ACORN
Respiratory sequence

Dr p.gh.shirazi

Educational objectives

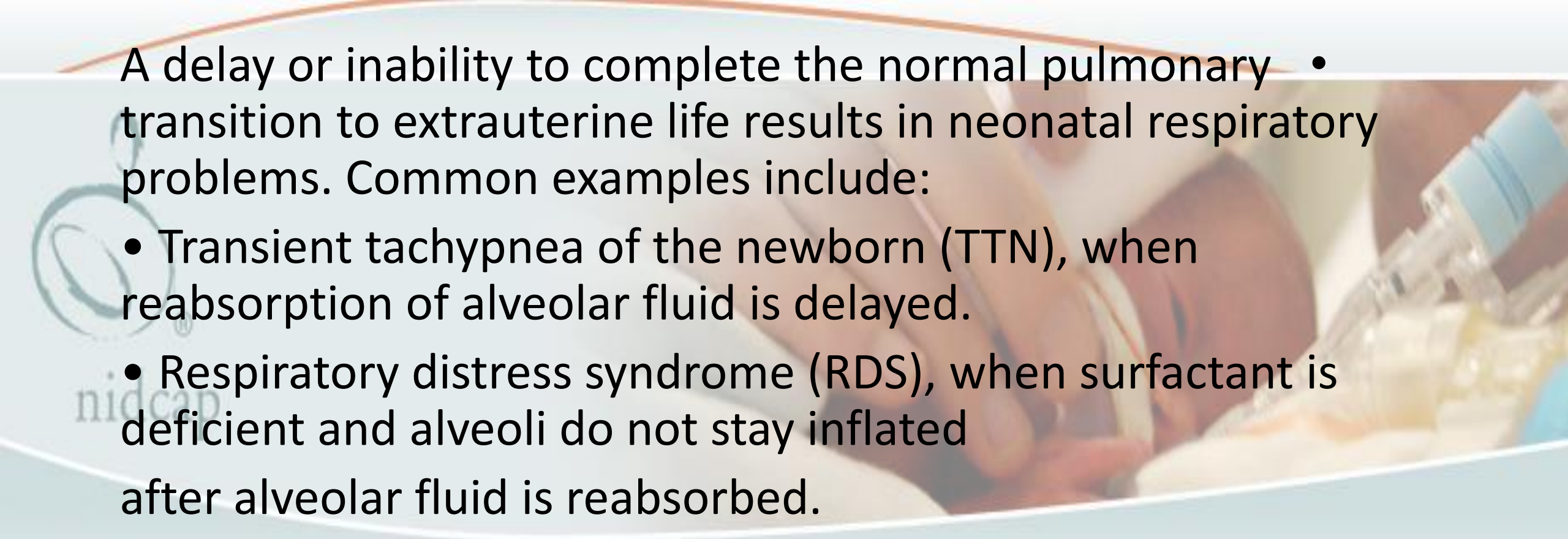
Upon completion of this chapter, you will be able to:

- Identify infants who require respiratory support or interventions.
- Apply the Acute Care of at- Risk Newborns (ACoRN) Respiratory Sequence.
- Determine oxygen requirements and select an appropriate oxygen delivery method.
- Use the Respiratory Score to organize care and monitor status on the basis of severity of respiratory distress.

- Recognize the need for, and how to initiate, respiratory support.
- Perform basic interpretation of chest radiographs and blood gas results.
- Recognize and manage the common causes of respiratory distress.
- Recognize when to exit the Respiratory Sequence to other ACoRN sequences

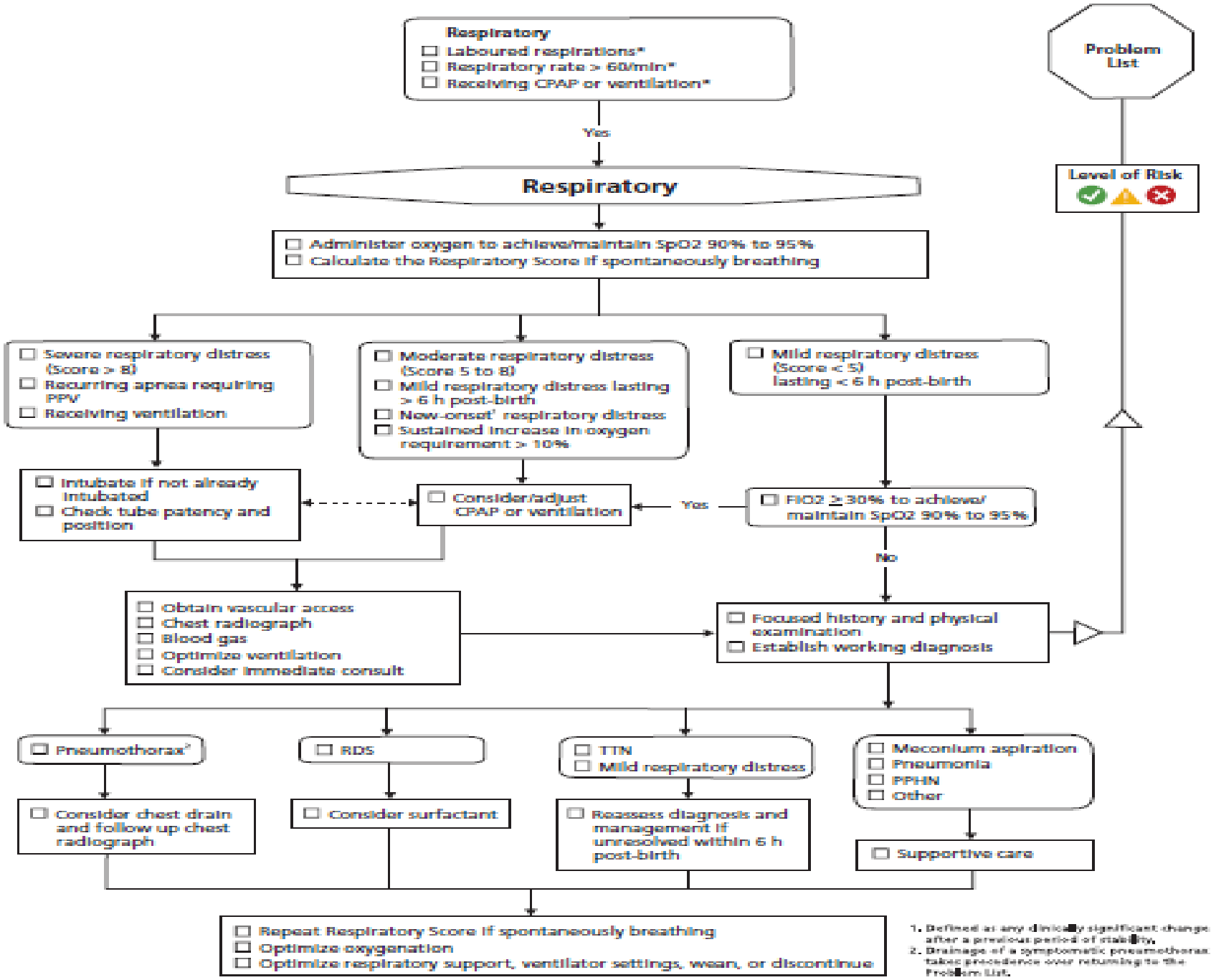
A delay or inability to complete the normal pulmonary transition to extrauterine life results in neonatal respiratory problems. Common examples include:

- Transient tachypnea of the newborn (TTN), when reabsorption of alveolar fluid is delayed.
- Respiratory distress syndrome (RDS), when surfactant is deficient and alveoli do not stay inflated after alveolar fluid is reabsorbed.
- Aspiration syndromes, when small airways and alveoli become obstructed by meconium, blood, or amniotic fluid.
- Pneumonia, when lungs become infected.



- Persistent pulmonary hypertension of the newborn (PPHN), when smooth muscle in the pulmonary vasculature does not relax and pulmonary pressures remain high, causing cyanosis from right to left or bidirectional shunting at the patent ductus arteriosus(PDA) and patent foramen ovale (PFO).
- Pneumothorax, when lungs suffer external compression from air trapped between the lung and chest wall.
- Pulmonary hypoplasia, when lungs are small and underdeveloped due to a space- occupying lesion in the chest (e.g., congenital diaphragmatic hernia [CDH]) or prolonged, severe oligohydramnios.

ACoRN Respiratory Sequence



1. Defined as any clinically significant change after a previous period of stability.
2. Drainage of a symptomatic pneumothorax takes precedence over returning to the Problem List.

Alerting Signs

- An infant who shows one or more of the following Alerting Signs enters the ACoRN Respiratory Sequence:

Respiratory
Laboured respirations*
Respiratory rate > 60/min*
Receiving CPAP or ventilation*

Laboured respirations* •

An infant with laboured respirations is also described as having respiratory distress, difficulty breathing, or increased work of breathing. The signs of laboured respirations are: •

• Nasal flaring— Outward flaring movements of the nostrils on inspiration in an attempt to move more air into the lungs.

• Grunting— Audible sounds produced as the infant exhales against a partially closed glottis in an effort to maintain end- expiratory pressure and increase functional residual capacity.

• Intercostal and subcostal retractions— Retractions of the intercostal and subcostal spaces due to increased negative pressure within the chest. Mild retractions involve the intercostal spaces only; moderate retractions involve the intercostal and subcostal spaces.

• — Paradoxical backward movements of the sternum on inspiration caused by increased negative pressure within the chest. Involvement of the intercostal, subcostal, and sternal spaces is considered severe.

- Gasping is an ominous sign of cerebral hypoxia characterized by deep, single or stacked, slow, and irregular breaths, indicating a terminal respiratory rhythm. Gasping is an Alerting Sign for the Resuscitation

Sequence and not an indication of laboured breathing.

Respiratory rate > 60 breaths / min*

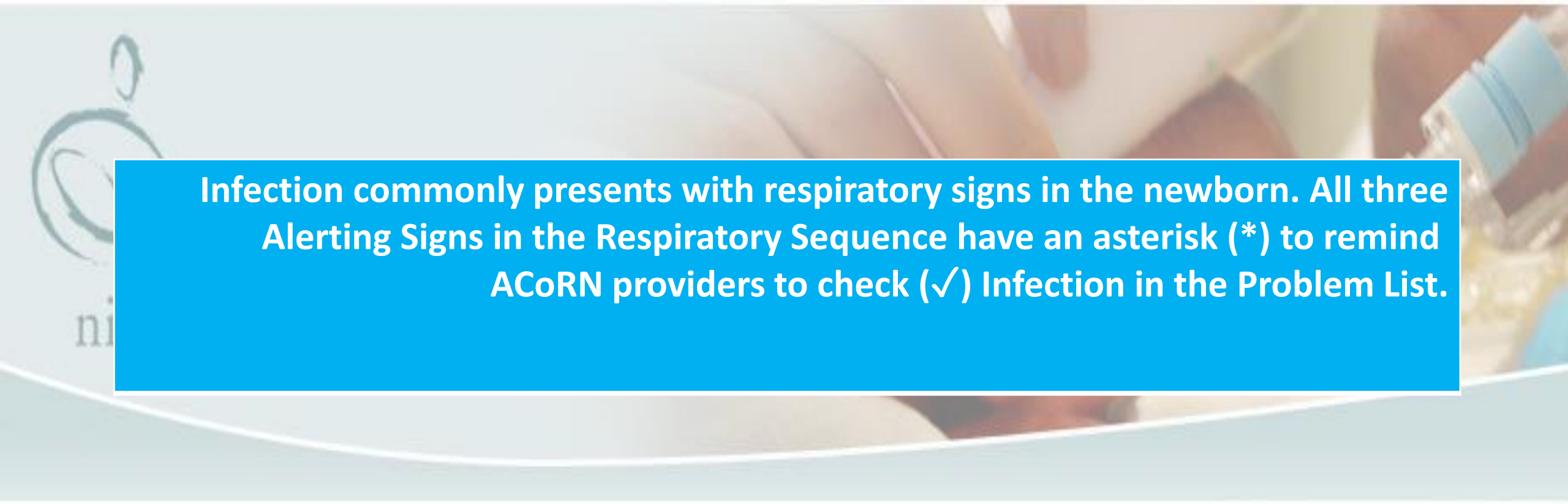
The normal newborn respiratory rate is 40 to 60 breaths/ min. Tachypnea, a respiratory rate greater than 60 breaths/ min, usually indicates an intrathoracic process causing respiratory difficulty or distress.

Tachypnea is often the first and most subtle sign of respiratory distress in infants with mildly decreased respiratory function

Receiving CPAP or ventilation* •

This Alerting Sign identifies infants who are receiving • ongoing respiratory support via either CPAP or ventilation. Any infant receiving positive inspiratory pressure (PIP) and positive end- expiratory

pressure (PEEP) qualifies as receiving ventilation, irrespective of whether it is being delivered manually or by ventilator, through an endotracheal tube (ETT; invasively) or via nasal prongs or mask (noninvasively).



Infection commonly presents with respiratory signs in the newborn. All three Alerting Signs in the Respiratory Sequence have an asterisk (*) to remind ACoRN providers to check (✓) Infection in the Problem List.

Core Steps:

Infants entering the Respiratory Sequence will have had monitoring established and some interventions performed as part of the Consolidated Core Steps. ACoRN providers should continually reassess airway patency and adequacy of respiratory drive in infants with respiratory Alerting Signs.

The Core Steps in the Respiratory Sequence are to:

- Administer oxygen as needed to achieve and maintain a SpO2 of 90% to 95%, based on pulse oximetry, and
- Calculate the Respiratory Score for all spontaneously breathing infants.

Oxygen administration

- Oxygen can be administered to a spontaneously breathing infant during resuscitation and stabilization in different ways (Figure 3.1).

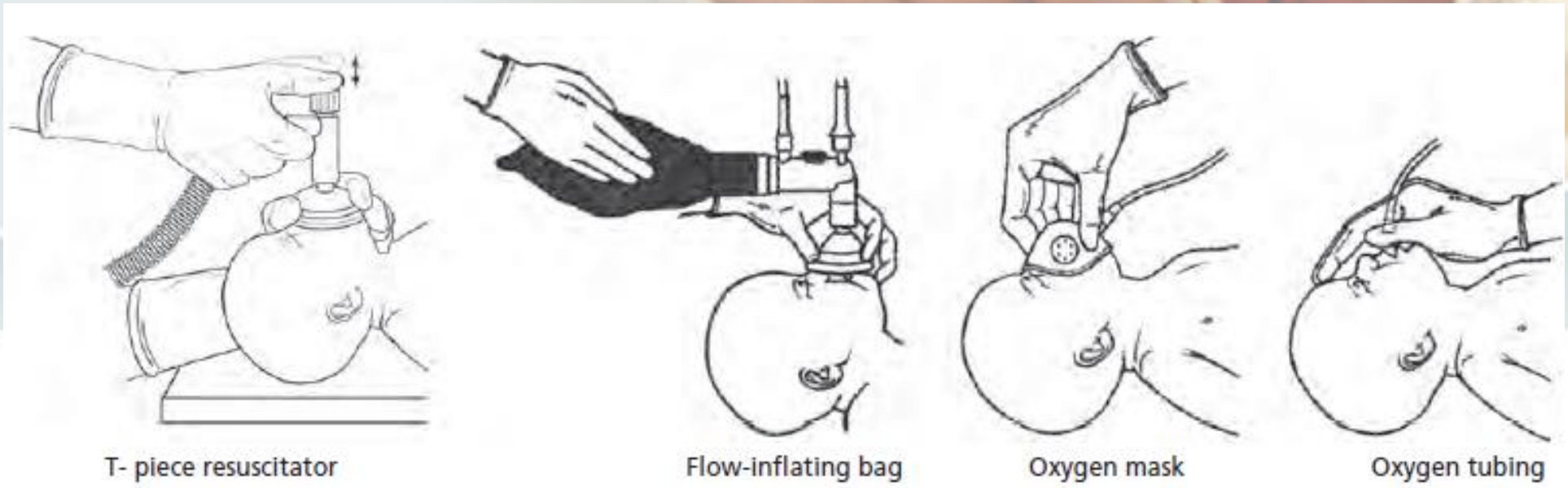


Figure 3.1. Oxygen administration.

Source: American Academy of Pediatrics, American Heart Association, Canadian Paediatric Society. Neonatal Resuscitation Textbook, 6th edition, 2011.

Oxygen administration during resuscitation

- Apply the T- piece resuscitator, flow- inflating bag, oxygen mask, or oxygen tubing/prongs to theinfant's face.
- Use a flow rate of 10 L/ min.
- A T- piece resuscitator or flow- inflating bag delivers the oxygen concentration set on the blender if there is a seal between the infant's face and the mask. When there is a seal, either device may also deliver CPAP, depending on the set flow rate.
- A T- piece resuscitator or flow- inflating bag held close to the infant's face but without a seal functions like an oxygen mask or oxygen tubing by diluting flow with room air and cannot deliver the oxygen concentration set on the blender.

Oxygen administration during stabilization

Administering oxygen to a spontaneously breathing infant during stabilization requires a less user-dependent system and, because these infants require oxygen for longer periods, it should, ideally, be humidified. Options include:

- Low- flow nasal prongs
- An oxygen hood or incubator
- Heated, humidified high- flow nasal cannula therapy (H3FNC)

Figure 3.2. Infant with nasal prongs.

An oxygen hood, placed over the infant's head, contains the breathing environment .

- Blended humidified oxygen/ air is administered at the concentration set on the blender.
- A gas flow greater than 7 L/ min prevents CO2 accumulation.
- An exact amount of delivered oxygen can be determined using an oxygen analyzer placed close to the

infant's mouth and adjusted via the oxygen blender to achieve the desired SpO2 between 90% and 95%.

