

Chronic Asthma

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THE STORY IN ONE SENTENCE

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“Asthma — calm the daily swelling with an inhaled steroid every day, and use a rescue inhaler (albuterol) only when symptoms flare.”

Normal anatomy — the body's job

Normally the airways—the bronchi (say BRON-kye, the bigger tubes) and bronchioles (the smallest branches)—stay open and their smooth muscle (a band of muscle wrapped around each tube) stays relaxed, so air moves freely into and out of the alveoli (say al-VEE-oh-lye, tiny air sacs where oxygen crosses into the blood). The airway lining is thin, makes just a light coat of mucus to trap dust, and shows no swelling. Keep that picture in mind, because every asthma finding is a change from this baseline.

BODY'S JOB

The airways must stay wide enough for air to flow in during a breath and out during an exhale without any drag, delivering oxygen to the alveoli and carrying away carbon dioxide.

1. Air enters through the nose or mouth and travels down the trachea (windpipe), which splits into two main bronchi—one tube for each lung.
2. Each bronchus divides into smaller and smaller branches, like a tree spreading into twigs, called bronchioles.
3. At the ends of the smallest bronchioles sit clusters of alveoli, tiny grape-like sacs wrapped in capillaries (the body's tiniest blood vessels).
4. Oxygen hops from the alveoli into the blood; carbon dioxide hops the other way and is breathed out.

5. The airway walls have three layers: an inner lining of cells that make a thin layer of mucus to trap dust, a middle layer of smooth muscle that can tighten or relax, and an outer support layer of cartilage and elastic tissue.
6. In health, the smooth muscle stays relaxed, the lining makes just enough mucus to catch germs, and the space inside the tube (the lumen) stays wide open.
7. This design lets air flow in and out without effort, keeping oxygen delivery steady and carbon dioxide removal running smoothly.

What breaks

Long-lasting swelling in the airway walls—driven by eosinophils (a type of white blood cell), mast cells, and lymphocytes—causes the lining to puff up, mucus glands to overproduce thick sticky gunk, and the smooth muscle to become jumpy and squeeze tight in response to small triggers that would not bother normal airways.

CLINICAL TERM

Airway inflammation mediated by type 2 helper T-cells (Th2), eosinophils, and mast cells leads to bronchial hyper-reactivity (airways tighten too easily), mucosal swelling (edema) (swelling of the inner lining), mucus plugging (thick plugs block small branches), and smooth-muscle hypertrophy (the muscle layer grows thicker over time). Common triggers include allergens (pollen, dust mites, pet dander), respiratory viruses (rhinovirus, RSV), exercise (especially in cold dry air), cold air alone, NSAIDs (aspirin, ibuprofen), sulfites (preservatives in wine and dried fruit), and gastroesophageal reflux (stomach acid washing back into the throat). This upstream break produces the downstream clinical picture: intermittent wheezing, trouble breathing (dyspnea) (shortness of breath), chest tightness, and cough—all reversible with bronchodilators or spontaneously between attacks.

How the body compensates

Early in an asthma attack, the patient may look and sound okay—normal oxygen level on the fingertip monitor, talking in full sentences—because the body compensates by breathing faster and recruiting extra muscles to move air through the narrow tubes. This stable appearance is dangerous because it hides how close the patient is to respiratory collapse.

1. The body compensates by increasing respiratory rate (tachypnea, breathing fast) and using accessory muscles—the sternocleidomastoid and scalenes in the neck, the intercostals between the ribs—to pull harder and move air through narrowed airways.
2. Oxygen saturation (SpO₂) often stays normal at first, around 95 to 100 percent, because faster breathing keeps oxygen delivery up, masking how severe the airflow blockage really is.

3. This is the dangerous middle: the patient may appear stable while silently exhausting their respiratory muscles, which can only work overtime for so long before they fail.
4. The crash point to watch for is a silent chest (no wheezing because almost no air is moving), inability to speak in full sentences, altered mental status (confusion or drowsiness from rising carbon dioxide), or a normal or rising PaCO₂ on a blood gas (carbon dioxide should be low from hyperventilation; normal or high means the patient is tiring and heading toward respiratory arrest).
5. Patients with a history of prior ICU admission for asthma, three or more emergency-department visits in the past year, hospitalization or emergency visit in the past month, use of more than one canister of short-acting beta-agonist per month, or poor perception of their own symptom severity are at highest risk of sudden collapse.
6. Watch the trend—worsening peak expiratory flow (how fast air rushes out when they blow hard into a meter), increasing accessory-muscle use, retractions (skin sucking in between ribs or above the collarbone), decreasing alertness—not just a single snapshot at one moment.
7. A patient who looks stable but is using more and more effort to breathe, whose sentences are getting shorter, or whose peak flow is dropping is not stable; they are on the downslope toward respiratory failure and need urgent escalation of treatment.

Domino effect

1. A trigger—allergen (pollen, dust mite, pet dander), viral cold (rhinovirus, RSV), exercise, cold air, NSAID (aspirin, ibuprofen), or acid reflux—activates mast cells and eosinophils already sitting in the airway walls. — These immune cells release histamine (causes itch and swelling), leukotrienes (powerful constricting signals), and cytokines (chemical messengers) that tell nearby blood vessels to leak fluid into the tissue (causing swelling (edema), or swelling) and smooth muscle to contract (bronchoconstriction, or tightening).
2. The airway lumen (the open space inside the tube) narrows from three changes happening at once: smooth-muscle constriction squeezing the tube, mucosal swelling puffing the lining inward, and thick mucus plugs blocking the passage. — Narrow airways crank up resistance to airflow, so the patient must work much harder to pull air in and push air out; exhaling becomes especially difficult because airways tend to collapse further under the pressure of breathing out, trapping air in the lungs.
3. Wheezing (a high-pitched musical whistling sound on breathing out), a prolonged expiratory phase (taking much longer to exhale than to inhale), trouble breathing (dyspnea) (shortness of breath, air hunger), and chest tightness appear. — Air forced through the narrowed tubes at high speed vibrates the walls and whistles (that is the wheeze); the patient feels starved for air because less oxygen reaches the alveoli with each breath, and the brain senses carbon dioxide starting to rise, triggering the feeling of suffocation.

