

# Acute Dyspnea

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## THE STORY IN ONE SENTENCE

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

“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).”

## Normal anatomy — the body's job

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Breathing moves air in and out through open airways—your windpipe (trachea) and the branching tubes (bronchi and bronchioles) inside your lungs. Blood flows through tiny vessels (capillaries) wrapped around millions of tiny air sacs (alveoli) deep in your lungs. Gas exchange—oxygen jumping into the blood, carbon dioxide jumping out—happens across the thin walls between air sacs and capillaries. A control center in your brainstem (the medulla, the breathing control center at the base of your brain) watches oxygen and carbon dioxide levels in your blood and adjusts how fast and how deep you breathe to keep them balanced. Picture this: air flows in, oxygen hops into the blood, carbon dioxide hops out, and air flows out—all automatic, all the time. Keep this picture in mind, because every abnormal finding in acute trouble breathing (dyspnea) (sudden trouble breathing) is a break in one of these jobs.

## BODY'S JOB

The airways stay open so air can flow freely. The alveoli (air sacs) stay dry and thin-walled so oxygen and carbon dioxide can jump across easily. The heart pumps blood through the lung capillaries at just the right speed—not too fast, not too slow. The medulla (the breathing control center in your brainstem) adjusts breathing rate and depth automatically to match what your body needs moment by moment. If you do not know these normal jobs first, the abnormal findings will seem random and confusing. They are not random—they are clues pointing back to which job broke.

1. Air flows in through the nose or mouth, down the windpipe (trachea), and into branching airways (bronchi and bronchioles) that reach every part of the lung.

2. Oxygen from the air diffuses (moves) across the thin walls of the alveoli (air sacs) into the blood flowing through capillaries wrapped around them.
3. At the same time, carbon dioxide (CO<sub>2</sub>, the waste gas cells make) diffuses out of the blood into the alveoli so you can breathe it out.
4. The heart pumps oxygen-rich blood from the lungs out to the body, and pumps oxygen-poor blood back to the lungs to pick up more oxygen.
5. The medulla (breathing control center in your brainstem) watches oxygen and carbon dioxide levels in your blood through special sensors (chemoreceptors) in your arteries and brain and adjusts how fast and how deep you breathe to keep them in the safe zone.

## What breaks

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One thing breaks upstream, and every abnormal finding you see downstream points back to that one break. The job is to read the clues backward to find it.

**CLINICAL TERM**

Four main categories cause acute trouble breathing (dyspnea) (sudden trouble breathing). **Airway obstruction** (something blocking the airway—foreign body stuck in the throat, swelling from anaphylaxis or infection like epiglottitis) or **bronchospasm** (airway muscles squeezing tight—asthma flare-up (exacerbation) triggered by allergens or infection, COPD flare-up from infection or irritants) prevents air from moving in and out. **Lung parenchymal problems** (parenchyma means lung tissue) disrupt gas exchange: **pneumonia** (bacterial or viral infection filling alveoli with pus and fluid), **pulmonary edema** (fluid flooding the lungs) (fluid leaking into alveoli from backed-up blood, usually from heart failure or ARDS), **pulmonary embolism** (a clot lodging in a lung artery) (blood clot blocking lung vessels so blood cannot pick up oxygen), **pneumothorax** (air leak into the chest cavity collapsing the lung), or **ARDS** (acute respiratory distress syndrome—severe inflammation damaging alveoli, often from sepsis, pneumonia, or aspiration of stomach contents). **Cardiac causes** reduce forward blood flow or cause blood to back up into the lungs: **acute heart failure** (left ventricle too weak to pump forward, so blood backs up into the lungs and leaks fluid into alveoli), **cardiac tamponade** (the heart squeezed by fluid around it) (fluid in the pericardial sac around the heart squeezing it so it cannot fill or pump), or **arrhythmia** (abnormal heart rhythm like atrial fibrillation (an irregular, quivering heartbeat) or ventricular a fast heart rate (tachycardia) dropping cardiac output). **Metabolic or systemic causes** increase the body's oxygen demand or acid load beyond what normal lungs can handle: **sepsis** (severe infection raising oxygen demand body-wide and causing lactic acidosis (acid from oxygen-starved tissues) from poor tissue blood flow (perfusion)), **diabetic ketoacidosis** (the acid crisis of missing insulin) (DKA) (dangerous acid buildup from uncontrolled diabetes forcing the lungs to blow off extra carbon dioxide to compensate), **severe anemia** (hemoglobin below 7 g/dL—too few red blood cells to carry oxygen, so the lungs must work harder to load what little hemoglobin is left), or **metabolic acidosis** (acid building up in the blood) (acid overload from kidney failure, poisoning, lactic acidosis from shock forcing the lungs to hyperventilate to blow off CO<sub>2</sub> and raise pH). That break does not stay local—it triggers a cascade of compensations. The downstream signal you will actually see at the bedside is **fast breathing (tachypnea)** (fast breathing rate over 20 breaths per minute) pushing more air per minute. Read all the abnormal findings backward from there—they all point to this one broken thing upstream.

