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NEONATAL RESUSCITATION

High-risk delivery — Infants who are more likely to require resuscitation can be identified by the presence of one or more of the following risk factors:

1). Maternal conditions – Advanced or very young maternal age, maternal diabetes mellitus or hypertension, maternal substance use disorder, or previous history of stillbirth, fetal loss, or early neonatal death.

2). Fetal conditions – Prematurity, postmaturity, congenital anomalies, intrauterine growth restriction, or multiple gestations.

3). Antepartum complications – Placental anomalies (eg, placenta previa or placental abruption), or presence of either oligohydramnios or polyhydramnios.

4). Delivery complications – Transverse lie or breech presentation, chorioamnionitis, foul-smelling or meconium-stained amniotic fluid, antenatal asphyxia with abnormal fetal heart rate pattern, maternal administration of a narcotic within four hours of birth, deliveries that require instrumentation (eg, forceps or vacuum deliveries) or cesarean delivery for maternal or fetal compromise.

Preterm infants — Preterm infants pose a greater challenge than term infants because they are more likely to require resuscitation and develop complications from the resuscitative process, particularly extremely low birth weight (ELBW) infants (BW <1000 g). If a preterm birth can be anticipated and time permits, it is preferable to transfer the mother prior to delivery to a perinatal center that has fully trained staff with expertise and experience in the care of preterm infants.

Begin with a rapid assessment of the neonate's clinical status based on the following questions:

- Is the infant full-term?
- Does the infant have good muscle tone?
- Is the infant breathing or crying?

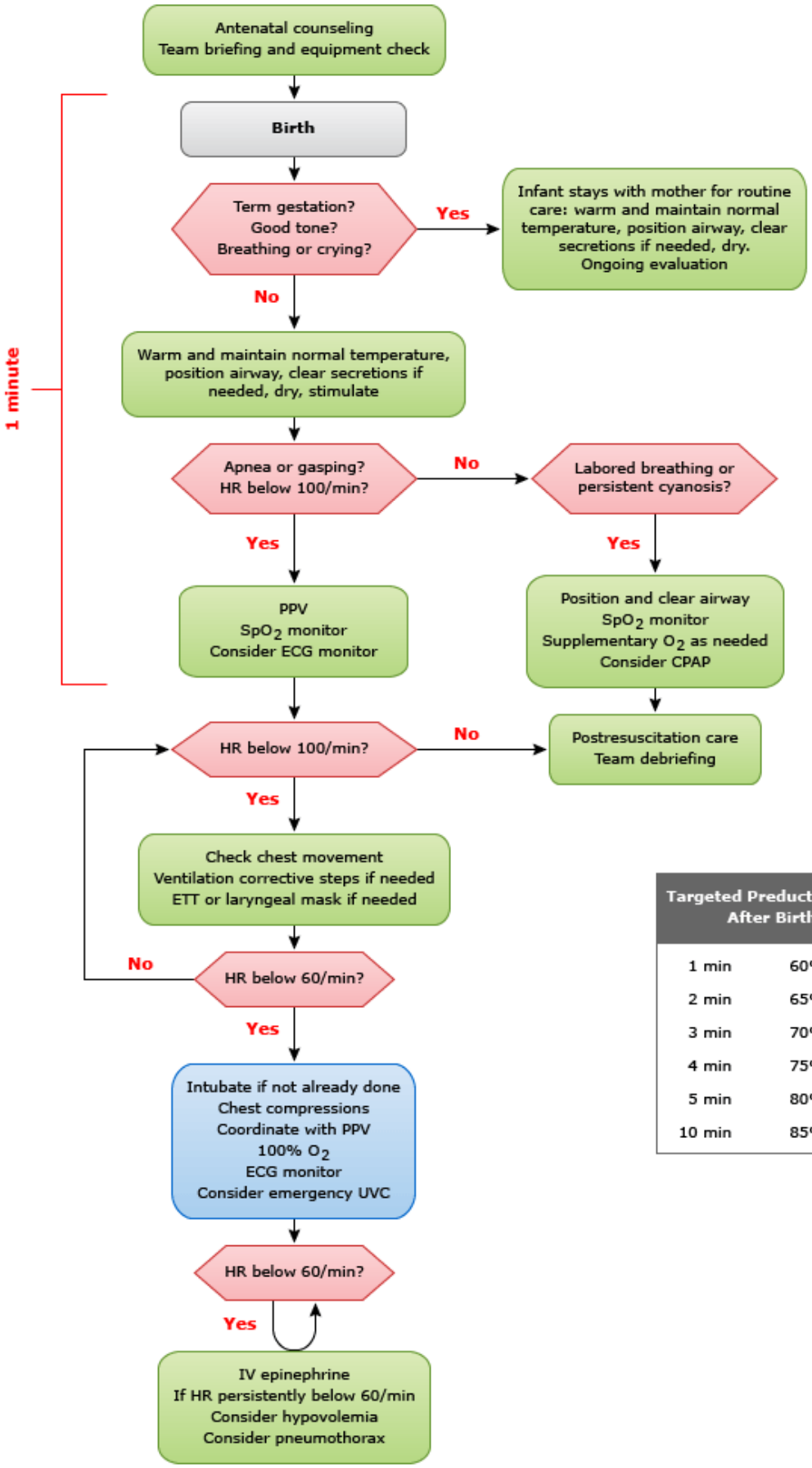
If the answer to all three questions is yes, the newborn does not need resuscitation, should not be separated from the mother, and is managed by routine neonatal care.