- Gas entering the hood should be warmed to 32°C to 34°C and humidified.

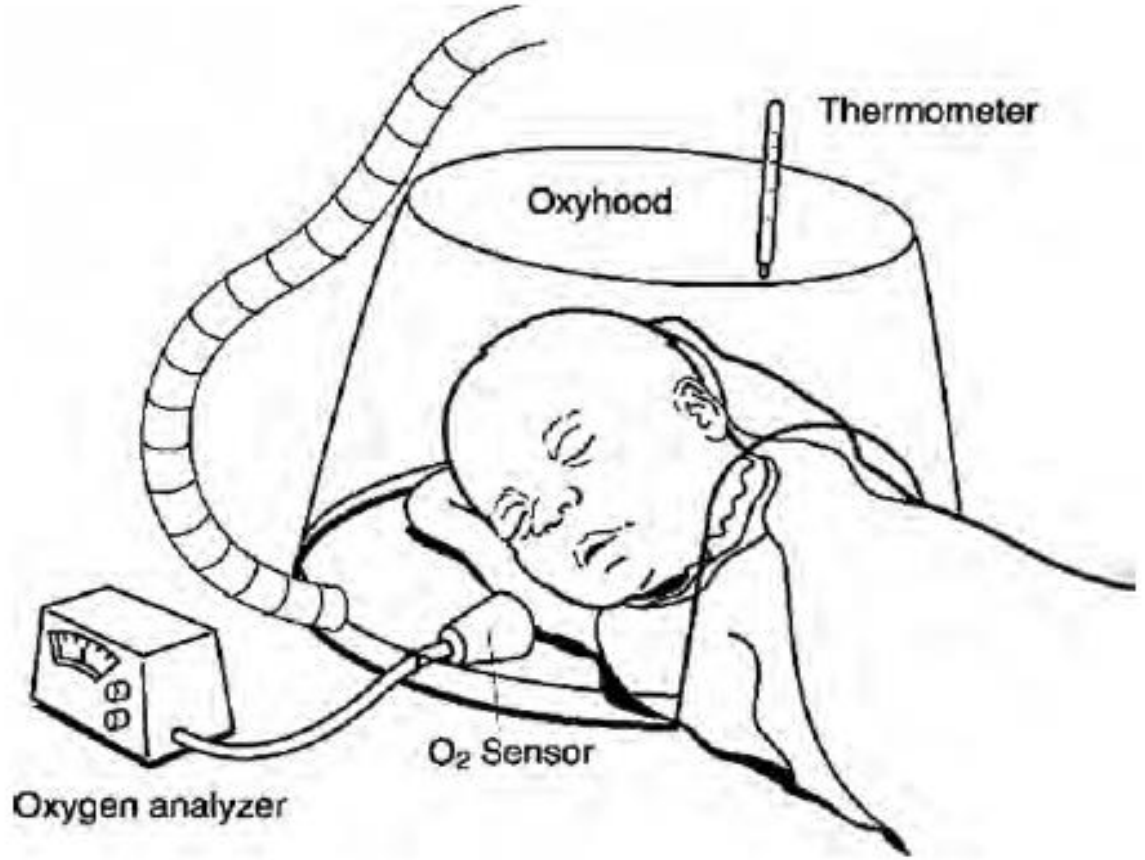


Figure 3.3. Infant in oxygen hood.

- Incubators manufactured after 1990 can administer high oxygen concentrations, humidity, and warmth and have built- in oxygen analyzers that continuously adjust the oxygen flow to maintain a preset concentration. Older incubators or incubators with the port- hole doors opened frequently are not able to maintain set oxygen concentrations.
- H3FNC administers blended oxygen at flows of 1 L/ min to 8 L/ min via special prongs.
- Gas is humidified to prevent irritation and drying of the nasal mucosa.
- Oxygen can be titrated up or down to maintain a SpO2 of 90% to 95%.
- Higher flow rates can wash out dead space in the infant's upper airway, which allows accurate assessment of the oxygen content being delivered to the alveoli.
- Theoretical concerns exist about the pressure generated by high flow rates, with current systems being unable to measure pressures delivered. High pressures may increase the risk of air leaks and become more likely when exhalation is impaired, such as when cannulae are greater than half the diameter of an infant's nares or migrate too far into the nares and when an infant's mouth is closed.

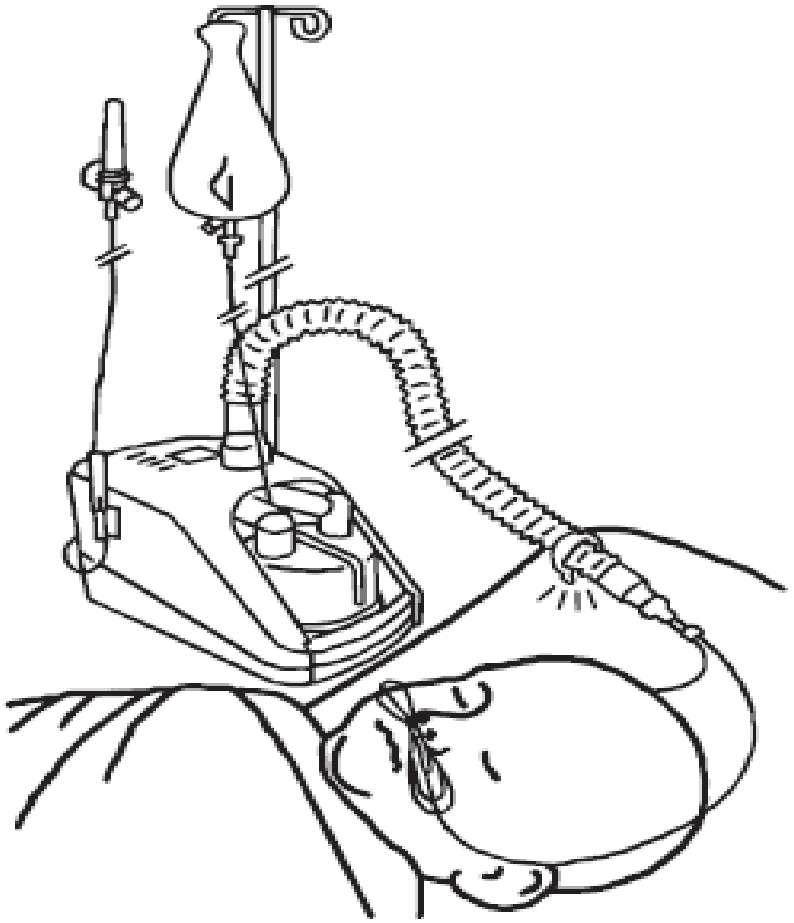


Figure 3.4. Infant with heated, humidified high-flow nasal cannula.

Oxygen delivery

Oxygen delivery to an infant's organs depends on cardiac output and the oxygen content (CaO_2) in the circulating blood. Factors affecting the blood's ability to carry oxygen include:

- Oxygen saturation (SpO_2 or SaO_2)
- Hemoglobin (Hb) concentration
- Oxygen dissolved in plasma (PO_2 or PaO_2)

If these factors are known, the amount of oxygen carried to the infant's tissues can be calculated using this formula:

$$CaO_2 = (SaO_2 \times Hb \times 1.34\ g/mL) + 0.003\ (PaO_2)$$

Most of the oxygen carried in blood is bound to Hb in an approximate ratio of bound to dissolved oxygen of 40:1. The amount of oxygen bound to Hb or the oxygen saturation is measured using pulse oximetry.

Pulse oximetry:

Pulse oximetry is frequently used to monitor an infant's oxygenation because it is noninvasive, easy to use, and provides immediate readings in a continuous display. SpO2 closely reflects SaO2. Applying the

probe to an infant's right hand or wrist measures pre- ductal oxygen saturation, which reflects the oxygen content of blood coming from the lungs and going to the brain.

Safe oxygen saturation levels for infants

While the ideal oxygen saturation range for infants, particularly preterm infants, is unclear, levels of 90% to 95% are generally recommended for newborns receiving supplemental oxygen. Oxygen saturation values greater than 95% are associated with oxygen toxicity, which can cause tissue and organ damage.

The lower an infant's gestational age (GA), the greater the risk for oxygen toxicity. Term infants with hypoxic ischemic injury are also at high risk. When Hb levels and cardiac function are normal, oxygen saturations in the normal range indicate adequate oxygen delivery to tissues and organs which, in turn, prevents the development of acidosis and pulmonary vasoconstriction. However, while oxygen saturation monitoring provides a good indication of how effective respiratory interventions have been in oxygenating the blood, it is not an indicator of effective breathing or ventilation

Hemoglobin concentration

Adequate Hb levels are essential for oxygen delivery. In the face of severe anemia, cyanosis may not be detectable because approximately 50 g of deoxygenated Hb is required for an infant to appear cyanotic on

inspection. An anemic infant with a Hb of 100 g/ L, for example, would not appear cyanotic until the SpO2 dropped to 50%. When significant anemia is present, hypoxemic infants may appear pale rather than cyanotic.

Blood transfusion can be lifesaving in cases with decreased oxygen carrying capacity secondary to anemia.

Partial pressure of oxygen

PO2 is a measure of the partial pressure exerted by oxygen molecules dissolved in plasma. As PO2 increases, more oxygen is bound to Hb, until saturation is complete. PaO2 indicates how well the lungs are transferring oxygen to blood. As PO2 increases, saturation also rises rapidly, resulting in more oxygen bound to Hb and available

for transport in circulating blood. When SO2 rises above 95%, large changes in PO2 cause minimal additional increases in saturation. This hyperoxic state increases risk for oxygen toxicity.

In newborns, fetal Hb binds oxygen with greater affinity than adult Hb. This is represented by a shift in the oxyhemoglobin dissociation curve to the left for fetal Hb

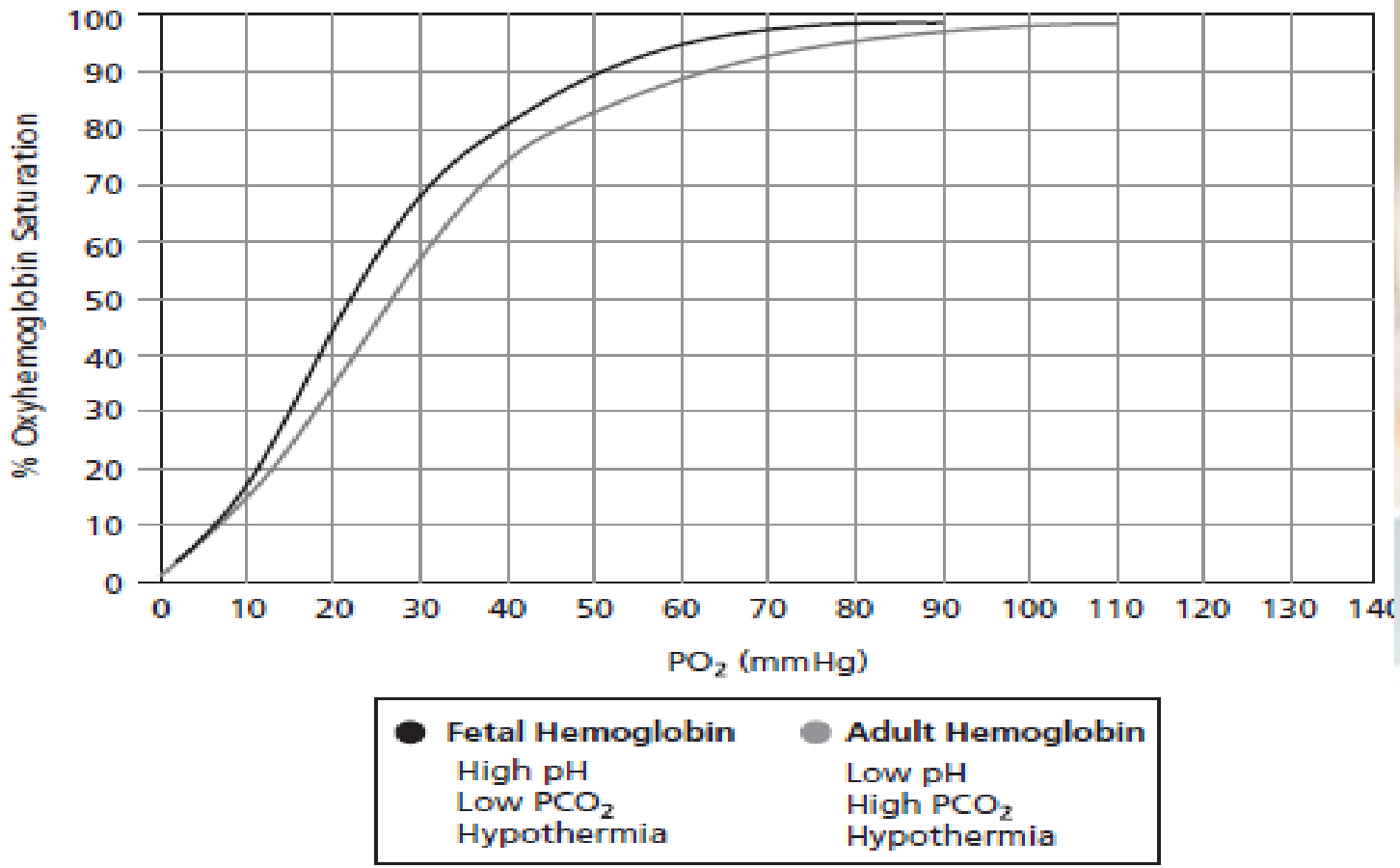
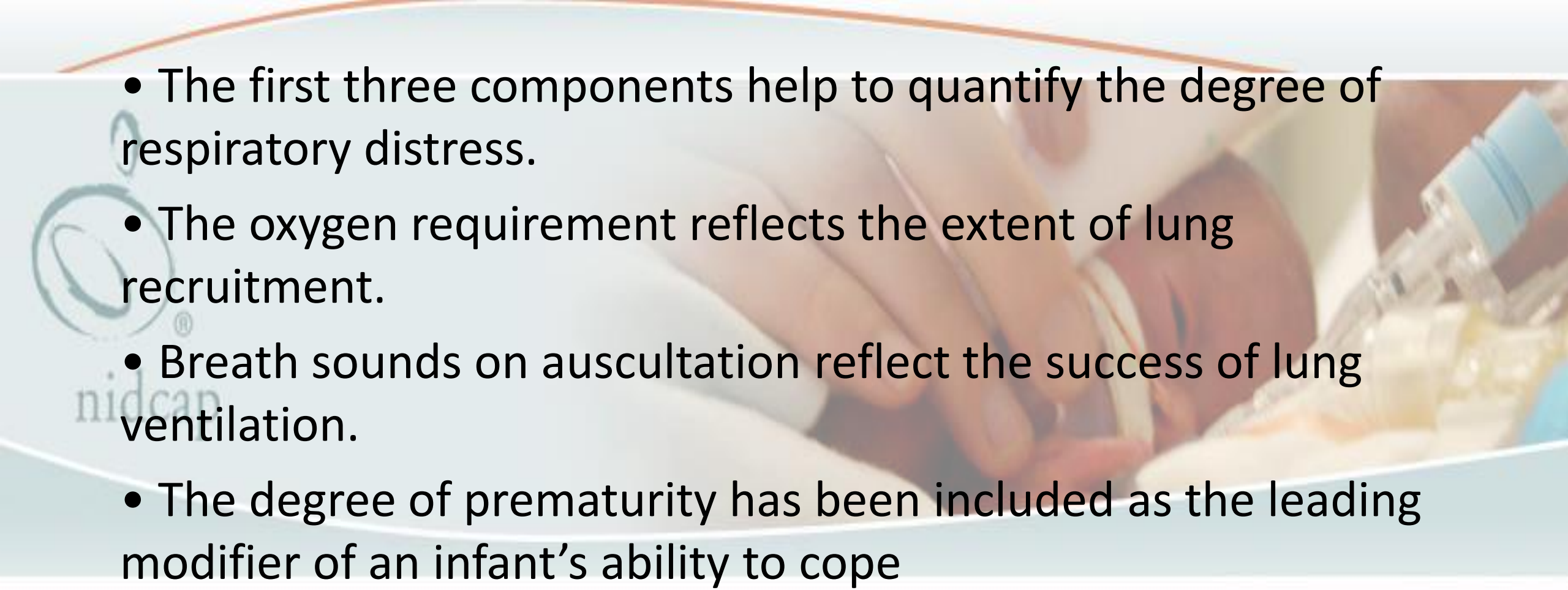


Figure 3.5. The oxyhemoglobin dissociation curve.

Calculation of the Respiratory Score

Infants who have difficulty inflating and ventilating their lungs show increased work of breathing and develop respiratory distress. Judging the severity of respiratory distress is a skill acquired with experience. The Respiratory Score (Table 3.1) assists ACoRN providers in recognizing the signs and symptoms of distress that need assessment. It is used in all infants who are breathing spontaneously, including those being treated with CPAP or noninvasive positive pressure ventilation (PPV). The Respiratory Score is not intended for infants who are intubated and receiving ventilation.