4. Cough develops, often dry at first or producing small gobs of thick, sticky, sometimes yellowish or white mucus (not a sign of infection unless fever and colored coughed-up mucus (sputum) persist). — Irritant receptors in the swollen, inflamed airway lining fire off signals that trigger the cough reflex, trying to clear mucus plugs and reopen blocked passages; coughing is the body's attempt to fix the problem but often just exhausts the patient further.
5. A short-acting beta-agonist (SABA) rescue inhaler—albuterol (also called salbutamol outside the U.S.) or levalbuterol—provides rapid but temporary relief within 5 to 15 minutes. — The beta-agonist chemical lands on beta-2 receptors sitting on airway smooth muscle, flipping a switch inside the muscle cell that causes relaxation (bronchodilation, or widening), opening the tubes fast—proving the obstruction is reversible—but it does nothing to calm the underlying swelling, so symptoms creep back in 3 to 4 hours and the patient needs another puff.
6. Without a daily inhaled corticosteroid (ICS), the patient becomes dependent on using the rescue inhaler again and again (sometimes many times a day), which paradoxically increases the risk of a severe, life-threatening asthma attack. — Using SABA alone—without an ICS to put out the fire of inflammation—allows chronic swelling to smolder unchecked; the airway walls remodel (thicken and stiffen permanently), hyper-reactivity worsens (airways become even twitchier), and one strong trigger can provoke a massive attack that the rescue inhaler cannot reverse in time, leading to respiratory failure or death.
7. A daily inhaled corticosteroid—fluticasone, budesonide, beclomethasone, mometasone, or ciclesonide—prevents attacks by shutting down the upstream inflammatory cascade at the gene level. — Corticosteroids slip into the nucleus of inflammatory cells and turn off the genes that code for cytokines, adhesion molecules, and enzymes that recruit more white blood cells; fewer eosinophils and mast cells pile up in the airway walls, swelling stays low, mucus production drops back to normal, and smooth muscle becomes much less twitchy—so airways stay open even when triggers show up, and attacks become rare or disappear entirely.
8. In moderate to severe asthma (needing ICS daily but still having symptoms or night waking more than twice a month), a long-acting beta-agonist (LABA)—salmeterol or formoterol—is added on top of the ICS in a single combination inhaler (never LABA alone), providing 12-hour bronchodilation. — The LABA keeps smooth muscle relaxed around the clock, cutting through breakthrough symptoms and letting the ICS work without interruption—but giving LABA without ICS has been shown to increase the risk of asthma death (the SMART trial proved this), so the combination in one device is now the standard and is mandatory for safety.

Symptoms · what the patient feels, and why

- **Wheezing:** Air is forced through narrowed, swollen airways and vibrates as it squeezes past — like wind whistling through a crack in a window. The sound is usually loudest when you breathe out because your chest squeezes the airways even tighter during exhalation.
- **Shortness of breath:** Narrowed airways make it harder to move air in and out, so less oxygen reaches the alveoli (the tiny air sacs in your lungs where oxygen jumps into your blood). Your brain senses the low oxygen and triggers a feeling of hunger for air.
- **Chest tightness:** Smooth muscle rings around your breathing tubes squeeze and clamp down — like a tight rubber band around a straw. Your chest has to work extra hard to push air through the narrow tubes, and that effort feels like a heavy band wrapped around your ribs.
- **Cough (often dry or with thick, sticky mucus):** The lining of your airways is swollen and irritated, and thick mucus plugs build up inside the tubes. This tickles the cough sensors in your airway walls, and your body tries to cough the mucus out to clear the path for air.
- **Waking up at night coughing or wheezing:** Your body's natural anti-swelling hormone cortisol (which tamps down inflammation like a fire extinguisher) drops to its lowest level between midnight and early morning. Without that protection, airway swelling and twitchiness peak, and symptoms break through and wake you up.
- **Symptoms triggered or worsened by exercise, cold air, or allergens (pollen, pet dander, dust mites):** Exercise makes you breathe fast and hard, drying out the airway lining and releasing chemicals that cause swelling. Cold air and allergens (things you are allergic to) directly wake up inflammatory cells called mast cells and eosinophils (white blood cells that are part of your immune defense) that dump histamine and leukotrienes (chemicals that make airways squeeze shut and swell).
- **Breathing out takes much longer than breathing in (prolonged expiratory phase):** Narrowed airways collapse under the pressure of breathing out — like a kinked garden hose. Air gets trapped inside your lungs because it takes a long time to squeeze it all out through the narrow, floppy tubes.
- **Using neck, shoulder, and belly muscles to breathe (accessory muscle use):** When your main breathing muscle the diaphragm (a dome-shaped sheet under your lungs) cannot pull in enough air on its own because the airways are so narrow, your body recruits extra muscles in your neck, shoulders, and abdomen to help lift and squeeze your chest. This is a sign your lungs are working very hard.
- **Cannot speak a full sentence without stopping to breathe:** Severe airway narrowing means you cannot take in enough air in one breath to power your voice through a whole sentence. You have to pause every few words to gasp for air — a warning that your breathing is dangerously weak and you may be heading toward respiratory failure (when your lungs cannot keep up with your body's oxygen needs).

- Silent chest (little or no wheezing even though the person looks very sick and is struggling to breathe): The airways are squeezed so tight that almost no air is moving in or out — not enough to make a wheeze sound. This is like a completely clogged pipe: no flow means no noise. It is a life-threatening emergency because breathing is about to stop completely.

Focused exam

- Wheezing on breathing out—a high-pitched whistling sound you can hear with or without a stethoscope: Long-term swelling and buildup of immune cells inside the lung's small breathing tubes (bronchioles) shrinks the airway opening. When air rushes through these narrowed passages during exhale, it vibrates like wind through a tiny crack in a window, making a whistle. The whistle is the sound of a half-closed airway struggling to push air out.
- Accessory muscle use—neck, shoulder, and rib muscles visibly tightening and pulling with each breath: When the airway narrows, breathing turns into hard work. The body calls in helper muscles beyond the main breathing muscle (the diaphragm) to drag air in. Seeing these extra muscles strain means the person is fighting to breathe and may run out of steam soon.
- Peak expiratory flow (PEF) drops 20% or more below the person's best reading, or stays below 80% of predicted value: Peak flow measures how fast air can shoot out of the lungs. In chronic asthma, inflammation and muscle squeezing shrink the airway opening like a kinked garden hose, so air crawls out slower. A 20% drop from personal best is a red flag that asthma is slipping out of control and needs treatment adjustment per NAEPP EPR-4 and GINA 2023 guidelines.
- Cough at night or cough triggered by running, cold air, or laughing hard: Swollen, irritated airways become jumpy (hyperreactive). Exercise makes you breathe faster, which cools and dries the airway lining, triggering the airway muscle to squeeze and extra mucus to pour out. Lying flat at night lets stomach acid creep up and mucus pool in the back of the throat. The cough is the body trying to clear the gunk and signals asthma is not well controlled.
- Using the rescue inhaler (short-acting beta-agonist like albuterol) more than 2 days per week, not counting doses taken before exercise: Needing the rescue puffer this often means the airways are tightening again and again despite daily controller medicine—or that daily medicine is not being taken at all. Current NAEPP EPR-4 and GINA 2023 rules say this pattern is uncontrolled asthma, and treatment must be stepped up to prevent worse attacks.