For infants who require further intervention, the basic steps ("ABCs") in resuscitation in any age group are still applicable. However, several aspects of neonatal resuscitation are unique and lead to differences in the initial resuscitative steps:

●Initial stabilization (provide warmth, clear **A**irway if necessary, dry, and stimulate)
●**B**reathing (ventilation and oxygenate)
●**C**hest compressions
●Administration of epinephrine and/or volume expansion

The decision to progress from one step to the next is determined by the response of the infant to the applied resuscitative intervention based upon the respiratory effort and heart rate (HR) (algorithm 1).

●No further resuscitative actions are required if the infant responds to initial intervention with adequate spontaneous respirations and a HR >100 beats per minute (bpm).



Targeted Preductal SpO_2 After Birth	
1 min	60%-65%
2 min	65%-70%
3 min	70%-75%
4 min	75%-80%
5 min	80%-85%
10 min	85%-95%

I. Initial steps — Initial steps in the delivery room are started within a few seconds of birth and should be applied throughout resuscitation

●**Dry the infant**, keep warm and maintain body temperature, preferably with skin-to-skin contact with mother, if the neonate's condition permits. After delivery, temperature should be measured and recorded. Temperature should be maintained between 36.5°C and 37.5°C. Neonatal hypothermia increases oxygen consumption and metabolic demands, which can impair subsequent resuscitative efforts, especially in the asphyxiated or preterm infant. Preterm infants are particularly prone to rapid loss of body heat because of their large body surface area relative to their mass, thin skin, and decreased subcutaneous fat.

●**Position airway** and clear secretions if needed. For infants requiring further intervention, the infant is positioned to open the airway by placing the infant flat on his/her back on a radiant warmer bed with the neck in a neutral to slightly extended position; the neck should not be hyperextended or flexed.

Suctioning immediately after birth is reserved for babies with obvious obstruction due to secretions and those who require positive pressure ventilation (PPV). Once the infant has been correctly positioned, the mouth and nose should be suctioned either with a bulb syringe or mechanical suction device. The mouth is suctioned first and then the nares to decrease the risk for aspiration. In the presence of meconium-stained amniotic fluid (MSAF), routine intrapartum nasopharyngeal suctioning and/or endotracheal suctioning post-delivery are not recommended.

●**Stimulation** – Tactile stimulation of the newborn is initiated promptly after birth to facilitate respiratory effort. Efforts at stimulating the infant should not be prolonged and should be no more than 30 seconds before initiating next resuscitative steps.