Table 3.1 lists the six components of the Respiratory Score with their descriptors. Each component is scored from 0 to 2



- The first three components help to quantify the degree of respiratory distress.
- The oxygen requirement reflects the extent of lung recruitment.
- Breath sounds on auscultation reflect the success of lung ventilation.
- The degree of prematurity has been included as the leading modifier of an infant's ability to cope

with respiratory distress. The more premature the infant, the will occur. earlier and quicker decompensation

Table 3.1.
The Respiratory Score

Score	0	1	2
Respiratory rate	40 to 60/min	60 to 80/min	> 80/min
Retractions	None	Intercostal or subcostal retractions (or both)	Intercostal, subcostal, and sternal retractions
Grunting	None	With stimulation	Continuous at rest
Oxygen requirement*	None	≤ 30%	> 30%
Breath sounds on auscultation	Easily heard throughout	Decreased	Barely heard
Prematurity	> 34 weeks	30 to 34 weeks	< 30 weeks
		Respiratory Score	__/12

Adapted from Downes JJ, Vidyasagar D, Boggs TR, Morrow GM. Respiratory distress syndrome of newborn infants. I. New clinical scoring system (RDS score) with acid-base and blood-gas correlations. Clin Pediatr 1970; 9(6):325–331.

**Note:* An infant receiving oxygen before an O2 analyzer is set up or in an open oxygen delivery system is assigned a score of 1.

The Respiratory Score is the sum of the six component items. It is used by itself or in conjunction with other clinical factors to organize care of the infant presenting with:

- Recurring apnea that requires PPV,
- Mild respiratory distress that lasts longer than 6 h post- birth,
- New- onset respiratory distress in a previously asymptomatic infant, and
- A sustained increase in oxygen requirement more than 10% above baseline, for 10 min or longer, to keep oxygen saturations within the target range.

The Respiratory Score is also used to track changes in the severity of the respiratory distress over time in infants who are not intubated.

Organization of Care

In the ACoRN Respiratory Sequence, Organization of Care is based on severity of respiratory distress

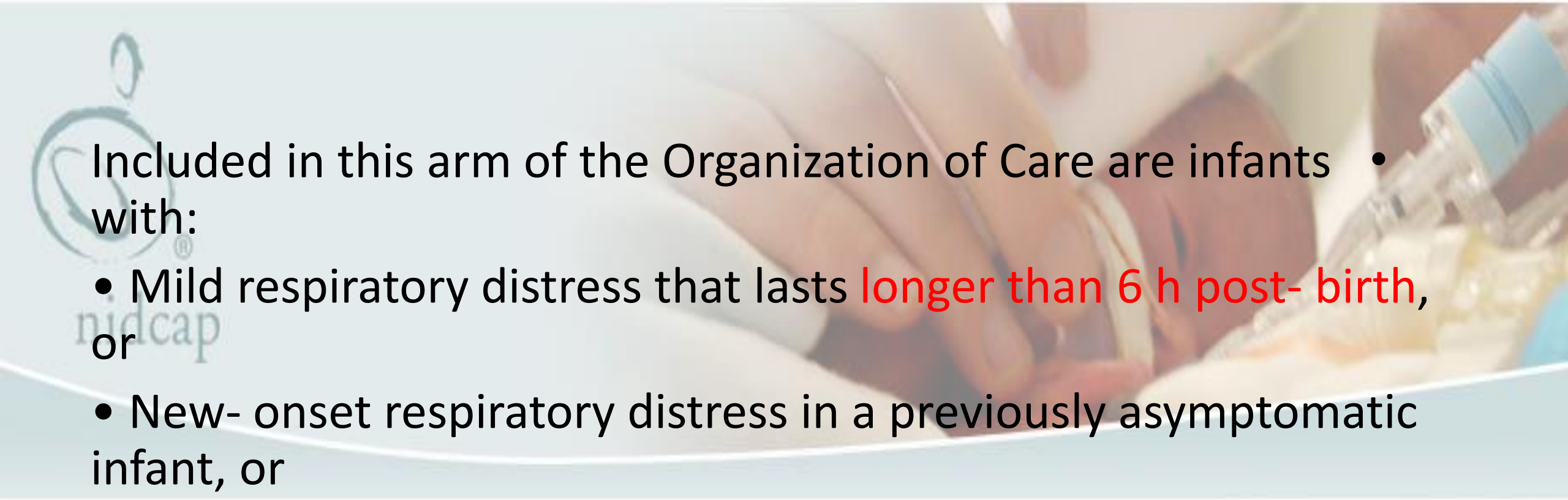
as defined by the Respiratory Score and is modified by the presence of additional clinical factors.

Mild respiratory distress is defined as:

- A Respiratory Score less than 5, with signs starting at birth and lasting less than 6 h.

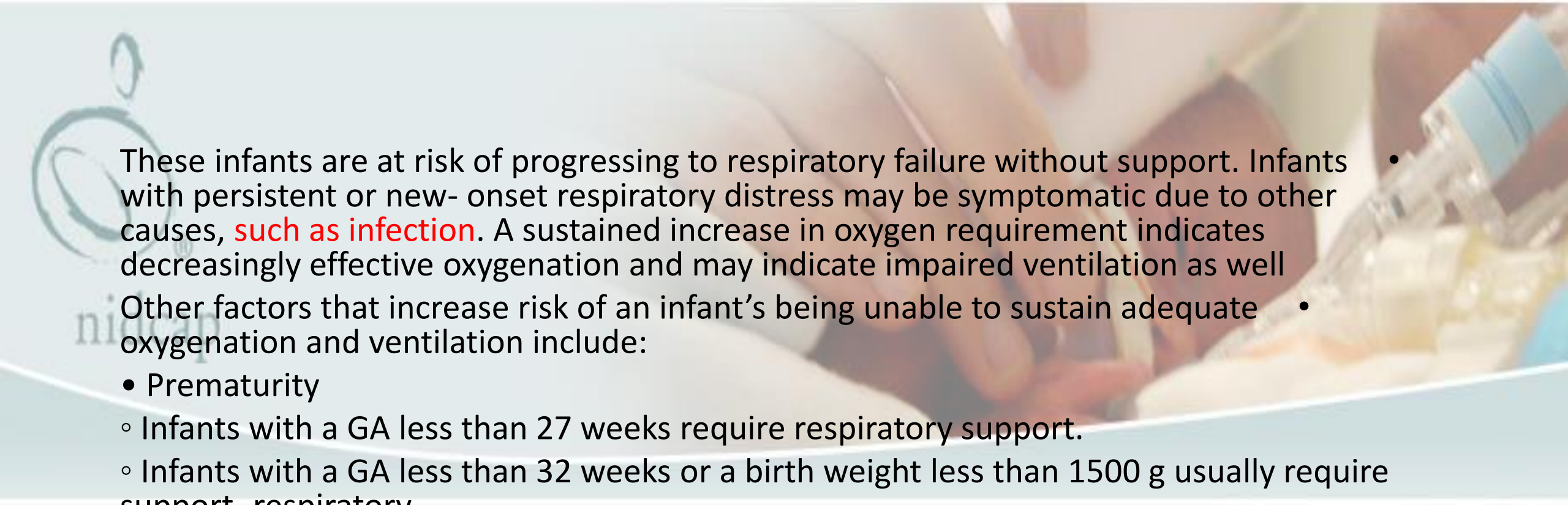
Moderate respiratory distress is defined as:

- A Respiratory Score of 5 to 8.



Included in this arm of the Organization of Care are infants with:

- Mild respiratory distress that lasts longer than 6 h post- birth, or
- New- onset respiratory distress in a previously asymptomatic infant, or
- A sustained increase in oxygen requirement more than 10% above baseline, for 10 min or longer, to keep oxygen saturations within the target range



These infants are at risk of progressing to respiratory failure without support. Infants with persistent or new- onset respiratory distress may be symptomatic due to other causes, such as infection. A sustained increase in oxygen requirement indicates decreasingly effective oxygenation and may indicate impaired ventilation as well

Other factors that increase risk of an infant's being unable to sustain adequate oxygenation and ventilation include:

- Prematurity
  - Infants with a GA less than 27 weeks require respiratory support.
  - Infants with a GA less than 32 weeks or a birth weight less than 1500 g usually require support. respiratory
  - Infants born less than 34 weeks GA, whose antenatal steroid coverage is incomplete, may respiratory support. require
- Baseline oxygen requirements greater than 30% indicate that an infant's respiratory reserves are low.

Severe respiratory distress is defined as:

- A Respiratory Score greater than 8.

Included in this arm of the Organization of Care are infants:

- With recurring apnea that requires PPV, or
- Who are already receiving ventilation due to respiratory failure during resuscitation or a previous

passage through the Respiratory Sequence.

These infants require immediate intervention to prevent further deterioration and even death.

Response

Mild respiratory distress:

Infants with mild respiratory distress lasting less than 6 h post- birth require close observation and

regular monitoring using the Respiratory Score. They may require supplemental oxygen to maintain

blood oxygen levels within the target SpO2 range of 90% to 95%.

Any of the following signs suggest that an infant is no longer meeting criteria for mild respiratory

distress and should prompt the ACoRN provider to reassess and increase respiratory support:

- Persistence of respiratory signs beyond 6 h of age
- A worsening Respiratory Score
- A sustained increase in oxygen requirement of greater than 10% from baseline for 10 min or more
- An oxygen requirement of 30% or greater to maintain saturations within the target range. In an

infant who is not receiving respiratory support, this indicates atelectasis (loss of lung volume) and

inability to sustain adequate oxygenation.

Moderate respiratory distress

Infants who are breathing spontaneously but experiencing moderate respiratory distress will benefit from support with CPAP, which prevents atelectasis by stabilizing the small airways and chest wall at end-expiration. CPAP can delay or prevent the progression to severe respiratory distress and eventual respiratory failure. A sustained increase in oxygen requirements in an infant with moderate respiratory distress usually indicates a loss of lung volume and the need to increase the level of respiratory support provided. Increasing the CPAP level should help to stabilize lung volumes and improve oxygenation.

Higher CPAP levels may increase the risk for pneumothorax. Administering CPAP pressures greater than 8 cmH2O or switching to noninvasive PPV (biphasic respiratory support) should be decided in consultation with your referral centre.

In the extreme preterm infant with RDS, improving lung function may require the administration of surfactant.

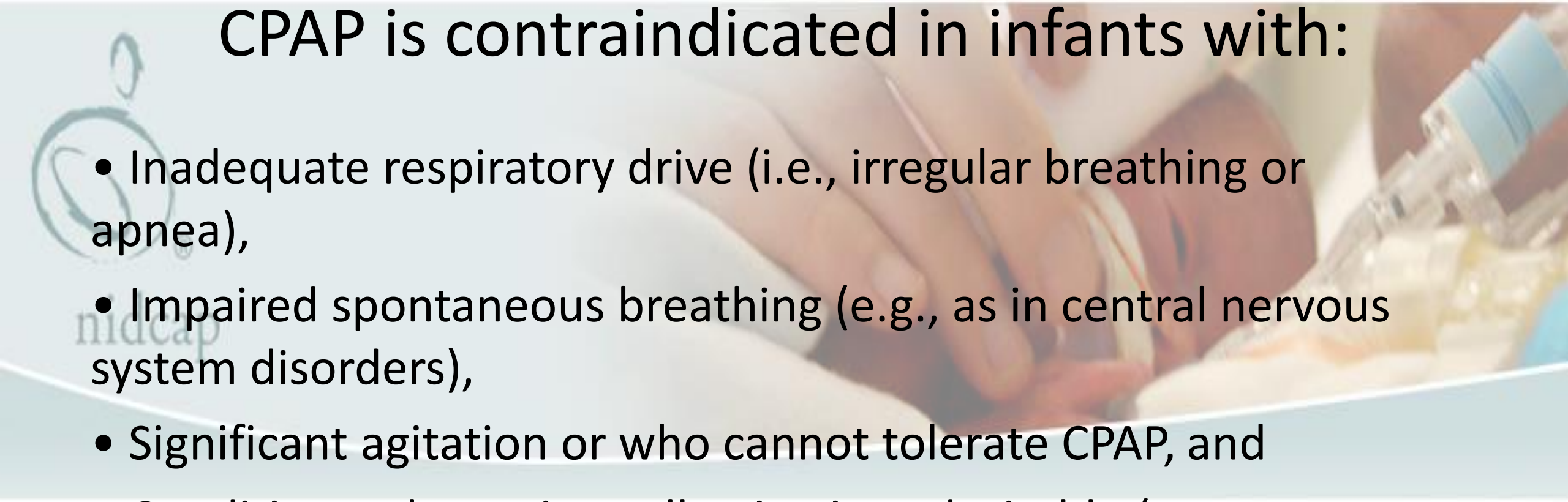
Continuous positive airway pressure

CPAP decreases the need for endotracheal intubation and mechanical ventilation in infants with moderate respiratory distress and good respiratory drive. CPAP must be administered and monitored by on- site, trained personnel, in settings resourced to provide additional respiratory support, especially if escalation in care is anticipated. The purpose of CPAP is to:

- Improve arterial PO2 to reduce inspired oxygen concentration in infants with respiratory distress who do not require mechanical ventilation,
- Stabilize respiratory function on extubation from mechanical ventilation, and
- Treat obstructive apnea in some preterm infants.

CPAP reduces mixed and obstructive apnea. It has no effect on central apnea.

CPAP is contraindicated in infants with:



- Inadequate respiratory drive (i.e., irregular breathing or apnea),
- Impaired spontaneous breathing (e.g., as in central nervous system disorders),
- Significant agitation or who cannot tolerate CPAP, and
- Conditions where air swallowing is undesirable (e.g., gastrointestinal obstruction, necrotizing enterocolitis, congenital diaphragmatic hernia [CDH]).

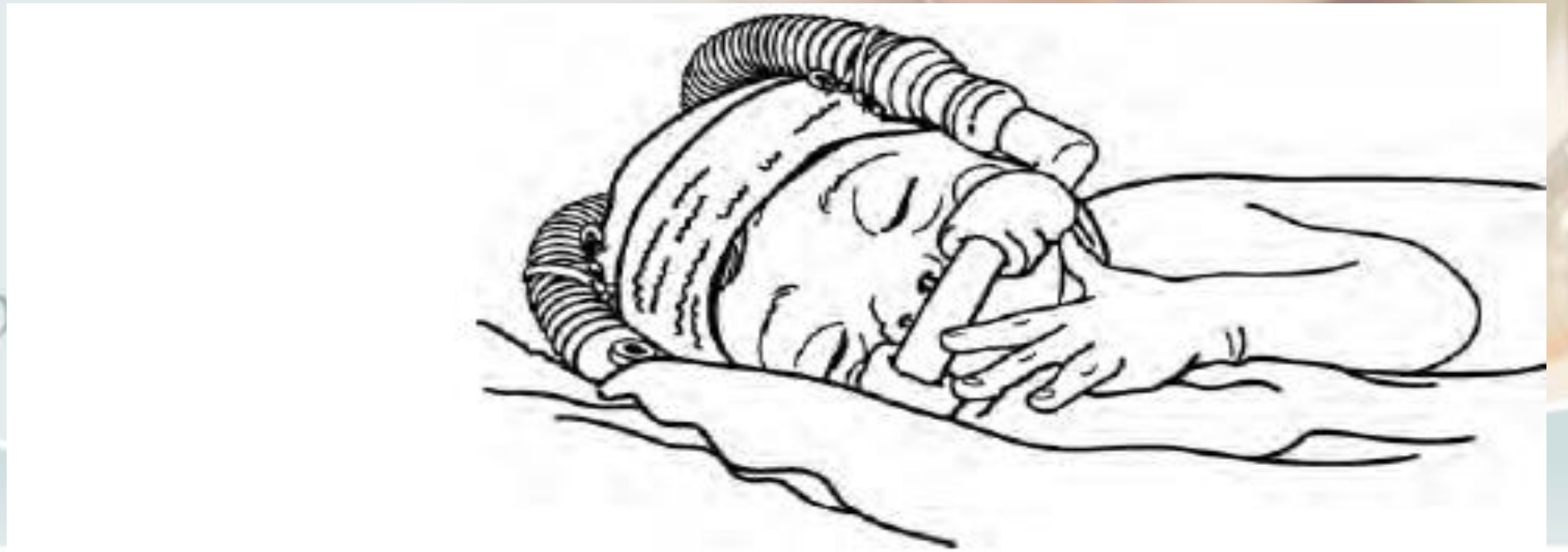


Figure 3.6. Infant on CPAP.

Severe respiratory distress

- Infants with severe respiratory distress, recurrent apnea, or gasping require immediate intubation andventilation to prevent respiratory failure. If a care provider with intubation skills is not present, use of a

laryngeal mask airway should be considered.

Intubation:

Intubation should not be delayed for intravenous (IV) access and premedication in infants requiring urgent or emergent care (e.g., during resuscitation). However, newborns should be premedicated whenever possible because laryngoscopy and intubation are painful procedures that can cause hypertension, increased intracranial pressure, bradycardia, and hypoxia. Premedication provides analgesia and can lessen hemodynamic consequences. A typical premedication regimen includes combining atropine and opiates (e.g., morphine or fentanyl) with a short- acting paralyzing agent (e.g., succinylcholine). The paralytic should only be administered by a care provider who is familiar with its use and skilled in neonatal intubation.

In the event of a sudden deterioration in an intubated infant, airway patency should be assessed.

The DOPE acronym can help identify and troubleshoot possible causes.

D . . . Displaced ETT? Has the infant accidentally extubated or is the ETT in the right main stem

bronchus? Ensuring that the ETT is not displaced from its proper position involves:

- Making certain that it is at the same measurement mark at the lip or nares as when inserted,
- Using an exhaled CO2 detector to determine whether the tube is inserted in the airway, not

the esophagus,

- Auscultating for presence and symmetry of breath sounds in the chest but not over the stomach

area (this may not be a reliable sign in small infants), and

- Inspecting the tube position using a laryngoscope

O . . . Obstructed airway or ETT? Kinked or blocked by secretions?

