CHAPTER

14

Approach to Respiratory Distress in the Newborn

Respiratory distress occurs in 4–7% of all neonates[1,2] and is the reason for 30–40% of all admissions to the NICU. It is more common among preterm (30%) and post-term (21%) than among term neonates (4.2%).[2] A neonate can develop respiratory distress due to various conditions, which are not limited to respiratory or cardiovascular causes alone. Appropriate management includes evaluating the neonate for the severity and cause of respiratory distress and initiating respiratory support when appropriate.

DEFINITION

The National Neonatal Perinatal Database of India (NNPD)[3] defines respiratory distress as the presence of any two of the following features:

1. Respiratory rate >60/minute
2. Subcostal/intercostal recessions
3. Expiratory grunt/groaning

In addition to the above features, the presence of nasal flaring, suprasternal retractions, and decreased air entry on auscultation of the chest also indicates the presence of respiratory distress. Gasping, choking, or stridor (signs of upper airway obstruction), apnea or poor respiratory effort or bradycardia, poor perfusion, and cyanosis are life-threatening signs that require prompt intervention.[4]

CAUSES

Respiratory distress can occur due to various conditions (Table 14.1), and the frequency of a given disease depends on multiple factors, of which gestation is the most important. In preterm neonates, respiratory distress syndrome (RDS) is the most common cause, while in late preterm and term neonates, transient tachypnea of the newborn (TTN) is the predominant cause.[2]

Table 14.1: Common causes of respiratory distress in the newborn

Airway
• Choanal atresia
• Pierre Robin sequence
• Tracheoesophageal fistula
• Laryngotracheomalacia
• Vocal cord paralysis
Pulmonary diseases
• Transient tachypnea of the newborn (TTN)
• Respiratory distress syndrome (RDS)
• Meconium aspiration syndrome
• Pneumothorax and other air-leak syndromes
• Persistent pulmonary hypertension of the newborn
• Pulmonary hypoplasia
• Lung malformations
• Congenital diaphragmatic hernia
Cardiac diseases
• Congenital heart disease
• Arrhythmia
• Congestive cardiac failure
• Cardiomyopathy
Thoracic causes
Chest wall deformity
Skeletal dysplasia
Neuromuscular diseases
• Central nervous system damage (birth trauma, hemorrhage), meningitis, asphyxia
• Medication (maternal sedation, narcotic withdrawal)
• Muscular disease (myasthenia gravis)
• Spinal cord injury
Others
• Sepsis
• Anemia
• Polycythemia
• Hypo- and hyperthermia

INVESTIGATION AND MANAGEMENT

Initial Assessment

An initial assessment of respiratory distress is done to rule out life-threatening conditions which require immediate management, such as inadequate respiratory efforts or obstructed airway (gasping,

choking, stridor or apnea) or circulatory collapse (bradycardia, hypotension, and poor perfusion). If such features are present, emergency measures such as oxygen administration, bag and mask ventilation, or intubation should be initiated as necessary.

General Examination

Look for dysmorphic features, craniofacial malformations, features of intrauterine growth restriction, scaphoid abdomen, and drooling of saliva. Note the color of the neonate (pink vs. *cyanosed*) and use a pulse oximeter to determine the oxygen saturation.

Assessment of Respiratory Distress

Inspection: Observe whether the neonate is breathing comfortably or if signs of respiratory distress are present. Some respiratory sounds are audible to the ears without a stethoscope. **Stridor** is a harsh "crowing" sound produced due to narrowing the upper airway at the level of the larynx or extrathoracic trachea. It is often inspiratory but can be expiratory or biphasic. Stridor can occur in newborns due to laryngomalacia, Pierre Robin sequence, vocal cord palsy, laryngeal or subglottic narrowing due to edema, web, or stenosis.[5] **Grunting** is an expiratory noise due to the closure of the glottis during expiration. By this mechanism, neonates can increase their functional residual capacity (FRC). **Stertor or snoring** is a low-pitched inspiratory noise produced due to obstruction at the level of the nasopharynx (adenoid hypertrophy) or oropharynx (micrognathia, macroglossia). This sound can be inspiratory, expiratory, or both.

Note the symmetry of chest excursions and synchrony with abdominal wall movement. A rounded chest wall with increased anteroposterior diameter is a marker of hyperinflation. Pectus carinatum or pigeon chest deformity may be noted in chronic conditions with an increased respiratory effort that causes the sternum to protrude. On the other hand, the pectus excavatum or funnel chest is a developmental deformity wherein the sternum and costal cartilage are depressed.