II. Apnea/gasping and heart rate <100 bpm — For neonates who are apneic or gasping and/or have HR <100 beats per minute (bpm), the following interventions are performed within 30 seconds after delivery:

●**Begin positive pressure ventilation** (PPV) with bag-mask ventilation (BMV) or T-piece resuscitator at a rate of 40 to 60 breaths per minute. During neonatal resuscitation, PPV can be administered by a T-piece resuscitator, self-inflating bag, or flow-inflating bag.

The self-inflating bag has a pressure-release valve, commonly called a pop-off valve that is set by the manufacturer to release at approximately 30 to 40 cm H2O pressure. It has been assumed that the delivered oxygen concentration using a bag without a reservoir is 40 percent when using a source of 100 percent oxygen.

The following steps are required to effectively and safely provide PPV:

1. Position – The infant should be positioned with the neck in a neutral to slightly extended position to ensure an open airway.

2. Suction – The nose and mouth should be suctioned as needed to clear any mucous to prevent aspiration prior to delivery of assisted breaths.

3. Seal – An airtight seal between the rim of the mask and the face is essential to achieve the positive pressure required to inflate the lungs. An appropriately sized mask is selected and positioned to cover the chin, mouth, and nose, but not the eyes of the infant. The mask is held on the face by positioning the hand of the clinician so that the little, ring, and middle fingers are spread over the mandible in the configuration of the letter "E" and the thumb and index are placed over the mask in the shape of the letter "C". The ring and fifth fingers lift the chin forward to maintain a patent airway.

! Breath rate and inflation pressure – PPV should be given at a rate of 40 to 60 breaths per minute. Term neonates usually require an inflation pressure of approximately 30 cm H2O initially to adequately inflate the lungs. In preterm infants, an initial inflation pressure of 20 to 25 cm H2O is usually adequate.

●**Place the neonate on a monitor**, including pulse oximetry and continuous ECG to monitor, which provide continuous assessment of HR and oxygen saturation (SpO_2) during resuscitation.

Further resuscitative efforts are based upon the neonate's response after 15 to 30 seconds of BMV.

●**Optimize PPV if HR is not increasing** – If the HR is **not** increasing, evaluate for chest rise with assisted breaths. If the chest is not rising appropriately with administered breaths, optimize PPV as follows :

•Adjust the mask to improve the seal
•Reposition airway to ensure correct head position
•Suction mouth and nose
•Open the mouth and tilt the jaw forward
•Increase the pressure administered using increments of 5 to 10 cm H_2O to maximum of 40 cm H_2O

●If the above measures fail, secure the airway by performing endotracheal intubation or placing a laryngeal mask airway

Laryngeal mask airway (LMA) — In term and preterm infants with birth weight >1500 g (≥34 weeks gestation), LMAs may be considered as an alternative to endotracheal intubation if BMV is unsuccessful in providing adequate ventilation, or if endotracheal intubation is unsuccessful or not feasible. An LMA is a soft mask with an inflatable cuff attached to a silicone rubber airway. The inflated cuff covers the laryngeal opening and its rim conforms to the contours of the hypopharynx, occluding the esophagus with a low-pressure seal.

The LMA is inserted through the mouth using the index finger to guide "blind" insertion along the hard palate.

Endotracheal intubation — Endotracheal intubation allows direct access to the upper trachea for delivery of invasive PPV. Intubation is a skill that must be learned and takes practice to become proficient.

Endotracheal intubation may be indicated if: 1). BMV is ineffective or prolonged. 2). Chest compressions are being performed. In addition, elective intubation may be performed in certain special circumstances, such as congenital diaphragmatic hernia, airway stabilization of the extremely low birth weight infant (ELBW; BW <1000 g), and for administration of surfactant.