- Pursed-lip breathing (breathing out through nearly closed lips) or tripod position (leaning forward, hands on knees or table): Pursed lips create back-pressure that props small airways open longer when air flows out, stopping them from collapsing like a straw when you suck too hard. The tripod position opens the rib cage wider and recruits extra muscles to help haul air in. Both are the body's emergency tricks when airflow is dangerously blocked.

Diagnostics · every test answers a question

- Spirometry with bronchodilator reversibility testing → Is the airway blocked, and does it open back up after the patient breathes in a rescue inhaler (short-acting beta-agonist)? This test shows whether the airway narrowing is at least partly reversible, the signature of asthma.
- Fractional exhaled nitric oxide (FeNO) → Is there Type 2 airway inflammation driven by eosinophils (white blood cells that fuel allergic swelling)? High FeNO points to this kind of inflammation and predicts strong response to inhaled corticosteroids.
- Home peak expiratory flow (PEF) monitoring for 1 to 2 weeks, morning and evening → Does airflow swing up and down from day to day or morning to night? Asthma causes airflow to yo-yo because inflammation and muscle tightness change throughout the day.
- Complete blood count (CBC) with differential → Is the blood eosinophil count elevated, suggesting Type 2 (eosinophilic) asthma? This helps pick the right biologic therapy if severe asthma does not respond to standard inhalers.
- Serum total IgE level → Is the total IgE (allergic antibody that coats mast cells and triggers allergic reactions) elevated, pointing to allergic asthma? This identifies people who may benefit from omalizumab (Xolair), a biologic that blocks IgE.
- Chest X-ray (if the diagnosis is unclear or to rule out complications like pneumonia or a collapsed lung) → Is there another cause of wheezing or shortness of breath, such as pneumonia (lung infection), heart failure (fluid backing up into the lungs), or a foreign object stuck in the airway?
- Allergy testing—skin prick test or specific IgE blood test to common environmental allergens (dust mites, pet dander, mold, cockroach, tree pollen, grass pollen, weed pollen) → Which specific allergens (triggers that the immune system mistakes for invaders) set off asthma symptoms? Knowing this guides avoidance strategies and helps decide if allergen immunotherapy (allergy shots or tablets) is worth trying.
- Methacholine challenge test (if spirometry is normal but symptoms strongly suggest asthma) → Are the airways jumpy (hyperreactive) even when they appear normal at rest? This test provokes the airway muscle to squeeze by having the patient breathe increasing doses of methacholine, a chemical that mimics the nerve signal acetylcholine.

Treatment logic

- Daily low-dose inhaled corticosteroid (ICS) such as fluticasone, budesonide, or beclomethasone—or as-needed ICS-formoterol (combined ICS and fast-acting bronchodilator) for mild asthma → Turns down the inflammatory alarm system inside the airway wall. ICS crosses into airway cells and binds glucocorticoid receptors in the nucleus, shutting off genes that make inflammatory messenger proteins (cytokines like IL-4, IL-5, IL-13) and blocking white blood cells called eosinophils and mast cells from gathering and releasing chemicals that swell and tighten the airway. This prevents the airway from becoming jumpy and swollen, stopping attacks before triggers hit. GINA 2023 and NAEPP EPR-4 call ICS the cornerstone of asthma control.
- Short-acting beta-2 agonist (SABA) such as albuterol or levalbuterol, taken at the first sign of symptoms (chest tightness, wheeze, cough, shortness of breath) or 15 minutes before exercise → Grabs onto beta-2 receptors on the surface of airway smooth muscle cells. This flips a switch inside the cell, raising a molecule called cyclic AMP that tells the muscle to relax and let go of its squeeze (bronchodilation). The airway opens back up within 5 to 15 minutes, letting air flow freely again.
- Identify and avoid known triggers—allergens (dust mites, pet dander, pollen, mold, cockroach droppings), irritants (tobacco smoke, wood smoke, strong perfumes, cleaning chemicals, air pollution), cold air, exercise without pre-treatment, respiratory infections, NSAIDs (aspirin, ibuprofen, naproxen), and untreated GERD (stomach acid backing up into the food pipe) → Stops the chain reaction before it starts. When triggers land on the airway lining, they activate mast cells and eosinophils, which dump histamine, leukotrienes, and other chemicals that squeeze the airway muscle and call in more inflammatory cells. Removing the trigger keeps those cells quiet, reducing the number of attacks.
- Teach and check correct inhaler technique at every clinic visit, using physical demonstration, teach-back, and a written asthma action plan with pictures → Ensures the medicine lands deep inside the lungs where the small airways sit, instead of coating the back of the throat or getting swallowed into the stomach where it does nothing. Studies show 70 to 90% of patients use inhalers wrong—shaking too little, breathing in too slow, not holding their breath after the puff—leading to treatment failure even when the drug choice is correct. Fixing technique improves lung function (FEV₁) and symptom control per ATS/ERS and GINA guidelines.

- Add long-acting beta-2 agonist (LABA) such as formoterol or salmeterol, always combined with ICS in a single inhaler (never LABA alone), for moderate to severe persistent asthma when low-dose ICS is not enough → Provides steady muscle relaxation (bronchodilation) around the clock for 12 to 24 hours by staying attached to beta-2 receptors longer than albuterol. Paired with ICS, this combo tackles both the swelling (inflammation) and the squeezing (bronchoconstriction), cutting the number of attacks and improving how much air you can blow out. GINA and NAEPP recommend ICS-LABA as the main step-up when symptoms happen more than twice a week or wake you at night despite low-dose ICS.
- Add-on biologic therapy—omalizumab (Xolair, blocks IgE), mepolizumab (Nucala) or benralizumab (Fasenra, both block IL-5 or its receptor), or dupilumab (Dupixent, blocks IL-4 receptor alpha)—for severe uncontrolled asthma despite high-dose ICS-LABA and proven good adherence → Targets specific arms of the Type 2 inflammatory chain reaction. Omalizumab grabs IgE (the allergic antibody) before it can glom onto mast cells, stopping them from exploding and dumping histamine. Mepolizumab and benralizumab cut the supply of eosinophils by blocking IL-5 (the signal that tells bone marrow to make eosinophils) or wiping out eosinophils already in the blood. Dupilumab blocks the receptors for IL-4 and IL-13 (two messengers that drive eosinophil recruitment, mucus overproduction, and airway thickening). These drugs slash asthma attacks by 40 to 70% in the right patients per GINA and ATS/ERS severe asthma guidelines.
- Written asthma action plan (daily medicine list, how to spot worsening—dropping peak flow, needing rescue inhaler more often, symptoms waking you at night—when to double ICS, when to start oral steroids, when to call the doctor, when to go to the emergency room) and vaccinations: annual influenza shot, COVID-19 series, and pneumococcal vaccine (PPSV23 or PCV series) per ACIP guidelines → Action plans put the patient in the driver's seat—they learn to catch flare-ups early and step up treatment before a full-blown attack lands them in the hospital. Studies show written plans cut emergency visits and hospital stays by teaching people what to do when asthma slips. Vaccines train the immune system's memory cells to recognize viruses (influenza, SARS-CoV-2) and bacteria (*Streptococcus pneumoniae*) so the body can fight them off fast, preventing respiratory infections that are the top trigger for asthma attacks.