## How the body compensates

The body keeps vital signs looking relatively stable for a while by ramping up breathing effort and heart rate. This is the dangerous middle zone, because stable vital signs can fool the team into missing how close the patient really is to respiratory failure. The job is to see **impending**

respiratory failure\*\*—when compensation is about to stop working—before the crash point arrives. Once compensation fails, the window to reverse course closes fast.

1. {'what': 'Fast breathing (Tachypnea) (fast breathing) increases minute ventilation (total air moved per minute).', 'why': 'The medulla (brainstem breathing center in the lower back of the brain) detects low oxygen through peripheral chemoreceptors (special sensors in the carotid bodies at the fork in the carotid arteries in the neck and in the aortic arch in the chest that sense oxygen levels in the blood) or high carbon dioxide through central chemoreceptors (sensors in the medulla itself that sense CO<sub>2</sub> and pH in cerebrospinal fluid) and tells the diaphragm and intercostal muscles (breathing muscles between the ribs) to contract faster. Breathing 30 times per minute instead of 12 moves more air in and out, buying time.', 'limit': 'Fast breathing (Tachypnea) is exhausting. The diaphragm and intercostal muscles (breathing muscles) fatigue because they are working nonstop without rest, and the patient cannot keep it up forever. Watch for slowing respiratory rate in a patient who was previously tachypneic—it may mean they are tiring out and about to stop breathing, not that they are improving. A respiratory rate dropping from 35 to 8 in a still-distressed patient is a dire sign—they are crashing.'}
2. {'what': 'Accessory muscles (neck, rib, and belly muscles) activate to pull in bigger breaths.', 'why': 'The diaphragm alone cannot keep up, so the body recruits sternocleidomastoid (thick neck muscles that lift the ribcage up), scalenes (small neck muscles that also lift the first ribs), intercostals (muscles between ribs that expand the chest outward), and abdominal muscles (that push the diaphragm up during exhale to empty the lungs faster and make the next inhale easier) to increase tidal volume (air per breath). You see this as retractions (skin sucking in above the collarbone, between ribs, or below the ribcage because the strong negative pressure inside the chest sucks soft tissue inward) and visible neck muscle strain—the sternocleidomastoid muscles pop out like thick ropes running from behind the ears down to the collarbone.', 'limit': 'Using accessory muscles is hard work and not sustainable. It is a sign of severe respiratory distress and means the patient is working extremely hard just to move air. These muscles were not designed to breathe nonstop—they fatigue quickly.'}

3. {'what': 'A fast heart rate (Tachycardia) (fast heart rate) compensates for low oxygen delivery.', 'why': 'If oxygen saturation drops, the heart races (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. Think of it like this: if each bucket of blood carries less oxygen (because hemoglobin saturation is low or there are fewer red blood cells in anemia), you need to move more buckets per minute to deliver the same total amount of oxygen.', 'limit': 'A fast heart rate (Tachycardia) increases the heart's own oxygen demand (the heart muscle itself needs oxygen to pump that fast), which can worsen myocardial starved blood flow (ischemia) (low blood flow to the heart muscle causing chest pain or heart attack) if the patient has underlying coronary artery disease. It is also not sustainable long-term because the heart cannot rest between beats—the filling time (diastole) gets shorter and shorter, which can drop cardiac output and blood pressure.'}]
4. {'what': 'The patient sits upright and leans forward (tripod position).', 'why': 'This position uses gravity to help the diaphragm move down (abdominal organs fall away from it, giving it more room to contract) and expands the chest cavity, making it easier to breathe. Splinting the arms on the knees or table lets the pectoralis muscles (chest muscles) and accessory neck muscles pull harder on the ribcage to lift it up and expand the chest. Patients with severe trouble breathing (dyspnea) refuse to lie flat because lying down makes it worse by pushing abdominal organs up against the diaphragm (reducing how far it can move) and letting fluid spread across more lung surface in pulmonary edema (fluid flooding the lungs).', 'limit': 'If a patient who was sitting bolt upright suddenly lies down or slumps over, it may mean they are exhausted and about to decompensate—not that they feel better. Always ask why the position changed.'}]

