

Noninvasive Versus Invasive Ventilation

NIV (a mask that pushes air in gently) helps awake patients whose lungs can still heal — like someone with a COPD flare-up or fluid in the lungs from heart failure — while invasive ventilation (a breathing tube down the throat) is for patients who cannot protect their airway, are too tired to breathe, or whose lungs are so sick they need full control.

THE STORY IN ONE SENTENCE

NIV (a mask that pushes air in gently) helps awake patients whose lungs can still heal — like someone with a COPD flare-up or fluid in the lungs from heart failure — while invasive ventilation (a breathing tube down the throat) is for patients who cannot protect their airway, are too tired to breathe, or whose lungs are so sick they need full control.

“NIV works for awake patients with fixable lung problems; invasive (intubation) is for patients who cannot stay safe on a mask or whose lungs need full machine control.”

Normal anatomy — the body's job

Your lungs pull oxygen from the air into your blood and push carbon dioxide (the body's waste gas) out with every breath. The diaphragm (a big muscle under your lungs that looks like an upside-down bowl) pulls down to suck air in, and when it relaxes, air flows back out naturally. At the same time, tiny air sacs called alveoli (like microscopic balloons at the tips of your airways) wrap around blood vessels and swap oxygen for CO₂ in a split second. Keep that normal picture in your head, because every weird finding in breathing failure makes sense only as a change from this smooth rhythm.

BODY'S JOB

The diaphragm and chest muscles pull air in, the alveoli swap oxygen for CO₂, and the brainstem (the base of your brain where it meets the spine) sets the breathing rhythm automatically by watching CO₂ levels in the blood — when CO₂ climbs, the brainstem signals you to breathe faster to blow it off. Oxygen gets carried on red blood cells to every cell in the body, and CO₂ (the waste from cells burning fuel) rides back to the lungs to be dumped out. If any piece — the airways, the alveoli, the breathing muscles, or the brain's signal — breaks down, gas exchange fails and you cannot keep oxygen up or CO₂ down on your own.

1. Air flows in through the nose or mouth, down the trachea (windpipe), and splits into the bronchi (the two main branches, one to each lung).
2. The diaphragm pulls down and the rib muscles lift, creating negative pressure (suction) inside the chest that pulls air into the lungs.
3. Oxygen crosses from the alveoli into the blood, and CO₂ crosses from the blood into the alveoli, all in the time of a heartbeat.
4. The diaphragm relaxes, the chest shrinks back, and air flows out, carrying the waste CO₂ away.
5. The brainstem senses CO₂ rising in the blood and sends a signal to breathe again, keeping the cycle smooth and automatic even while you sleep.

What breaks

When the lungs cannot swap oxygen and CO₂ well — because airways are squeezed shut (COPD, asthma), alveoli are flooded (pneumonia, heart failure fluid, ARDS), alveoli are collapsed (atelectasis), or the breathing muscles are too weak or tired — oxygen in the blood drops (hypoxemia) and CO₂ builds up (hypercapnia). The body tries to fix this by breathing faster and harder, but that work burns energy fast and the muscles fatigue, pushing the patient toward respiratory failure.

CLINICAL TERM

Acute hypoxemic or hypercapnic respiratory failure from impaired gas exchange (ventilation-blood flow (perfusion) mismatch, shunt, hypoventilation, or diffusion defect) or increased work of breathing that exhausts respiratory muscles. Downstream effects you see at the bedside are low oxygen in the blood (hypoxemia) ($\text{SpO}_2 < 90\%$ or $\text{PaO}_2 < 60$ mmHg), hypercapnia ($\text{PaCO}_2 > 45$ – 50 mmHg and rising), fast breathing (tachypnea) (fast breathing > 24 – 30 /min), accessory muscle use (neck and shoulder muscles pulling to help breathe), and altered mental status from CO_2 retention (CO_2 narcosis) or low oxygen to the brain. All point upstream to the same broken step: lungs that cannot meet the body's oxygen demand or clear CO_2 , forcing a choice between noninvasive support (NIV) to assist spontaneous breathing or invasive ventilation (intubation) to take over the work completely.

How the body compensates

The body tries to keep oxygen up and CO_2 down by breathing faster and harder, pulling in more air with every breath. For a while, vital signs may look acceptable — oxygen saturations stay near 90%, heart rate rises to pump more blood, and blood pressure holds steady. But this is the dangerous middle: the breathing muscles are burning energy fast and will fatigue, and the patient is one step away from crashing. Your job is to see that crash point coming before it arrives, because once the muscles give out, the patient stops breathing and the window to help safely closes fast.