Medications

Albuterol (short-acting beta-agonist, SABA) · Short-acting bronchodilator (rescue inhaler)

Albuterol relaxes the tight muscle wrapped around your breathing tubes within minutes. It lands on tiny switches called beta-2 receptors sitting on the surface of airway muscle cells and flips them on. This raises a chemical messenger inside the cell—cyclic AMP (like turning up the volume on a 'relax' signal)—which tells the muscle to unwind and let air flow through. The

effect lasts 4 to 6 hours. This medicine stops an asthma attack in its tracks by opening the airway fast.

1. Albuterol molecules land on beta-2 receptors sitting on the surface of airway muscle cells.
2. The receptor changes shape and activates an enzyme called adenylyl cyclase inside the cell.
3. The enzyme makes more cyclic AMP (cAMP), a molecule that acts like a 'stop squeezing' signal.
4. Rising cAMP lowers calcium inside the muscle cell (calcium is what makes muscle contract).
5. With less calcium, the muscle fibers slide apart and the airway opens wider, letting you breathe easier.

SAFETY WATCH

Tremor (shaking hands), especially at higher doses, because beta-2 receptors also sit on skeletal muscle. · Fast heart rate (tachycardia, over 100 beats per minute) and palpitations (feeling your heartbeat), since some drug reaches beta receptors in the heart. · Low potassium (hypokalemia) when used frequently or at high doses; potassium shifts into cells as cAMP rises. · Nervousness, headache, and throat irritation from the inhaled powder or mist. · Using your rescue inhaler more than 2 days a week (except before exercise) signals poor control—call your doctor to step up controller therapy.

Inhaled corticosteroid (ICS): budesonide, fluticasone, beclomethasone, mometasone, ciclesonide · Inhaled anti-inflammatory steroid (daily controller)

An inhaled corticosteroid is a steroid medicine you breathe straight into your lungs every day to calm down the ongoing immune attack in your airways. The drug enters the cells lining your breathing tubes and travels to the nucleus (the control center of the cell). There it turns off the genes that tell the cell to make inflammatory chemicals (proteins called cytokines and chemokines). Fewer inflammatory chemicals means less swelling, less mucus, fewer immune cells crowding the airway, and airways that are less twitchy and reactive. Over days to weeks, the airway lining heals and becomes less likely to clamp shut when you meet a trigger.

1. ICS particles land on the wet surface of the airway and dissolve.
2. The steroid molecule crosses into epithelial cells and binds to a glucocorticoid receptor (the steroid's docking site) in the cell's cytoplasm (the gel inside the cell).
3. The receptor-steroid pair moves into the nucleus (the cell's command center).
4. Inside the nucleus, the pair lands on DNA and blocks genes that code for inflammatory proteins (interleukins, tumor tissue death (necrosis) factor, and leukotrienes).
5. At the same time, it turns on genes that make anti-inflammatory proteins (such as lipocortin-1, which stops an enzyme that makes inflammation chemicals).

6. Fewer inflammatory signals mean fewer eosinophils, mast cells, and T-cells migrate into the airway, mucus production drops, and the airway lining stops swelling.
7. Over 2 to 4 weeks, airway responsiveness (how easily the airway clamps shut) improves and symptom-free days increase.

SAFETY WATCH

Oral thrush (white yeast patches in mouth and throat) and hoarse voice (dysphonia)—rinse your mouth with water and spit after every dose to wash steroid off the throat. · At high doses for many years: possible adrenal suppression (your adrenal glands—the stress-hormone glands above your kidneys—slow down), thinner bones (lower bone mineral density, raising fracture risk), cataracts (clouding of the eye lens), and mild rise in blood sugar. · In children, high doses may slow growth velocity slightly during the first year; final adult height is usually not affected, and the benefit of controlled asthma outweighs this risk. · Bruising more easily and thinner skin are rare with inhaled doses but can happen at very high doses. · Use the lowest dose that keeps asthma controlled; step down dose once control is stable for 3 months.

Inhaled corticosteroid + formoterol (ICS-formoterol): budesonide-formoterol, beclomethasone-formoterol, mometasone-formoterol · Combination anti-inflammatory and long-acting bronchodilator (controller and reliever in one inhaler)

This inhaler combines an anti-inflammatory steroid (to calm the swollen airway lining) with formoterol, a long-acting muscle relaxer (to keep the airway open). Formoterol starts working within 1 to 3 minutes—fast enough to relieve symptoms—and lasts 12 hours. The steroid reduces the immune attack that causes swelling and mucus. When taken daily, the combination prevents symptoms and attacks. When taken as needed at the first sign of tightness, it both relieves the attack and delivers anti-inflammatory medicine right when inflammation is flaring. This dual action is safer and more effective than using a short-acting bronchodilator alone, because every puff treats both the muscle spasm and the underlying swelling.

1. Formoterol binds beta-2 receptors on airway muscle, raising cyclic AMP inside the cell and lowering calcium, so the muscle relaxes within 1 to 3 minutes.
2. The effect lasts 12 hours because formoterol's chemical shape lets it stick to the receptor and re-bind repeatedly.
3. At the same time, the inhaled steroid (budesonide, beclomethasone, or mometasone) enters epithelial cells, binds glucocorticoid receptors, and moves to the nucleus.
4. In the nucleus, the steroid turns off genes for inflammatory proteins (cytokines, chemokines) and turns on genes for anti-inflammatory proteins.

5. Over hours to days, immune-cell traffic into the airway drops, mucus production falls, and the airway lining heals.
6. The combination prevents the need to increase short-acting rescue medicine, which is a sign of worsening inflammation.

SAFETY WATCH

Rinse and spit after every dose to prevent thrush and hoarse voice from the steroid. · Tremor (shaking), fast heartbeat, and low potassium can occur from the formoterol, especially if you take extra doses. · Using more than 8 to 12 puffs in 24 hours signals severe worsening—seek urgent care. · High steroid doses over years carry the same risks as ICS alone: adrenal suppression, bone thinning, cataracts, and mild blood-sugar rise. · Never use formoterol (or any long-acting beta-agonist) without an inhaled steroid—studies show that long-acting bronchodilators alone can mask worsening inflammation and increase the risk of asthma death. · Check your technique: breathe in slowly and deeply as you press the inhaler, hold your breath for 5 to 10 seconds, then exhale; wrong technique means the medicine lands in your mouth instead of your lungs.