5. {'what': 'Watch for signs that compensation is failing: altered mental status (confusion, agitation, then drowsiness), rising CO<sub>2</sub> (PaCO<sub>2</sub> above 50 mmHg) on arterial blood gas, silent chest (no breath sounds because almost no air is moving), and paradoxical breathing (belly moves in during inhale instead of out).', 'why': 'These are late signs of impending respiratory failure. Confusion or agitation means the brain is not getting enough oxygen (hypoxemia affects the cerebral cortex—the thinking and awareness part of the brain—and causes confusion, poor judgment, and anxiety). Drowsiness or lethargy means carbon dioxide is building up and sedating the brain (CO<sub>2</sub> crosses the blood-brain barrier easily and acts like an anesthetic on neurons, causing CO<sub>2</sub> narcosis). A silent chest means the patient is barely moving air despite working hard—airway obstruction is severe or the breathing muscles are so fatigued they cannot generate enough force to move air. Paradoxical breathing (abdominal paradox—belly sucks in when they try to breathe in instead of pushing out) means the diaphragm is so fatigued it is moving the wrong way—it gets sucked up by negative pressure in the chest instead of pulling down to expand the lungs.', 'limit': 'Once you see these signs, the patient is minutes away from respiratory arrest (breathing stops completely). The decision to intubate (place a breathing tube through the mouth into the trachea and connect the patient to a mechanical ventilator) is based on clinical signs of failing compensation—worsening work of breathing, rising CO<sub>2</sub>, altered mental status—not just one oxygen number. Do not wait for oxygen saturation to hit zero; act when the trend shows compensation is running out.'}
6. {'what': 'Look at the trend over time, not just one snapshot.', 'why': 'A patient with stable vital signs on a downhill trend—oxygen saturation slowly dropping from 94 to 92 to 89 percent despite increasing supplemental oxygen flow, respiratory rate slowly climbing from 24 to 28 to 32 breaths per minute, mental status slowly worsening from alert to confused to drowsy—is not truly stable. They are compensating now, but compensation is running out. Serial assessments (checking respiratory rate, work of breathing, accessory muscle use, ability to speak, mental status, and oxygen saturation every 15 to 30 minutes) are critical to catch the downhill slide before the crash.', 'limit': 'Do not wait for the numbers to crash. Act when you see the trend heading toward failure, because once the crash happens—respiratory rate suddenly drops, patient becomes unresponsive, oxygen saturation plummets—the patient may suffer cardiac arrest before you can secure the airway. It is easier and safer to intubate a tired, awake patient than to intubate during a full code (cardiac arrest resuscitation).'} }

## Domino effect

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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. — 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  $\times$  tidal volume). — The medulla (brainstem breathing center at the base of the brain) detects low oxygen (hypoxemia— $\text{PaO}_2$  below 60 mmHg or  $\text{SaO}_2$  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— $\text{PaCO}_2$  above 45 mmHg) through central chemoreceptors ( $\text{CO}_2$  sensors in the medulla itself that actually sense pH changes in cerebrospinal fluid caused by  $\text{CO}_2$ ) 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  $\text{CO}_2$ . 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). — 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,  $\text{SaO}_2$  below 90 percent or  $\text{PaO}_2$  below 60 mmHg on arterial blood gas) causes a fast heart rate (tachycardia) (fast heart rate) and altered mental status (confusion, agitation, then drowsiness). — 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,  $\text{PaCO}_2$  above 45 mmHg on arterial blood gas) causes drowsiness, confusion, headache, and depressed mental status ( $\text{CO}_2$  narcosis). — 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)— $\text{CO}_2$  builds up in the blood.  $\text{CO}_2$  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  $\text{CO}_2$  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  $\text{CO}_2$ ), 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. — 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, CO<sub>2</sub> rises, mental status worsens, breathing slows or becomes shallow—respiratory arrest (breathing stops) follows within minutes. — 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, CO<sub>2</sub> climbs sharply (PaCO<sub>2</sub> may reach 70, 80, or higher), the rising CO<sub>2</sub> 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 CO<sub>2</sub> 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. — 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 (CO<sub>2</sub> 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.

## Symptoms · what the patient feels, and why

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- 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 CO<sub>2</sub>.

- 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.
- Crackles or crackles in the lungs (rales) (popping or crackling sounds when breathing in): Tiny air sacs (alveoli) or small airways that were stuck shut suddenly pop open as air rushes in. This happens when the sacs fill with fluid (from heart failure pushing fluid into the lungs, or pneumonia filling them with pus) or when scar tissue from chronic lung disease makes the lungs stiff and collapsed sacs snap open.
- Silent chest (very quiet or no breath sounds even though the person is trying hard to breathe): The airways are squeezed so tightly shut—or plugged so completely with thick mucus—that almost no air moves in or out. No air movement means no sound. This is a life-threatening emergency because the lungs can't exchange oxygen and carbon dioxide at all, and breathing may stop completely within minutes.
- Bluish skin (Cyanosis) (lips, tongue, or nail beds turn blue or purple): The blood doesn't have enough oxygen (usually oxygen saturation drops below 85 to 90 percent). Hemoglobin (the protein inside red blood cells that carries oxygen) without oxygen looks dark blue instead of bright red. You see the blue color first in thin-skinned areas like the lips, tongue, and under the fingernails.

