

Neonatal Respiratory Distress

Newborn breathing trouble — give oxygen, check for surfactant deficiency (missing lung coating) in preemies, clear fluid or meconium in term babies, and treat infection fast.

THE ONE-LINE TRUTH

A newborn who breathes fast, pulls in the chest with each breath, grunts, or turns blue is showing that the lungs are not opening or swapping oxygen the way they should.

The chain — how it unfolds

1. Baby is born (preterm or stressed term pregnancy) → lungs either lack surfactant, have leftover fluid, inhale meconium, or are infected. — why: Preterm birth (<37 weeks, especially <34 weeks) means the lungs have not finished making surfactant, the coating that keeps air sacs open. In term babies, C-section without labor skips the squeeze that pushes fluid out, stress in the womb triggers meconium release, or bacteria/viruses cross the placenta or birth canal and infect the lungs.
2. Air sacs collapse, fill with fluid/meconium/pus, or stay stiff → oxygen cannot cross the thin wall into the blood. — why: Without surfactant, every exhale lets the sacs collapse like deflating sticky balloons, and the next inhale has to pop them open again—exhausting work. Fluid, meconium, or infection physically blocks the sacs or thickens the wall so oxygen cannot diffuse through. Either way, the blood leaving the lungs is low in oxygen.
3. Chemoreceptors (oxygen sensors in the brainstem and major arteries) detect low oxygen and high carbon dioxide → send emergency signals to breathe faster and harder. — why: The carotid bodies (sensors in the neck arteries) and the medulla (brainstem respiratory center) are wired to keep oxygen and carbon dioxide in a tight range. When oxygen drops or CO₂ rises, they fire rapid signals to the diaphragm, intercostal muscles (ribs), and accessory muscles (neck, belly) to increase the breathing rate and depth—this is fast breathing (tachypnea) (>60 breaths/min) and increased work of breathing.

4. Baby retracts (chest pulls in), flares nostrils, and grunts (closes vocal cords at the end of exhale) to trap pressure and prop sacs open. — why: Retractions mean the baby is generating huge negative pressure to suck air into stiff or collapsed lungs—the soft spots between ribs and above collarbones cave in visibly. Nasal flaring lowers airway resistance. Grunting closes the glottis (voice box) at the end of exhale, trapping air and pressure inside (positive end-expiratory pressure, PEEP) so sacs do not collapse completely—it is the body's natural CPAP (continuous positive airway pressure). These are emergency compensations, not the disease itself.
5. Compensation keeps oxygen borderline for minutes to hours, but the baby is burning energy and tiring out the breathing muscles. — why: The diaphragm and intercostals are working at maximum effort, using up glucose and oxygen reserves. As long as the baby can keep up the work, oxygen saturation may stay 85–92% and the baby looks "stable"—but this is a false stability. The baby is on a steep downhill slope, and the window to intervene is closing.
6. If the root cause is not fixed, the baby exhausts → breathing slows or stops (apnea), carbon dioxide climbs (respiratory too much acid in the blood (acidosis)), oxygen drops, skin turns blue (cyanosis), and vital organs (brain, heart, kidneys) start to fail. — why: Tired muscles cannot maintain the work of breathing. Carbon dioxide (a waste acid) builds up in the blood, lowering pH (acidosis), which weakens the heart and blood vessels. Low oxygen (hypoxemia) damages the brain (can cause seizures, cerebral palsy, or death) and triggers the lungs' blood vessels to clamp shut (PPHN), creating a vicious cycle. The baby may stop grunting and go limp—this is a crash, not improvement.
7. Complications accelerate the crash: pneumothorax (air leaks into chest, lung collapses), PPHN (lung vessels squeeze shut, blood bypasses lungs), or sepsis (infection spreads, blood pressure drops). — why: High pressures from hard breathing or mechanical ventilation can pop a hole in the lung (pneumothorax), suddenly collapsing it and needing emergency needle or chest tube. PPHN happens when the lungs' blood vessels stay in fetal mode (high resistance), so blood takes the path of least resistance through the ductus arteriosus and foramen ovale, bypassing the lungs entirely—oxygen saturation plummets even with high oxygen. Sepsis overwhelms the immune system, causes shock, and shuts down organs.

- 8.** Treatment targets the broken step: surfactant (for RDS), oxygen and CPAP (for TTN, mild RDS, pneumonia), suctioning $\pm$ intubation (for meconium), antibiotics (for pneumonia/sepsis), and inhaled nitric oxide $\pm$ high-frequency ventilation (for PPHN). — why: Surfactant replacement (beractant, poractant alfa) is given through a breathing tube directly into the lungs of preterm babies with RDS—it coats the sacs and prevents collapse, often improving oxygen within minutes. CPAP (continuous positive airway pressure) delivers steady pressure through nasal prongs to hold sacs open without a breathing tube. Mechanical ventilation takes over the work of breathing when the baby is too tired. Oxygen is titrated carefully (target saturation 90–95% in preterm, 95–100% in term) to avoid both low oxygen in the blood (hypoxemia) and oxygen toxicity (too much oxygen damages lungs and eyes). Antibiotics (ampicillin + gentamicin) are started early if pneumonia or sepsis is suspected. Inhaled nitric oxide relaxes lung blood vessels in PPHN. Each treatment fixes a specific broken step, not just the symptom.