Palpation and percussion: While palpation and percussion are done sparingly in neonates, one can get valuable information by feeling the tracheal position, locating the apex beat, palpating for crepitus or murmurs, and percussing for normal resonance. Dullness on percussion may be noted over areas of consolidation or collapse,

stony dullness over pleural effusion, and hyper-resonance over pneumothorax or bulla.

Auscultation: Assess whether the breath sounds are heard equally and symmetrically in all areas, whether there is a prolongation of the inspiratory or expiratory phase, and presence of added sounds like rales, rhonchi, wheeze, and stridor.

Respiratory distress score: The severity of respiratory distress can be objectively graded using respiratory distress scores. The two commonly used scores are the Silverman–Anderson Score[5] (Table 14.2) and Downes' Vidyasagar Score[6] (Table 14.3). The signs of respiratory distress are assigned a numerical value (0 indicating normal and 2 indicating the worst). The final summary score grades the severity of distress into none (score = 0) or mild (<3), moderate (4–6), and severe (≥7). Using these scores, nurses and clinicians can monitor the progression of respiratory distress and initiate optimal treatment. Neonates with mild to moderate distress can be managed using positive distending pressure like CPAP, while those with severe distress may need intubation and mechanical ventilation.

Respiratory distress score: Evidence

Silverman score does not include assessment of skin color, breath sounds, or tachypnea but correlates well with mortality.

The Downe's score shows a good correlation with physiological parameters such as pH, A-a DO_2 and PaO_2.

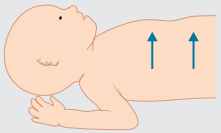
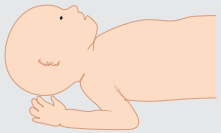
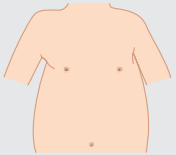
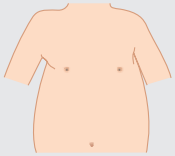
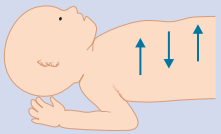
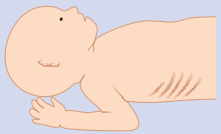
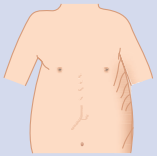
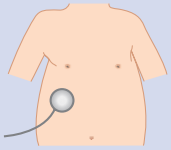
A score of 7 or more at age 24–30 hours, when the score is usually at its peak is associated with higher mortality compared to no mortality when the score is less than 6.

Serial monitoring is more important than one-time assessment. In impending respiratory failure, the score can be falsely low due to apnea, poor perfusion, and failure to maintain body temperature. The choice of one score over the other is often based on the unit's preference and training received by health care professionals.

Oxygen Saturation

Oxygen saturation measured using pulse oximetry is considered the sixth vital sign. Saturation below 90% indicates hypoxia.[9] It should be checked in preductal (right hand) and postductal sites (right/left leg). A pre-postductal difference of more than 5–10% indicates probable right-to-left shunt through patent ductus arteriosus (PDA) in the setting of persistent pulmonary hypertension of the newborn (PPHN). Central cyanosis is bluish discoloration of the

Table 14.2: Silverman–Anderson score for grading the severity of respiratory distress

Component	*Upper chest retractions*	*Lower chest retractions*	*Xiphoid retractions*	*Alar nasi flaring*	*Expiratory grunt*
Observation	Observe the synchrony of the upper chest with abdomen movement during inspiration	Observe the retractions between the ribs below the mid-axillary line	Retraction below the xiphoid process	Observe the nasal flaring before application of nasal interface	
Score 0	 Upper chest synchronized with abdomen	 None	 None	 None	 None
Score 1	 Upper chest lags compared to abdomen	 Just visible	 Just visible	 Just visible	 Audible with stethoscope

(*Contd.*)