1. {'step': 'Fast breathing (Tachypnea) (fast breathing > 24 – 30 breaths/min) and accessory muscle use (neck, shoulder, and belly muscles pulling to help the diaphragm).', 'why': 'The brainstem senses rising CO_2 or low oxygen and signals the body to breathe faster and deeper to blow off CO_2 and pull in more oxygen. Using extra muscles is a red flag that the diaphragm is struggling and the work is unsustainable.'}
2. {'step': 'A fast heart rate (Tachycardia) (fast heart rate > 100 – 120 bpm) and mild high blood pressure (hypertension) (slightly high blood pressure).', 'why': 'The heart speeds up to pump more blood to the lungs and body, trying to deliver what little oxygen is available and clear CO_2 faster. This is the sympathetic nervous system (the body's fight-or-flight system) kicking in with adrenaline.'}
3. {'step': 'Low oxygen in the blood (Hypoxemia) partially corrected by supplemental oxygen (nasal cannula, face mask, high-flow nasal cannula).', 'why': 'Giving extra oxygen raises the amount in each breath, so even if the lungs are not swapping gases well, more oxygen molecules get across into the blood. This buys time but does not fix the underlying problem — if oxygen needs keep climbing or the patient tires, NIV or intubation is next.'}

4. {'step': 'Agitation, confusion, or somnolence (drowsiness) from hypercapnia (CO₂ narcosis) or low oxygen in the blood (hypoxemia).', 'why': 'High CO₂ acts like a sedative on the brain, making the patient sleepy and clouding thinking; low oxygen makes the brain fuzzy and the patient restless or confused. Either is a red flag that compensation is failing and the brain is not getting what it needs.'}
5. {'step': 'Retractions (skin pulling in between ribs or above the collarbone with each breath) and nasal flaring.', 'why': 'These are signs the body is working as hard as it can to pull air in; they are common in children but also seen in adults in severe distress. They signal that the breathing muscles are near exhaustion.'}
6. {'step': 'The crash point: respiratory muscle fatigue, worsening low oxygen in the blood (hypoxemia) or hypercapnia despite maximal noninvasive support, altered mental status, or inability to protect the airway.', 'why': 'Once the muscles fatigue, breathing slows and shallows (paradoxical breathing — the belly sucks in instead of pushing out), CO₂ spikes, oxygen drops, and the patient may stop breathing (apnea) or aspirate. This is the moment intubation must happen immediately, because waiting risks cardiac arrest or brain injury from prolonged low oxygen.'}

Domino effect

1. Lungs fail to swap oxygen for CO₂ — airways collapse, alveoli flood or collapse, or inflammation thickens the walls. — The upstream break: gas exchange stops working. Oxygen cannot get into the blood, and CO₂ cannot get out, so blood oxygen (PaO₂) drops and CO₂ (PaCO₂) climbs. Every downstream domino starts here.
2. Low oxygen in the blood (Hypoxemia) (low blood oxygen) and hypercapnia (high blood CO₂) trigger the brainstem to signal faster, deeper breathing. — The brainstem has chemoreceptors (sensors in the carotid arteries and aorta) that watch CO₂ and oxygen levels nonstop. When CO₂ rises or oxygen drops, they send a panic signal: breathe harder and faster to fix it. This is automatic — you cannot stop it.
3. Fast breathing (Tachypnea) (fast breathing), accessory muscle use, and increased work of breathing. — The diaphragm and chest muscles are now working overtime, pulling harder and faster to move more air. The body recruits neck, shoulder, and belly muscles to help. This burns energy (ATP) and oxygen fast — the muscles themselves start running out of fuel.
4. Sympathetic nervous system activates: a fast heart rate (tachycardia) (fast heart), high blood pressure (hypertension) (high blood pressure), and agitation or anxiety. — The body releases adrenaline (epinephrine) and norepinephrine to pump more blood, deliver more oxygen, and keep the brain and heart fed. The patient looks restless, sweaty, and panicked — this is the fight-or-flight response trying to buy time.