- Altered mental status (confusion, agitation, or unusual sleepiness): The brain needs a steady supply of oxygen to think clearly and stay awake. Low oxygen (hypoxemia) makes people confused, restless, or agitated. High carbon dioxide (hypercapnia, which piles up when you can't breathe out enough) acts like a sedative and makes people drowsy, sluggish, or even unresponsive. Both mean the lungs are failing to do their job and the body can't keep up.

## Focused exam

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- Oxygen saturation below target for the patient type: In acute trouble breathing (dyspnea) (sudden shortness of breath), low oxygen is the first sign that the body is not swapping gases well. COPD (chronic obstructive pulmonary disease, long-term lung damage that blocks air flow) patients need 88–92%; everyone else needs 94–98%. Below target means the broken step is stealing oxygen from the blood.
- Fast breathing (respiratory rate above 20 breaths per minute in adults): Fast breathing is the body's main defense when something is broken upstream—it pushes more air to try to fix low oxygen, high carbon dioxide, or blood flow trouble. In acute trouble breathing (dyspnea), this is the central finding that all others build on.
- Use of neck and rib muscles (accessory muscles), skin pulling in with each breath (retractions), or belly breathing backwards (paradoxical abdominal motion): When fast breathing is not enough, the body recruits extra muscles to push harder. In acute trouble breathing (dyspnea), this means the lungs are working against something—stiff lungs, blocked airways, or a heart that cannot pump well. This is a warning that the body is tiring and running out of backup fixes.
- Unable to speak full sentences, only words or phrases: Speaking requires breath. If a patient in acute trouble breathing (dyspnea) cannot finish a sentence, the breathing muscles are so busy keeping oxygen up that they have nothing left for talking. This tells you the body is at its limit.
- Crackles (rales), wheezing (whistling sounds), a high-pitched noise on breathing in (stridor) (high-pitched noise), or silent chest in severe asthma: In acute trouble breathing (dyspnea), the type of sound tells you which broken step: crackles mean fluid in alveoli (tiny air sacs where oxygen crosses into blood)—from heart failure or pneumonia; wheezing means lower airways are narrowed (asthma, COPD flare-up); a high-pitched noise on breathing in (stridor) means upper airway is swollen (anaphylaxis, epiglottitis, foreign body). Silent chest in severe asthma is a red flag—air is too thick to move and almost no breathing is happening.

- Heart rate above 100 beats per minute (tachycardia), confusion (altered mental status), or sleepiness (somnolence): Low oxygen makes the heart race and the brain foggy. High carbon dioxide makes you sleepy. In acute trouble breathing (dyspnea), these signs mean the broken step is serious enough to harm the brain and heart, not just the lungs.

## Diagnostics · every test answers a question

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- Pulse oximetry (oxygen saturation measurement) → Is the body getting enough oxygen into the blood? Does the patient meet target saturation for their condition?
- Arterial blood gas (ABG) → Is carbon dioxide building up? Is the blood too acidic (acidosis, pH below 7.35) or too basic (alkalosis, pH above 7.45)? Is the body compensating (adjusting pH by breathing or kidney work)?
- Chest X-ray → Is there fluid, infection, air, or mass in the chest? Is the heart enlarged?
- B-type natriuretic peptide (BNP or NT-proBNP) → Is the heart stretched and failing? Is heart failure causing the trouble breathing (dyspnea)?
- Cardiac troponin (I or T) → Is heart muscle dying (heart attack or acute coronary syndrome, ACS, where a blocked artery starves heart muscle)? Is the heart under severe stress?
- D-dimer → Is there a blood clot breaking down somewhere in the body? Should I look for PE or a clot in a deep leg vein (deep vein thrombosis) (DVT, clot in leg veins)?
- CT pulmonary angiography (CTPA, computed tomography scan with contrast dye to see lung blood vessels) or ventilation-blood flow (perfusion) (V/Q) scan → Is there a clot blocking the pulmonary arteries (PE)?
- Electrocardiogram (ECG, heart tracing showing electrical rhythm) → Is the heart in a dangerous rhythm? Are there signs of heart attack or right heart strain from PE?

## Treatment logic

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- 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).

- 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.
- 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.
- 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 H<sub>2</sub>O for CPAP, 10/5 cm H<sub>2</sub>O 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.
- 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.