P . . . Pneumothorax or other critical diagnosis? Other possible causes include pulmonary interstitial

emphysema, or atelectasis.

E . . . Equipment working and ventilation optimized?

Mechanical ventilation

Administering mechanical ventilation (cycled positive inspiratory pressure [PIP] with PEEP) stabilizes

the infant's small airways and chest wall, expands the lungs during inspiration by delivering a tidal

volume at a set respiratory rate, and prevents atelectasis at end-expiration.

Ventilation is delivered via an ETT:

- Manually, using a T- piece resuscitator or self- or flow- inflating bag, or
- Using a ventilator.

The indications for ventilation include:

- Ineffective respiration with decreased respiratory drive (irregular breathing or apnea),
- Severe respiratory distress (Respiratory Score greater than 8),
- Moderate respiratory distress (Respiratory Score 5 to 8) with unsatisfactory blood gases (pH ≤ 7.25 and PCO2 ≥ 55),
- Increasing Respiratory Score, increasing oxygen requirements despite CPAP (or both),
- The decision to administer surfactant,
- Use as an alternative to CPAP for infants requiring transport.

Mechanical ventilation must be administered and monitored by on- site, trained personnel in settings resourced to provide respiratory support, especially if escalation in care is anticipated.

Next Steps

The Next Steps in the Respiratory Sequence should include obtaining a focused history and conducting a physical examination.

For infants with moderate to severe respiratory distress, Next Steps also include obtaining vascular access, ordering diagnostic tests and imaging (e.g., blood gases and a chest radiograph), optimizing ventilation, and requesting an expert consult.

Focused history:

Important information to gather for a focused respiratory history includes:

Antepartum

- Confirm GA and accuracy of dates
- Prenatal ultrasound findings, for evidence of conditions that impair lung function, such as CDH,

congenital pulmonary airway malformation (congenital cystic adenomatoid malformation),

abnormal lung echotexture, and oligohydramnios

- Maternal diabetes, as a risk factor for RDS or cardiac outflow tract anomalies
- Risk factors for congenital pneumonia, including maternal Group B Streptococcus (GBS) status,premature rupture of membranes, maternal fever, or chorioamnionitis
- Receipt of antenatal steroids for pulmonary maturation
- Maternal use of medications (e.g., selective serotonin reuptake inhibitors) which may cause tachypnea or PPHN, or illicit substances, particularly narcotics, which can suppress respiratory drive

Intrapartum:

- Presence of atypical or abnormal fetal heart rate during labour and delivery
- Meconium- stained amniotic fluid
- Time of rupture of membranes
- Signs of chorioamnionitis (maternal fever, uterine tenderness, fetal tachycardia, foul-smelling vaginal discharge)
- Nature of labour and mode of delivery
- Medications used
- Intrapartum antibiotics for GBS prophylaxis

Neonatal:

- Umbilical cord blood gas results (arterial and venous)
- Condition at birth, including Apgar score
- Need for resuscitation, and response
- Timing for onset of symptoms (i.e., present at birth or after a period of normal respiratory function)
- GA and birth weight

Focused physical examination:

A focused respiratory examination includes:

Observation

- Laboured respirations (e.g., nasal flaring, retractions, or grunting)
- Colour of skin and mucous membranes (i.e., peripheral versus central cyanosis)
- Respiratory support (e.g., oxygen requirements, size and position of ETT, ventilator settings)

Examination:

- Auscultate both lung fields for equality and nature of breath sounds. Diminished breath sounds unilaterally

may signal intubation of the right bronchus, pneumonia, atelectasis, or a pneumothorax or

other space- occupying lesion (e.g., a diaphragmatic hernia).

- Listen for grunting, inspiratory stridor, expiratory wheeze, crackles.
- Check for cleft palate or micrognathia (small jaw).
- Pass a nasogastric tube through each nare to rule out choanal atresia.

Vascular access:

• Obtain vascular access to administer dextrose solution. Provide fluids and glucose during stabilization

and early management. Infants with moderate to severe respiratory distress and those receiving respiratory

support cannot be orally fed because of poor oral– motor coordination and risk of aspiration.

• Consider risks for infection and need for antibiotics

Chest radiograph:

Order a chest radiograph to aid diagnosis, help optimize PEEP/ CPAP levels for optimal lung volume, and guide further management.

Blood gas:

Blood gases help monitor oxygenation (SaO2 and PaO2), ventilation (PaCO2), and acid base status.

- pH estimates the blood total acid load, which mostly reflects dissolved CO2 but may also include

metabolic acids (e.g., lactic acid).

- PCO2 indicates how well the lungs are removing CO2 from blood (ventilation).
- PaO2 (arterial PO2) indicates how well the lungs are transferring oxygen to blood (oxygenation) in

relation to % inspired oxygen.

- Base deficit (BD) estimates how much metabolic acid is present in the blood. Base excess (BE), the

negative value of BD, and bicarbonate are also used to describe acid- base status.

Arterial, capillary, or venous samples are nearly equally useful for determining PCO_2, pH, and BD.

In acute respiratory illness, blood gases are considered satisfactory when the pH is 7.25 to 7.40 and the PCO_2 is 45 mmHg to 55 mmHg. An acidosis ($pH \leq 7.25$) with PCO_2 at or above 55 indicates poor ventilation (respiratory acidosis).

Optimize ventilation:

Optimizing ventilation involves ensuring that:

- The infant is connected to the ventilator,
- The ventilator or manual ventilation equipment is delivering the settings indicated and is not malfunctioning,
- Chest expansion can be observed, and breath sounds are equal and symmetrical,
- The infant breathes in synchrony with the ventilator and work of breathing has decreased, and
- Pulse oximetry and blood gases are within the target range.

Consider immediate consult

Consideration should be given to obtaining immediate consultation for guidance depending on expertise with managing neonatal respiratory disease and the resources available on site. When an infant's needs exceed or are anticipated to exceed site capabilities, early recognition and planning for transport can be lifesaving.

While waiting for diagnostic test results, the ACoRN provider should establish a working diagnosis based on the presentation of mild, moderate, or severe respiratory distress and a Level of Risk category for this infant.

In a spontaneously breathing infant, the ACoRN Respiratory Score is used to monitor an infant's condition and response to interventions over time. It is used to guide the organization of care according

to changes in severity

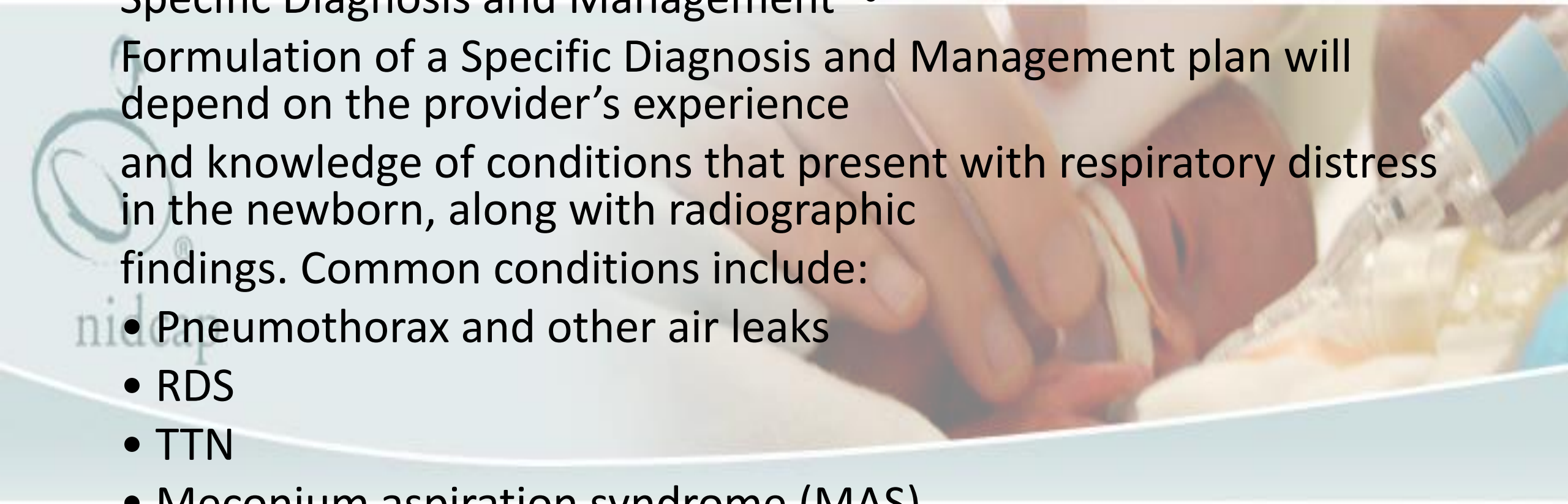
Specific Diagnosis and Management •

Formulation of a Specific Diagnosis and Management plan will depend on the provider's experience

and knowledge of conditions that present with respiratory distress in the newborn, along with radiographic

findings. Common conditions include:

- Pneumothorax and other air leaks
- RDS
- TTN
- Meconium aspiration syndrome (MAS)
- Pneumonia
- PPHN



Pneumothorax

A pneumothorax occurs when overdistension of an infant's lung results in rupture of the alveoli or terminal bronchioles and release of intrapulmonary air into the pleural space ('air leak').

Clinically, a pneumothorax may occur when there is:

- rapid improvement in lung compliance after surfactant therapy,
- delivery of excessive airway pressures, or
- plugging of small airways causing a ball- valve effect, as in meconium aspiration.

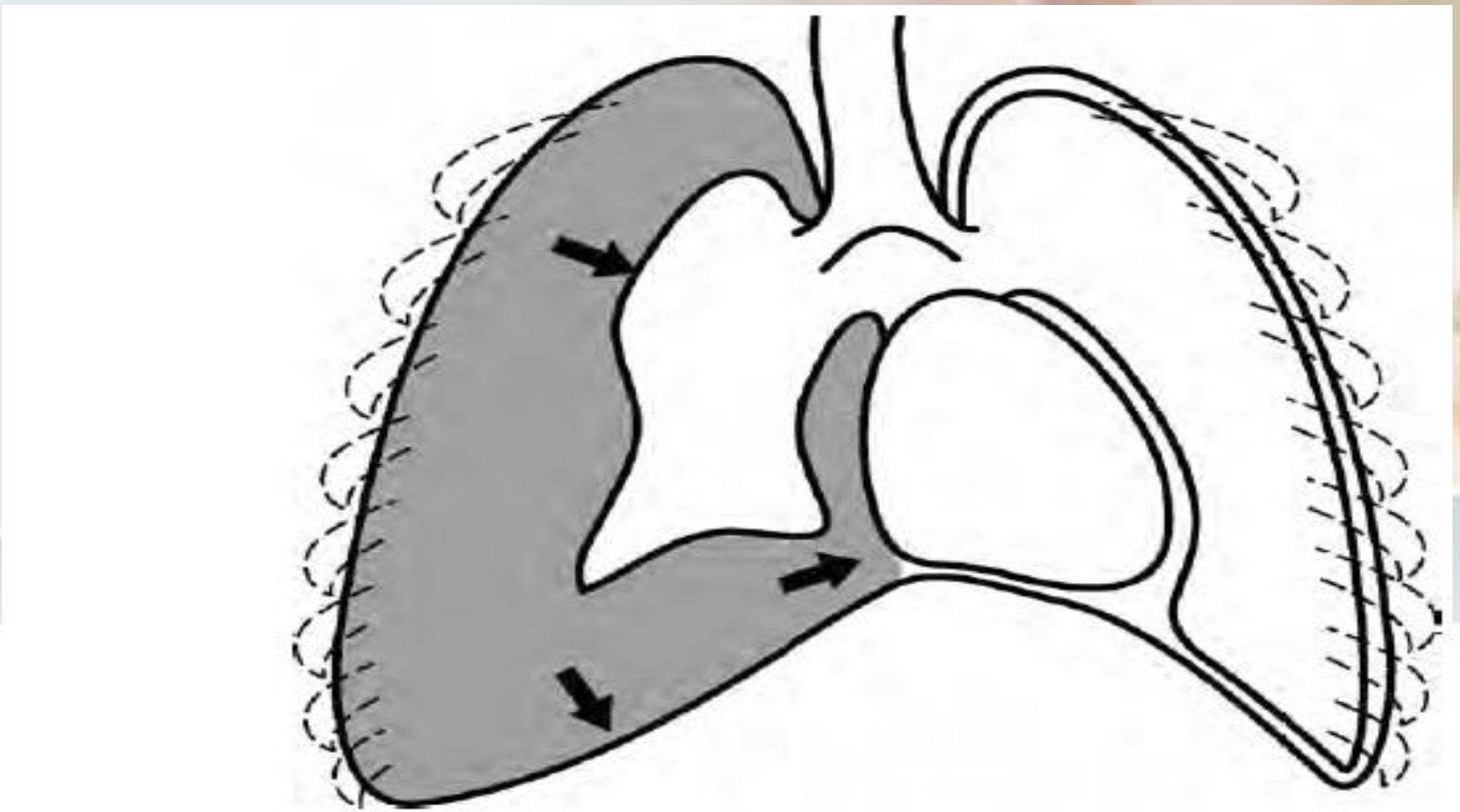


Figure 3.7. Pneumothorax.

Infants at high risk for pneumothorax include those:

- with lung disease (RDS, aspiration syndromes, or hypoplastic lungs), or
- receiving respiratory support, especially high levels of CPAP or mechanical ventilation.

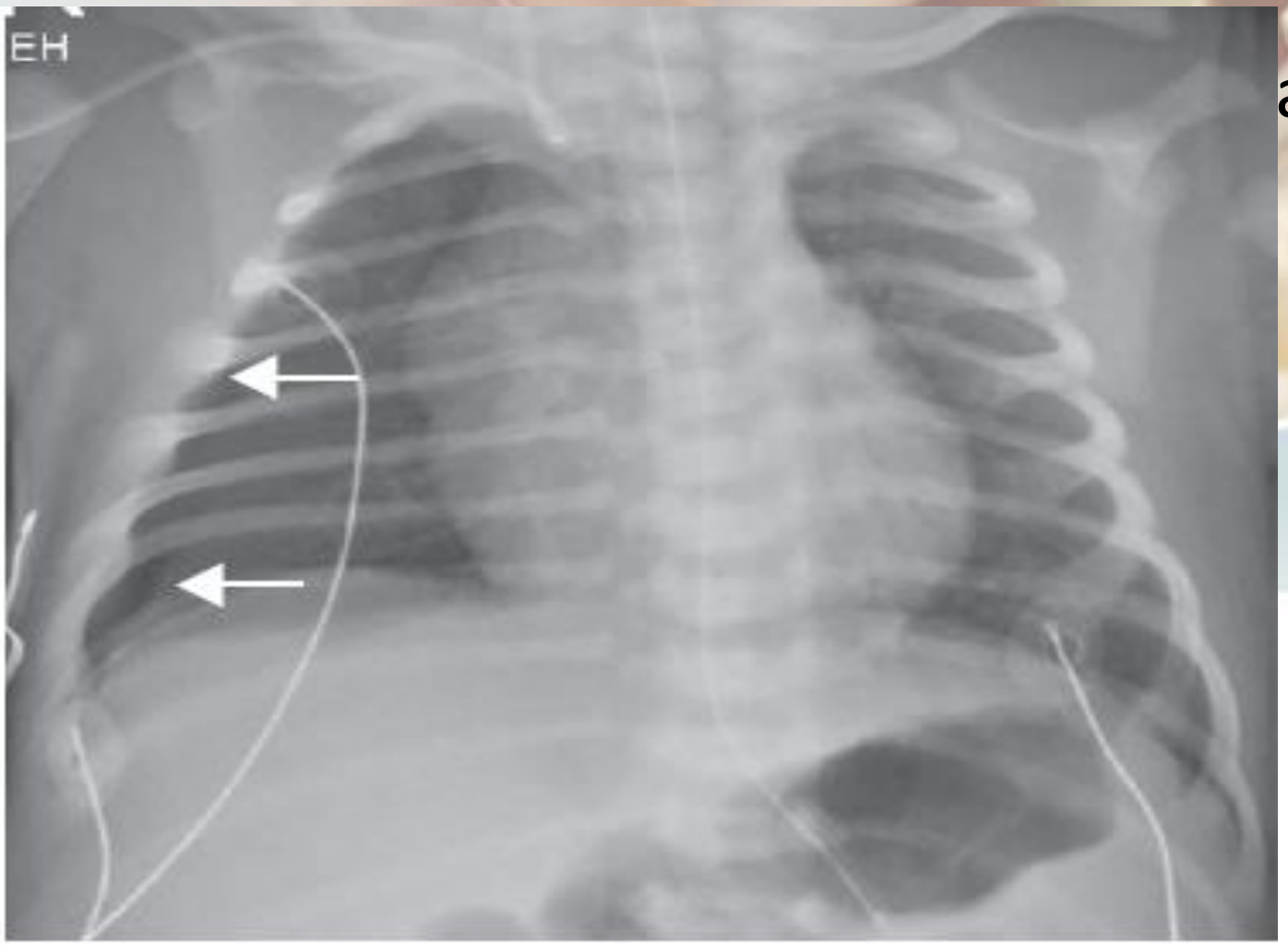
Risk for pneumothorax is highest in the first 24 to 48 h post- birth.
A ‘spontaneous pneumothorax’

can occur in spontaneously breathing infants not receiving respiratory support, usually around the time of initial lung inflation. Pneumothoraces usually present with an acute increase in respiratory distress,

Oxygen requirements, and CO2 retention.