Inhaled corticosteroid + long-acting beta-agonist (ICS-LABA): fluticasone-salmeterol, budesonide-formoterol, mometasone-formoterol, fluticasone-vilanterol, beclomethasone-formoterol · Combination daily controller (anti-inflammatory steroid plus 12- or 24-hour bronchodilator)

This combination inhaler delivers an anti-inflammatory steroid to shut down the immune attack in your airways and a long-acting bronchodilator (salmeterol, formoterol, or vilanterol) to keep airway muscle relaxed for 12 to 24 hours. The steroid reduces swelling, mucus, and the number of activated immune cells. The long-acting beta-agonist (LABA) binds to beta-2 switches on muscle cells, raising the 'relax' signal inside and keeping airways open all day or all night. Together they prevent symptoms, cut the number of attacks, improve lung function, and reduce the need for rescue inhalers. This is the backbone of treatment for moderate to severe asthma (GINA steps 3 to 5; NAEPP steps 3 to 6).

1. The LABA (salmeterol, formoterol, or vilanterol) binds beta-2 receptors on airway muscle and raises cyclic AMP, which lowers intracellular calcium and relaxes the muscle.
2. Salmeterol and vilanterol have a slow onset (10 to 20 minutes) but last 12 to 24 hours; formoterol starts in 1 to 3 minutes and lasts 12 hours.
3. The inhaled steroid enters epithelial cells, binds glucocorticoid receptors, moves to the nucleus, and switches off genes that make inflammatory chemicals.
4. Over days, fewer eosinophils, mast cells, and T-cells enter the airway; mucus glands shrink; and the airway lining becomes less swollen and less twitchy.

5. Peak anti-inflammatory effect takes 1 to 2 weeks of daily use; lung function continues to improve for 3 to 6 weeks.

SAFETY WATCH

Never use a LABA without an inhaled steroid in the same inhaler—LABA monotherapy increases the risk of asthma-related death by masking worsening inflammation. · Rinse mouth and spit after each dose to prevent oral thrush and hoarse voice. · Tremor, fast heart rate, and low potassium from the LABA, especially at higher doses. · Long-term high-dose inhaled steroid risks: adrenal suppression (adrenal glands stop making enough cortisol, your body's stress hormone), thinner bones, cataracts, mild rise in blood sugar, and easy bruising. · In children, high doses may slow growth rate slightly in the first year, but final adult height is usually normal and the benefit of asthma control is greater. · Check inhaler technique at every visit—70 to 80 percent of patients use inhalers wrong, wasting medicine. · Step down to the lowest effective dose once asthma is well controlled for 3 months; step up if control is lost.

Long-acting muscarinic antagonist (LAMA): tiotropium, umeclidinium · Long-acting anticholinergic bronchodilator (add-on controller at step 4 to 5)

A LAMA blocks the chemical signal (acetylcholine) that tells airway muscle to squeeze shut. Normally, nerves release acetylcholine onto muscle cells, where it lands on muscarinic receptors (especially the M3 type) and triggers contraction. Tiotropium and umeclidinium sit on those M3 receptors and block acetylcholine from binding, so the muscle stays relaxed for 24 hours. This adds another layer of airway opening on top of the ICS-LABA combination, especially helpful in people whose airways still tighten despite steroids and long-acting beta-agonists. LAMAs are now recommended as add-on therapy before increasing steroid dose.

1. Tiotropium or umeclidinium is inhaled and lands on muscarinic-3 (M3) receptors on the surface of airway muscle cells.
2. The drug molecule binds tightly and blocks the receptor so acetylcholine (the nerve signal to contract) cannot attach.
3. Without acetylcholine stimulation, calcium does not rise inside the muscle cell and the muscle stays relaxed.
4. The drug also blocks M3 receptors on mucus-gland cells, reducing mucus production.
5. The effect lasts 24 hours, allowing once-daily dosing.
6. Adding a LAMA to ICS-LABA improves lung function and reduces attacks (exacerbations) more than doubling the steroid dose.

SAFETY WATCH

Dry mouth (most common; sip water throughout the day). · Urinary retention (difficulty emptying bladder fully), especially in older men with enlarged prostate—tell your doctor if you cannot urinate. · Blurred vision if powder gets in eyes; close eyes during inhalation and rinse hands after. · Worsening of narrow-angle glaucoma (sudden eye pressure rise causing pain, blurred vision, halos around lights)—use with caution if you have glaucoma. · Rare paradoxical bronchospasm (sudden airway tightening right after inhaling)—stop the medicine and call your doctor. · Not for use in people allergic to atropine or ipratropium.

Omalizumab · Anti-IgE monoclonal antibody biologic (step 5 add-on for allergic asthma)

Omalizumab is an injected antibody medicine that grabs and neutralizes IgE, the immune protein that triggers allergic reactions. In allergic asthma, your body makes too much IgE when it sees an allergen (pollen, dust mite, pet dander). That IgE sticks to the surface of mast cells and basophils (immune cells full of inflammatory chemicals). When the allergen appears again, it bridges two IgE molecules on the cell surface, and the cell explodes, dumping histamine (the itch-and-swallow chemical), leukotrienes (chemicals that tighten airways and make mucus), and other chemicals that cause airway swelling, mucus, and muscle tightness. Omalizumab binds to free-floating IgE in your blood before it can attach to cells, so allergen exposure no longer triggers the allergic cascade. Over weeks, mast cells lose their IgE coat and become less reactive. This cuts down allergic attacks, reduces steroid needs, and improves asthma control.

1. Omalizumab is injected under the skin every 2 or 4 weeks.
2. The antibody circulates in blood and binds to the part of the IgE molecule that would normally attach to cell receptors (the Fc portion).
3. With its receptor-binding site blocked, IgE cannot stick to mast cells or basophils.
4. Free IgE levels in blood drop by more than 95 percent within hours.
5. Over 8 to 12 weeks, mast cells that already have IgE on their surface gradually lose those receptors (down-regulation), becoming less reactive.
6. When you breathe in allergen, there is no IgE on the cell surface to cross-link, so the cell does not release histamine and leukotrienes, and the allergic airway reaction is blocked.

SAFETY WATCH

Anaphylaxis (severe allergic reaction with throat swelling, trouble breathing, low blood pressure, shock) can occur within 2 hours of injection or rarely days later—first three doses are given in a clinic with a 2-hour observation; you will be prescribed an epinephrine auto-injector to carry at all times. · Injection-site reactions (redness, swelling, pain) in about 45 percent of patients. · Rare increased risk of heart attack, stroke, or blood clots (seen in some trials); tell your doctor if you have heart disease. · Do not stop inhaled steroids suddenly when starting omalizumab—taper slowly under your doctor's guidance. · Check total IgE level and body weight before starting (dose is based on both); recheck asthma control at 16 weeks—if no improvement, consider stopping. · Avoid live vaccines during treatment.

