

Neonatal Respiratory Distress

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

At birth, the baby's first cry pushes fluid out of the lungs and fills millions of tiny air sacs (alveoli) with air. A slippery coating called surfactant—made by cells in the lungs starting around 24 weeks of pregnancy—keeps those sacs open between breaths so oxygen can cross into the blood and carbon dioxide can leave. The umbilical cord is clamped, the placenta is gone, and now the baby's lungs must do all the oxygen work on their own.

BODY'S JOB

The lungs' job is to swap gases: pull oxygen from the air into the blood and push carbon dioxide (the body's waste gas) out. Every breath fills the alveoli, oxygen crosses a paper-thin wall into tiny blood vessels (capillaries), and the heart pumps that oxygen-rich blood to the brain, heart, and every cell. Surfactant is the secret ingredient—it lowers surface tension (the stickiness that makes bubbles collapse) so the sacs stay open with very little effort. Without surfactant, or if the sacs are blocked or filled with fluid, the baby cannot get enough oxygen no matter how hard they breathe.

1. Baby takes first breath → air rushes into the nose and mouth, down the windpipe (trachea), and into millions of branching airways that end in grape-like clusters of air sacs (alveoli).
2. Surfactant coats the inside of each air sac → keeps it open between breaths, like a non-stick spray keeping a balloon from gluing shut.

3. Oxygen crosses the sac wall → moves from the air inside the sac into the blood inside the capillary wrapped around it (takes less than one second).
4. Heart pumps oxygen-rich blood → out to the brain, heart, kidneys, and every organ; carbon dioxide (waste) travels back to the lungs and is breathed out.
5. Cycle repeats with every breath → in a healthy newborn, this happens 40–60 times per minute, smooth and quiet, no grunting or chest pull-in.

What breaks

The air sacs either collapse after every breath (because surfactant is missing or not working), stay filled with fluid or meconium, or get infected—so oxygen cannot cross into the blood.

CLINICAL TERM

Respiratory distress syndrome (RDS) in preterm infants due to surfactant deficiency; transient fast breathing (tachypnea) of the newborn (TTN) from delayed clearance of fetal lung fluid; meconium aspiration syndrome (MAS) causing airway obstruction and chemical pneumonitis; neonatal pneumonia (bacterial or viral); congenital anomalies (diaphragmatic hernia, cystic adenomatoid malformation); or persistent pulmonary high blood pressure (hypertension) of the newborn (PPHN) with right-to-left shunting.

How the body compensates

The baby's body tries to force oxygen in and keep the air sacs open by breathing faster, pulling chest muscles inward, flaring the nostrils, and grunting to trap pressure. These efforts can keep oxygen levels borderline for a while, but if the root cause is not fixed, the baby tires out, carbon dioxide rises, and the system crashes—so the job is to recognize distress early and intervene before exhaustion or complications (pneumothorax, PPHN) tip the baby over the edge.

1. {'what': 'Fast breathing (Tachypnea) (fast breathing >60 breaths/min)', 'why': 'The baby senses low oxygen and high carbon dioxide and the brainstem (respiratory center in the medulla) fires faster signals to the diaphragm and rib muscles, trying to pull in more air per minute to boost oxygen.'}
2. {'what': 'Retractions (chest, ribs, and belly pull inward with each breath)', 'why': 'When air sacs are stiff or collapsed, the baby has to generate huge negative pressure to suck air in; this pulls the soft tissue between the ribs (intercostal), above the collarbones (supraclavicular), below the ribs (subcostal), and at the notch above the breastbone (suprasternal) inward—visible proof the baby is working extra hard.'}

