

Acute Dyspnea

Sudden trouble breathing—give oxygen first, then figure out which of four main systems broke: airway (blocked or squeezed shut), lung tissue (filled, collapsed, or clotted off), heart (too weak to pump), or body chemistry (demanding more oxygen than the lungs can deliver).

THE ONE-LINE TRUTH

When someone suddenly cannot breathe, give oxygen right away, then figure out why.

The chain — how it unfolds

1. One upstream break—airway blocked, alveoli filled or collapsed, heart failing to pump, or body demanding more oxygen than healthy lungs can deliver—triggers the cascade. — why: Every abnormal finding you see downstream is the body's attempt to compensate for that one break. Think mechanistically: what broke, and what is the body doing to try to fix it? The compensations are not random—they follow predictable cause-and-effect chains.
2. Fast breathing (Tachypnea) (fast breathing, over 20 breaths per minute) pushes more air per minute (minute ventilation = respiratory rate × tidal volume). — why: The medulla (brainstem breathing center at the base of the brain) detects low oxygen (hypoxemia— PaO_2 below 60 mmHg or SaO_2 below 90 percent) through peripheral chemoreceptors (oxygen sensors in the carotid bodies in the neck and aortic arch in the chest) or high carbon dioxide (hypercapnia— PaCO_2 above 45 mmHg) through central chemoreceptors (CO_2 sensors in the medulla itself that actually sense pH changes in cerebrospinal fluid caused by CO_2) and tells the diaphragm and intercostal muscles to contract faster. Breathing 30 times per minute instead of 12 triples the amount of air moving in and out, trying to pull in more oxygen and blow off more CO_2 . This is the first and most consistent sign of respiratory distress—always check and document the respiratory rate.

3. Accessory muscles—sternocleidomastoid (thick neck muscles running from behind the ears to the collarbone), scalenes (small neck muscles that lift the first two ribs), intercostals (muscles between the ribs), and abdominal muscles—activate to pull in bigger breaths (increased tidal volume). — why: Fast breathing (Tachypnea) alone is not enough if each breath is shallow. The body recruits extra muscles to suck harder and expand the chest more, pulling in bigger gulps of air. You see this as retractions (skin pulling in above the collarbone—supraclavicular, between the ribs—intercostal, or below the ribcage—subcostal—because the strong negative pressure inside the chest sucks soft tissue inward) and visible neck muscle strain—the sternocleidomastoid muscles stand out like thick ropes. The patient may flare their nostrils (trying to pull in more air through the nose) and use abdominal muscles to force air out faster. These are signs of severe distress—the patient is working as hard as they can to breathe.
4. Low oxygen in the blood (Hypoxemia) (low oxygen saturation, SaO_2 below 90 percent or PaO_2 below 60 mmHg on arterial blood gas) causes a fast heart rate (tachycardia) (fast heart rate) and altered mental status (confusion, agitation, then drowsiness). — why: If oxygen saturation stays low despite fast breathing (tachypnea), the heart races (tachycardia—often 110 to 140 beats per minute or higher) to pump blood faster, delivering whatever oxygen is available to the brain and vital organs more quickly. The sympathetic nervous system (the body's fight-or-flight system) releases epinephrine (adrenaline) and norepinephrine (stress hormones that speed the heart and constrict blood vessels to shunt blood to the brain and heart). Low oxygen in the blood (Hypoxemia) also directly impairs brain function—mild low oxygen in the blood (oxygen saturation 85 to 90 percent) causes confusion, poor judgment, and anxiety because the cerebral cortex (the thinking and awareness part of the brain) is very sensitive to low oxygen; severe low oxygen in the blood (oxygen saturation below 80 percent) causes lethargy, stupor, and loss of consciousness because the brainstem and deeper brain structures shut down when starved for oxygen. The progression is predictable: first agitation and confusion, then drowsiness and lethargy, then unresponsiveness.