Spot it — what you see, and why

- Fast breathing (tachypnea) — more than 60 breaths in one minute: The baby's lungs are not exchanging oxygen and carbon dioxide well, so the brain tells the body to breathe faster to try to get more air in and out.
- Grunting sounds with each breath out: The baby closes the vocal cords partway when breathing out to keep air trapped in the lungs a little longer, which helps the tiny air sacs (alveoli) stay open instead of collapsing — it is the body's emergency trick to keep more lung working.
- Flaring nostrils (nasal flaring): The baby is working hard to pull air in, so the muscles around the nose open the nostrils wider to let more air through.
- Chest pulls in between the ribs or under the ribcage with each breath (retractions): The baby's chest muscles have to work extra hard to pull air into stiff or blocked lungs, so the soft spots between the ribs get sucked inward.
- Blue or gray color of the lips, tongue, or skin (cyanosis): Not enough oxygen is getting from the lungs into the blood, so the blood turns darker and the skin looks blue or gray instead of pink.
- Floppy body, weak muscle tone, or poor response: The brain and muscles are not getting enough oxygen to work normally, so the baby becomes limp and less alert.

Red flags — act now

- Blue or gray color that does not improve with oxygen
- Breathing stops for more than 15 to 20 seconds, or the heart rate drops below 100 beats per minute

- Baby becomes floppy, unresponsive, or cannot cry
- Grunting and chest retractions get worse quickly
- Baby was born before 34 weeks or breathed in thick meconium-stained fluid

Treat it — in this order

1. Exogenous surfactant (beractant, poractant alfa, or calfactant) given through an endotracheal tube into the lungs → Surfactant is the soapy lipid-protein mix that lines the inside of the alveoli and keeps them from collapsing when the baby breathes out. Preterm babies (especially before 34 weeks) do not make enough surfactant, so their alveoli stick shut and gas exchange fails. Giving surfactant replacement coats the alveoli, drops the surface tension, recruits collapsed air sacs, improves lung compliance (how easily the lungs stretch), and lets oxygen cross into the blood. Breathing becomes easier within minutes to hours.
2. Continuous positive airway pressure (CPAP, typically 5–8 cm H₂O pressure delivered through nasal prongs or mask) → CPAP holds the airways and alveoli open with gentle constant pressure. It recruits collapsed air sacs, splints the floppy upper airway, reduces the work of breathing, and improves oxygenation. The baby can often avoid intubation and mechanical ventilation if CPAP is started early. In mild RDS or TTN, CPAP alone may be enough.
3. Supplemental oxygen (delivered by hood, nasal cannula, CPAP, or ventilator, titrated to keep oxygen saturation 90–95%) → Low oxygen in the blood (hypoxemia) damages the brain, heart, and other organs. Giving oxygen raises the partial pressure of oxygen in the alveoli, drives more oxygen across the alveolar-capillary membrane into the bloodstream, and prevents tissue low oxygen (hypoxia). The goal is to match oxygen delivery to the baby's oxygen saturation target—too little causes harm, too much causes oxygen toxicity and retinopathy of prematurity in preterm babies.
4. Empiric intravenous antibiotics (ampicillin plus gentamicin, started within the first hour if sepsis or pneumonia is suspected) → Bacterial pneumonia and early-onset sepsis (usually group B Streptococcus or Escherichia coli) cause inflammation, fluid leakage into the alveoli, and impaired gas exchange that look identical to RDS on exam. Ampicillin covers group B Strep and Listeria; gentamicin covers gram-negative rods. Starting antibiotics early kills the bacteria, reduces lung inflammation, prevents septic shock, and can save the baby's life if infection is the real cause.

5. Endotracheal intubation and mechanical ventilation (pressure-control or volume-control modes with gentle settings to protect the lungs) → When CPAP fails and the baby cannot maintain oxygen saturation or clear carbon dioxide, or when pauses in breathing (apnea) or severe retractions signal exhaustion, mechanical ventilation takes over the work of breathing. The ventilator delivers positive pressure breaths, opens collapsed alveoli, improves gas exchange, and prevents respiratory arrest. It also allows safe delivery of surfactant.

Your top priorities at the bedside

1. Attach pulse oximeter probe and monitor oxygen saturation continuously, keeping SpO₂ between 90 and 95 percent in preterm babies or 95 to 100 percent in term babies. — why: Too little oxygen harms the brain and other organs; too much oxygen in preterm babies damages the retina (back of the eye) causing retinopathy of prematurity and can also injure the lungs, so we match oxygen to the baby's exact need.
2. Position the baby with the head in neutral or slightly extended position and suction the mouth and nose gently only if you see secretions blocking the airway. — why: Neutral head position keeps the airway open without kinking the soft trachea (windpipe); suctioning only when needed prevents vagal stimulation (triggering the vagus nerve) that slows the heart rate and worsens oxygen levels.
3. Keep the baby warm under a radiant warmer or in an incubator, maintaining skin temperature between 36.5 and 37.5 degrees Celsius. — why: Cold stress increases oxygen consumption and metabolic demand, making respiratory distress worse because the baby has to work even harder to breathe and stay warm at the same time.