A tension pneumothorax may present with sudden onset of
to the heart is impaired. cardiovascular collapse as venous return

Chest radiograph



ax:

- Hemithorax volume is similar in the affected (right) and unaffected (left) side.
- The affected side (right) is more lucent, and a black rim of air, which contains no lung markings,

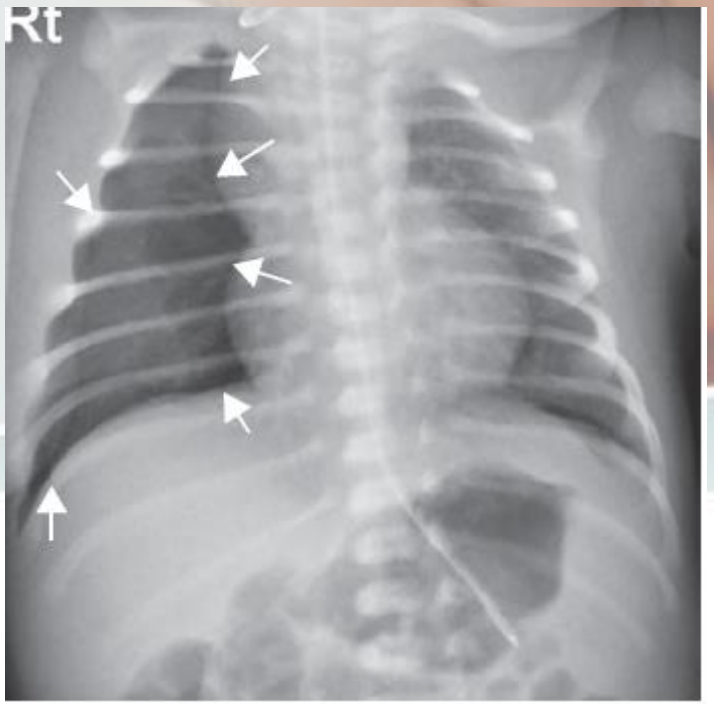
(arrows) may be noted between the chest wall and the lung tissue.

- The cardiac silhouette is not shifted toward the unaffected side.

A small pneumothorax with minimal respiratory distress and no cardiovascular deterioration can be

observed until it resolves spontaneously.

Moderate pneumothorax:



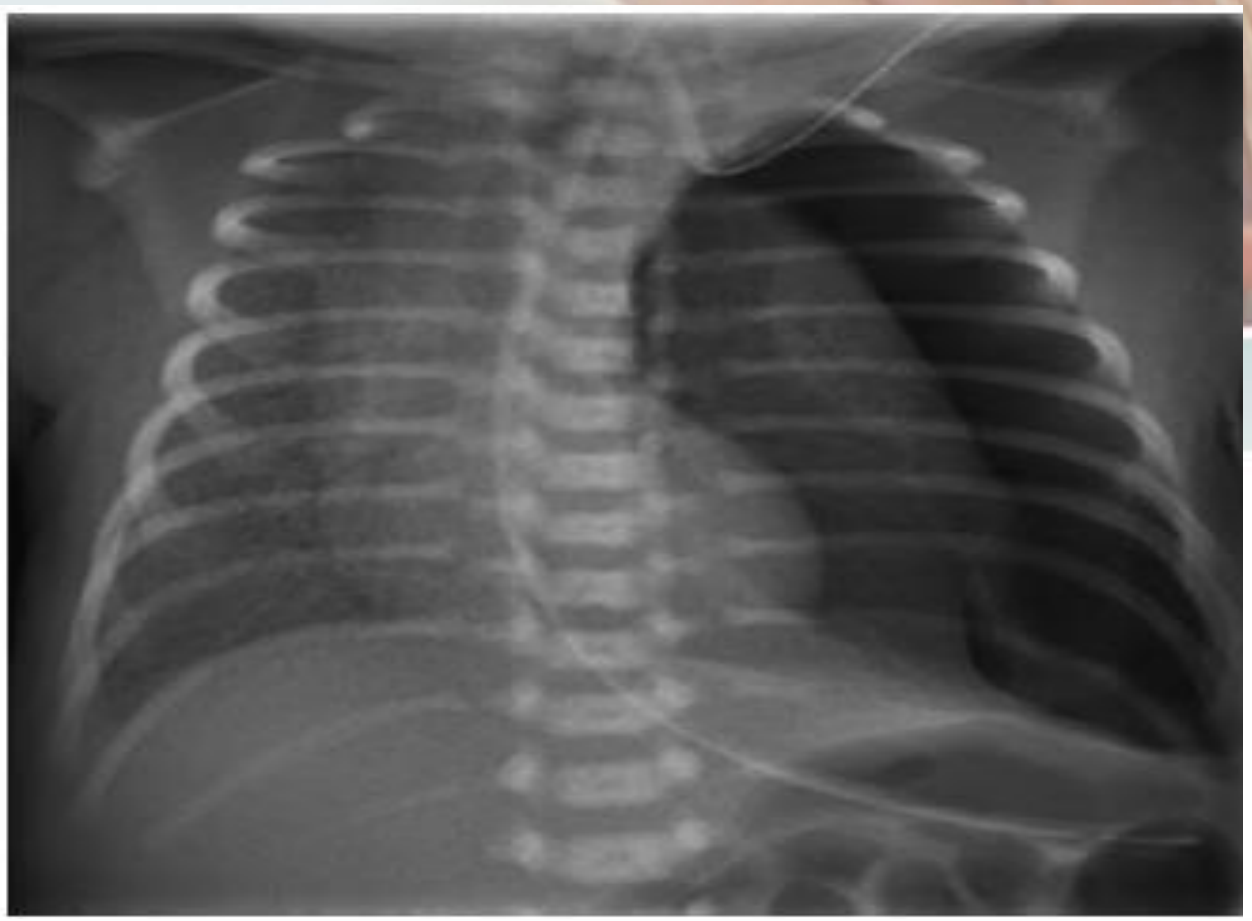
• Hemithorax volume is slightly larger in the affected (right) side than in the unaffected (left) side.

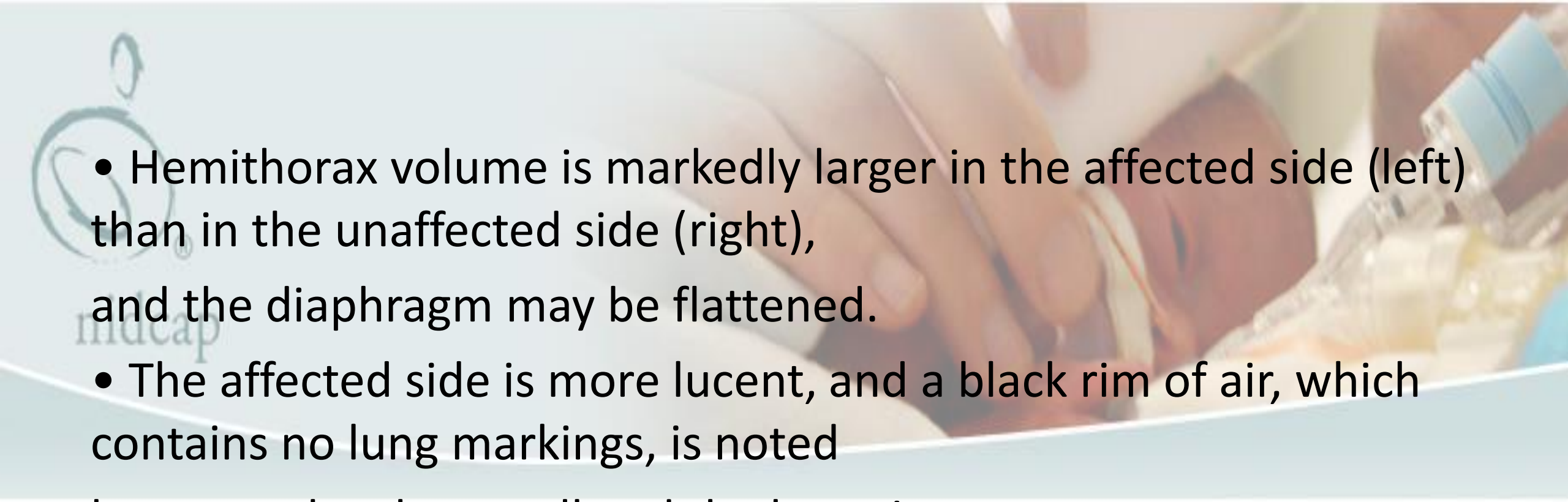
• The affected side (right) is more lucent, and a black rim of air (arrows) is noted between the chest

wall, diaphragm, mediastinum, and the lung tissue.

• The cardiac silhouette is slightly shifted toward the unaffected side.

Large (tension) pneumothorax— anteroposterior view: •





• Hemithorax volume is markedly larger in the affected side (left) than in the unaffected side (right),

and the diaphragm may be flattened.

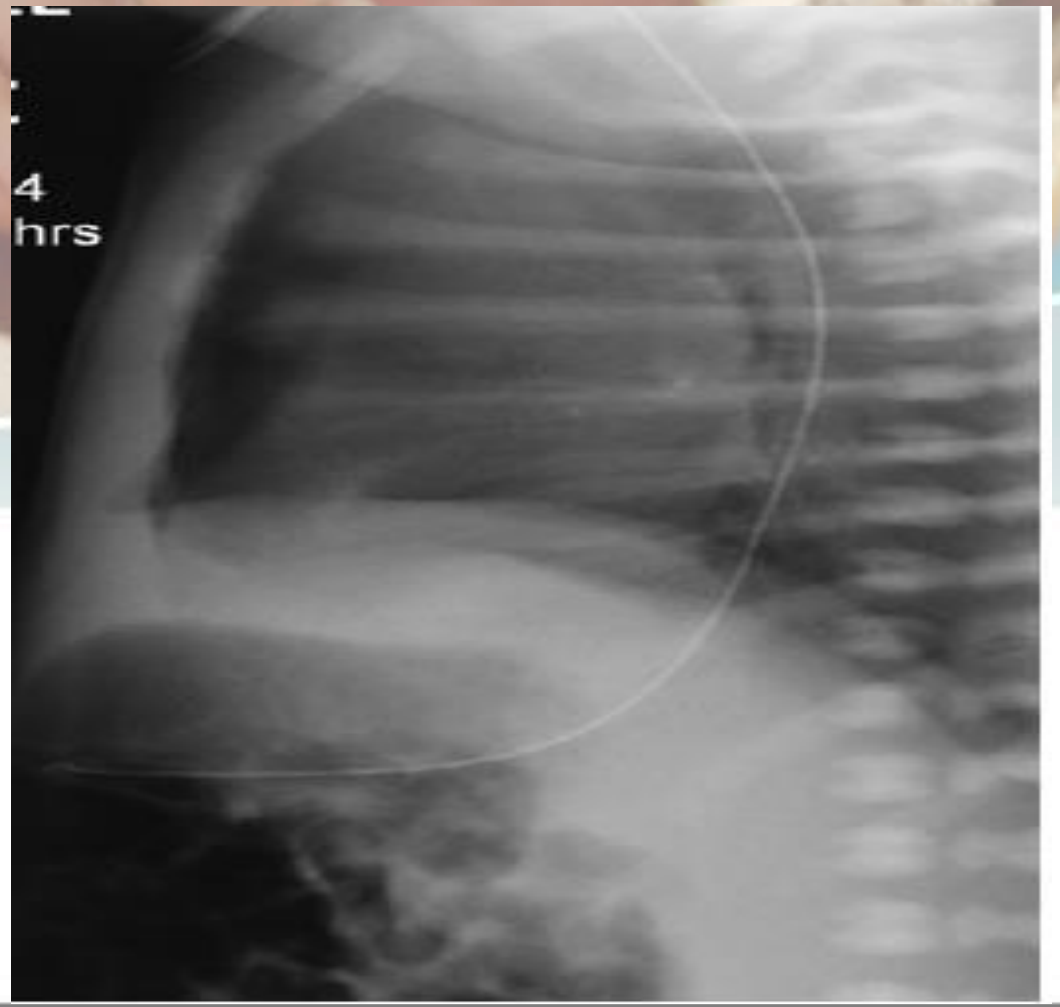
• The affected side is more lucent, and a black rim of air, which contains no lung markings, is noted

between the chest wall and the lung tissue.

• The cardiac silhouette is markedly shifted toward the unaffected side.

Large (tension) pneumothorax— lateral view:

- The pneumothorax is represented by a large anterior lucency and lucency at the level of the diaphragm.
- The diaphragm may be flattened.



Pneumomediastinum and left- sided pneumothorax:

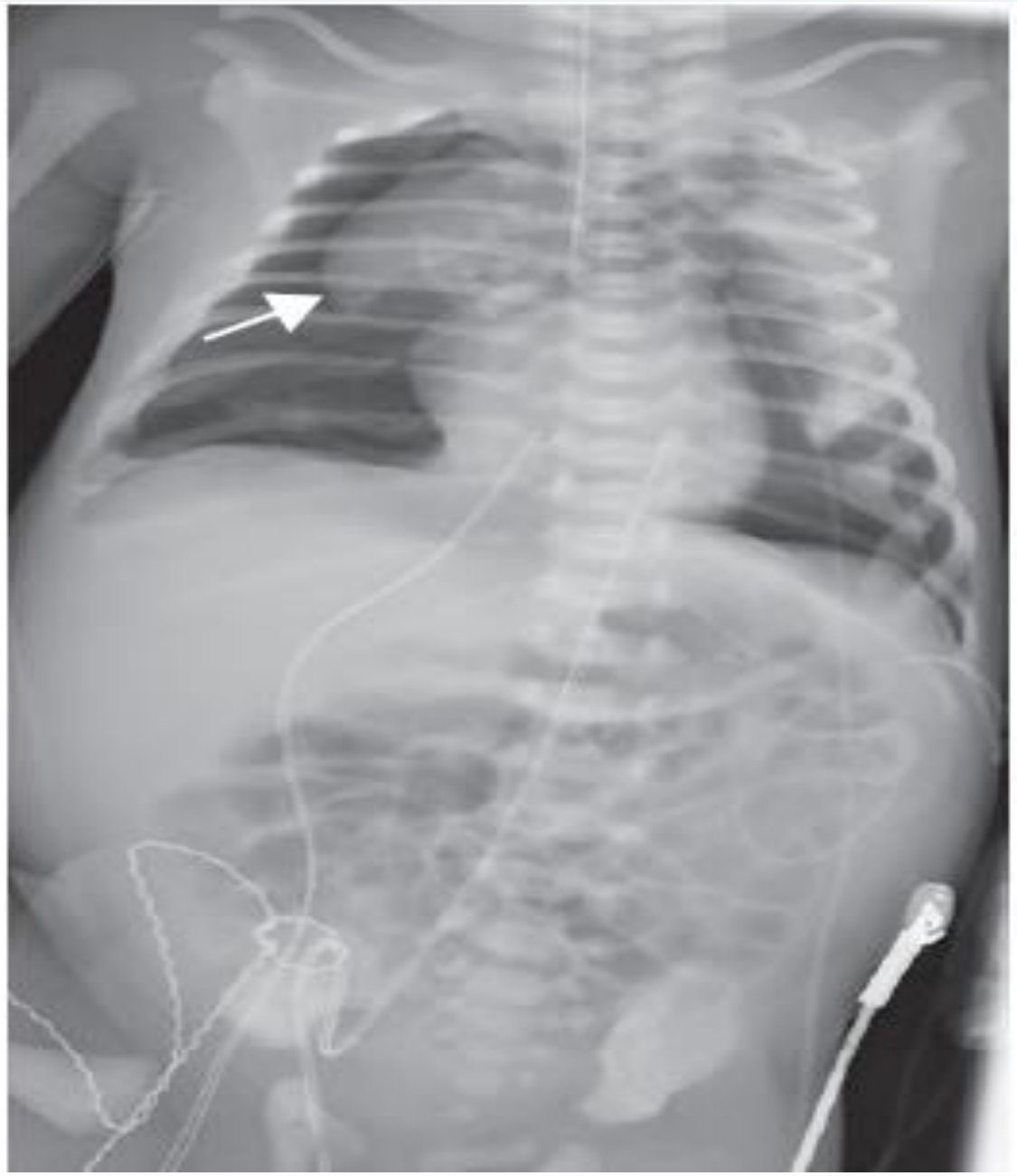
Signs of pneumomediastinum:

- The arrow shows a thymus outlined by air and clearly lifted off the cardiac surface, radiologically referred to as the ‘sail’ or ‘butterfly’ sign.
- The heart borders are sharp, the diaphragms are flattened and sharply outlined, indicating air tracking inferiorly down the mediastinum and out along the right and left sides of the diaphragm.

Signs of pneumothorax:

- The entire right hemithorax is hyperlucent, as is the lower edge of the left hemithorax.
- Most of the air is anterior to the lung (the lung ‘rim’ can only be seen easily inferiorly on the right).
- If a pneumopericardium were present, it would present as a halo seen around the heart.

nidcap



Chest transillumination:

Comparative transillumination of the chest may be useful when an infant is deteriorating rapidly and a

chest radiograph cannot be obtained quickly. Transillumination is late preterm infants. less sensitive when used in term and

Transillumination must be performed in a darkened environment, using a fibreoptic device

(transilluminator) capable of delivering high illumination.

- The transilluminator must be in direct contact with the infant's chest.
- A unilateral pneumothorax is suspected when the halo around larger on one side of the chest the point of contact is significantly compared with the other.

Point- of- care lung ultrasound:

Ultrasound assessment for pneumothorax is emerging as a new application of point- of- care ultrasound

(POCUS) in the neonatal period. POCUS should be performed by trained individuals. A pneumothorax on ultrasound is characterized by a series of patterns including prominent A-lines, absence of B- lines, absent lung sliding, and possibly a lung point sign.

