locks onto the receptor, it starts a chain reaction inside the cell that raises a molecule called cyclic AMP (cAMP).
4. Higher cAMP tells the muscle to relax and stop squeezing.
5. The airway opens wider, air flows more easily, and you can breathe better within minutes.

SAFETY WATCH

Can make your heart race or feel like it's pounding (called a fast heart rate (tachycardia)).
· May cause shaky hands (tremor), especially at higher doses. · Can lower potassium levels in the blood if used repeatedly in high doses, which can affect heart rhythm. · Use carefully in people with heart disease or irregular heartbeat – the drug can stress the heart. · Too much albuterol can make you feel jittery or anxious.

Ipratropium (short-acting muscarinic antagonist, SAMA) · Bronchodilator - anticholinergic (muscarinic antagonist)

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.

1. You breathe in the ipratropium mist through a nebulizer or inhaler.
2. The drug lands on muscarinic receptor switches on the airway muscle cells.
3. Normally, a nerve chemical called acetylcholine locks onto these receptors and tells the muscle to squeeze and the glands to make more mucus.
4. Ipratropium sits on the receptor and blocks acetylcholine from getting through – like putting a cap on a plug.
5. With the signal blocked, the muscle stays relaxed and less mucus is made, so the airways stay open and clearer.

SAFETY WATCH

Can cause dry mouth because it reduces saliva production. · May blur your vision or cause eye pain if the mist accidentally sprays into your eyes – always use a mouthpiece, not a face mask, if possible. · Can make it harder to urinate (urinary retention), especially in older men with prostate problems. · Rarely can trigger narrow-angle glaucoma (a type of eye pressure problem) if mist gets in the eyes. · Generally very safe with few heart effects, unlike albuterol.

Prednisone or methylprednisolone (systemic corticosteroid) · Corticosteroid (anti-inflammatory hormone)

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.

1. You swallow a pill (prednisone by mouth) or receive an injection or IV dose (methylprednisolone) if you can't swallow.

2. The drug travels through your bloodstream and enters cells all over your body.
3. Inside the cell, it binds to a receptor and moves into the cell's control center (the nucleus).
4. It tells the cell to stop making chemicals that cause inflammation (like cytokines and prostaglandins) and to make proteins that calm things down.
5. Over hours to days, swelling in the airways shrinks, mucus production drops, and breathing becomes easier.

SAFETY WATCH

Short courses (5 days) are generally safe, but even brief use can raise blood sugar, so watch glucose levels closely in people with diabetes. · Can cause trouble sleeping (insomnia), mood swings, or increased appetite. · May raise blood pressure temporarily. · Can increase the risk of stomach irritation or ulcers, especially if taken with NSAIDs like ibuprofen. · Long-term or repeated use can weaken bones, suppress the immune system, or cause weight gain – but a single 5-day course rarely causes these problems. · Do NOT stop suddenly after weeks of use – the dose must be tapered – but a 5-day burst usually does not require a taper.

Azithromycin, amoxicillin-clavulanate, or doxycycline (antibiotic) · Antibiotic (kills or stops bacteria from growing)

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.

1. You swallow the antibiotic pill (or receive it by IV if very sick).
2. The drug travels through your bloodstream and reaches the infected lung tissue.
3. It enters or attacks the bacteria: azithromycin stops the bacteria from making proteins they need to survive; amoxicillin-clavulanate breaks open the bacteria's protective wall; doxycycline blocks the bacteria from reading its genetic instructions.
4. The bacteria die or stop multiplying.
5. Your immune system cleans up the dead bacteria, and the infection-driven inflammation calms down.

SAFETY WATCH

Azithromycin can rarely cause irregular heart rhythms (QT prolongation), especially in people with existing heart problems or low potassium/magnesium. · Amoxicillin-clavulanate can cause diarrhea or stomach upset; rarely causes liver irritation. · Doxycycline can make your skin very sensitive to sunlight (photosensitivity) – wear sunscreen and avoid tanning beds. · All antibiotics can cause allergic reactions – rash, hives, or in rare cases severe swelling or trouble breathing (anaphylaxis). · Overuse or unnecessary use of antibiotics can lead to antibiotic-resistant bacteria, making future infections harder to treat.

Supplemental oxygen · Medical gas therapy

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.