5. Respiratory muscle fatigue sets in — the diaphragm and accessory muscles run out of ATP and oxygen, and breathing becomes shallow and slow. — Muscles can only work at max effort for so long before they fatigue, just like sprinting until your legs give out. When the diaphragm fatigues, breaths get smaller and slower (hypoventilation), CO₂ spikes further, and oxygen drops dangerously. This is the tipping point where the patient is about to crash.
6. Altered mental status (confusion, somnolence, or agitation) from CO₂ narcosis (high CO₂ acting like a sedative) or low oxygen in the blood (hypoxemia) (low oxygen to the brain). — The brain needs steady oxygen and hates high CO₂. When CO₂ climbs above 50–60 mmHg, it crosses into the brain and acts like anesthesia, making the patient drowsy and slow to respond (CO₂ narcosis). Low oxygen makes the brain fuzzy, confused, or combative. Either way, the patient cannot protect their airway safely anymore, and intubation is urgent.
7. Without support (NIV or intubation), pauses in breathing (apnea) (stopping breathing), cardiac arrest (heart stops from low oxygen or high CO₂), or aspiration (breathing in vomit or secretions, causing pneumonia or airway blockage). — Once the muscles fatigue completely, the patient stops breathing. Low oxygen (hypoxia) and high CO₂ (respiratory too much acid in the blood (acidosis)) make the heart rhythm unstable, leading to arrest. If the patient is drowsy and vomits, they can aspirate (breathe it into the lungs), which blocks the airway or causes severe pneumonia. This is why we intervene before the crash: NIV if the patient is awake and the problem is fixable, intubation if they cannot protect their airway or are too far gone.
8. The fix: NIV (mask with CPAP or BiPAP) for awake patients with a reversible cause (COPD, heart failure, mild pneumonia); invasive ventilation (intubation) for patients who cannot stay safe on a mask, are too tired, or need full control while the lungs heal. — Both are bridges. NIV pushes air in gently to keep airways and alveoli open, making each breath easier and buying time for medications (steroids, diuretics, antibiotics) to work. Intubation takes over the work completely, protecting the airway and letting the machine deliver high oxygen and pressure while the lungs rest and heal. The goal is always to get the patient breathing on their own again as soon as the upstream break is fixed.

Symptoms · what the patient feels, and why

- Working hard to breathe — fast breathing, using neck and belly muscles to pull air in, flaring nostrils: The lungs are stiff, flooded, or blocked, so the person has to pull harder and faster to get enough oxygen in and carbon dioxide out
- Low oxygen level on the monitor (SpO₂ below 90% even with extra oxygen): Damaged or collapsed air sacs in the lungs cannot pick up oxygen from the air and load it onto red blood cells
- Fast heart rate (over 100–120 beats per minute): The heart tries to pump faster to deliver what little oxygen is in the blood to the organs that need it

- Confusion, sleepiness, or trouble staying awake: The brain is not getting enough oxygen, or carbon dioxide is building up in the blood and acting like a sedative
- Very fast breathing (over 25–30 breaths per minute): The body tries to blow off extra carbon dioxide or pull in more oxygen by speeding up breathing
- Sweating and looking anxious or exhausted: The work of breathing is like running a marathon — muscles tire out and the body releases stress hormones like adrenaline
- Speaking only in short bursts or single words instead of full sentences: The person cannot take a deep enough breath to speak a whole sentence without pausing for air
- Blue or gray color to the lips, fingernails, or skin (cyanosis): So little oxygen is loaded onto red blood cells that the blood itself looks dark and shows through the skin

Focused exam

- Alert and awake versus drowsy or confused: The mask (noninvasive ventilation, or NIV) only works if the person can stay awake, follow directions, and keep the mask on their face. If they are getting sleepy or confused, it means their brain is not getting enough oxygen or carbon dioxide (the trash gas the body needs to breathe out) is piling up in the blood. That tells you NIV is failing and you need to put a breathing tube down their throat (intubation) to take over the work of breathing completely.
- Work of breathing: are they using neck muscles, belly muscles, or pulling their ribs in to breathe?: The body only pulls out these backup muscles when the main breathing muscle (the diaphragm, the sheet of muscle below the lungs) cannot do the job alone. Mild extra work may get better with NIV. Severe extra work with exhaustion means the muscles will tire out fast — you need to intubate before they stop altogether.
- Oxygen level (SpO₂) on room air, then on NIV or extra oxygen: If oxygen stays low even with help, the lungs are badly damaged — either NIV is not strong enough, or the problem is not what you thought (like a blood clot in the lung or a collapsed lung). This number tells you if NIV can work or if you need intubation right away.
- Carbon dioxide level (end-tidal CO₂ monitor or arterial blood gas pCO₂) in the first hour of NIV: If CO₂ is high (above 45 mmHg) and not dropping in the first 1 to 2 hours on NIV, the lungs cannot blow off the trash gas fast enough — the person is tiring out. This is the number one sign NIV will fail and intubation is coming.
- Ability to cough and clear spit and mucus (secretions): NIV works only if the person can cough hard enough to spit out their own gunk. If they are too weak or too drowsy to cough, secretions pile up in the airways and block air from reaching the lung tissue. That makes NIV fail. Intubation with a tube that reaches into the lungs lets you suction the gunk out.