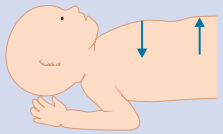
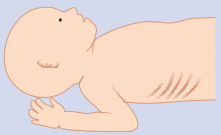
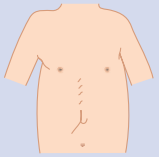
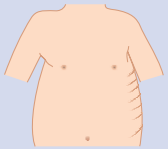
Table 14.2: Silverman–Anderson score for grading the severity of respiratory distress (*Contd.*)					
Component	*Upper chest retractions*	*Lower chest retractions*	*Xiphoid retractions*	*Alar nasi flaring*	*Expiratory grunt*
Score 2	 See-saw movement of the chest and abdomen	 Marked	 Marked	 Marked	 Audible without stethoscope

Table 14.3: Downe's score for grading severity of respiratory distress			
Feature	*Score 0*	*Score 1*	*Score 2*
Cyanosis of lips and oral mucosa	None	In-room air	In 40% FiO_2
Retractions (intercostal, subcostal and suprasternal)	None	Mild	Severe
Expiratory grunt	None	Audible with stethoscope	Audible without stethoscope
Air entry	Normal	Decreased	Barely audible
Respiratory rate	<60	60–80	>80 or apnea

entire body, including the skin, mucous membranes, and tongue. It is an important indicator of hypoxia. The deoxyhemoglobin level should be at least 2.5 g/dl in the blood for cyanosis to manifest. Polycythemic neonates with high hemoglobin may manifest cyanosis when oxygen saturation is 88%, while anemic neonates may not appear cyanosed until saturation drops below 65%.[8] Hence pulse oximeter is an essential tool to identify hypoxia. Peripheral cyanosis can be due to cold exposure, polycythemia, or potentially serious conditions like hypoglycemia, sepsis, or decreased left ventricular output like hypoplastic left heart syndrome or coarctation of aorta. The uses and limitations of pulse-oximetry are listed below:

- **Diagnosis:** Pulse-oximetry is the best tool for the continuous and noninvasive diagnosis of hypoxia. The normal range of SpO_2 at sea level is 97–99%, with a lower limit of 94%. Hypoxia is considered in neonates if SpO_2 is less than 90%.
- **Grading the severity of hypoxic respiratory failure:** The oxygen saturation index provides non-invasive and continuous monitoring of oxygenation status. It is calculated as MAP X FiO_2 divided by SpO_2. OSI correlates well with oxygenation index (OI = 2 × OSI for OI between 5 and 25).
- **Diagnosis of PPHN:** Preductal–postductal oxygen saturation difference >5% and labile oxygen saturation should raise the suspicion of PPHN.
- **Screening of critical congenital heart disease in neonates:** Universal predischarge pulse oximetry screening reduces early cardiac deaths due to critical congenital heart disease by one-third. The screening is done at 24 hours of age or before hospital discharge. Saturations are recorded in the right hand (preductal) and either leg (postductal). Saturation of at least 95% on both sites and a difference of 3% or less between the two readings is considered a PASS. A saturation of less than 90% on any site mandates immediate assessment, while intermediate values need re-testing.

Limitations of Pulse-oximetry

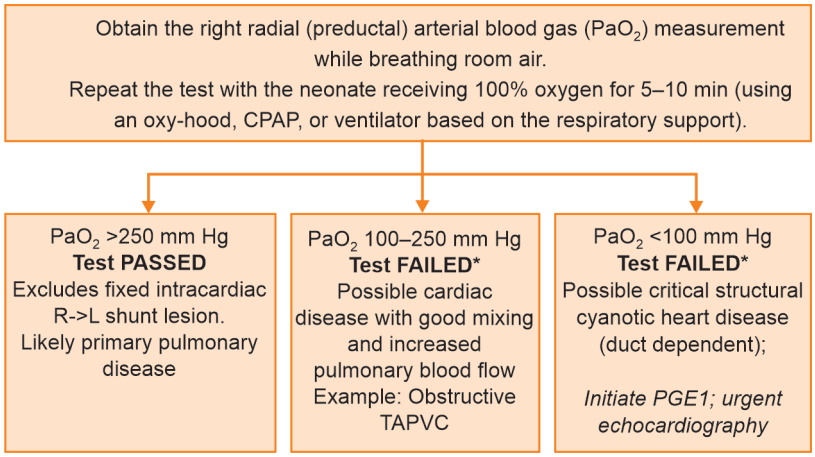
- Not a good tool to detect hyperoxia, as SpO_2 readings above 95% may occur with PaO_2 values of 80–300 mm Hg (sigmoidal oxygen dissociation curve).
- Not reliable below SpO_2 <70%.
- Ambient light, dyshemoglobinemia (methemoglobin and carboxyhemoglobin), low peripheral perfusion states, and movement of limbs interfere with the measurement.