Management of a symptomatic pneumothorax:

A symptomatic pneumothorax, especially one under tension, needs to be drained urgently and takes precedence over returning to the Problem List to address the next ACoRN Sequence.

There are two ways to drain a pneumothorax: needle aspiration or chest tube insertion.

Needle aspiration: .1

This procedure should only be undertaken as an emergency in an infant with significant compromise and a positive transillumination or chest radiograph. In severely symptomatic infants, it may be necessary to proceed with needle aspiration before a confirmatory chest radiograph but after ensuring the ETT has not been displaced or obstructed. The needle or vascular catheter can be held in place for ongoing removal of air or be connected to an underwater seal. Needle aspiration is usually an interim measure, pending placement of a chest tube.

Chest tube insertion:

Inserting a chest tube provides continuous drainage of a pneumothorax, allowing re- expansion of the ipsilateral collapsed lung and release of pressure on the heart and other mediastinal structures.

A chest tube should never be left open to the environment because this would allow air to be drawn back into the pleural space with spontaneous, negative- pressure breathing. Before unclamping

the chest tube, it should be connected to a drainage system with an underwater seal or a flutter (Heimlich) valve to prevent air re- entry.

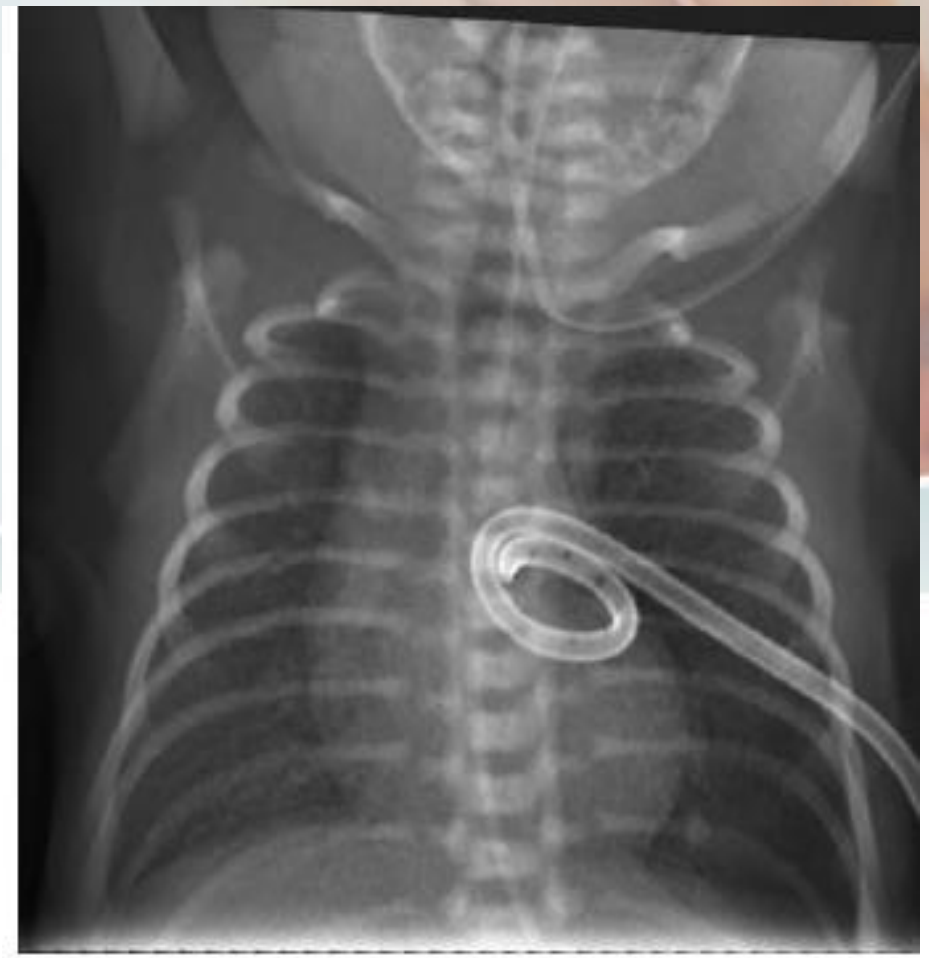
Initial indications that a chest tube is within the pleural space are:

- Palpation of the tube between the ribs rather than up the chest wall after the tube insertion
- Bubbling in the underwater seal at the time of unclamping
- Condensation and serous drainage in the tube
- Fluid meniscus moving along the tube
- Signs of improvement in the infant's oxygenation and perfusion

Drainage of frank blood is unusual and may indicate that a blood vessel has been ruptured.

Definitive confirmation that the chest tube is in the pleural space and the pneumothorax has been drained is obtained by chest .radiograph

Tension pneumothorax, post- drain insertion:



Respiratory distress syndrome

RDS is a condition caused by lack of pulmonary surfactant, a soapy substance that is normally present in mature lungs. Surfactant reduces surface tension within the alveoli, preventing their collapse and

allowing them to inflate more easily. Without surfactant, widespread atelectasis (alveolar collapse) results in decreased lung volume and increased work of breathing. The lung surface area for gas exchange is

reduced, causing hypoxemia and hypercarbia

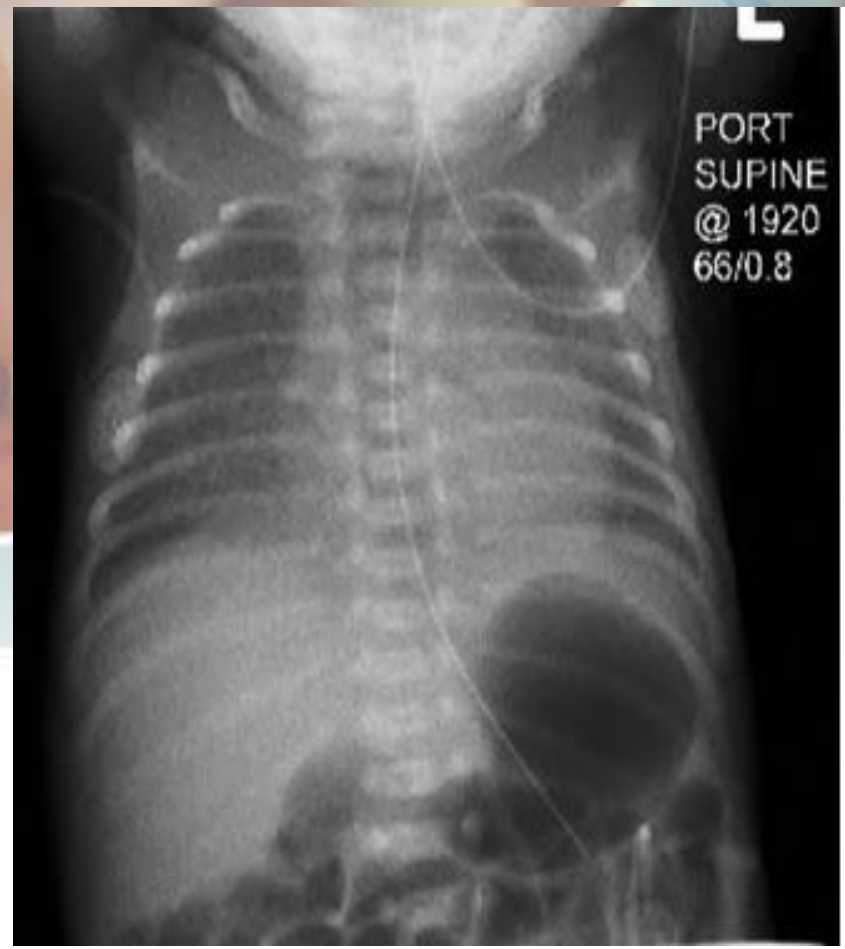
RDS is primarily a disease of preterm infants and its incidence increases with decreasing GA. Risk for RDS also increases in late preterm infants born to mothers with poorly controlled diabetes.

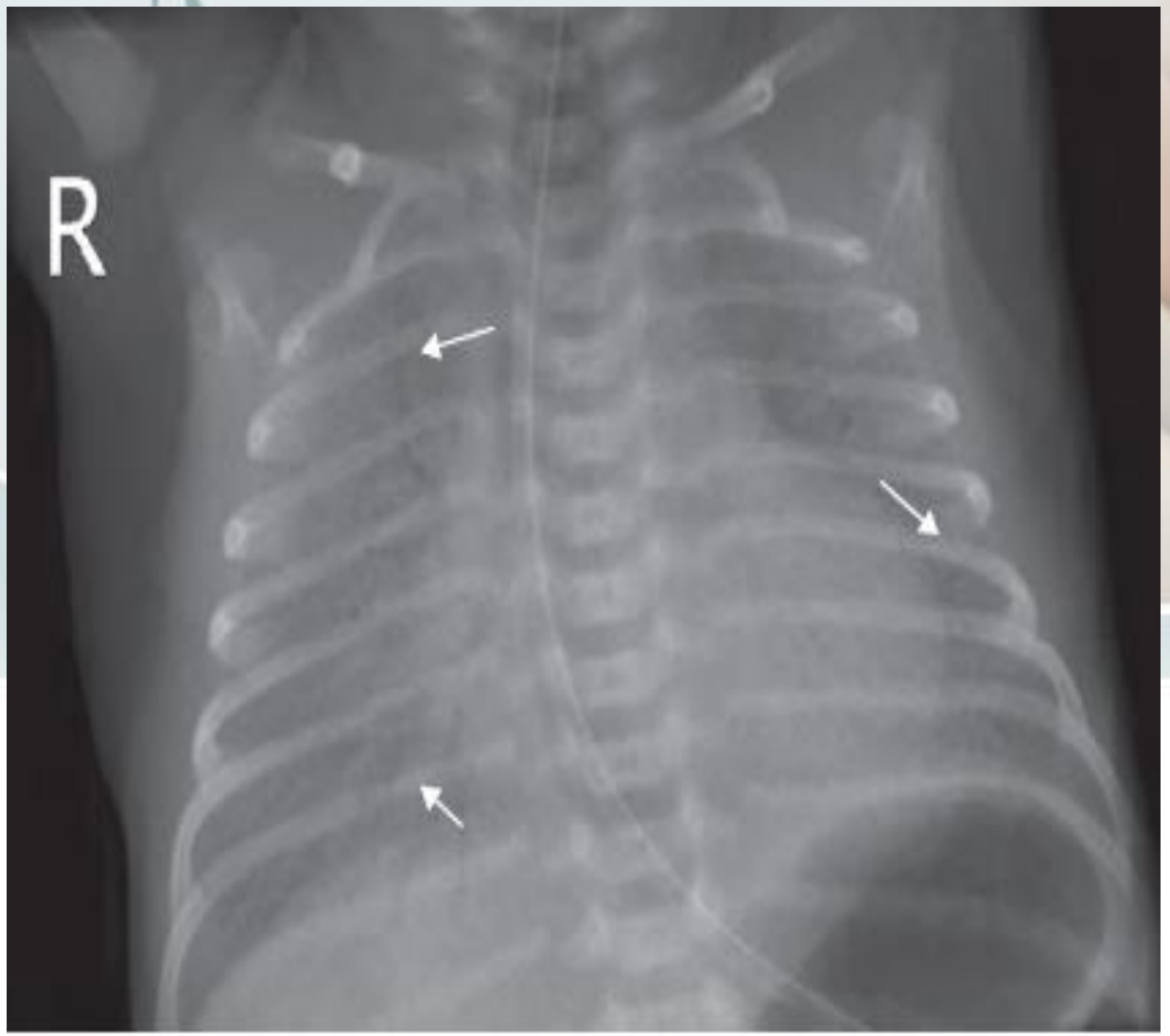
Infants with RDS can present with any degree of respiratory distress and oxygen requirement.

Symptoms usually appear shortly after birth and become progressively more severe in the first 72 h if the infant's own respiratory efforts, or the respiratory supports provided, are unable to prevent progressive

lung collapse. The natural course of RDS, if not treated with exogenous surfactant, is to slowly improve after 72 h to 5 days. During this period, endogenous surfactant production and release established. become

- Normal or slightly decreased lung inflation
- Fairly clear lung fields with slight diffuse haziness
- Diaphragm and heart borders mildly obscured
- Few air bronchograms (airways containing air become visible against collapsed lung) appear longitudinally

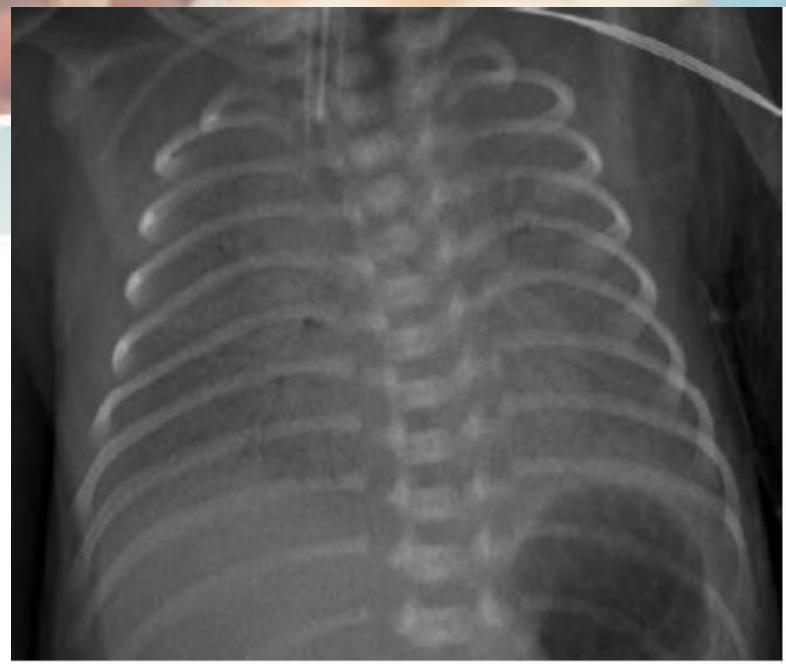




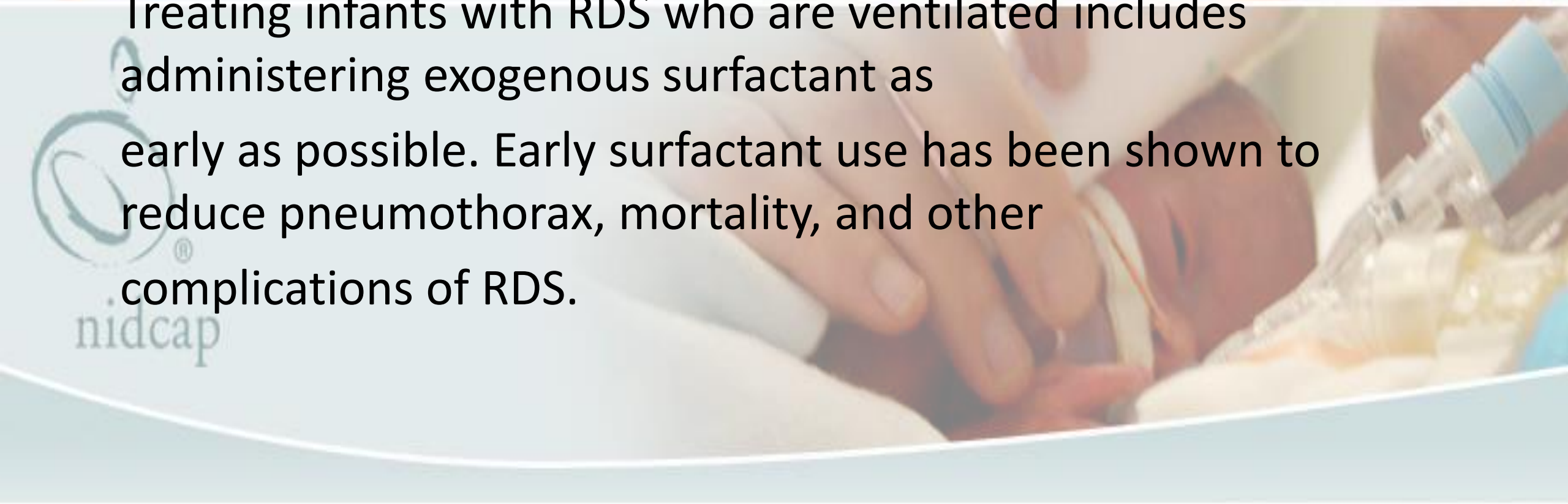
- Moderately decreased lung inflation
- Lung fields diffusely hazy, with a ‘ground glass’ appearance
- Heart borders and diaphragm greater than 50% obscured
- Air bronchograms more widely seen in upper and).lower lobes (arrows

Severe RDS:

- Decreased lung inflation
- Lung fields diffusely hazy, with a total or near- total ‘whiteout’ appearance
- Heart borders and diaphragm greater than 50% obscured
- Air bronchograms in upper and lower lobes



Treating infants with RDS who are ventilated includes administering exogenous surfactant as early as possible. Early surfactant use has been shown to reduce pneumothorax, mortality, and other complications of RDS.



Surfactant administration:

Exogenous surfactant is administered directly into the infant's trachea and bronchial tree via the

endotracheal tube (ETT) or a thin catheter, as shown in Figure 3.8. The usual dose (depending on the brand) is 2.5 mL/kg to 5 mL/ kg.

During surfactant administration, infants may develop transient desaturation, bradycardia (or both),

due to airway obstruction or vagal stimulation. These effects usually resolve with slowing or halting administration until the infant improves. Sometimes the ventilator settings and the percent of inspired

oxygen need to be increased temporarily.