- Reason for lung failure: COPD flare, heart failure with lung fluid (pulmonary swelling (edema)), pneumonia, after removing a breathing tube (post-extubation), weakened immune system: The cause tells you which people NIV will help and which ones need intubation from the start. COPD flare-ups and heart failure with fluid in the lungs often improve with NIV. Unconsciousness, severe shock (very low blood pressure that organs are not getting enough blood), or risk of breathing in stomach contents (aspiration) means intubation now.
- Response at 1 to 2 hours: Is work of breathing less? Is oxygen better? Is the person calmer?: This is the moment of truth for NIV versus intubation. If the person is not improving, keeping NIV going wastes time and lets them get worse. You must intubate before exhaustion sets in and they stop breathing.

Diagnostics · every test answers a question

- Arterial blood gas (ABG) → Is oxygen getting into the blood, and is carbon dioxide being blown off? Is the blood turning acidic from too much CO₂ (respiratory too much acid in the blood (acidosis))?
- Chest X-ray → What is breaking the lungs — fluid, infection, collapse, or air leak?
- Oxygen saturation (SpO₂) by pulse oximeter → Is oxygen reaching the blood and tissues right now?
- End-tidal CO₂ (capnography monitor) → Is the person blowing off carbon dioxide with each breath?
- Brain natriuretic peptide (BNP or NT-proBNP) → Is heart failure the cause of the lung fluid and breathing trouble?
- Complete blood count (CBC) → Is there infection or bleeding causing the lung problem?
- Lactate → Are the tissues getting enough oxygen, or is the body going into shock?

Treatment logic

- Bilevel positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP) via face mask → Pushes air into the lungs without a tube down the throat, so the person stays awake and can cough, swallow, and talk. BiPAP pushes harder when they breathe in (helps move air) and softer when they breathe out (helps keep airways open). CPAP pushes steady pressure all the time (keeps fluid-filled air sacs open in heart failure). This buys time while the lungs heal from COPD flare-up or fluid backup from heart failure.
- Oxygen delivery (nasal cannula, face mask, or non-rebreather, titrated to target SpO₂ 88 to 92% in COPD, 94 to 98% in other causes) → Fills the blood with oxygen molecules so tissues stop screaming for air, which slows the breathing rate and stops exhaustion. Stops the cascade of low oxygen (hypoxia) driving panic, fast heart rate, and organ damage.

- Intubation with mechanical ventilation (endotracheal tube down the throat plus ventilator machine) → Takes over all the work of breathing by pushing air in and out on a set rhythm, so the exhausted respiratory muscles rest completely. The machine handles both oxygen delivery and CO₂ removal, which NIV cannot always do. Protects the airway from spit, vomit, or blood. Lets you suction out secretions deep in the lungs.
- Treat the upstream cause: bronchodilators (albuterol, ipratropium) and steroids for COPD; diuretics (furosemide) for pulmonary edema (fluid flooding the lungs); antibiotics for pneumonia → Fixes the actual broken part — opens airways in COPD by relaxing the muscle rings around them and calming inflammation, drains fluid in heart failure by making the kidneys pee out extra water, fights germs in pneumonia. This shrinks the work the lungs have to do so NIV or rest becomes enough.
- High-flow nasal cannula (HFNC) if low oxygen alone, no CO₂ buildup → Delivers warm, humidified, high-flow oxygen (up to 60 liters per minute) through soft prongs in the nose — easier to tolerate than a mask. Washes out the dead space (the first part of the airway that does not trade oxygen) so each breath counts more. Gives a little positive pressure to keep airways open.
- Head-of-bed elevation to 30 to 45 degrees, sitting upright, reduce or avoid sedation → Gravity helps fluid drain from the lungs, the diaphragm (main breathing muscle) works better when sitting up because the belly organs are not pushing on it, and staying alert keeps the throat muscles tight so the airway stays open and the person can cough out spit.
- Monitor for NIV failure and intubate early if no improvement in 1 to 2 hours → Prevents the dangerous wait-and-see trap where NIV fails slowly and the person crashes suddenly. Early intubation (before total exhaustion) is safer than late intubation (during a code). Guideline-supported thresholds: worsening mental status, rising CO₂, falling oxygen despite NIV, worsening work of breathing.