Cardiac examination: Examination of the respiratory system is incomplete without simultaneous assessment of the cardiac system. Neonates with cardiac disease who manifest tachypnea without significant retractions may have poor perfusion, cyanosis, and abnormal heart sounds or murmurs on auscultation. Table 14.4 shows the differences in manifestation between respiratory and cardiac disorders. Monitor pulse rate, blood pressure, and capillary refill time to identify hypoperfusion secondary to prolonged hypoxemia. Hyperoxia test (Flowchart 14.1) is a simple bedside test used to distinguish cyanotic heart disease from pulmonary disease. The arterial blood gas is obtained when the neonate receives room

Table 14.4: Differences in respiratory and cardiac disorders in the newborn

	Respiratory disorders	*Cardiac disorders*
Clinical findings	• Cyanosis • Severe retractions • Normally split second heart sound • Liver may be pushed down with lung hyperexpansion but liver span will be normal	• Cyanosis, gallop rhythm, or murmur • Effortless tachypnea or mild distress • Single second heart sound • Large liver when associated with cardiac failure
Chest X-ray	• Normal heart size • Abnormal pulmonary parenchyma, such as total whiteout or patches of consolidation in pneumonia, fluid in the fissures in TTN or ground glass • Appearance in RDS	• Cardiomegaly • Decreased pulmonary vascularity and normal cardiac size—pulmonary oligemia. The blood flow to the pulmonary circulation is decreased due to right ventricular outflow obstruction with an associated right-to-left shunt • Increased pulmonary vascularity due to left-to-right shunt lesions and in cyanotic lesions such as transposition of the great vessels or total anomalous pulmonary venous return
Blood gas analysis	Increased $PaCO_2$ and decreased PaO_2	Normal $PaCO_2$ and decreased PaO_2. However, $PaCO_2$ may be raised with pulmonary congestion or an associated lung infection
Hyperoxia test	PaO_2 >150 mm Hg (except in severe PPHN)	PaO_2 <150 mm Hg

Flowchart 14.1: Hyperoxia test and its interpretation



*Persistent Pulmonary hypertension of the newborn (PPHN) can also result in a failed hyperoxia test. Unlike infants with a duct-dependent cardiac disease, those with PPHN show a pre-to-post ductal saturation difference greater than 10% on pulse- oximetry (where shunting is at ductal level) and labile saturations with agitation or clinical interventions (e.g. suctioning). Infants with cyanotic heart disease have fixed low oxygen saturations.

air (FiO_2 = 21%) and while on 100% FiO_2 for 10 minutes. Neonates with cyanotic heart disease do not have an increase in PaO_2 above 100 mm Hg after 100% oxygen administration.

Approach to Management

Specific management depends on the underlying condition. History, clinical examination, and necessary investigations help establish the diagnosis.

History

A detailed history is essential in assigning a cause of respiratory distress (Table 14.5).

Evolution of Respiratory Distress

The risk factors and evolution of respiratory distress can give a clue to diagnosis (Table 14.6).

Investigations

The common investigations performed on a neonate with respiratory distress are provided below. The selection of tests depends on the clinical condition, availability, and whether a particular test would

Table 14.5: Relevant history in a neonate with respiratory distress
Antenatal
Maternal history of
Diabetes mellitus: TTN, RDS
Pregnancy-induced hypertension or Intrauterine growth retardation: Polycythemia, hypoglycemia
Asthma: TTN
Fever or urinary tract infection in the preceding two weeks of delivery: neonatal early onset sepsis
Substance abuse: Narcotic drug withdrawal
Polyhydramnios: Tracheoesophageal fistula, neuromuscular disorders
Oligohydramnios: Pulmonary hypoplasia
Rh isoimmunization: Hydrops fetalis
Antenatal steroids: non-receipt predisposes to RDS
Previous sibling with respiratory distress: Surfactant protein B deficiency
Intranatal
Prolonged rupture of membranes, intrapartum fever, or chorioamnionitis: Sepsis
Meconium-stained liquor: Meconium aspiration syndrome, asphyxia
Fetal distress: Asphyxia
C-section without labor: TTN, RDS, PPHN
Breech presentation, instrumental delivery: Trauma, Erb's with phrenic nerve palsy
Postnatal
Onset of respiratory distress: Onset at birth: TTN, RDS, pneumothorax or air leak, MAS, congenital malformations
Onset hours or days later: Congenital heart disease, sepsis

TTN: Transient tachypnea of the newborn; *RDS:* Respiratory distress syndrome; *UTI:* Urinary tract infection; *Rh:* Rhesus; *PPHN:* Persistent pulmonary hypertension of the newborn; *MAS:* Meconium aspiration syndrome.