5. Hypercapnia (high blood carbon dioxide, PaCO₂ above 45 mmHg on arterial blood gas) causes drowsiness, confusion, headache, and depressed mental status (CO₂ narcosis). — why: If the lungs cannot blow off carbon dioxide fast enough—because air is not moving (severe airway obstruction from asthma or COPD, shallow breathing from exhausted muscles), alveoli are not working (ARDS filling air sacs with fluid, severe pneumonia clogging them with pus), or the breathing drive itself is suppressed (opioid overdose depressing the medulla)—CO₂ builds up in the blood. CO₂ crosses the blood-brain barrier easily and acts like a sedative on neurons (brain cells), making the patient sleepy, confused, and eventually unresponsive. This is respiratory acidosis (acid building up from trapped carbon dioxide) (blood pH drops below 7.35 because CO₂ reacts with water to form carbonic acid, which breaks down into hydrogen ions that lower pH), and it is a sign that ventilation (air movement in and out of the lungs) is failing. Early signs are headache (from dilated blood vessels in the brain caused by high CO₂), flushed skin, and confusion; late signs are severe drowsiness, stupor, and coma.
6. Positioning, speech pattern, and breath sounds are diagnostic clues pointing back to which system broke. — why: How the patient sits tells you how hard they are working: tripod position (leaning forward with hands on knees or table) means severe distress, often seen in COPD flare-up (exacerbation) or severe asthma because it recruits accessory muscles (pectoralis, sternocleidomastoid) and opens the airways by pulling the ribcage up and forward. Whether they can speak full sentences tells you how much air they are moving: one-word answers (the patient says 'help' then gasps, 'me' then gasps) mean severe trouble breathing (dyspnea) because they have to stop and breathe after every word; two- to three-word sentences mean moderate distress; full sentences without stopping mean mild or no distress. What sounds you hear on lung exam tells you what is in the alveoli: wheezing (musical squeaky whistling sounds, louder on exhale, mean squeezed small airways from bronchospasm in asthma or COPD), a high-pitched noise on breathing in (stridor) (harsh high-pitched sound on inhale, heard best over the neck, means upper airway obstruction from swelling in anaphylaxis, epiglottitis, or foreign body), crackles (wet popping or crackling sounds like velcro tearing, heard on inhale, mean fluid in alveoli from pulmonary edema (fluid flooding the lungs) or pus from pneumonia), or silent chest (barely any breath sounds at all because almost no air is moving—extremely dangerous, means severe obstruction or exhaustion and the patient is about to stop breathing). Retractions (skin sucking in) tell you they are working hard to pull air in against high resistance—the harder they suck, the worse the obstruction or stiffness.

7. If compensation fails—respiratory muscles fatigue, CO₂ rises, mental status worsens, breathing slows or becomes shallow—respiratory arrest (breathing stops) follows within minutes. — why: Fast breathing (Tachypnea) and accessory muscle use are exhausting. The diaphragm and intercostal muscles run out of ATP (adenosine triphosphate—the energy molecule muscles need to contract), oxygen debt builds up in the muscle cells, and they simply cannot contract anymore. Breathing becomes shallow and slow (bradypnea—respiratory rate drops below 10 breaths per minute), tidal volume (air per breath) plummets, CO₂ climbs sharply (PaCO₂ may reach 70, 80, or higher), the rising CO₂ sedates the medulla (brainstem breathing center) and shuts down the breathing drive, and the patient stops breathing (respiratory arrest). Oxygen saturation crashes to 50, 40, or lower, the heart slows (bradycardia from severe low oxygen in the blood (hypoxemia) damaging the heart's electrical system), and cardiac arrest follows within minutes. This is why you intubate (place a breathing tube) and start mechanical ventilation ****before**** the patient crashes, not after. The goal is to see impending respiratory failure—tiring out (accessory muscle use, retractions, paradoxical breathing where the belly sucks in instead of pushing out), rising CO₂ on blood gas, worsening confusion or new drowsiness—and act before the arrest. Once the patient stops breathing, your chances of a good outcome drop sharply.