Medications

Non-Invasive Ventilation (NIV) — BiPAP or CPAP · Positive-pressure ventilation device (not a drug; a breathing support tool)

NIV blows pressurized air into the lungs through a tight-fitting mask so the patient does not have to work as hard to breathe. It pushes oxygen in and helps push carbon dioxide out. The pressure props open collapsed air sacs (alveoli) so gas exchange can happen. The patient stays awake, protects their own airway, and can take the mask off if needed.

1. The machine delivers air at two pressures: higher pressure during inhale (IPAP) and lower pressure during exhale (EPAP).
2. The positive pressure splints open collapsed or fluid-filled alveoli so oxygen can cross into the blood.

3. It lowers the work of breathing by assisting each breath, giving the tired respiratory muscles a chance to rest.
4. In COPD with hypercapnia (too much CO₂), NIV helps blow off carbon dioxide so it does not build up and make the blood acidic.
5. In cardiogenic pulmonary edema (fluid flooding the lungs) (fluid backing up into the lungs from a weak heart), the positive pressure reduces the fluid leak and improves oxygen delivery.

SAFETY WATCH

Watch for skin breakdown on the nose and face from the tight mask; reposition or pad the mask edges. · Monitor for gastric distension (air swallowed into the stomach) which can cause discomfort and rarely aspiration. · Check arterial or venous blood gases 1–2 hours after starting NIV to confirm CO₂ is dropping and pH is improving; if not, the patient needs intubation. · Do NOT use NIV if the patient is unconscious, cannot protect their airway, has severe low oxygen in the blood (hypoxemia) despite high oxygen, is in shock, has facial trauma that prevents a seal, or cannot clear secretions. · Claustrophobia and mask discomfort are common; coaching and a calm environment help, but if the patient cannot tolerate the mask, NIV will fail.

Endotracheal Intubation with Invasive Mechanical Ventilation · Invasive airway management and mechanical ventilation (not a drug; a life-support procedure)

A breathing tube is placed through the mouth into the trachea (windpipe) and connected to a ventilator machine. The machine takes over breathing completely. The patient is usually sedated. The tube seals the airway so the machine can deliver precise volumes of oxygen and pressure directly into the lungs, protect the airway from vomit or secretions, and allow suctioning of mucus.

1. The endotracheal tube is passed between the vocal cords into the trachea and the cuff is inflated to seal the airway.
2. The ventilator delivers breaths at a set rate, volume, and pressure, taking over the work of breathing entirely if needed.
3. Positive end-expiratory pressure (PEEP) keeps alveoli open at the end of each breath so they do not collapse.
4. The sealed tube prevents aspiration of stomach contents or blood and allows suctioning of secretions that the patient cannot cough up.
5. Oxygen concentration (FiO₂) and ventilator settings are adjusted based on blood gas results to normalize oxygen and carbon dioxide levels.

SAFETY WATCH

Intubation requires deep sedation and often paralysis; the patient cannot speak, eat, or protect their airway without the tube. · Risk of ventilator-associated pneumonia (VAP) rises after 48 hours; use lung-protective ventilation, elevate the head of the bed, and practice daily sedation breaks and spontaneous breathing trials. · Monitor for barotrauma (lung rupture causing pneumothorax) if pressures are too high; use low tidal volumes (6 mL/kg ideal body weight) in ARDS per ARDSNet protocol. · Prolonged intubation (>7–10 days) may require tracheostomy to reduce sedation needs and allow safer long-term ventilation. · Hemodynamic instability can occur during intubation due to sedative medications and positive pressure reducing venous return; have vasopressors ready in shock patients. · Do NOT delay intubation if the patient is unconscious, cannot protect the airway, is in severe shock, has refractory low oxygen in the blood (hypoxemia), or has failed NIV after 1–2 hours; delaying intubation in a crashing patient increases mortality.

High-Flow Nasal Cannula (HFNC) · High-flow oxygen delivery device (not a drug; a respiratory support tool)

HFNC delivers heated, humidified oxygen at very high flow rates (up to 60 liters per minute) through soft nasal prongs. The high flow creates a small amount of positive pressure in the upper airway, washes out carbon dioxide from the nose and throat (dead space), and delivers a precise, stable oxygen concentration. It is more comfortable than a mask and can deliver more oxygen than a standard nasal cannula.