Mepolizumab, reslizumab · Anti-IL-5 monoclonal antibody biologic (step 5 add-on for eosinophilic asthma)

Mepolizumab and reslizumab are injected antibodies that block interleukin-5 (IL-5), a protein signal that tells the bone marrow to make eosinophils and tells eosinophils to stay alive and active in the airways. Eosinophils are white blood cells full of toxic granules; when they crowd into airway tissue, they release proteins that damage the lining, thicken mucus, and keep inflammation going. In severe asthma, high eosinophil counts drive attacks and steroid dependence. These biologics grab IL-5 in the blood before it reaches eosinophils, so the bone marrow makes fewer eosinophils and those already in the tissue die off faster. Within weeks, blood and airway eosinophil counts drop, attacks become less frequent, and many patients can reduce or stop daily oral steroids.

1. Mepolizumab (under-skin injection every 4 weeks) or reslizumab (intravenous infusion every 4 weeks) enters the bloodstream.
2. The antibody binds tightly to IL-5 molecules, forming an inactive complex.
3. IL-5 can no longer reach its receptor on eosinophil precursors in the bone marrow, so fewer eosinophils mature.
4. IL-5 also cannot signal mature eosinophils in tissue to survive, so existing eosinophils undergo programmed cell death (apoptosis).
5. Within 4 weeks, blood eosinophil count drops by 70 to 90 percent; airway eosinophils fall over 8 to 12 weeks.
6. With fewer eosinophils, airway damage decreases, mucus plugs thin, and the frequency of severe attacks (exacerbations) drops by about 50 percent.

SAFETY WATCH

Injection-site reactions (redness, swelling, itch) with mepolizumab; infusion reactions (headache, flushing, muscle aches) with reslizumab (given slowly over 30 to 60 minutes). · Headache, back pain, and fatigue reported in trials. · Rare anaphylaxis—reslizumab carries a black-box warning; infusions are given in a clinic with observation. · Do not stop oral or inhaled steroids suddenly; taper dose slowly over months under your doctor's watch to avoid adrenal crisis (life-threatening drop in cortisol, the stress hormone). · Watch for eosinophilic granulomatosis with polyangiitis (EGPA, formerly Churg-Strauss syndrome)—a rare disease with rash, nerve pain, sinus inflammation, and organ damage that may unmask when steroids are lowered; not caused by the biologic but revealed as steroid dose drops. · Avoid live vaccines during treatment. · Parasite infections (worms) may be harder to clear, because eosinophils help fight them—treat any parasites before starting.

Benralizumab · Anti-IL-5 receptor alpha monoclonal antibody biologic (step 5 add-on for eosinophilic asthma)

Benralizumab is an injected antibody that binds to the IL-5 receptor alpha sitting on the surface of eosinophils and their parent cells. Once attached, it marks those cells for destruction by recruiting natural killer cells (a type of immune cell that kills tagged targets). This not only blocks the IL-5 signal (like mepolizumab and reslizumab do) but actively wipes out eosinophils already in the blood and tissue within hours to days. Blood eosinophils drop to near zero, airway eosinophils vanish, and the cascade of damage they cause—mucus plugs, epithelial injury, and ongoing inflammation—stops. Attacks become rare, lung function improves, and many patients can reduce or eliminate daily oral steroids. The effect lasts 8 weeks, so dosing is every 4 weeks for the first three doses, then every 8 weeks.

1. Benralizumab is injected under the skin every 4 weeks for three doses, then every 8 weeks.
2. The antibody binds to IL-5 receptor alpha (IL-5R α) on the surface of eosinophils, basophils, and their bone-marrow precursors.
3. The Fc tail of the antibody (the constant region) recruits natural killer (NK) cells, which recognize the antibody-coated eosinophil as a target.
4. NK cells release toxic proteins (perforin and granzymes) that punch holes in the eosinophil and trigger its death (antibody-dependent cell-mediated cytotoxicity, or ADCC).
5. Within 24 hours, blood eosinophil counts drop to near zero; tissue eosinophils fall over the next few weeks.
6. Without eosinophils releasing damage proteins (major basic protein and eosinophil peroxidase), the airway lining heals, mucus production drops, and attack frequency falls by about 50 to 70 percent.

SAFETY WATCH

Injection-site reactions (pain, redness, swelling). · Headache and sore throat (pharyngitis) reported in trials. · Rare anaphylaxis (severe allergic reaction)—first dose may be given in clinic with observation. · Do not stop oral steroids abruptly; taper slowly over months to avoid adrenal insufficiency (your adrenal glands may have slowed down from long steroid use). · Watch for eosinophilic granulomatosis with polyangiitis (EGPA) unmasking as steroids are reduced—symptoms include rash, numbness or tingling in hands or feet (nerve damage), sinus pain, and kidney or heart problems. · Because eosinophils help fight parasites (worms), treat any helminth infection before starting benralizumab. · Avoid live vaccines during treatment. · Very low eosinophil counts are expected and safe; eosinophils recover slowly if the drug is stopped.

Dupilumab · Anti-IL-4 receptor alpha monoclonal antibody biologic (step 5 add-on for type-2 inflammatory asthma with elevated eosinophils or FeNO)

Dupilumab blocks the receptor for two inflammatory signals, interleukin-4 (IL-4) and interleukin-13 (IL-13), which drive type-2 inflammation in asthma. These two proteins tell immune cells to make more IgE, recruit eosinophils, thicken mucus, and keep the airway lining inflamed. Dupilumab binds to the shared part of the receptor (IL-4 receptor alpha) on the surface of immune cells, airway cells, and mucus glands, stopping both IL-4 and IL-13 from sending their signals. This shuts down multiple inflammatory pathways at once: IgE production drops, eosinophil counts fall, mucus becomes thinner, and the airway lining calms. Patients have fewer attacks, better lung function, and many can reduce oral steroid doses. Dupilumab also treats atopic dermatitis (eczema) and nasal polyps, common partners of severe asthma.

1. Dupilumab is injected under the skin every 2 weeks (after a loading dose).
2. The antibody binds to IL-4 receptor alpha (IL-4R α), a protein chain shared by the receptors for IL-4 and IL-13.
3. With the receptor blocked, IL-4 cannot signal B cells to make IgE (the allergic antibody) or push T-cells toward a type-2 pattern.
4. IL-13 cannot signal airway cells to produce thick mucus, recruit eosinophils, or thicken the layer under the epithelium (subepithelial fibrosis).
5. Over 2 to 4 weeks, blood eosinophil counts drop (though not as low as with anti-IL-5 biologics), IgE levels fall, and mucus plugs thin.
6. Airway inflammation decreases, lung function improves, and severe attacks drop by about 50 to 70 percent.