- Anticoagulation (unfractionated heparin 80 units/kg IV bolus then 18 units/kg/hour drip targeting aPTT 1.5–2.5 times control, or low-molecular-weight heparin such as enoxaparin 1 mg/kg subcutaneously twice daily, or direct oral anticoagulant such as apixaban 10 mg twice daily for 7 days then 5 mg twice daily) plus thrombolytic therapy (alteplase 100 mg IV over 2 hours) if massive or high-risk PE with hemodynamic instability (systolic BP below 90 mmHg or signs of shock) → Anticoagulation stops the clot from growing by blocking the coagulation cascade (clotting protein chain reaction—heparin activates antithrombin III, a natural brake protein, which then blocks thrombin and factor Xa, two key clotting proteins). Thrombolytic (alteplase activates plasminogen to plasmin, an enzyme that breaks fibrin strands holding the clot together) dissolves the clot so blood can flow back through pulmonary arteries and gas exchange restarts. Blood pressure and oxygen saturation improve within hours.
- Antibiotics (ceftriaxone 1–2 g IV daily plus azithromycin 500 mg IV or by mouth daily, or levofloxacin 750 mg IV or by mouth daily) for community-acquired pneumonia causing acute trouble breathing (dyspnea) → Antibiotics kill bacteria filling the alveoli (air sacs) with pus and inflammatory fluid. Ceftriaxone (a cephalosporin, cell-wall-busting antibiotic) covers *Streptococcus pneumoniae* (most common pneumonia bacteria) and *Haemophilus influenzae* (common bacteria, especially in COPD patients); azithromycin (a macrolide) or levofloxacin (a fluoroquinolone) adds coverage for atypical bacteria (*Mycoplasma pneumoniae*, *Legionella pneumophila*, *Chlamydia pneumoniae*, bacteria without standard cell walls). Within 24–48 hours, fever drops, white blood cell count falls, and oxygen exchange improves as alveoli clear.

## Medications

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### Oxygen (titrated to target saturation) · Supplemental gas

Pure oxygen raises the amount of oxygen in every breath so more can cross from your lungs into your blood. It does not fix what caused the low oxygen, but it buys time while doctors treat the real problem. Think of it like turning up the volume when the radio signal is weak — it helps you hear better, but the real fix is fixing the antenna.

1. Oxygen flows through a mask or nasal prongs into your nose and throat.
2. It travels down the airways into the alveoli.
3. Higher oxygen levels in the alveoli push more oxygen across the thin membrane into blood.
4. Red blood cells pick up oxygen and carry it to every tissue in your body.

**SAFETY WATCH**

In patients with COPD (lungs damaged by smoking or pollution), aim for oxygen saturation 88–92%. Too much oxygen can turn off their breathing drive because their brain learned to trigger breaths based on low oxygen instead of high carbon dioxide. · In all other patients, aim for saturation 94–98%. Higher levels waste oxygen and can hurt the lungs over days. · Watch for worsening carbon dioxide buildup (confusion, extreme sleepiness) in COPD patients on oxygen. · Oxygen helps things burn — keep it away from open flames and do not smoke near oxygen equipment.

**Albuterol · Short-acting beta-2 agonist (SABA) bronchodilator**

Albuterol flips a switch on the muscle cells that wrap around your airways, telling them to relax. When the muscle relaxes, the airway opens wider so air can flow in and out easily. Think of it like loosening a tight belt around your chest — you can suddenly take a deeper breath.

1. Albuterol is breathed in as a mist from an inhaler or nebulizer machine.
2. It lands directly on airway muscle cells.
3. Albuterol locks onto beta-2 receptors (tiny docking sites) on the muscle surface.
4. This triggers a chemical chain reaction inside the cell that raises cyclic AMP (a messenger molecule).
5. Cyclic AMP lowers calcium levels inside the muscle cell.
6. Low calcium tells the muscle to relax.
7. The airway widens and air flows freely.

**SAFETY WATCH**

Can cause fast heart rate (palpitations), shaking hands, and nervousness because beta-2 receptors also sit on heart and nerve cells. · May raise blood sugar slightly in people with diabetes. · Using it more than every 4 hours regularly is a red flag that asthma or COPD is not controlled and you need a daily controller medicine. · Safe in pregnancy when needed — untreated asthma is more dangerous to the baby than the drug.

**Ipratropium · Short-acting anticholinergic bronchodilator**

Ipratropium blocks acetylcholine (a chemical messenger that normally tells airway muscles to squeeze). By blocking the signal, the muscle stays relaxed and the airway stays open. It works alongside albuterol but uses a different route, so together they open airways better than either alone.

1. Ipratropium is breathed in as a mist, often mixed with albuterol in one nebulizer treatment.
2. It lands on airway muscle cells.

3. Ipratropium blocks muscarinic receptors (docking sites for acetylcholine).
4. Without acetylcholine's signal, the muscle cannot squeeze.
5. The airway stays open and air flows easily.

**SAFETY WATCH**

Can cause dry mouth and throat because it blocks saliva production. · Avoid spraying mist into the eyes — it can blur vision and raise eye pressure (dangerous in glaucoma). · Use carefully in men with enlarged prostate — it can make urination harder. · Safe in pregnancy and breastfeeding.

