

Asthma Exacerbation

Every breath is a fight. The pipes squeeze shut, puff up, and plug with gunk.

THE ONE-LINE TRUTH

The airways clamp shut (smooth muscle spasm), swell up (inflammation led by IgE, mast cells, eosinophils, and leukotrienes), and fill with thick sticky mucus—air cannot get through.

The chain — how it unfolds

1. Trigger contacts the airway — why: An allergen (pollen, pet dander, dust mite), virus (rhinovirus is the top cause), cold air, exercise, or irritant (smoke, strong smell) lands on the airway lining. In asthma, the immune system is already primed to overreact to harmless things.
2. Mast cells pop open and dump histamine and leukotrienes — why: Mast cells in the airway lining are coated with IgE antibodies that recognize the trigger. When the trigger binds, the mast cells degranulate (burst open and spill their chemicals into the tissue)—this is a type 1 hypersensitivity reaction, the same kind that happens in hay fever or a bee-sting allergy.
3. Smooth muscle around the airway spasms (bronchospasm) — why: Histamine and leukotrienes lock onto receptors on smooth muscle cells and make them contract fast and hard—like a fist squeezing a straw shut. The airway narrows within seconds.
4. Airway lining swells and mucus pours out — why: The inflammatory cascade kicks in—eosinophils (white blood cells) swarm into the airway wall, Th2 helper T-cells release cytokines (IL-4, IL-5, IL-13), and blood vessels get leaky (increased vascular permeability), so fluid oozes into the tissue and the lining puffs up. Mucus glands go into overdrive and fill the tube with thick sticky mucus.
5. Air gets trapped behind the narrowed airways — why: Breathing out is harder than breathing in when airways are squeezed—air can sneak in past the narrow spot, but it cannot easily escape. Each breath stacks more air inside, and the lungs overinflate (air trapping).
6. Wheeze, cough, chest tightness, and breathlessness — why: Air rushing through the tight pipes makes a high whistling sound (wheeze). Stretch receptors and irritant receptors in the airway sense the swelling and mucus and trigger coughing. The chest feels squeezed because the airways are narrow and the lungs are overinflated.

7. Muscle exhaustion leads to silent chest and respiratory failure — why: The breathing muscles—diaphragm, intercostals, accessory muscles—can only work at maximum effort for so long. When they tire out, breathing slows and shallows (hypoventilation), CO₂ climbs, oxygen drops, and the patient may stop breathing. A silent chest (no wheeze because almost no air is moving) and confusion are late danger signs.

Spot it — what you see, and why

- Wheeze (mostly when breathing out): Air squeezes through narrowed small airways on the way out. The tight space makes the air spin and whistle—just like blowing through pursed lips—and that creates the musical wheeze sound you hear with a stethoscope.
- Cough (often at night or after exercise): Swollen airway walls and sticky mucus irritate the nerves that trigger coughing. Tightened airway muscles also pull on those nerves, making the urge to cough even stronger.
- Chest tightness or pressure: The rings of smooth muscle wrapped around each airway squeeze into a spasm—like a fist clenching—and that creates a squeezing or band-like feeling across the chest.
- Shortness of breath (hard to get enough air): Narrowed airways slow the flow of air in and out. At the same time, old air gets trapped in the lungs because it cannot escape fast enough, so the lungs overinflate like balloons that never fully empty. Both problems make breathing feel hard.
- Prolonged breathing-out phase (takes longer to exhale): Airways naturally collapse a bit when you breathe out, because pressure outside the airway becomes higher than inside. When airways are already narrowed by asthma, they collapse even more, so it takes much longer to push all the air out.
- Using neck, shoulder, and rib muscles to breathe: The diaphragm—the main breathing muscle under the lungs—cannot move enough air on its own. So the body recruits extra muscles in the neck, shoulders, and between the ribs to help lift and expand the chest with each breath.

Red flags — act now

- Silent chest, speaking only one word at a time, blue skin, or confusion—life-threatening asthma; breathing may stop within minutes
- Carbon dioxide level holding steady or rising during a severe attack—the breathing muscles are exhausting and the lungs are starting to fail
- Little or no improvement after one hour of strong inhaled medicine and steroids—the attack is not responding to standard treatment

- History of being put on a breathing machine for asthma before, or any hospital admission for asthma in the past year—marks the person as high-risk for another severe attack
- Cannot lie flat or can only say one word per breath—the airways are severely blocked

Treat it — in this order

1. Supplemental oxygen by nasal cannula or mask to keep SpO₂ at least 90%, targeting 93–95% → Raises the amount of oxygen in the blood right away, protecting the heart, brain, and other organs from damage while other drugs work to open the airways.
2. Short-acting beta-agonist—albuterol (salbutamol)—by nebulizer or metered-dose inhaler with spacer: 4–8 puffs every 20 minutes for the first hour, then every 3–4 hours; or continuous nebulization if the flare-up (exacerbation) is severe → Albuterol turns on beta-2 receptors on the smooth muscle wrapped around the airways, causing the muscle to relax within 5–15 minutes. This opens the breathing tubes (bronchodilation) and makes it easier to move air in and out—like releasing a pinched straw.
3. Ipratropium bromide (a short-acting anticholinergic) added to albuterol in moderate to severe exacerbations → Ipratropium blocks muscarinic receptors (the docking sites for acetylcholine, a nerve signal chemical) on airway smooth muscle, preventing signals that tell the muscle to tighten. Together with albuterol, it produces more bronchodilation than either drug alone and reduces hospital admissions (Cochrane 2013, GINA 2024).
4. Systemic corticosteroids: prednisone or prednisolone 40–50 mg by mouth, or methylprednisolone 60–125 mg intravenously if the patient cannot swallow or is vomiting → Shuts down the inflammatory cascade in the airway walls over 4–6 hours by blocking genes that produce inflammatory proteins (cytokines). This reduces swelling, mucus production, and airway twitchiness (hyperresponsiveness), cuts the risk of relapse, and speeds recovery (GINA 2024, Cochrane reviews, NAEPP EPR-3). Oral and IV routes work equally well when the gut is functioning.
5. Intravenous magnesium sulfate 2 grams infused over 20 minutes for severe exacerbations not responding to initial therapy (PEF or FEV₁ less than 40 percent predicted after 1 hour of maximum treatment) → Magnesium blocks calcium channels in airway smooth muscle cells. Since calcium makes muscle squeeze, blocking it causes the muscle to relax a little more (bronchodilation). This gives a modest extra benefit and reduces hospital admissions in truly severe cases (Cochrane 2014, GINA 2024).

Your top priorities at the bedside

1. Assess airway and work of breathing every 15 minutes during acute phase — why: Silent chest or single-word speech = intubate now.

2. Continuous pulse ox + peak flow trend after each treatment — why: Objective response tracking.
3. Give steroids EARLY (first hour) — why: Prevents rebound and cuts relapse in half.