8. Treat the upstream break, not just the compensatory response. — why: Fast breathing (Tachypnea), accessory muscle use, a fast heart rate (tachycardia), and altered mental status are the body's emergency attempts to maintain oxygenation (oxygen delivery to tissues = cardiac output $\times$ oxygen content in blood) and ventilation (CO₂ removal from the blood). They are compensations, not the disease itself. You must fix the root cause: open the airway (intubate if complete obstruction, give epinephrine 0.3 to 0.5 mg intramuscular for anaphylaxis to reverse airway swelling by constricting blood vessels and relaxing smooth muscle, remove foreign body with laryngoscopy and Magill forceps or emergent cricothyrotomy if cannot intubate), reverse bronchospasm (albuterol nebulizer for asthma—a beta-agonist that relaxes airway smooth muscle, plus ipratropium—an anticholinergic that blocks airway constriction, and systemic corticosteroids like methylprednisolone to reduce inflammation), drain the fluid (furosemide 40 mg IV for heart failure pulmonary edema (fluid flooding the lungs)—a loop diuretic that makes the kidneys dump fluid to reduce blood volume and lower pressure in the lungs, chest tube for pneumothorax to let trapped air escape and re-expand the lung), support the heart (inotropes like dobutamine to strengthen the pump by increasing contractility, treat life-threatening arrhythmias with synchronized cardioversion—controlled shock to reset the heart rhythm—or antiarrhythmic medications), kill the infection (antibiotics like ceftriaxone plus azithromycin for community-acquired pneumonia covering *Streptococcus pneumoniae* and atypicals, vancomycin plus piperacillin-tazobactam for hospital-acquired pneumonia covering MRSA and *Pseudomonas*, source control for sepsis—drain abscesses, remove infected catheters), or correct the metabolic problem (insulin infusion plus IV fluids for DKA to shut off ketoacid production and dilute the acid, sodium bicarbonate if pH is severely low below 7.1 and causing life-threatening arrhythmias, transfuse packed red blood cells for severe anemia with hemoglobin below 7 g/dL to restore oxygen-carrying capacity). Giving supplemental oxygen (nasal cannula, high-flow nasal cannula, non-rebreather mask, or bag-valve-mask ventilation) supports the patient and buys time while you fix the break, but oxygen alone does not fix the break. Always ask: what broke upstream, and how do I fix it? The compensations will stop once you fix the root cause—fast breathing slows, accessory muscles relax, heart rate comes down, mental status clears—but only if you treat the disease, not just the symptoms.

Spot it — what you see, and why

- Fast breathing (Tachypnea) (breathing faster than 20 breaths per minute in adults): The brainstem (the part at the base of your brain that runs breathing on autopilot) has sensors that watch oxygen and carbon dioxide levels in your blood. When oxygen drops or carbon dioxide climbs, the brainstem speeds up breathing to pull in more oxygen and blow off extra CO₂.

- Accessory muscle use (you can see neck, shoulder, or belly muscles working hard during breathing): The diaphragm (the big dome-shaped muscle under your lungs that does most of the breathing work) can't pull in enough air by itself. Extra muscles in your neck (the sternocleidomastoids), between your ribs (the intercostals), and in your belly jump in to help expand the chest and drag more air into the lungs with each breath.
- Intercostal or supraclavicular retractions (skin sucks in between the ribs or above the collarbone when you breathe in): When you struggle to pull air in, the pressure inside your chest drops so low that it pulls the soft skin and tissue inward like a vacuum. This shows the lungs are working very hard—either the airways are blocked, the lungs are stiff and won't stretch, or something is squeezing them from outside.
- Inability to speak full sentences (can only get out 2 to 3 words per breath): Breathing is so fast and takes so much effort that there's no air left over to push through your vocal cords and make sound. Every bit of oxygen you pull in is needed just to keep your body and brain alive, so you can only squeeze out a few words before you have to gasp again.
- A high-pitched noise on breathing in (Stridor) (a loud, high-pitched squeaky sound when breathing in): Air rushes through a narrowed upper airway—like your larynx (voice box) or trachea (windpipe)—making a squeaky noise. The narrowing can come from swelling (like in anaphylaxis or croup), something stuck in the throat, or a tumor or infection pressing on the airway.
- Wheezing (a musical, whistling sound when breathing out): Air squeezes through narrowed small airways (the bronchioles) deep in the lungs. The narrowing happens when smooth muscle around the airways tightens (like in asthma or anaphylaxis) or thick mucus clogs the tubes (like in COPD or severe asthma). The fast, squeezed airflow vibrates the airway walls and makes a whistle.

Red flags — act now

- Silent chest (no breath sounds) in someone struggling to breathe or having an asthma or COPD attack—means almost no air is moving and breathing may stop completely any second.
- Confusion, drowsiness, or not responding—means the brain isn't getting enough oxygen or carbon dioxide is building up dangerously high in the blood.
- Can only speak 2 to 3 words per breath—means breathing is taking every bit of energy and the person is in severe respiratory distress.
- A high-pitched noise on breathing in (Stridor) (high-pitched squeaky sound when breathing in)—means the upper airway is blocked or swelling shut and could close off completely, cutting off all air.

- Blue lips, tongue, or nail beds even with oxygen being given—means oxygen levels are dangerously low (usually below 85 percent) and the lungs can't deliver enough oxygen to the blood.