SAFETY WATCH

Injection-site reactions (redness, swelling, pain) are common. · Conjunctivitis (red, itchy, watery eyes) in 10 to 15 percent of patients; usually mild and managed with lubricating eye drops, but see an eye doctor if severe. · Rarely keratitis (inflammation of the cornea—the clear front of the eye—causing eye pain, blurred vision, light sensitivity)—report eye symptoms promptly. · Eosinophil count may rise transiently in some patients (usually not a problem unless very high, above 1500 cells per microliter, which raises risk of organ damage). · Do not stop oral or high-dose inhaled steroids suddenly—taper slowly to avoid adrenal crisis. · Watch for eosinophilic granulomatosis with polyangiitis (EGPA) unmasking as steroids are reduced. · Do not give live vaccines during treatment. · Parasite (helminth) infections should be treated before starting, because type-2 immunity helps clear worms. · Dupilumab improves asthma control in patients with coexisting eczema or nasal polyps, making it a good choice when all three are present.

Oral corticosteroid (prednisone, prednisolone, methylprednisolone) - short course for flare-up (exacerbation) · Systemic steroid burst (rescue treatment for asthma attack)

A short course (3 to 7 days) of oral steroid pills shuts down the whole-body and airway immune attack during a severe asthma flare-up (exacerbation). Prednisone and its cousins enter every cell, move to the nucleus, and turn off hundreds of inflammatory genes at once—stopping production of cytokines, chemokines, and enzymes that feed swelling and mucus. At the same time, steroids stabilize the walls of tiny blood vessels so they leak less fluid into the airway tissue, and they tell immune cells to leave the area. Within hours to a few days, airway swelling drops, mucus thins, and the lungs can move air again. Oral steroids speed recovery, cut the need for hospitalization, and prevent relapses in the two weeks after an attack. They do not replace inhaled controllers, which must be stepped up to prevent the next attack.

1. You swallow prednisone (or prednisolone, methylprednisolone); it absorbs from the gut into the bloodstream within 30 to 60 minutes.
2. The steroid crosses into cells throughout the body and binds glucocorticoid receptors.
3. Receptor-steroid pairs enter the cell nucleus and block genes that code for inflammatory proteins (IL-1, IL-5, IL-13, TNF-alpha, leukotrienes).
4. Steroids also turn on genes for anti-inflammatory proteins (such as lipocortin, which stops the enzyme phospholipase A2 from making inflammatory chemicals).
5. Blood vessels in the airway become less leaky, so swelling (edema) drops.
6. Eosinophils, neutrophils, and lymphocytes stop migrating into the airway and existing cells die off or leave.
7. Mucus glands shrink and produce less mucus.

8. Symptom improvement begins in 4 to 6 hours; peak effect at 1 to 2 days.

SAFETY WATCH

Short-term (3 to 7 days): increased appetite, trouble sleeping (insomnia), mood swings (irritability, anxiety, or feeling 'wired'), mild fluid retention, stomach upset, and high blood sugar (especially if you have diabetes—check glucose). · Courses longer than 2 weeks or repeated bursts over the year raise risk of weight gain, high blood pressure, thinning bones (osteoporosis), cataracts, glaucoma, easy bruising, poor wound healing, and increased infection risk. · Long-term daily steroids (maintenance) can suppress your adrenal glands (they stop making cortisol, your body's stress hormone); suddenly stopping can cause adrenal crisis (dangerous drop in blood pressure, confusion, collapse)—always taper long-term steroids slowly. · In children, repeated or prolonged courses may slow growth; use the shortest course that works. · People on steroids for more than 3 weeks should avoid live vaccines (measles, varicella, nasal flu vaccine) until steroids are stopped for at least 1 month. · Watch blood sugar if diabetic; adjust diabetes medicines during the steroid course. · Take with food to protect the stomach; a single morning dose reduces sleep trouble.

Leukotriene receptor antagonist (montelukast, zafirlukast) · Oral anti-inflammatory (mild daily controller, alternative to low-dose ICS)

Montelukast and zafirlukast block receptors for leukotrienes, inflammatory chemicals made by immune cells that cause airway muscle to tighten, blood vessels to leak fluid, and mucus glands to turn on. Leukotrienes (especially leukotriene D4 or LTD4) are released by mast cells and eosinophils when they are triggered by allergens, exercise, or cold air. These drugs sit on the leukotriene receptor (called cysteinyl leukotriene receptor 1, or CysLT1) on airway muscle and other cells and prevent leukotrienes from binding. This stops one branch of the inflammatory cascade. Leukotriene blockers are less powerful than inhaled steroids but can help with allergic asthma, exercise-induced symptoms, and aspirin-sensitive asthma. They are taken once or twice daily as a pill, making them easier for people who struggle with inhalers. They also treat allergic rhinitis (hay fever).

1. You swallow the pill; it absorbs into the bloodstream and travels to all tissues.
2. The drug binds to CysLT1 receptors on the surface of airway muscle cells, blocking the site where leukotrienes (LTC4, LTD4, LTE4) would attach.
3. Without leukotriene signaling, calcium does not rise inside the muscle cell, so the muscle stays relaxed.
4. The drug also blocks leukotriene receptors on blood-vessel cells, reducing fluid leak and swelling.
5. Mucus gland activity drops.

6. Over days to weeks, symptoms improve modestly; attacks may become less frequent.
7. In aspirin-exacerbated respiratory disease (AERD), blocking leukotriene receptors blunts the surge of leukotrienes caused by aspirin or NSAIDs (non-steroidal anti-inflammatory drugs such as ibuprofen).

SAFETY WATCH

Headache, stomach upset, and upper respiratory infection reported in trials. · Neuropsychiatric events (FDA black-box warning): agitation, aggression, mood swings, depression, suicidal thoughts, sleep disturbances (vivid dreams, sleepwalking), and rarely hallucinations or obsessive-compulsive behaviors—monitor mood closely, especially in children and teens; stop the drug if these occur. · Rare eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome) unmasking when oral steroids are reduced—not caused by the drug but revealed as steroid dose drops. · Rarely, elevated liver enzymes (transaminases)—check liver tests if symptoms of liver trouble (yellow skin, dark urine, severe fatigue) appear. · Drug interactions: zafirlukast can raise blood levels of warfarin (a blood thinner), increasing bleeding risk—monitor INR (a measure of blood clotting) closely if taking both.

Theophylline · Oral bronchodilator and mild anti-inflammatory (rarely used add-on)

Theophylline is a pill that relaxes airway muscle and may have mild anti-inflammatory effects at low doses. It blocks an enzyme (phosphodiesterase) that breaks down cyclic AMP, the 'relax' messenger inside muscle cells, so muscle stays relaxed longer. It also blocks adenosine receptors, which can reduce inflammation slightly. Theophylline has a narrow safety window—the dose that helps is close to the dose that causes side effects—so blood levels must be checked regularly. It is rarely used today because inhaled steroids and LABAs work better and are safer, but theophylline may be added when other medicines are not enough and cost is a concern (it is inexpensive).