3. {'what': 'Nasal flaring (nostrils widen with each breath)', 'why': 'The baby opens the nostrils as wide as possible to lower airway resistance and let more air rush in—like opening a door wider to let a crowd through faster.'}
4. {'what': 'Grunting (short, forced sound at the end of each breath)', 'why': 'The baby half-closes the vocal cords (glottis) at the end of exhale to trap air and pressure inside the lungs for a split second longer—this positive end-expiratory pressure (PEEP) keeps collapsing sacs propped open and buys a tiny bit more time for oxygen to cross; it is the body's built-in CPAP.'}
5. {'what': 'Bluish skin (Cyanosis) (blue color of lips, tongue, or skin)', 'why': 'When oxygen saturation drops below ~85%, the blood does not carry enough oxygen and looks dark blue instead of bright red; central bluish skin (cyanosis) (tongue and lips) means the lungs or heart are not oxygenating blood, while peripheral bluish skin (only hands/feet) can be from poor circulation or cold.'}
6. {'what': 'The danger: compensation hides the severity for a while', 'why': 'A baby can grunt, retract, and breathe fast for hours and keep oxygen borderline—so vital signs may look "okay" even though the baby is exhausting the breathing muscles and heading toward respiratory failure. The crash point is when the baby gets too tired to keep up the work, carbon dioxide climbs (respiratory too much acid in the blood (acidosis)), oxygen plummets despite maximum effort, and the heart and brain start to fail. Watch the trend—worsening retractions, rising oxygen needs, or a baby who stops grunting and goes limp is crashing, not improving.'}
7. {'what': 'Complications that accelerate the crash', 'why': 'Pneumothorax (air leak into the chest cavity, collapsing the lung)—sudden worsening, absent breath sounds on one side, needs urgent needle decompression or chest tube. PPHN (persistent pulmonary high blood pressure (hypertension))—lung blood vessels clamp shut, blood bypasses the lungs, severe bluish skin (cyanosis), needs inhaled nitric oxide and high oxygen. Pauses in breathing (Apnea) (breathing stops)—preterm or exhausted babies stop breathing for >20 seconds, need stimulation or mechanical ventilation. Sepsis (infection spreads to blood)—shock, low blood pressure, organ failure, needs antibiotics and ICU support.'}

Domino effect

1. Baby is born (preterm or stressed term pregnancy) → lungs either lack surfactant, have leftover fluid, inhale meconium, or are infected. — 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. — 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. — 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. — 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. — 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. — 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). — 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 ± intubation (for meconium), antibiotics (for pneumonia/sepsis), and inhaled nitric oxide ± high-frequency ventilation (for PPHN). — 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.

Symptoms · what the patient feels, 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.
- Pauses in breathing (Apnea) — pauses in breathing that last more than 15 to 20 seconds: The breathing control center in the brainstem may be immature or exhausted from working so hard, or oxygen levels drop so low that the drive to breathe weakens.
- Foamy white or pink fluid at the mouth or nose: Fluid has leaked into the air sacs or the baby has breathed in amniotic fluid or meconium (the baby's first stool), and the lungs are trying to push it out, mixing it with air to make foam.

Focused exam

- Fast breathing (Tachypnea) (breathing faster than 60 breaths per minute in a newborn): The baby's lungs are stiff or partly collapsed, so the body tries to fix low oxygen by breathing faster. This is usually the first sign that the lungs are in trouble.
- Chest wall retractions (the skin between and below the ribs sucks inward with each breath): Stiff or collapsed lungs create strong negative pressure when the baby tries to pull air in. The chest wall caves in because the lungs resist opening. This tells you the work of breathing is dangerously high.
- Expiratory grunting (a soft groaning sound at the end of each breath out): The baby closes the vocal cords partway during exhale to trap air inside the lungs and keep the alveoli (tiny air sacs) from collapsing completely. Grunting is the body's self-rescue trick when surfactant (the slippery coating that holds air sacs open) is missing or the lungs are full of fluid.
- Nasal flaring (the nostrils widen with every breath in): The baby is recruiting every muscle to open the airway and pull in more oxygen. This is a classic sign the body is fighting to breathe.
- Central bluish skin (cyanosis) (blue color on the lips, tongue, or trunk): When oxygen saturation drops below roughly 85–90%, not enough oxygen reaches the blood, and the skin turns blue. This is a red flag that gas exchange in the lungs is failing.
- Decreased or absent breath sounds on listening with a stethoscope (auscultation) (very quiet or silent lungs in one or more areas): Collapsed alveoli, thick fluid, or plugged airways block air from moving. A silent chest means whole sections of lung are not working. In pneumothorax (air leak), one side goes completely quiet.

