

Anaphylaxis

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally, mast cells sit quietly in your tissues—skin, lungs, gut wall, blood vessel walls—wearing IgE antibodies on their surface like guards with specific alarms. Each IgE molecule recognizes only one threat, so when a mast cell meets its target, it releases chemicals like histamine (tells blood vessels to widen and leak) and leukotrienes (squeeze airways shut) in one small, controlled patch, then the reaction stops. In anaphylaxis, that control breaks down. An allergen—peanuts, shellfish, a drug like penicillin, a bee sting, or latex—locks onto IgE on mast cells everywhere in your body at once. Instead of a small local response, mast cells dump their chemical cargo all at the same moment: histamine, leukotrienes (chemicals that squeeze airways and make vessels leaky), tryptase (an enzyme that breaks down tissue), and prostaglandins (chemicals that drive swelling) flood your bloodstream. Blood vessels relax and widen throughout your body, so blood pressure drops—like opening a dam and letting water spread into a wide, shallow pool. Fluid leaks out of vessel walls into tissues, causing swelling in your throat, face, lips, and gut. Airways tighten, making it hard to breathe. Your skin breaks out in raised, itchy welts called hives and turns red. This cascade happens within minutes to two hours after you touch, breathe, or swallow the allergen. Your body tries to fight back—your heart speeds up to keep blood flowing, you feel anxious or dizzy as your brain senses trouble—but without an immediate shot of epinephrine (a medicine that squeezes blood vessels back tight and opens airways), the reaction can spiral into life-threatening shock or airway closure. In five to twenty out of every hundred cases, a second wave hits four to twelve hours later even after symptoms improve. Every sign traces back to that first moment when the allergen triggered a whole-body mast-cell explosion.

What you might feel

- Hives (urticaria) and skin flushing
- Angioedema (swelling of face, lips, tongue, or throat)
- Difficulty breathing, wheezing, or a high-pitched noise on breathing in (stridor) (high-pitched sound when breathing in)
- Low blood pressure (Hypotension) (low blood pressure) and dizziness or fainting

- A fast heart rate (Tachycardia) (fast heart rate)
- Nausea, vomiting, belly cramping, and diarrhea
- Sense of impending doom or severe anxiety
- No skin findings in up to 20 out of 100 cases

Call the doctor right away if...

- A high-pitched noise on breathing in (Stridor), hoarseness, or trouble swallowing—signs the upper airway is swelling and may close off completely
- Low blood pressure (Hypotension) or fainting—the cardiovascular system is collapsing into distributive shock
- Severe trouble breathing, blue skin (cyanosis), or oxygen saturation below 90 percent—the lungs are failing to get enough oxygen into the blood
- Confusion, loss of consciousness, or seizure—the brain is not getting enough blood or oxygen
- Symptoms coming back 4 to 12 hours after the first wave improves (biphasic reaction)—up to 20 out of 100 people have a second round that can be just as dangerous

What the care team will do

- Give intramuscular epinephrine 0.3 to 0.5 mg (1:1000 concentration) in the outer mid-thigh immediately, then prepare to repeat every 5 to 15 minutes if symptoms do not improve.
- Assess and support the airway—look for a high-pitched noise on breathing in (stridor), hoarseness, drooling, or tongue swelling; sit the patient upright if they are having trouble breathing, and prepare suction and bag-valve-mask ventilation equipment at the bedside.
- Start two large-bore intravenous lines and give a rapid bolus of 1 to 2 liters of normal saline in adults (20 mL per kg in children), repeating boluses if blood pressure stays below 90 systolic.
- Lay the patient flat and elevate the legs unless they are vomiting or having severe trouble breathing, in which case keep the head of the bed up 30 to 45 degrees.
- Place the patient on continuous pulse oximetry and cardiac monitoring, checking oxygen saturation and heart rate and rhythm every 1 to 2 minutes during the emergency phase.
- Give supplemental oxygen by non-rebreather mask at 15 liters per minute if oxygen saturation is below 94 percent or the patient looks short of breath.
- Prepare second-line medications at the bedside—H1 antihistamine (diphenhydramine 25 to 50 mg IV), H2 antihistamine (famotidine 20 mg IV), corticosteroid (methylprednisolone 125 mg IV or equivalent), and if the patient takes a beta-blocker, draw up glucagon 1 to 2 mg IV.

- Monitor the patient for at least 4 to 6 hours after symptoms resolve, and arrange longer observation or admission if the reaction was severe, involved low blood pressure (hypotension), or required more than one dose of epinephrine.

Medicines you may be given — and why

Epinephrine (adrenaline) 0.3-0.5 mg IM into mid-outer thigh, repeat every 5-15 minutes if needed

Epinephrine is the only life-saving drug in anaphylaxis because it fixes the three deadly problems at once. First, it squeezes blood vessels by turning on alpha-1 adrenergic receptors (switches on the muscle layer inside artery and vein walls). This pushes blood pressure back up and stops shock. Second, it opens airways by turning on beta-2 adrenergic receptors (switches on the muscle wrapped around breathing tubes). This relaxes the muscle so air can move freely again. Third, it tells mast cells—the cells that dumped the allergic chemicals—to stop releasing histamine (the main chemical that causes itching, swelling, and leaking) and other chemicals by turning on beta-2 adrenergic receptors on those cells. No other drug works this fast or fixes all three problems. It must go into muscle (IM), not under the skin, because muscle has more blood vessels. The drug reaches the bloodstream in 3 to 5 minutes instead of 20 to 30. The outer thigh (vastus lateralis muscle, the big muscle on the outside of your thigh) is the best spot because the muscle is big, close to the surface, and has great blood flow.