**Methylprednisolone or Prednisone · Corticosteroid (steroid anti-inflammatory)**

Corticosteroids turn down the entire alarm system causing swelling in the airways. They shrink swelling, reduce mucus, and make airways less twitchy. Think of swelling like a fire in the airway walls — steroids are the fire extinguisher that calms everything down over hours to days.

1. Steroid enters the bloodstream (oral pill or IV) or is breathed in directly onto airway surfaces.
2. It crosses into cells and locks onto glucocorticoid receptors inside the cell nucleus.
3. The steroid-receptor pair turns off genes that make swelling proteins (cytokines, leukotrienes).
4. It also turns on genes that make anti-swelling proteins.
5. Over 4 to 6 hours, swelling shrinks, mucus decreases, and airways become less sensitive to triggers.

**SAFETY WATCH**

Short courses (5 days) are usually safe. Watch for high blood sugar in people with diabetes (steroids raise glucose). · Can cause upset stomach, mood swings, trouble sleeping, and bigger appetite. · Long-term use (weeks to months) causes bone thinning, weight gain, high blood pressure, and stops the body from making its own steroids. · Never stop steroids suddenly after long-term use — taper slowly to let the adrenal glands wake up. · Safe in pregnancy when needed — untreated severe asthma is more dangerous.

**Furosemide · Loop diuretic**

Furosemide makes the kidneys dump salt and water into the urine. This shrinks the total fluid volume in the body, which takes pressure off the heart and pulls fluid back out of the lungs.

Think of the bloodstream like an overfilled water balloon — furosemide opens a drain so the balloon shrinks and stops leaking.

1. Furosemide enters the bloodstream (IV or oral pill).
2. It travels to the kidneys and is pushed into the urine tubes.
3. It blocks the sodium-potassium-chloride cotransporter (NKCC2 pump) on the thick ascending limb of the loop of Henle.
4. Salt (sodium) and water cannot be pulled back into the blood, so they stay in the urine.
5. Large volumes of urine are made (diuresis means peeing more).
6. Blood volume drops, pressure in the veins falls, and fluid is pulled back out of the lungs into the bloodstream, then out through urine.

#### **SAFETY WATCH**

Can cause low potassium (hypokalemia), which triggers dangerous heart rhythms. Check potassium levels and replace as needed. · Can cause dehydration and low blood pressure (dizziness, fainting) if too much fluid is removed. · Worsens kidney function if you are already dehydrated. · Can cause ringing in the ears (ototoxicity) at very high IV doses. · Watch urine output — if you do not urinate within 1 hour of the IV dose, the kidneys may not be responding.

### **Nitroglycerin · Nitrate vasodilator**

Nitroglycerin relaxes the smooth muscle in blood vessel walls, especially veins. When veins widen, blood pools there instead of returning to the heart. This takes pressure off the heart and lungs. Think of it like opening a relief valve on a pressure cooker — the steam (fluid) spreads out and the pressure drops.

1. Nitroglycerin is given under the tongue (sublingual tablet), as a paste on the skin, or through an IV.
2. It is changed inside blood vessel cells into nitric oxide (a gas that signals muscle to relax).
3. Nitric oxide turns on guanylate cyclase, an enzyme that makes cyclic GMP (a messenger molecule).
4. Cyclic GMP lowers calcium in the muscle cells.
5. The muscle relaxes, the vessel widens, and blood pressure drops.
6. Blood pools in the veins, reducing the volume of blood returning to the heart (preload).
7. The heart does not have to work as hard, and fluid backs up less into the lungs.

**SAFETY WATCH**

Can cause severe drop in blood pressure (hypotension) leading to dizziness, fainting, or shock. Monitor blood pressure every 5 to 10 minutes when starting IV nitroglycerin. · Causes headache in most patients (from widening of brain blood vessels). · Never give to patients taking erectile dysfunction drugs (sildenafil, tadalafil, vardenafil) — the combination can cause life-threatening blood pressure drop. · Do not give if systolic blood pressure is below 90 or if the patient has right ventricular heart failure (fluid backs up differently and nitroglycerin can worsen it). · The body gets used to it quickly (within 24 hours of continuous use), so on-and-off dosing or breaks are needed.

**Heparin (unfractionated) or Enoxaparin (low-molecular-weight heparin) · Anticoagulant (blood thinner)**

Heparin does not dissolve clots that already exist. Instead, it stops the clot from growing bigger and prevents new clots from forming while the body's own enzymes slowly dissolve the clot over days to weeks. Think of it like putting out a campfire — heparin does not remove the burnt logs, but it stops new wood from catching fire.

1. Heparin is injected under the skin (subcutaneous) or into a vein (IV).
2. It locks onto antithrombin III in the blood.
3. The heparin-antithrombin team grabs and shuts down thrombin (factor IIa) and factor Xa (two key enzymes in the clotting chain).
4. Without thrombin, fibrin threads cannot form, so no new clot builds.
5. The existing clot stays the same size or slowly shrinks as the body's plasmin enzyme digests it.