Diagnostics · every test answers a question

- Chest X-ray (anteroposterior view) → Which lung disease pattern is causing the distress—collapsed air sacs, fluid still in the lungs, air leak, or infection?

- Arterial or capillary blood gas (pH, PaCO₂, PaO₂, bicarbonate) → Is the baby getting enough oxygen and clearing carbon dioxide, and is the blood turning acidic from poor gas exchange?
- Pulse oximetry (continuous oxygen saturation monitoring with a sensor on the hand or foot) → Is oxygen reaching the blood, and is the baby responding to treatment?
- Complete blood count with differential and blood culture (if infection suspected) → Is bacterial pneumonia or sepsis driving the respiratory distress, especially if the mother had fever, prolonged rupture of membranes, or chorioamnionitis?
- C-reactive protein (CRP) and procalcitonin (optional inflammatory markers) → Does the baby have systemic inflammation that points toward infection rather than pure surfactant deficiency?
- Lecithin-to-sphingomyelin (L/S) ratio or phosphatidylglycerol level in amniotic fluid (if available before delivery) → Did the mother's amniotic fluid show that the baby's lungs were mature enough to make surfactant?

Treatment logic

- 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.
- 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.
- 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.

- 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.
- 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.
- Gentle handling, thermoregulation (keeping the baby warm in an incubator or radiant warmer), and minimal stimulation → Cold stress forces the baby to burn energy and oxygen to stay warm, worsening low oxygen in the blood (hypoxemia) and too much acid in the blood (acidosis). Keeping the baby at a neutral thermal environment (skin temperature 36.5–37.5°C) reduces metabolic demand and lets all available oxygen go to the brain and vital organs. Gentle handling prevents agitation that spikes oxygen needs.
- Fluid management (restricting IV fluids to 60–80 mL/kg/day in the first days to avoid fluid overload) → Excess IV fluid can leak into the lungs and worsen pulmonary edema (fluid flooding the lungs), especially in babies with immature kidneys or a patent ductus arteriosus (PDA) that shunts extra blood to the lungs. Careful fluid balance keeps the lungs dry and improves oxygenation.

Medications

Exogenous surfactant (beractant, poractant alfa, calfactant) · Pulmonary surfactant replacement

Surfactant is a slippery coating that normally lines the inside of the air sacs in the lungs (the alveoli) and keeps them from collapsing when the baby breathes out. Preterm babies often don't make enough surfactant yet, so their air sacs stick shut and breathing becomes exhausting. Giving surfactant directly into the baby's windpipe coats the air sacs so they can stay open with each breath, letting oxygen in and carbon dioxide out much more easily.

1. The medicine is dripped through a breathing tube directly into the baby's windpipe and spreads down into the small airways.
2. Surfactant spreads out as a thin film over the inside walls of the air sacs.

3. This film lowers surface tension (the stickiness that makes wet surfaces cling together), so the air sacs don't collapse at the end of each breath.
4. With air sacs staying open, oxygen can cross into the bloodstream and carbon dioxide can leave, so the baby's blood oxygen rises and breathing becomes less work.

SAFETY WATCH

The baby must be watched closely during the dose because surfactant can briefly block the airway or cause the oxygen level to spike or drop. · Too much oxygen right after surfactant can harm the eyes and lungs, so the oxygen is turned down carefully as the baby improves. · Surfactant can cause a short-lived drop in blood pressure or heart rate, so monitors stay on throughout.

Supplemental oxygen (nasal cannula, CPAP, or ventilator) · Respiratory support gas

Oxygen is the gas in air that cells need to make energy. When a baby's lungs are sick or immature, not enough oxygen reaches the blood. Giving extra oxygen (more than the 21% found in room air) raises the amount available in each breath so the blood can carry enough to the brain, heart, and other organs even when the lungs aren't working perfectly.