IV normal saline or lactated Ringer's, 1 to 2 liters rapid bolus (adults); 10 to 20 mL per kilogram bolus (children)

In anaphylaxis, histamine and other chemicals punch holes in capillaries (the tiniest blood vessels). Fluid leaks out of the bloodstream into the surrounding tissues. This is called third-spacing or capillary leak. The result is that the blood volume drops sharply. Blood pressure falls and organs do not get enough oxygen (distributive shock). Giving a large volume of IV fluid fast refills the blood vessels, raises the circulating volume, and brings blood pressure back up. Normal saline (0.9% sodium chloride, salt water with the same saltiness as blood) and lactated Ringer's (a balanced salt solution with sodium, chloride, potassium, calcium, and lactate) are isotonic. This means they have the same concentration of particles as blood, so they stay in the vessels longer. They do not rush into cells or out of the body right away. Epinephrine tightens the vessels and IV fluids fill them. Together they reverse shock.

Diphenhydramine 25 to 50 mg IV or IM (adults); 1 mg per kilogram IV or IM, max 50 mg (children)

Diphenhydramine blocks H1 histamine receptors on blood vessels, airways, and skin. Histamine—released by mast cells during anaphylaxis—normally locks onto these receptors and causes blood vessels to widen and leak, smooth muscle in airways to tighten, and skin to

itch and swell. By sitting in the receptor and blocking histamine from binding, diphenhydramine reduces itching, hives, swelling, and some of the airway tightness. However, it works slowly (peak effect in 1 to 3 hours). It does NOT reverse life-threatening shock or severe airway closure. This is why it is only a helper drug and never replaces epinephrine.

Methylprednisolone 125 mg IV or prednisone 40 to 60 mg by mouth (adults); methylprednisolone 1 to 2 mg per kilogram IV (children)

Corticosteroids enter immune cells (mast cells, basophils, T cells, macrophages) and travel into the nucleus (the cell's control center). There they turn on anti-inflammatory genes and turn off pro-inflammatory genes. This reduces the production of cytokines (inflammatory messenger chemicals such as interleukin-5 and tumor tissue death (necrosis) factor-alpha), leukotrienes (fat chemicals that tighten airways and make vessels leak), and prostaglandins (chemicals that cause fever, pain, and swelling). The result is less inflammation over the next 4 to 12 hours. Corticosteroids are given in anaphylaxis to reduce the chance of a biphasic reaction (a second wave of symptoms 4 to 12 hours later, which happens in 5 to 20% of cases) and to calm down long-lasting inflammation. However, steroids take 4 to 6 hours to start working. They do NOT treat the immediate life-threatening symptoms and never replace epinephrine.

Albuterol 2.5 to 5 mg nebulized or 4 to 8 puffs via inhaler with spacer, repeat every 20 minutes if needed

Albuterol is added if wheezing or trouble breathing continues after epinephrine. It locks onto beta-2 adrenergic receptors on the smooth muscle that wraps around the breathing tubes (bronchioles). This turns on a pathway inside the muscle cell that raises cyclic AMP (cAMP, a chemical messenger inside the cell). This lowers calcium and causes the muscle to relax. The airways widen (bronchodilation), air flows more easily, and wheezing goes down. Albuterol works only in the lungs. It does NOT raise blood pressure or stop mast cells from releasing histamine, so it is never a substitute for epinephrine. It is used as an add-on when airway tightness persists even after you give IM epinephrine.

Glucagon 1 to 2 mg IV or IM, repeat every 5 minutes if needed (for patients on beta-blockers who do not respond to epinephrine)

Glucagon is used when a patient on a beta-blocker (such as metoprolol, atenolol, propranolol) does not respond to epinephrine. Beta-blockers sit on beta-1 (heart) and beta-2 (lung, vessel) adrenergic receptors and stop epinephrine from binding, so epinephrine cannot raise heart rate, boost the heart's pumping strength, or fully open airways. Glucagon works through a completely different receptor—the glucagon receptor—which turns on the same downstream pathway (raising cyclic AMP) without needing the beta-receptor. This allows glucagon to speed up the heart, strengthen heart squeezes, and relax airway smooth muscle even when beta-receptors are blocked. Glucagon also raises blood sugar by telling the liver to break down

glycogen (stored sugar) into glucose. This can help if the patient has low blood sugar (hypoglycemia) from long-lasting shock.

Famotidine 20 mg IV (H2 antihistamine, optional add-on)

About 10 to 15% of histamine's effects in anaphylaxis happen through H2 histamine receptors (most happen through H1 histamine receptors). H2 histamine receptors are found on blood vessels, heart, and stomach. When histamine binds to H2 histamine receptors on blood vessels, it causes the vessels to widen and leak, which drops blood pressure. Blocking H2 histamine receptors with famotidine reduces this effect and may help steady blood pressure a bit when added to an H1 antihistamine blocker (such as diphenhydramine). However, H2 antihistamine blockers work slowly and are not essential. They are optional add-ons, never replacements for epinephrine or fluids.

Where this information comes from

- World Allergy Organization Anaphylaxis Guidance 2020
- American Academy of Allergy, Asthma & Immunology Practice Parameter 2020
- Resuscitation Council UK Emergency Treatment of Anaphylaxis Guidelines 2021
- UpToDate

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