Table 14.6: Risk factors and evolution of respiratory distress in common neonatal conditions	
Disorder	*Evolution of the condition*
Respiratory distress syndrome	Infants with RDS are preterm and typically present within the first several hours of life, mostly immediately after delivery. The distress is marked by tachypnea, nasal flaring, grunting, and subcostal and intercostal retractions. In infants who do not receive exogenous surfactant therapy, the endogenous surfactant synthesis results in recovery of the distress in the first week.
TTNB	Risk factors include cesarean section without labor, precipitous delivery, late preterm or early term births, and gestational diabetes. Respiratory distress has onset immediately after birth, but it is mild and self-limited, with resolution in 72 hours.

(*Contd.*)

Table 14.6: Risk factors and evolution of respiratory distress in common neonatal conditions (*Contd.*)

Disorder	*Evolution of the condition*
Pneumonia	Risk factors for early onset sepsis include group *B Streptococcus* carriage in pregnant mothers, chorioamnionitis, maternal fever, prolonged rupture of membranes, preterm premature rupture of membranes, and spontaneous onset of preterm labor. Neonatal pneumonia presents as a generalized sepsis illness. Onset can be delayed by a few hours and is generally indistinguishable from RDS.
Meconium aspiration	Risk factors include meconium-stained amniotic fluid, post-term gestation, fetal distress, or perinatal depression. Onset is at birth with progressive worsening.
Pulmonary hypoplasia	Risk factors include severe oligohydramnios, renal dysplasia or agenesis, lower urinary tract obstruction, premature rupture of membranes in early gestation, diaphragmatic hernia, neuromuscular disorder. Respiratory distress has onset at birth and is severe.

be helpful in the given scenario. For example, in a neonate with suspected tension pneumothorax, it would be wise to do a transillumination of the thorax and proceed with treatment rather than wait for a chest X-ray.

1. **Gastric aspirate shake test:** The gastric aspirate shake test is a simple bedside test to predict the risk of RDS. It is advantageous in areas where radiography is unavailable. The test involves mixing 0.5 mL of gastric aspirate obtained within one hour of birth and an equal volume of 95% ethyl alcohol in a clean glass test tube (size 10 mm × 110 mm). Cork the test tube, shake it vigorously for 15 seconds, and allow it to stand for 15 minutes before examining the liquid-air interface for the stability of bubbles.[9] The presence of an entire rim of bubbles is considered a positive test, the absence of bubbles is considered negative, and the incomplete rim of bubbles is an intermediate test. The test is highly specific but has a sensitivity of only 70%. In one study, no infant with a positive test developed RDS, while 66% of those with a negative test result had RDS.
2. **Transillumination:** A fiber-optic bright light source applied to the chest wall can be used to promptly identify air leaks like pneumothorax, pneumomediastinum, pneumopericardium, and pneumoperitoneum. Severe pulmonary interstitial emphysema

(PIE) and emphysematous bullae may also transilluminate. The room must be dark while performing this test, and one must also differentiate the slight normal halo of light around the probe from increased transillumination noted from air collection. When used in small neonates, care must be exercised to avoid skin burning from the light source.