1. Extra oxygen is delivered through a soft tube in the nose, a mask, or a breathing machine.
2. The higher concentration of oxygen in each breath increases the amount that dissolves in the blood inside the lung capillaries.
3. Hemoglobin in red blood cells picks up this oxygen and carries it throughout the body.
4. Organs get the oxygen they need to function, preventing damage from low oxygen (hypoxia).

SAFETY WATCH

Too much oxygen for too long can damage the retina (back of the eye), leading to retinopathy of prematurity and possible blindness, especially in very premature babies. · High oxygen can also injure delicate lung tissue, causing chronic lung disease (bronchopulmonary dysplasia). · Oxygen levels are monitored continuously with a pulse oximeter (a clip on the hand or foot) and kept in a safe target range, usually 90–95% for preterm infants.

Continuous positive airway pressure (CPAP) · Non-invasive respiratory support device

CPAP gently pushes air into the baby's nose at a steady, low pressure throughout the whole breathing cycle. This constant push acts like a brace that holds the airways and air sacs open,

preventing them from collapsing when the baby breathes out. It makes each breath easier and helps oxygen get into the blood without needing a breathing tube in most cases.

1. Soft prongs or a mask deliver a continuous stream of pressurized air or oxygen-enriched air into the baby's nose.
2. The pressure travels down the airways and splints them open, keeping them from pinching shut.
3. Air sacs at the end of the airways stay inflated even at the end of a breath, so more surface area is available for oxygen to move into the blood.
4. The baby breathes on their own but with less effort because the lungs don't have to be re-opened with every breath.

SAFETY WATCH

The pressure can push air into the stomach, causing bloating; a small tube may be placed in the stomach to vent the air. · Too much pressure can tear delicate lung tissue and let air leak into the chest (pneumothorax), so pressures are carefully adjusted. · The prongs can irritate or injure the inside of the nose if not positioned gently and checked often.

Mechanical ventilation (via endotracheal tube) · Invasive life-support device

A breathing machine takes over the work of breathing when a baby is too sick, too weak, or too premature to breathe well enough on their own. A soft tube is placed through the mouth or nose into the windpipe, and the machine pushes measured breaths of air and oxygen into the lungs at a set rate and pressure. This keeps oxygen flowing into the blood and carbon dioxide flowing out when the baby cannot do it alone.

1. A small breathing tube is placed into the baby's windpipe after sedation or when the baby is too ill to resist.
2. The ventilator pushes a set volume or pressure of gas into the lungs with each breath, opening the air sacs.
3. Oxygen from the breath crosses into the blood, and carbon dioxide is pushed out when the machine lets the lungs deflate.
4. The rate, pressure, and oxygen level are adjusted continuously based on blood gas measurements and the baby's response.

SAFETY WATCH

The tube can slip out of position or become blocked by mucus, cutting off air; nurses suction and check position frequently. · Too much pressure or volume can tear lung tissue, causing air leaks (pneumothorax) that compress the lung and heart. · Prolonged ventilation increases the risk of chronic lung injury (bronchopulmonary dysplasia) and infection. · Babies on ventilators need continuous monitoring in a neonatal intensive care unit.

Antibiotics (ampicillin + gentamicin, or other combinations) · Antimicrobial agents

Antibiotics kill or stop bacteria from multiplying. When a newborn has respiratory distress and infection is suspected (especially early-onset sepsis or pneumonia from bacteria picked up during birth), antibiotics are started right away because newborn infections can spiral out of control in hours. Ampicillin covers common bacteria from the mother's birth canal (like Group B Streptococcus and Listeria), and gentamicin covers many gram-negative bacteria (like E. coli). Together they target the most likely germs while lab cultures are growing.

1. Antibiotics are given through an IV line so they enter the bloodstream immediately.
2. Ampicillin interferes with bacterial cell-wall construction; without a sturdy wall, the bacteria burst and die.
3. Gentamicin blocks the bacteria's protein-making machinery (ribosomes); without proteins, bacteria cannot grow or reproduce.
4. As bacteria are killed or stopped, the immune system clears the debris and inflammation in the lungs drops, allowing the baby to breathe more easily.