**SAFETY WATCH**

Risk of bleeding anywhere (gums, urine, stool, brain). Watch for bruising, blood in urine or stool, severe headache, or sudden weakness. · Unfractionated heparin requires blood tests (aPTT) every 6 hours to adjust dose. Enoxaparin dose is based on weight and usually does not need monitoring (except in kidney failure or pregnancy). · Heparin-induced a low platelet count (thrombocytopenia) (HIT) is a rare but life-threatening reaction where platelets drop and clots form. Check platelet count at baseline and every 2 to 3 days. If platelets drop by half, stop heparin right away and switch to a different blood thinner. · Antidote for unfractionated heparin is protamine sulfate (reverses it within minutes). Enoxaparin is harder to reverse. · Use carefully in patients with recent surgery, bleeding ulcers, or brain bleeding (hemorrhage).

## Alteplase (tPA) · Thrombolytic (clot-dissolving agent)

Alteplase is a copy of the enzyme your body uses to dissolve old clots. It wakes up plasminogen (a sleeping protein in the blood) and turns it into plasmin (the active enzyme), which chews up the fibrin threads holding the clot together. Think of it like pouring drain cleaner on a clog — it actively breaks down the blockage so blood can flow again.

1. Alteplase is given through an IV over 15 minutes to 2 hours (depending on the condition).
2. It locks onto fibrin threads in the clot.
3. It changes plasminogen (inactive) into plasmin (active enzyme).
4. Plasmin cuts the fibrin threads into small pieces.
5. The clot dissolves and blood flow is restored within minutes to hours.

### SAFETY WATCH

High risk of bleeding, including brain bleeding (hemorrhage) (1 to 2% risk). Absolutely cannot be given if you have had recent surgery, trauma, or bleeding in the brain. · Do not give if blood pressure is above 185/110 (risk of brain bleed). · Do not give if you had a stroke or head injury in the past 3 months, active bleeding, or bleeding disorder. · Monitor thinking and strength every 15 minutes during and after the drug. Any sudden headache, confusion, or weakness means stop the drug right away and get a head CT. · Used only in ICU or emergency settings where the risk of death from the clot is higher than the risk of bleeding.

## Epinephrine · Sympathomimetic (adrenaline)

Epinephrine is the body's emergency hormone. It does three things at once: (1) squeezes blood vessels to raise blood pressure, (2) relaxes airway muscles to open breathing passages, and (3) stops mast cells from dumping histamine (the chemical that causes allergic swelling). It is the only drug that can reverse anaphylaxis (life-threatening allergic reaction). Think of it as flipping the body's fight-or-flight switch to maximum.

1. Epinephrine is injected into the thigh muscle with an auto-injector (EpiPen) or given IV in the hospital.
2. It enters the bloodstream within seconds.
3. It locks onto alpha and beta receptors throughout the body.
4. Blood vessels tighten, raising blood pressure and pushing blood to vital organs.
5. Airway muscles relax, opening breathing passages.
6. Mast cells stop dumping histamine, so swelling and hives begin to reverse.
7. Effects peak in 3 to 5 minutes and last 10 to 20 minutes.

**SAFETY WATCH**

Causes fast heart rate, pounding heart, shaking, nervousness, and sweating (normal side effects). · Can trigger dangerous heart rhythms in patients with heart disease. · Raises blood pressure — can cause stroke or heart attack if pressure goes too high. · Can raise blood sugar. · Never give into a vein unless in a hospital with continuous monitoring — it can cause severe high blood pressure or heart rhythm problems. · Safe in pregnancy — anaphylaxis kills mother and baby, so epinephrine is always given when needed.

**Antibiotics (varies by pathogen and setting) · Antimicrobial agents**

Antibiotics kill bacteria or stop them from multiplying. Each class targets a different part of the bacterial cell: some punch holes in the cell wall (beta-lactams like amoxicillin-clavulanate), some stop protein assembly (macrolides like azithromycin), and some break bacterial DNA (fluoroquinolones like levofloxacin). The right antibiotic depends on which bacteria are most likely in that setting (community, hospital, nursing home) and your allergies and kidney function.

1. Antibiotic is swallowed (oral pill) or given through a vein (IV).
2. It enters the bloodstream and reaches the lungs.
3. It gets into infected tissue and locks onto bacterial targets (cell wall enzymes, ribosomes, or DNA machinery).
4. Bacteria stop multiplying or die.
5. Your immune system cleans up dead bacteria and debris over 2 to 5 days.