1. You swallow theophylline (usually an extended-release tablet); it absorbs and circulates in blood.
2. The drug enters airway muscle cells and blocks phosphodiesterase, the enzyme that breaks down cyclic AMP.
3. With cAMP staying high, the muscle remains relaxed and the airway stays open.
4. Theophylline also blocks adenosine receptors on immune cells and neurons, which may dampen inflammation and stimulate breathing drive.
5. Peak effect takes several hours; steady blood levels are reached after a few days of regular dosing.

SAFETY WATCH

Narrow therapeutic window—blood level must stay between 5 and 15 micrograms per milliliter; levels above 20 cause serious toxicity. · Common side effects even at therapeutic levels: nausea, stomach upset, headache, insomnia, and jitteriness (like too much caffeine—theophylline is chemically similar to caffeine). · Toxic levels cause vomiting, rapid heartbeat (tachycardia), irregular heart rhythm (arrhythmias), seizures, and rarely death. · Drug interactions are common: smoking, rifampin (an antibiotic), phenytoin (a seizure medicine), and phenobarbital lower theophylline levels (dose must be increased); ciprofloxacin (an antibiotic), erythromycin (an antibiotic), cimetidine (a stomach-acid drug), and viral infections raise levels (dose must be decreased). · Liver or heart disease slows theophylline clearance, raising the risk of toxicity. · Check blood theophylline level regularly (draw 4 to 6 hours after morning dose for extended-release forms); adjust dose to keep level in safe range. · Not recommended as first-line therapy; reserve for patients who cannot use or afford inhalers.

Cromolyn sodium (rarely used) · Mast-cell stabilizer (inhaled preventive, now mostly historical)

Cromolyn is an inhaled powder that prevents mast cells from releasing their load of histamine and leukotrienes when triggered by allergens or exercise. It stabilizes the mast-cell membrane so the cell does not 'degranulate' (dump its contents). This blocks the early allergic reaction that starts airway tightening and swelling. Cromolyn must be inhaled 15 to 30 minutes before exposure to a known trigger (e.g. before exercise, before visiting a home with pets) and must be used four times daily for ongoing prevention. It is very safe (almost no side effects) but is weak compared to inhaled steroids, requires frequent dosing, and has been largely replaced by better medicines. It is still sometimes used in children or people who cannot tolerate any other controller.

1. Cromolyn powder is inhaled and lands on the surface of mast cells in the airway.
2. The drug enters the cell and blocks calcium channels in the membrane, preventing calcium influx.
3. Without the calcium surge, the mast cell cannot fuse its granules (sacs of histamine, leukotrienes, and other chemicals) to the cell membrane.
4. The granules stay inside, so no inflammatory chemicals are released even when allergen cross-links IgE on the cell surface.
5. This prevents the immediate allergic reaction—no histamine-driven swelling, no leukotriene-driven muscle tightening.

SAFETY WATCH

Very few side effects—one of the safest asthma drugs. · Throat irritation, cough, or bad taste from the powder (less common with nebulized solution). · Rare paradoxical bronchospasm (sudden airway tightening right after inhaling)—stop if this happens. · Requires four-times-daily dosing and pre-treatment before known triggers, making it hard to use correctly. · No longer widely available in the United States; nebulizer solution still used occasionally in young children.

Red flags

- Silent chest on exam (no wheezing heard) despite obvious severe breathing distress — means airways are nearly completely blocked and breathing may stop
- Cannot speak full sentences, or mental confusion, agitation, or extreme drowsiness — signs of dangerously low oxygen or rising carbon dioxide in the blood
- Normal or rising carbon dioxide level (PaCO_2) on a blood gas test — should be low because fast breathing blows off CO_2 ; normal or high means the breathing muscles are getting tired and about to fail
- History of a previous ICU admission or being put on a breathing machine (intubated) for asthma — marks the person as high-risk for a life-threatening attack
- Poor symptom awareness (the person does not feel how bad their breathing is getting until very late) — delays getting help and raises the risk of sudden collapse

Nursing priorities

1. Sit the patient fully upright (high Fowler position) and apply oxygen by nasal cannula or mask to keep oxygen saturation above 90 percent. — Upright position uses gravity to help the diaphragm (breathing muscle) move down and expand the lungs; supplemental oxygen prevents dangerous heart rhythm problems and brain damage from low oxygen levels.
2. Give the first dose of short-acting beta-2 agonist (albuterol 2.5 to 5 milligrams) by nebulizer or 4 to 8 puffs by metered-dose inhaler with spacer, and reassess breathing within 15 to 20 minutes. — Albuterol relaxes the smooth muscle wrapped around swollen airways within minutes, opening them so air can move; quick reassessment detects patients who do not improve and need systemic corticosteroids or emergency escalation.
3. Monitor respiratory rate, oxygen saturation, peak flow (if able), and work of breathing (use of neck muscles, flaring nostrils, pulling between ribs) every 15 to 30 minutes during an acute episode. — Worsening respiratory rate over 30, dropping oxygen saturation, declining peak flow, or increasing muscle use signals failing lungs and the need for higher-level care before respiratory arrest occurs.

4. Prepare to give or administer systemic corticosteroid (prednisone 40 to 60 milligrams by mouth, or methylprednisolone 40 to 80 milligrams intravenously) for moderate to severe exacerbations that do not quickly improve with albuterol. — Systemic corticosteroids reduce the inflammation (swelling) inside the airways over 4 to 6 hours, preventing the flare-up (exacerbation) from continuing or returning after bronchodilator wears off.
5. Assess and document the patient's ability to speak in full sentences, mental alertness, and presence or absence of wheezing sounds. — Speaking only short phrases, confusion, or silent chest despite distress are signs of severe airway obstruction and impending respiratory failure requiring immediate physician notification and possible intubation.
6. Keep the patient calm, reduce environmental triggers (strong smells, cold air, known allergens), and avoid sedatives or anxiolytics unless ordered by a physician. — Anxiety increases breathing effort and oxygen demand; however, sedatives can dangerously suppress the respiratory drive in someone already struggling to breathe, leading to respiratory arrest.
7. Teach correct inhaler technique using teach-back method—shake, exhale fully, press and inhale slowly, hold breath for 10 seconds—and confirm the patient can demonstrate it before discharge. — Up to 70 percent of patients use inhalers incorrectly, so medicine stays in the mouth instead of reaching deep into the swollen airways; proper technique ensures the drug deposits where inflammation exists.
8. Review the difference between daily controller medicine (inhaled corticosteroid taken every day to reduce swelling) and rescue medicine (albuterol taken only for sudden symptoms), and confirm the patient has both at home. — Many patients stop controller inhalers when they feel better, leaving the airway inflammation untreated; this leads to repeated emergency visits and higher risk of life-threatening asthma attacks.

Sources

- GINA 2024 Global Strategy for Asthma Management and Prevention
- NHLBI Expert Panel Report 4 (EPR-4) 2020
- Cochrane Airways Group
- ATS/ERS Standards for Asthma

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