3. **Chest radiography:** Radiography is the primary diagnostic tool for respiratory distress in neonates. The commonly taken view is anteroposterior, while lateral and cross-table lateral views can be used to evaluate air leaks, pleural effusions, and placement of tubes or catheters (*see* Chapter 38: Neonatal Chest X-ray).
4. **Ultrasound:** Point of care lung ultrasonography can be used to diagnose various pulmonary conditions like RDS, TTN, pleural and pericardial effusions, detection of pneumothorax, evaluation of mediastinal and thoracic masses, assess the position and movement of the diaphragm as in eventration and diaphragmatic palsy, and confirm the position of intravascular catheters.
5. **Fluoroscopy:** Fluoroscopy is like a continuous X-ray study and can be used to evaluate vascular, tracheal, and esophageal anomalies, tracheoesophageal fistula, diaphragmatic activity, and swallowing studies.
6. **Arterial blood gas analysis (ABG):** ABG provides a snapshot of the patient's respiratory condition and must always be interpreted in the clinical context.
   a. Normal values are pH 7.35–7.45, PaO_2 50–70 mm Hg, PCO_2 35–45 mm Hg, bicarbonate 20–24 mEq/L, and base deficit of 3–5.
   b. Respiratory failure is present when there is hypoxemia (PaO_2 <50), hypercarbia ($PaCO_2$ >60), and acidosis (pH <7.2).
   c. Hypoxemia may result from both cardiac and respiratory causes.
   d. Hypercarbia is a better indicator of respiratory failure. Rising $PaCO_2$ ($PaCO_2$ >45) in the presence of falling pH (pH <7.25) denotes failure of gas exchange.
   e. The goal of ventilation is not to make the blood gases entirely normal but to keep them within acceptable target ranges.
7. **Oxygenation indices:** These indices give an idea about the severity of respiratory illness and help institute appropriate therapy and predict death and adverse respiratory outcomes. The three commonly used oxygenation indices are:

a. **Alveolar-arterial oxygen pressure difference (A–a DO_2):** This can be calculated using the formula: $AaDO_2 = (713 \times FiO_2) - (PaCO_2/0.8) - (PaO_2)$, where 0.8 indicates respiratory quotient on a mixed diet and 713 is derived from 760 mm Hg (atmospheric pressure at sea level)- 47 mmHg (alveolar water vapor pressure). In healthy infants, $AaDO_2$ is less than 20 in room air. In the face of hypoxia, normal $AaDO_2$ indicates alveolar hypoventilation or a low FiO_2. A high $AaDO_2$ may result from ventilation-perfusion (V/Q) mismatch or shunt. If one were to increase the FiO_2 to 100% and observe an increase in PaO_2, then V/Q mismatch might be operating, while no change in PaO_2 means shunt. The normal $AaDO_2$ is highly dependent on FiO_2 (for each 10% increase in FiO_2, the $AaDO_2$ value increases by 5–7 points), so the value should not be interpreted without FiO_2.
b. **Arterial-to-alveolar oxygen tension ratio (a/A ratio):** The a/A ratio should be close to 1 in a healthy infant. A ratio of less than 0.3 indicates disturbances in oxygen transfer.
c. **Oxygenation index:** OI = [mean airway pressure × FiO_2/PaO_2 (mm Hg)] × 100. OI is a very sensitive indicator of the severity of respiratory illness as it factors in the pressure cost of achieving oxygenation, namely MAP. It indicates the severity of hypoxemic respiratory failure (HRF) in neonates. An OI >15 indicates a ventilation-perfusion mismatch, and OI ≥40 is associated with a poor prognosis with mortality approaching 80%. Infants with hypoxic respiratory failure and OI >25 may benefit from inhaled nitric oxide (iNO); when OI exceeds 40, ECMO therapy is indicated. Limitations include the need for an indwelling arterial catheter for frequent sampling. OI provides only an intermittent measurement of oxygenation status.

8. **Other investigations:** Send sepsis screen, blood culture, and CSF examination as indicated. Monitor blood sugars and electrolytes. Consider echocardiography to rule out congenital heart disease and to evaluate PPHN.

Treatment of Respiratory Conditions

The management of individual conditions is discussed in subsequent chapters. The basic principles of treatment include:

- **Supportive care:** Assess temperature, airway, breathing, and circulation.

- Correct hypothermia
- **Airway:** Assess the airway for the presence of secretions. Suction the airway, if needed, and place a shoulder roll.
- **Breathing:** Assess for respiratory distress using an objective scoring system. Check SpO_2. Intubate if respiratory distress is severe or infant apneic.
- **Circulation:** Assess heart rate, blood pressure, and urine output.

Check blood glucose level, and initiate intravenous fluids if distress is severe.

- **Respiratory support:** Respiratory support provided to the infant depends on the severity of respiratory distress, hemodynamic stability, the presence of spontaneous efforts, the underlying condition, and the presence of complications. The objective is to ensure adequate oxygenation and ventilation and thereby decrease the work of breathing.
- **Monitoring for and management of complications:** Monitor for worsening distress using Silverman's score, watch for hemodynamic instability, features of PPHN, acute kidney injury due to hypoxia and complications due to mechanical ventilation, etc.

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