1. You breathe in a higher concentration of oxygen through a nasal tube (nasal cannula) or face mask.
2. The extra oxygen fills the air sacs in your lungs.
3. More oxygen molecules cross the thin membrane into your blood and attach to hemoglobin.
4. Your blood oxygen level (SpO₂) rises into the target range of 88–92%.
5. Your organs and tissues get enough oxygen to function without the blood oxygen going dangerously high.

SAFETY WATCH

Always target oxygen saturation of 88–92% in COPD patients, NOT the normal 94–98% or 100% – over-oxygenation can cause CO₂ retention (hypercapnia) and make breathing failure worse. · Monitor CO₂ levels with blood gas tests if the patient is very sick or getting worse. · Oxygen supports combustion – keep away from open flames, smoking, or sparks. · Dry oxygen can irritate the nose and throat – use a humidifier if needed. · Long-term oxygen therapy is only prescribed for patients with chronic low oxygen (PaO₂ ≤55 mmHg or SpO₂ ≤88% at rest); it does NOT help everyone and should not be used casually.

Bilevel positive airway pressure (BiPAP or NIPPV) · Non-invasive ventilation device (not a drug, but a breathing support machine)

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.

1. You wear a snug mask connected to the BiPAP machine.
2. When you start to breathe in, the machine senses it and delivers air at a higher pressure (inspiratory positive airway pressure, or IPAP) to push your lungs open and fill them.
3. When you breathe out, the pressure drops to a lower level (expiratory positive airway pressure, or EPAP) so you can exhale more easily and push out carbon dioxide.
4. The pressure support keeps airways from collapsing, recruits more lung tissue, and takes the load off your tired breathing muscles.
5. Your oxygen level rises and carbon dioxide level falls without needing a breathing tube down your throat.

SAFETY WATCH

The mask must fit snugly, which can feel claustrophobic or uncomfortable – coaching and reassurance help. · Can cause skin breakdown or sores on the nose or face if the mask presses too hard for too long. · May cause dry mouth, nose irritation, or bloating from swallowing air. · Rarely, air can leak into the stomach (gastric distension) or even into the chest cavity outside the lungs (pneumothorax) if pressures are too high. · Requires close monitoring – if the patient gets worse or can't tolerate the mask, they may need intubation and mechanical ventilation. · BiPAP reduces the need for intubation and lowers mortality in acute hypercapnic respiratory failure (high CO₂) – it is first-line therapy in this setting.

Red flags

- 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

Nursing priorities

1. Position the patient sitting upright at 60 to 90 degrees, leaning slightly forward with arms resting on the overbed table. — This position (called tripod position) lets the diaphragm (main breathing muscle) drop down fully and uses gravity to help air get into the lower lung areas, which decreases the work of breathing.

2. 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. — COPD patients can lose their normal drive to breathe if given too much oxygen because their bodies rely on low oxygen (hypoxia) as the trigger to breathe; too much oxygen can cause hypercapnia (carbon dioxide buildup) and respiratory failure.
3. 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. — These bronchodilators (medicines that relax and widen the airway muscles) work by different mechanisms and together they open the narrowed airways faster and more completely, making it easier to move air in and out.
4. Give systemic corticosteroids (prednisone 40 mg by mouth daily for 5 days, or IV methylprednisolone if the patient cannot swallow) as ordered. — Corticosteroids reduce inflammation (swelling and irritation) in the airways, which decreases mucus production and airway narrowing; giving them early shortens recovery time and prevents treatment failure.
5. 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. — These vital signs show if the patient is tiring out or developing hypercapnia (carbon dioxide retention) or severe low oxygen (hypoxia) (low oxygen), which can progress quickly to respiratory arrest (stopping breathing) without intervention.
6. 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). — Controlled coughing helps move thick mucus out of the small airways without collapsing them, which prevents mucus plugging (blockage) and atelectasis (collapsed lung areas) that worsen gas exchange (oxygen in, carbon dioxide out).
7. 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. — Adequate hydration thins the mucus secretions so they are easier to cough up and clear from the airways; thick, dry mucus can plug airways and worsen obstruction.
8. 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. — Bacterial infections are a common trigger for exacerbations, and antibiotics kill the bacteria, reduce airway inflammation, and shorten recovery time when infection is present based on these clinical signs.

Sources

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- American Thoracic Society
- NEJM COPD Review

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