As lung compliance improves following surfactant administration, the pressure required to inflate

the lungs (and produce an easy rise of the chest), along with the oxygen required to maintain target

saturations, often decrease dramatically. It is important to:

- Closely monitor changes in oximetry readings and serial blood gases,
- Reduce the peak ventilator pressures, as needed, to avoid complications of over- inflation, including

pneumothorax, and

- Reduce the oxygen to maintain SpO2 within the desired range.

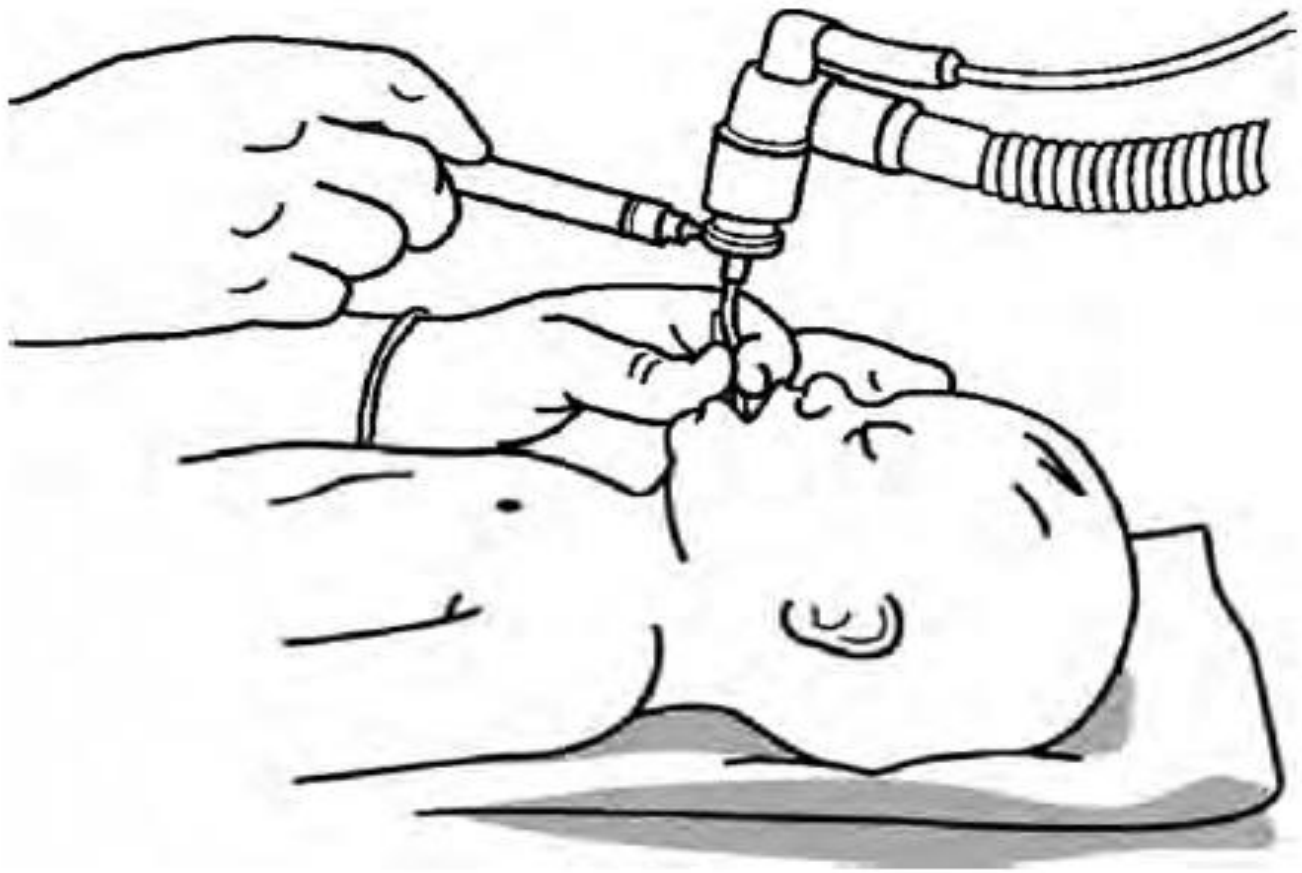


Figure 3.8. Surfactant delivery through endotracheal tube.

An improvement in respiratory status may be temporary and does not eliminate the need for transport to a centre capable of providing higher level neonatal care.

Health care providers administering surfactant must be skilled in neonatal intubation and prepared to deal with the rapid changes in lung compliance and oxygenation during and after surfactant is

given. They must also be aware of the potential complications of this treatment.

Transient tachypnea of the newborn

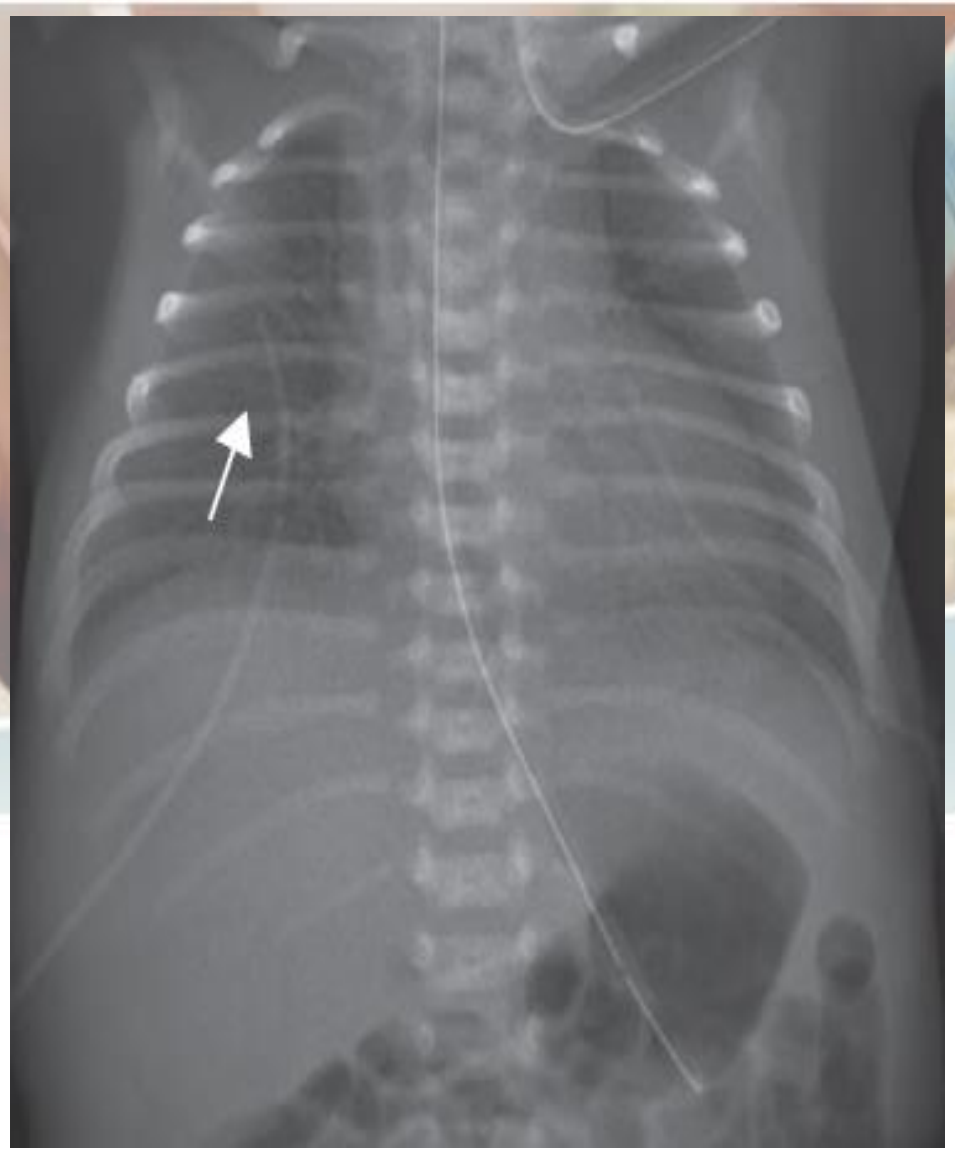
TTN occurs when lung fluid production does not cease before birth, and there is a delay in clearance of residual lung fluid after birth. It is a primary cause of respiratory distress in term or late preterm infants.

TTN is more common in newborns born by Caesarean section (especially when there has been no labour) or after a precipitous vaginal delivery.

Infants with TTN present with mild to moderate respiratory distress and oxygen requirements typically less than 40%. Respiratory distress due to TTN will often improve or resolve over the first few hours post- birth, as residual lung fluid is reabsorbed. TTN has been reported to last as long as 48 to 72 h,but usually shows improvement well before that. When TTN persists beyond 6 h post- birth or worsens within that time frame, other diagnoses need to be considered, including infection and surfactant deficiency or inactivation.

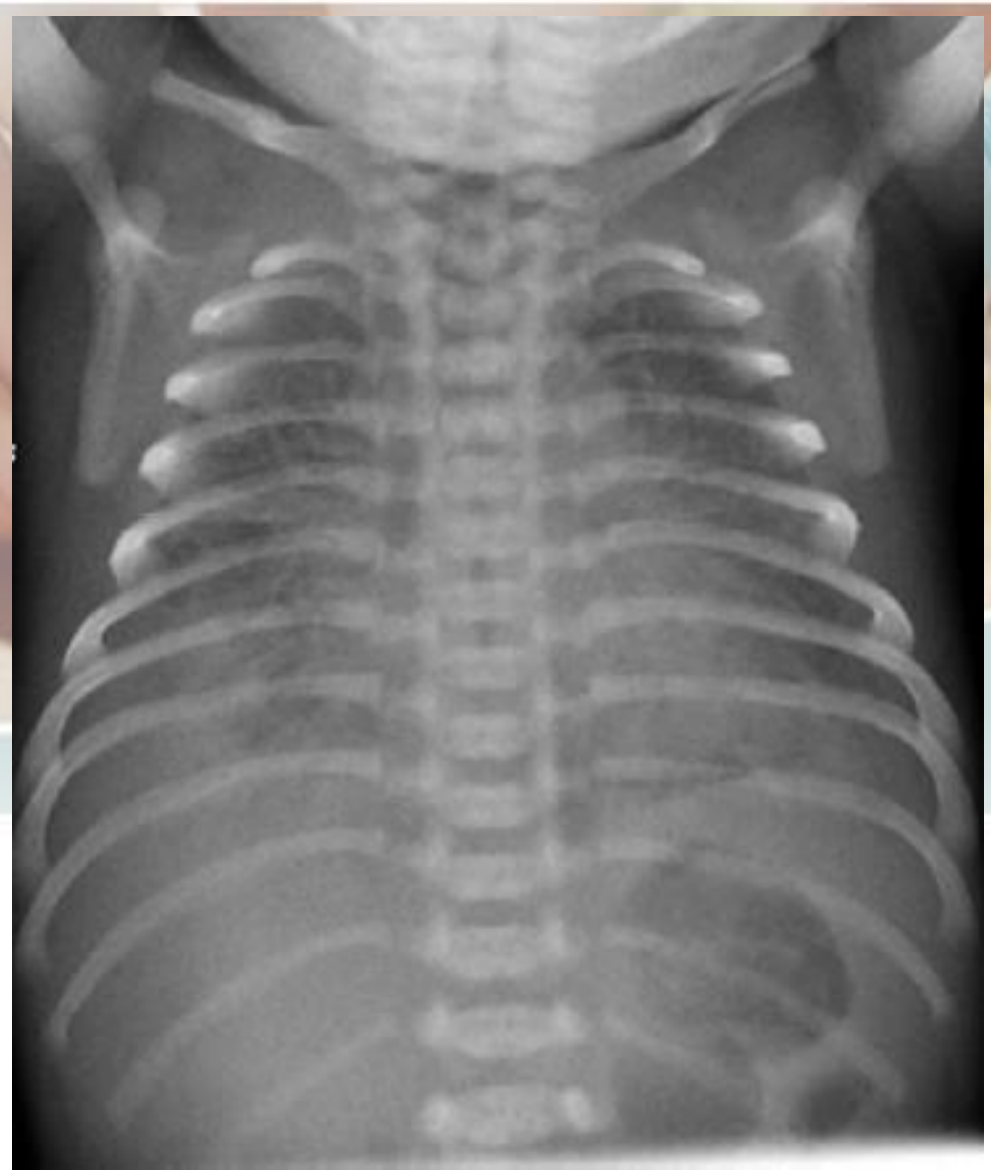
Mild TTN:

- Normal or increased lung inflation (normal in this example)
- Fairly clear lung fields
- Diaphragm and heart borders are seen throughout
- Increased vascular markings near the heart shadow give lungs a streaky appearance
- Fluid in the major fissure (arrow) may include a small amount of pleural fluid



Severe TTN:

- Same appearance as for mild TTN but with increased haziness of lung fields
- Diaphragm and heart borders are not well seen



Management of TTN is primarily supportive. The goals are to maintain adequate oxygenation and ventilation while lung fluid is reabsorbed. Natural resolution occurs with diuresis. Fluid overload should be avoided with judicious use of IV fluids, if parenteral nutrition and hydration are necessary. Diuretics do not play a role in typical TTN resolution.

Meconium aspiration syndrome

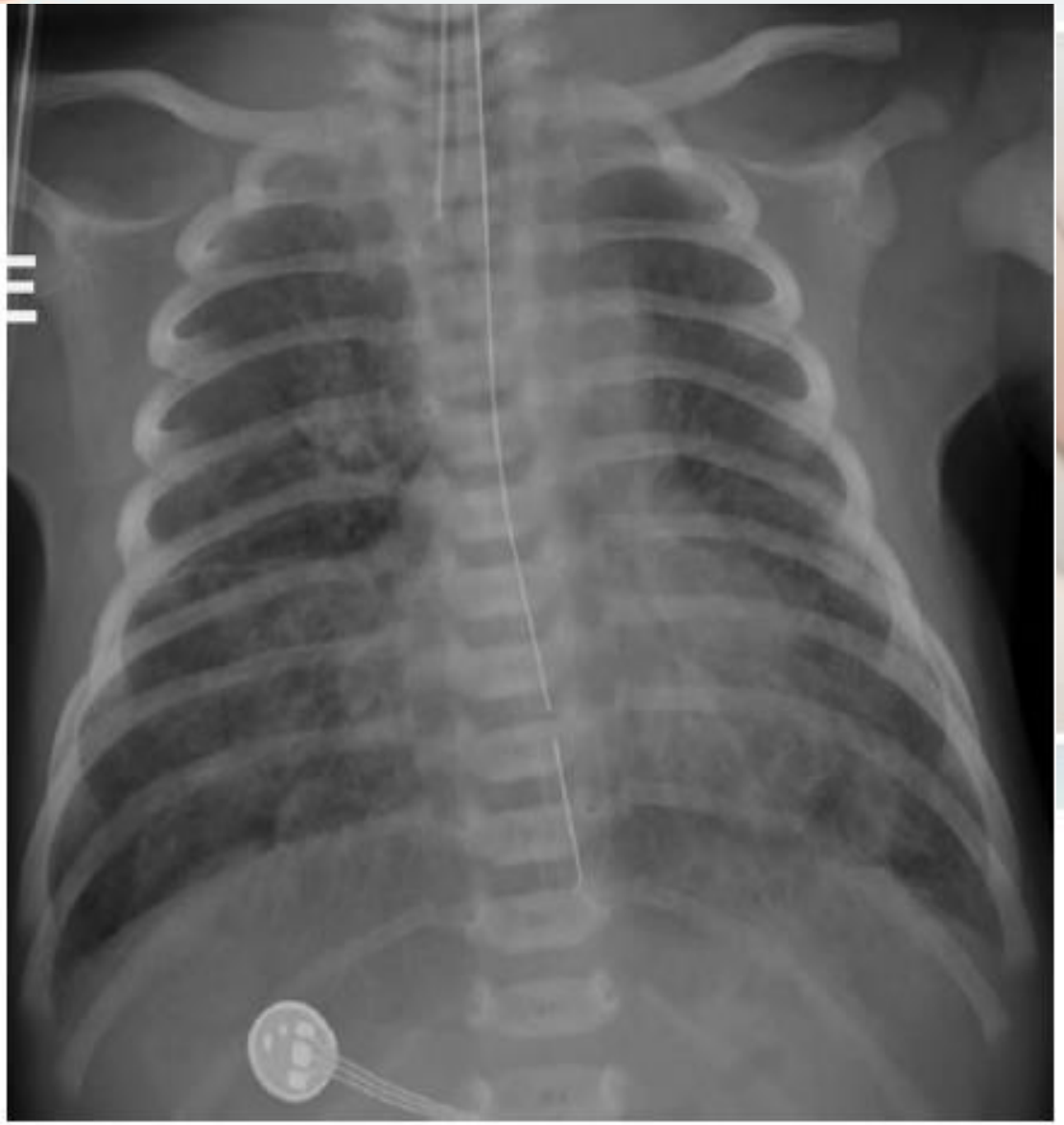
MAS is a spectrum of illness that occurs in post- term, term and, a functionally mature sometimes, late preterm infants (with gastrointestinal tract), who aspirate meconium before or at the time of birth.

Respiratory distress in MAS can be mild and transient or severe, leading to respiratory failure with hypoxemia, acidosis, and PPHN. MAS always warrants close observation because even infants who are stable initially can deteriorate and develop refractory PPHN.