Treat it — in this order

1. Supplemental oxygen titrated to target saturation (88–92% for COPD, 94–98% for all others) → Fills the broken gas exchange step by getting oxygen into the blood. This stops the low-oxygen chain reaction that makes the heart race and brain confused, and prevents tissue low oxygen (hypoxia) (oxygen starvation, where cells cannot make energy and start to die).
2. Epinephrine intramuscular (0.3–0.5 mg in thigh) for anaphylaxis, or nebulized albuterol (2.5–5 mg) plus ipratropium (0.5 mg) for asthma or COPD flare-up → Epinephrine stops the immune-driven swelling (angioedema, tissue puffing from leaked fluid) and airway tightening (bronchoconstriction, squeezing of airway smooth muscle) that blocks the airway in anaphylaxis by binding alpha-1 receptors (squeezes blood vessels, shrinks swelling) and beta-2 receptors (relaxes airway smooth muscle). Bronchodilators—albuterol relaxes airway smooth muscle by activating beta-2 receptors; ipratropium blocks acetylcholine (a tightening messenger released by the vagus nerve) via muscarinic receptors on smooth muscle—open the lower airways so air moves easier and breathing slows.
3. Systemic corticosteroids (methylprednisolone 125 mg IV, or prednisone 40–60 mg by mouth, or dexamethasone 6–12 mg IV or by mouth) for asthma flare-up, COPD flare-up, or anaphylaxis → Steroids suppress the inflammatory response (immune system overreaction that calls in white blood cells and causes swelling) by blocking the production of inflammatory mediators like leukotrienes and prostaglandins and reducing white blood cell migration. This reduces airway lining swelling (mucosal swelling (edema)) and mucus production so breathing gets easier over 4–6 hours. Effect peaks at 4–6 hours and lowers relapse risk.
4. Loop diuretic (furosemide 40–80 mg IV) plus nitroglycerin (sublingual 0.4 mg or IV starting 5–10 mcg/min) plus noninvasive positive pressure ventilation (NIPPV: BiPAP or CPAP) for acute cardiogenic pulmonary edema (fluid flooding the lungs) → Diuretic (furosemide) blocks the Na-K-2Cl cotransporter in the thick ascending limb of the loop of Henle (the kidney tube that recycles salt and water), pulling fluid out of lungs back into blood and then into urine. Nitroglycerin dilates veins (venodilation, widening of veins that carry blood back to the heart), reducing preload (blood volume returning to heart) so the heart does not overfill. BiPAP (bilevel positive airway pressure) or CPAP (continuous positive airway pressure) pushes air in with positive pressure (typically 5–10 cm H₂O for CPAP, 10/5 cm H₂O inspiratory/expiratory for BiPAP), opening collapsed alveoli and forcing oxygen across the alveolar-capillary membrane (thin wall between air sacs and blood vessels) faster, reducing work of breathing.

5. Needle decompression (14- or 16-gauge catheter at 2nd rib space midclavicular line or 5th rib space front of armpit line) or chest tube (thoracostomy) for tension pneumothorax → Releases the trapped air pressing on the lung and great vessels (superior vena cava and aorta, the main blood return and output pipes), letting the lung re-expand so air movement and venous return (blood returning to heart) work again and oxygen can enter blood. Needle decompression works in seconds; blood pressure and oxygen saturation improve right away.

Your top priorities at the bedside

1. Place the patient on continuous pulse oximetry (the finger or toe clip that measures oxygen) and apply supplemental oxygen immediately to keep saturation 88 to 92 percent if the patient has COPD or 94 to 98 percent for everyone else. — why: Low oxygen in the blood (Hypoxemia) (low blood oxygen) damages the brain, heart, and kidneys within minutes. Oxygen fixes low oxygen in the blood fast while you investigate the cause. COPD patients need a lower target because too much oxygen can shut down their drive to breathe.
2. Sit the patient completely upright at 90 degrees or let them lean forward with arms propped on the bedside table. — why: Upright positioning uses gravity to pull the diaphragm (breathing muscle) down and lets the lungs expand more fully. This increases tidal volume (the amount of air moving in and out with each breath) and lowers the work of breathing.
3. Obtain a full set of vital signs including respiratory rate, heart rate, blood pressure, temperature, and oxygen saturation, and repeat every 5 to 15 minutes until the patient is stable. — why: Fast breathing (Tachypnea) (fast breathing), a fast heart rate (tachycardia) (fast heart rate), low blood pressure (hypotension) (low blood pressure), and fever tell you if the patient is in shock, has sepsis, or is decompensating (getting worse). Trends show if treatment is working or if you need to escalate care.