**SAFETY WATCH**

Watch for allergic reactions: rash, hives, swelling, or anaphylaxis (rare but life-threatening with penicillins and cephalosporins). · Fluoroquinolones (levofloxacin, moxifloxacin) can cause tendon rupture, nerve damage, and mental health side effects — FDA black box warnings recommend avoiding them for mild infections. · Macrolides (azithromycin) can cause heart rhythm problems (QT prolongation) and are avoided in patients with heart disease. · All antibiotics can cause diarrhea and *Clostridioides difficile* (C. diff) infection (severe diarrhea from killing good gut bacteria). · Doses must be adjusted for kidney or liver disease. · Always finish the full course (usually 5 to 7 days for pneumonia) to prevent resistance, even if you feel better.

**Noninvasive Positive Pressure Ventilation (NPPV: BiPAP or CPAP) · Mechanical respiratory support (not a drug)**

NPPV is a tight-fitting mask that pushes air into the lungs under pressure. BiPAP delivers two pressures: higher when you breathe in (to inflate collapsed air sacs and push fluid out) and

lower when you breathe out (to make exhaling easier). CPAP delivers steady pressure. Think of it like inflating a partly deflated basketball — pressure pushes the walls open so air can fill it again.

1. A mask is strapped snugly over the nose or nose and mouth.
2. A machine pushes air in at a set pressure (usually 8 to 20 cm H<sub>2</sub>O).
3. Positive pressure inflates collapsed alveoli so oxygen can cross into the blood.
4. In heart failure, the pressure pushes fluid back out of the air sacs into blood vessels.
5. In COPD with high carbon dioxide, the pressure helps weak breathing muscles and blows off excess CO<sub>2</sub>.
6. You breathe along with the machine, reducing the effort needed to inflate the lungs.

#### SAFETY WATCH

Can cause skin breakdown or pressure sores on the bridge of the nose or forehead from the tight mask. Check skin every 2 hours. · Risk of aspiration (breathing in stomach contents) if you vomit while wearing the mask — use only in awake, cooperative patients. · Can cause dry mouth, stuffy nose, and bloating (air swallowed into the stomach). · If you become more confused, sleepy, or stop breathing with the mask on, you need immediate intubation (breathing tube inserted into the airway). · Not effective in massive pulmonary embolism (a clot lodging in a lung artery), severe pneumonia with shock, or conditions that need surgery (tension pneumothorax).

## Red flags

- 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.

## Nursing priorities

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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. — 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. — 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. — 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.
4. Establish intravenous access with at least one large-bore IV (18-gauge or bigger) and draw blood for labs including complete blood count, basic metabolic panel, troponin (heart enzyme), B-type natriuretic peptide or BNP (heart failure marker), and D-dimer (clotting marker) if ordered. — A large IV lets you give emergency fluids, medications, or blood products quickly if the patient crashes. Labs help confirm or rule out heart attack, heart failure, pulmonary embolism (a clot lodging in a lung artery), infection, and kidney failure.
5. Prepare to give nebulized albuterol 2.5 to 5 milligrams plus ipratropium 0.5 milligrams for wheezing or known asthma or COPD, or prepare intramuscular epinephrine 0.3 to 0.5 milligrams in the thigh if you see hives, lip swelling, or a high-pitched noise on breathing in (stridor) (high-pitched squeaky breathing sound). — Bronchodilators (albuterol and ipratropium) relax and open tight airways in asthma and COPD so air can move in and out again. Epinephrine reverses anaphylaxis by stopping airway swelling and raising blood pressure before the patient goes into respiratory or cardiac arrest.
6. Perform a focused lung assessment every 15 to 30 minutes, listening for wheezes (high-pitched whistling), crackles (popping or bubbling sounds), absent breath sounds, or a silent chest (no sounds even though the patient is working hard to breathe). — Wheezes mean bronchospasm. Crackles mean fluid in the lungs from heart failure or pneumonia. Absent breath sounds on one side mean pneumothorax (collapsed lung). A silent chest in someone struggling to breathe means almost no air is moving and they may stop breathing completely within seconds.

7. Keep suction equipment, a bag-valve mask (BVM), and intubation supplies at the bedside and notify the provider immediately if the patient becomes confused, drowsy, cannot speak, or has oxygen saturation below 90 percent despite high-flow oxygen. — Confusion and drowsiness mean the brain is not getting enough oxygen or carbon dioxide is building up to dangerous levels. These patients can stop breathing suddenly and need advanced airway management (intubation and mechanical ventilation) to stay alive.
8. Monitor for accessory muscle use (neck and shoulder muscles working with each breath), intercostal retractions (skin sucking in between the ribs), and inability to speak full sentences, and document these findings every 15 minutes. — These signs tell you the patient is using every muscle available to breathe and is at very high risk for respiratory failure (when breathing stops or becomes too weak to supply oxygen). Worsening signs mean you need to escalate to intensive care or prepare for intubation.

## Sources

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- American College of Emergency Physicians (ACEP) Clinical Policy on Acute Dyspnea
- American Heart Association / American College of Cardiology (AHA/ACC) Heart Failure Guidelines 2022
- Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024
- Global Initiative for Asthma (GINA) 2024

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