

Chronic Asthma

A plain-English guide for you and your family

WHAT IS HAPPENING

In healthy lungs, the airways stay open and relaxed so air flows smoothly in and out. In asthma, the airways become swollen all the time—white blood cells called eosinophils (say ee-oh-SIN-oh-fills), mast cells, and lymphocytes pile up in the airway walls and pour out chemicals that cause swelling, extra thick mucus, and tightness in the ring of muscle around each breathing tube. Triggers like pollen, a cold virus, exercise, chilly air, smoke, or even acid bubbling up from the stomach can set off a flare. During an attack, the airways squeeze so narrow that air whistles on the way out (that is the wheeze), breathing becomes hard work, and coughing tries to clear the gunk. A rescue puff (a short-acting beta-agonist like albuterol) relaxes the tight muscle fast, proving the narrowing can reverse. But the relief only lasts a few hours because the swelling underneath never left. That is why a daily controller medicine—an inhaled corticosteroid, or steroid mist—is essential: it quiets the white-cell alarm at the source, keeps swelling low, and stops attacks before they start. Without it, leaning on the rescue inhaler again and again actually raises the risk of a life-threatening attack.

What you might feel

- Wheezing
- Shortness of breath
- Chest tightness
- Cough (often dry or with thick, sticky mucus)
- Waking up at night coughing or wheezing
- Symptoms triggered or worsened by exercise, cold air, or allergens (pollen, pet dander, dust mites)
- Breathing out takes much longer than breathing in (prolonged expiratory phase)
- Using neck, shoulder, and belly muscles to breathe (accessory muscle use)
- Cannot speak a full sentence without stopping to breathe
- Silent chest (little or no wheezing even though the person looks very sick and is struggling to breathe)

Call the doctor right away if...

- 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

What the care team will do

- Sit the patient fully upright (high Fowler position) and apply oxygen by nasal cannula or mask to keep oxygen saturation above 90 percent.
- 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.
- 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.
- 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.
- Assess and document the patient's ability to speak in full sentences, mental alertness, and presence or absence of wheezing sounds.
- Keep the patient calm, reduce environmental triggers (strong smells, cold air, known allergens), and avoid sedatives or anxiolytics unless ordered by a physician.
- 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.
- 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.

Medicines you may be given — and why

Albuterol (short-acting beta-agonist, SABA)

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.

Inhaled corticosteroid (ICS): budesonide, fluticasone, beclomethasone, mometasone, ciclesonide

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.

Inhaled corticosteroid + formoterol (ICS-formoterol): budesonide-formoterol, beclomethasone-formoterol, mometasone-formoterol

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.

Inhaled corticosteroid + long-acting beta-agonist (ICS-LABA): fluticasone-salmeterol, budesonide-formoterol, mometasone-formoterol, fluticasone-vilanterol, beclomethasone-formoterol

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).

Long-acting muscarinic antagonist (LAMA): tiotropium, umeclidinium

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.

Omalizumab

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.

Mepolizumab, reslizumab

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.

Benralizumab

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.

Dupilumab

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.

Oral corticosteroid (prednisone, prednisolone, methylprednisolone) - short course for flare-up (exacerbation)

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.

Leukotriene receptor antagonist (montelukast, zafirlukast)

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).

Theophylline

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).

Cromolyn sodium (rarely used)

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.

Where this information comes from

- 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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