1. The device heats and humidifies the oxygen so it does not dry out or irritate the airway.
2. High flow rates (30–60 L/min) create a small positive end-expiratory pressure (a few cm H₂O) that gently splints the upper airway open.
3. The high flow flushes out carbon dioxide that collects in the nose and throat, improving the efficiency of each breath.
4. Stable FiO₂ (fraction of inspired oxygen) is delivered because the high flow exceeds the patient's own inspiratory demand, unlike a standard nasal cannula where room air dilutes the oxygen.

SAFETY WATCH

Monitor oxygen saturation and work of breathing closely; if the patient is using accessory muscles, has rising respiratory rate, or desaturates despite 100% FiO₂, they need NIV or intubation. · HFNC can give a false sense of security because the oxygen saturation may look acceptable while CO₂ is silently rising; check a blood gas if you suspect hypercapnia. · Do NOT use HFNC as the sole support in hypercapnic respiratory failure (COPD flare-up (exacerbation), obesity hypoventilation) where NIV is proven superior. · HFNC is best for hypoxemic respiratory failure without significant CO₂ retention, such as pneumonia, early ARDS, or post-extubation support in low-risk patients. · If the patient cannot tolerate nasal prongs, is a mouth breather, or has nasal obstruction, HFNC efficacy drops and another modality is needed.

Red flags

- Person stops breathing, gasps for air, or suddenly becomes unresponsive
- Oxygen level stays below 88-90% even with high-flow oxygen or noninvasive support
- Blood pressure drops sharply or heart rate climbs above 130-140
- Confusion or sleepiness gets worse — cannot follow simple commands or keep eyes open
- Breathing gets slower and shallower instead of faster (a sign the muscles are giving out)

Nursing priorities

1. Assess mental status and ability to follow commands every 15 to 30 minutes — The brain needs oxygen; worsening confusion or sleepiness means not enough oxygen is reaching the brain or CO₂ is building up to dangerous levels, signaling NIV is failing and the patient needs intubation before they stop breathing or their heart stops.
2. Monitor respiratory rate, oxygen saturation, and work of breathing continuously — Rising respiratory rate above 35, dropping SpO₂ (oxygen level in blood) despite NIV, or signs of exhaustion like using neck muscles to breathe mean the lungs are failing and NIV cannot keep up; catching this early prevents cardiac arrest from lack of oxygen.
3. Position the patient sitting upright at 45 to 90 degrees — Sitting upright uses gravity to pull the diaphragm (breathing muscle) down and shifts fluid away from the lungs, making each breath easier and reducing the work the heart and lungs must do; lying flat makes breathing harder and can worsen heart failure or aspiration risk.
4. Ensure proper NIV mask fit and check for skin breakdown on the nose and face every 2 hours — A loose mask leaks air and makes the pressure therapy fail; a too-tight mask causes pressure ulcers (skin wounds) on the bridge of the nose within hours, leading to pain, infection, and the patient refusing to wear the mask.

5. Keep suction and intubation equipment at the bedside and ready to use — Patients on NIV can deteriorate in minutes; if they vomit, lose consciousness, or stop breathing, immediate intubation and suctioning save their life by protecting the airway and providing full ventilator support before the heart stops from lack of oxygen.
6. Titrate oxygen to maintain SpO₂ 88 to 92 percent in COPD patients and 94 to 98 percent in others — Too much oxygen in COPD patients shuts down their breathing drive (because their bodies rely on low oxygen to trigger breaths) and causes CO₂ to build up to lethal levels; too little oxygen in other conditions starves the heart and brain.
7. Administer prescribed bronchodilators, diuretics, or antibiotics on time — NIV buys time but does not cure the disease; bronchodilators open airways in COPD and asthma, diuretics remove fluid from lungs in heart failure, and antibiotics kill infection; fixing the root cause lets the patient come off NIV safely.
8. Communicate frequently with the patient and reassure them during NIV therapy — The tight mask feels suffocating and causes panic, which increases breathing rate and work of breathing; calming the patient lowers their oxygen demand, improves cooperation with the mask, and prevents them from ripping it off before NIV has time to work.

Sources

- American Thoracic Society (ATS) Clinical Practice Guideline: Noninvasive Ventilation for Acute Respiratory Failure, 2017
- European Respiratory Society (ERS) / ATS Guidelines on NIV, 2017
- British Thoracic Society (BTS) / Intensive Care Society (ICS) Guideline for Ventilatory Support in Adults, 2016
- Cochrane Database: Noninvasive Ventilation for Acute Respiratory Failure

Last reviewed: 2026-07-07