SAFETY WATCH

Gentamicin can damage the kidneys or inner ear (causing hearing loss) if levels get too high, so doses are calculated carefully by weight and blood levels are monitored. · Prolonged or unnecessary antibiotic use can select for resistant bacteria and disturb the normal gut bacteria (microbiome). · If cultures come back negative and the baby is improving, antibiotics are usually stopped after 48–72 hours to avoid unnecessary exposure.

Caffeine citrate · Methylxanthine stimulant

Caffeine wakes up the breathing control center in the brainstem (the medulla) so that preterm babies remember to take regular breaths. Very premature infants often have pauses in breathing (apnea) of prematurity—they pause breathing for more than 20 seconds because their brain's breathing rhythm is still immature. Caffeine blocks adenosine receptors in the

brain; adenosine normally makes neurons quieter, so blocking it keeps the breathing neurons more active and the baby breathing more steadily.

1. Caffeine is given by mouth or IV and crosses into the brain quickly.
2. It plugs adenosine receptors on neurons in the breathing control centers, preventing adenosine from quieting them down.
3. With adenosine blocked, the neurons fire more consistently, sending regular signals to the diaphragm to contract and breathe.
4. Breathing becomes more regular, pauses in breathing (apnea) episodes drop, and the baby needs less support from CPAP or a ventilator.

SAFETY WATCH

Caffeine can make the heart race (tachycardia) or cause jitteriness if the dose is too high. · It can worsen feeding intolerance or reflux in some babies because it relaxes the muscle between the stomach and esophagus. · Blood levels are usually not monitored unless toxicity is suspected; the drug has a wide safety margin in the typical dose range.

Red flags

- 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

Nursing priorities

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. — 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. — 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. — 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.
4. Monitor respiratory rate, retractions (chest pulling in between ribs or below the rib cage), grunting, and nasal flaring every 15 to 30 minutes and report worsening signs immediately. — Increasing work of breathing signals that the lungs are getting stiffer or the baby is tiring out, meaning the current support is not enough and the team may need to escalate to CPAP (continuous positive airway pressure), intubation, or surfactant.
5. Delay feeding and keep the baby NPO (nothing by mouth) until respiratory rate is consistently below 60 breaths per minute and work of breathing improves. — Babies breathing fast cannot coordinate sucking, swallowing, and breathing, so feeding increases aspiration risk (milk going into the lungs); also, a full stomach pushes up on the diaphragm (breathing muscle) and makes breathing even harder.
6. Place an intravenous line and start maintenance fluids at 60 to 80 milliliters per kilogram per day, adjusting based on urine output and weight change. — Babies in respiratory distress cannot feed safely, so IV fluids prevent dehydration and low blood sugar; however, too much fluid leaks into the lungs and worsens respiratory distress, so we monitor intake and output closely.
7. Draw blood cultures and start ampicillin and gentamicin antibiotics within one hour if sepsis is suspected based on maternal fever, prolonged rupture of membranes, or clinical instability. — Early-onset sepsis (infection in the first 72 hours) looks identical to respiratory distress syndrome on exam and chest X-ray, and delaying antibiotics allows bacteria to multiply in the bloodstream, leading to shock and death.
8. Prepare for surfactant administration if the baby is intubated and chest X-ray shows ground-glass appearance (hazy white lung fields) with low lung volumes. — Surfactant replaces the missing lung coating in preterm babies, reducing surface tension so air sacs stay open; giving it early (within 2 hours of birth) improves survival and reduces the need for long-term ventilator support.

Sources

- AAP Guidelines for Perinatal Care, 8th Edition
- American Academy of Pediatrics (AAP) Textbook of Neonatal Resuscitation
- WHO recommendations on interventions to improve preterm birth outcomes
- Cochrane Neonatal Group reviews

Last reviewed: 2026-07-07