Aspirating meconium has three main effects on pulmonary mismatch: function, resulting in ventilation: perfusion

- Large and small airway obstruction
- Surfactant inactivation
- Chemical pneumonitis

Because MAS is a disease that begins with fetal compromise in utero, most cases are not preventable with interventions at birth, such as endotracheal suctioning. Severe MAS is life-threatening and requires prompt, specialized care. The main complications of MAS are respiratory failure, PPHN, and pulmonary air leaks, especially pneumothorax



Moderate to severe MAS:

- Normal or increased lung inflation (increased in this example)
- Lung fields show fluffy, patchy, and coarse infiltrates, and asymmetric areas of hyperinflation interposed with areas of atelectasis
- Heart borders and diaphragm are usually significantly obscured by areas of atelectatic lung

Avoiding respiratory failure:

The best strategy to avoid respiratory failure is to maintain optimal lung inflation and prevent atelectasis. In infants with moderate respiratory distress associated with MAS, this is best accomplished using CPAP.

Mechanical ventilation for MAS is usually reserved for infants with:

- Escalating oxygen requirements greater than 40% to 50% to maintain SpO2 greater than 90%
- PCO2 greater than 55 or pH less than 7.25.

The acts of intubation and initiation of mechanical ventilation can cause an infant with MAS to react adversely and PPHN to worsen. Premedication for intubation is recommended and sedation during mechanical ventilation may be needed. Infants with MAS often have low lung compliance and may require high ventilation pressure, increasing the risk for air leaks. Muscle

paralysis may be necessary to achieve control of ventilation and oxygenation.

Avoiding PPHN:

Infants with MAS are predisposed to PPHN and may have labile pulmonary circulation, dropping their

oxygenation in response to stimuli such as excessive handling, painful procedures, bright lights, or loud noises. Once established, PPHN is difficult to treat and may be life-threatening.

The following interventions can help promote stability:

- Shielding the infant's eyes from light
- Speaking in a low voice, and away from the infant's bed
- Minimizing handling
- Nesting
- Providing adequate pain relief
- Considering the need for sedation
- Maintaining pre- ductal SpO2 in the 90% to 95% range
- Documenting and reporting unstable SpO2 and a pre- ductal– post- ductal SpO2 difference

Avoiding pulmonary air leaks:

The best strategy to avoid pulmonary air leaks is to prevent lung overdistension. For infants receiving

mechanical ventilation, this is best accomplished by:

- Limiting initial CPAP levels to 5 to 8 cmH2O,
- Avoiding overventilation by maintaining PCO2 above 40 mmHg,
- Considering surfactant administration, and
- Avoiding suctioning of the ETT as much as possible.

Surfactant therapy:

Meconium aspiration causes secondary surfactant deficiency. Clinical trials have shown that surfactant replacement therapy can be a beneficial intervention in MAS . However, surfactant must be administered cautiously, avoiding hypoxia and excessive stimulation. The decision to administer surfactant in infants with MAS should be taken with great caution, because these infants can deteriorate due to labile pulmonary hypertension. Consultation with the referral centre is advised.

Pneumonia:

Neonatal pneumonia is an infectious infiltrate of the lungs. Pneumonia is usually interstitial and diffuse rather than lobar in appearance. It is more likely to occur in the presence of risk factors for sepsis (e.g., premature rupture of membranes, maternal colonization with GBS, or chorioamnionitis). Infants may or may not be systemically ill at onset, but the clinical course can be fulminant.

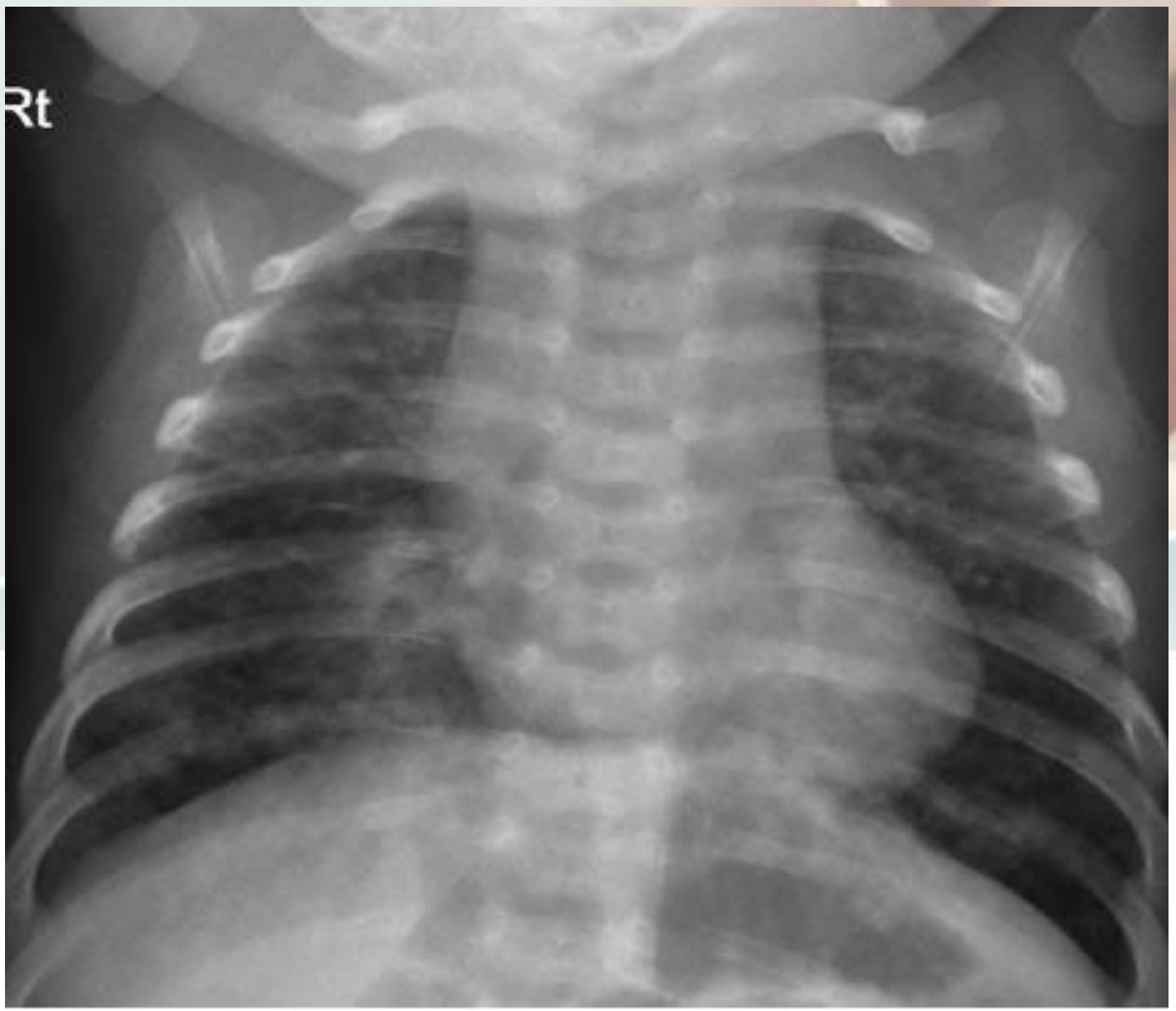
Pneumonia:

- Radiographic diagnosis is always tentative because the chest radiograph may mimic RDS, TTN, and,

occasionally, MAS.

- There may be moderately to markedly increased lung inflation.
- Lung fields may show patchy densities and various degrees of ‘whiteout’ and air bronchograms.
- Diaphragm and heart borders may be obscured.
- Discrete segmental or lobar involvement may be present but is not common.

nidcap



- The inability to rule out pneumonia by clinical or radiographic appearance gives rise to the recommendation
- to treat all respiratory disease in the newborn lasting longer than 6 h with IV antibiotics. Specific management is guided by the ACoRN Infection Sequence

Persistent pulmonary hypertension of the newborn:

PPHN is a disorder of the pulmonary vasculature characterized by failure of the normal drop in pulmonary vascular resistance after birth. PPHN leads to decreased pulmonary blood flow and persistence of the right-to-left shunts that exist in utero. When an infant's pulmonary arterial pressure exceeds systemic blood pressure, and the ductus arteriosus is open, right-to-left shunting is seen clinically as a pre- to post- ductal SpO2 difference with post- ductal hypoxemia . When right ventricular strain results in tricuspid valve incompetence and regurgitation of blood into the right atrium, this leads to right-to-left shunting through the foramen ovale. This is seen clinically as pre- and post- ductal hypoxemia.

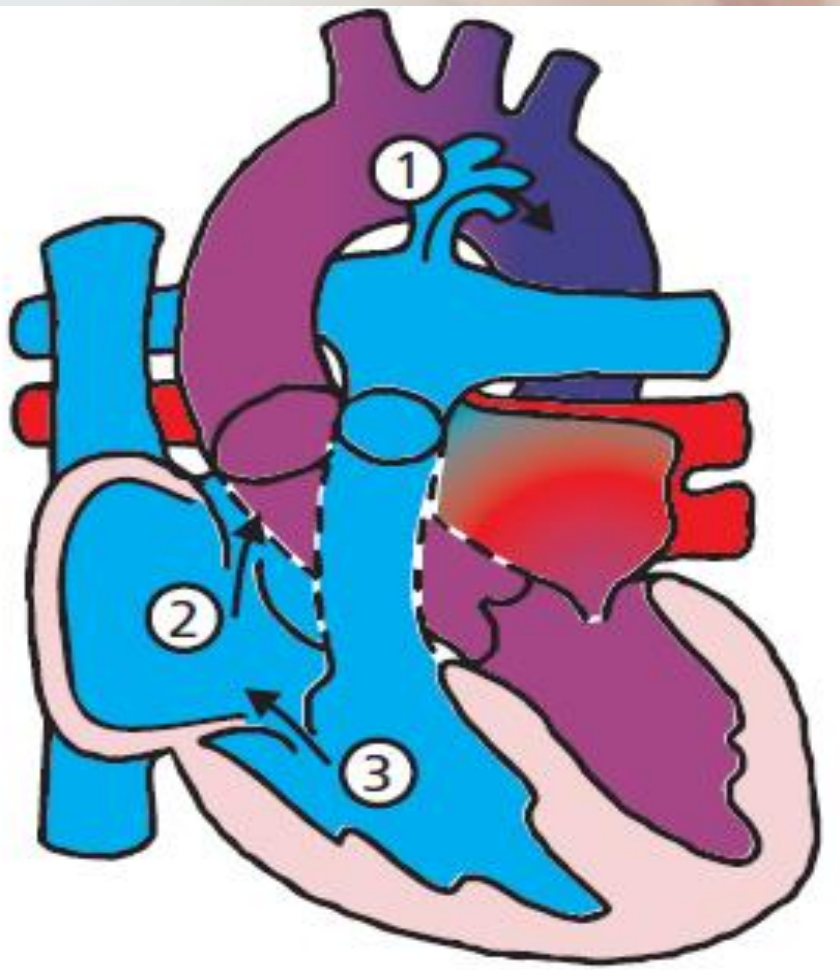


Figure 3.9. Blood flow in persistent pulmonary hypertension of the newborn. 1. Right-to-left shunt via the PDA. 2. Right-to-left shunt via the patent foramen ovale. 3. Functional tricuspid insufficiency and regurgitation due to right ventricular dysfunction

PPHN may have an underlying vascular component of prenatal origin, which includes increased

vascular muscularization and reactivity, decreased pulmonary vascularization, or abnormal vascular distribution.

PPHN may also be triggered by intra- and post- partum events that cause fetal hypoxia and acidosis. While PPHN is more commonly associated with respiratory conditions such as RDS, MAS, pneumonia, or CDH, it may also occur as a primary disturbance of transition in the absence of parenchymal lung disease. The diagnosis of PPHN should always be confirmed by an echocardiography to rule out abnormal

cardiovascular anatomy.

PPHN is life- threatening and requires prompt, specialized care. Interventions that help decrease the pulmonary arterial pressures during or following transition in infants prone to PPHN include:

- Minimizing handling and disturbance
- Use of sedation when necessary
- Use of paralysis, in consultation, when necessary to meet ventilation needs
- Maintaining oxygenation in the high end of the normal range:
  - PO2 70 mmHg to 90 mmHg, and pre- ductal SpO2 approximately 90% to 95%
- Minimizing atelectasis while avoiding overdistension in infants with moderate to severe respiratory distress, by using CPAP, mechanical ventilation, and/ or surfactant therapy in consultation with your referral centre, and
- Avoiding and correcting respiratory and metabolic acidosis to maintain:
  - PH 7.30 to 7.40, and
  - PCO2 40 mmHg to 50 mmHg

Other conditions

Pulmonary hypoplasia

Pulmonary hypoplasia refers to conditions where an infant's lungs are incompletely developed such that the number of patent airways and gas- exchange spaces (alveoli), and corresponding vascular supply, are

greatly reduced. Pulmonary hypoplasia may be seen in infants who experienced insufficient lung inflation in utero during early gestation, such as with:

- Severe oligohydramnios, caused by
  - Rupture of the membranes in the second trimester,
  - Renal agenesis, or
  - Urinary outflow obstruction;
- CDH; or
- Neuromuscular disease, with decreased fetal respiration.

Infants with pulmonary hypoplasia present at birth with profound respiratory failure. They have

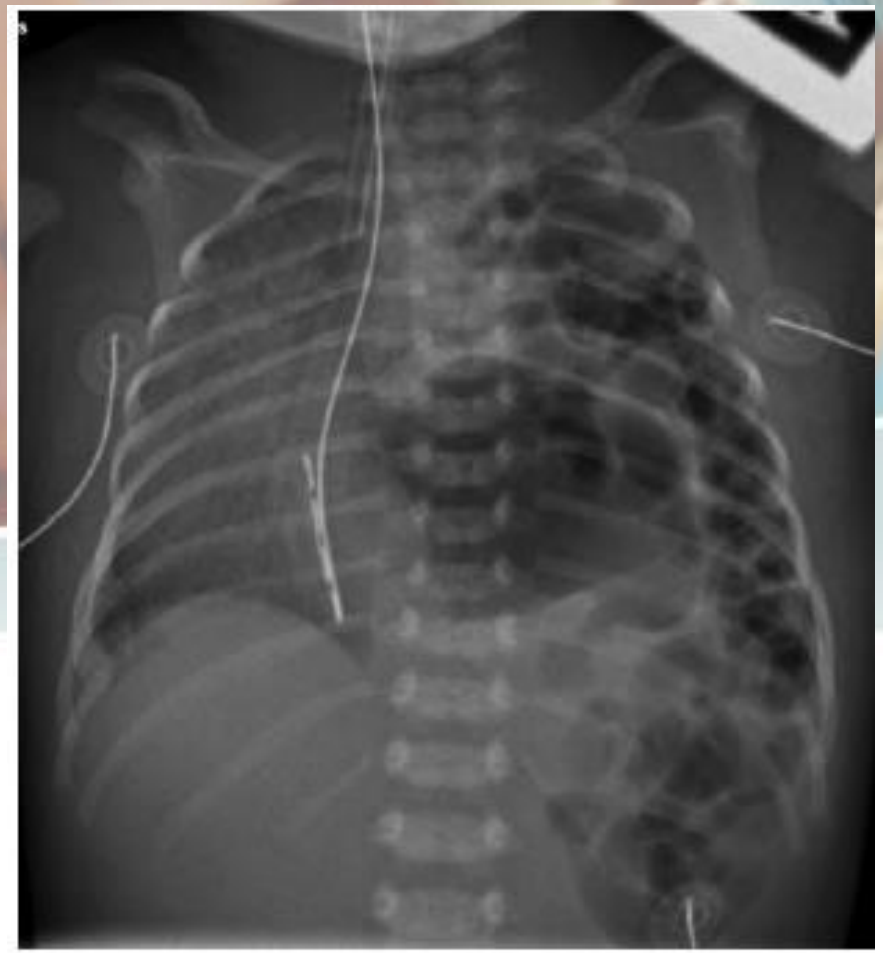
increased risk for PPHN and pneumothorax. This condition is life- threatening and requires prompt, specialized care as advanced forms of ventilation and intensive care result in improved outcomes.

Radiographic findings:

- Small but sometimes clear lung fields
- Bell- shaped chest
- Evidence of a space- occupying lesion (i.e., CDH)

Left- sided CDH:

- Stomach bubble is in the abdomen (gastric tube coiled in the chest).
- There is mediastinal shift to the right.
- Air is present in the bowel located within the left chest (diaphragm defect is more common on the left)



Level of Risk: Respiratory

In the ACoRN Respiratory Sequence, level of risk is based on the severity of respiratory distress, oxygen requirements, and the level of respiratory support being administered.

Green:

- Infant has mild respiratory distress lasting less than 6 hours post- birth, oxygen requirement less than 30% to maintain SpO2 of 90% to 95%, and ongoing monitoring requirements do not exceed site capability

Yellow:

- Infant is clinically stable but has mild respiratory distress lasting longer than 6 hours post-birth, and oxygen requirements less than 30%, **OR**
- Infant is stable, on CPAP of 8 cmH2O or less, and oxygen requirements less than 30%, **OR**
- Infant is clinically stable with new-onset respiratory distress and oxygen requirements less than 30%, **AND**
- Site can provide appropriate management, investigations, and monitoring for the infant's condition

Infants at a Yellow Level of Risk require increased levels of attention and consultation.

Red:

- Infant consistently requires more than 30% oxygen to meet target saturations, **OR**
- Infant has sustained increase in oxygen requirement greater than 10% above baseline, which does not improve with stabilization, **OR**
- Infant requires or is receiving ventilation beyond site capacity to manage or monitor safely

Infants at a Red Level of Risk require level 3 care. Transfer is required if needs exceed site capabilities.