Procedure – Two care providers are required for endotracheal intubation, one to perform the procedure and the other to assist and monitor the status of the neonate during the procedure. To minimize hypoxemia, time needed for intubation should be limited to 30 seconds, and free flowing oxygen is administered during the procedure.

The following steps are required for successful intubation of the neonate:

1. Initial stabilization – Unless contraindicated, the patient should be stabilized by BMV.
2. Positioning – The infant is placed on his/her back with the head in the midline and the neck slightly extended .
3. Insertion – The laryngoscope is held in the left hand of the clinician between the thumb and the first two or three fingers, with the blade pointing away from the clinician. The right hand stabilizes the head of the infant. The laryngoscope blade is inserted over the right side of the tongue pushing the tongue to the left and is advanced until the blade lies in the vallecula, just beyond the base of the tongue. The entire blade is lifted in the direction of the laryngoscope handle to allow visualization of the vocal cords. It is important not to torque the laryngoscope forward like a lever (the so-called "can opener" maneuver) as this can elevate the vocal cords out of view and can damage the alveolar ridge. Once the vocal cords are visualized, an appropriate-sized ETT is passed through them with the right hand until the vocal cord guide mark (heavy black line near the tip of the tube) is at the level of the vocal cords.

Some individuals prefer to use a stylet to provide rigidity and curvature to the tube; if a stylet is used, care should be taken that it does not protrude out of the tip of the tube, and when it is removed the tube is not inadvertently dislodged.

•*Assessment of successful intubation* – Successful endotracheal intubation is confirmed with all of the following while providing PPV through the ETT:

-Prompt increase in HR (if the HR was low at time of intubation)
-Adequate oxygenation as demonstrated by pulse oximetry
-Audible breath sounds over both lung fields
-Symmetrical chest movement
-Detection of exhaled carbon dioxide (CO2) using a colorimetric device or capnography
-Vapor condensation inside the ETT during exhalation

In addition, chest radiography is needed to confirm that the ETT is correctly placed above the carina of the trachea.

•*Insertion depth and securing ETT* – The depth of insertion is determined by gestational age, birth weight or the nasal-tragus length (NTL, distance between the nasal septum and tragus of the ear). Correct

placement is initially confirmed by the presence of equal breath sounds on both sides detected by auscultation using a stethoscope.

Tube size, mm, inside diameter	Gestational age, weeks	Weight, grams
2.5	<28	<1000
3	28 to 34	1000 to 2000
3.5	34 to 38	2000 to 3000
3.5 to 4	>38	>3000

Gestation (weeks)	Endotracheal tube insertion depth at lips (cm)	Baby's weight (g)
23 to 24	5.5	500 to 600
25 to 26	6.0	700 to 800
27 to 29	6.5	900 to 1000
30 to 32	7.0	1100 to 1400
33 to 34	7.5	1500 to 1800
35 to 37	8.0	1900 to 2400
38 to 40	8.5	2500 to 3100
41 to 43	9.0	3200 to 4200

III. Start chest compressions if HR is <60 bpm despite adequate PPV for 30 seconds

Chest compressions apply pressure to the lower one-third of the sternum visualized as an imaginary line between the nipples and the xiphoid process. In neonates, chest compressions can be applied using the two-thumb or two-finger technique. We generally prefer the two thumb technique because it generates higher systolic and coronary perfusion pressure, and it allows better access for umbilical line insertion.

a). *Two-thumb technique* – In this method, both hands encircle the infant's chest with the thumbs on the sternum and the fingers under the infant. If the infant is intubated, the person performing chest compressions should move to the head of the bed to perform chest compression. This will allow another team member access to the infant to insert an umbilical line should it be deemed necessary.

b). *Two-ginger technique*- In this method, the tips of the first two fingers, or the middle and ring finger, are placed in a perpendicular position over the sternum.

With both techniques, pressure is applied downward perpendicular to the chest wall sufficient to depress the sternum approximately one-third of the anteroposterior diameter of the chest, and then pressure is released to allow the heart to refill. Care should be taken to avoid applying pressure directly over the xiphoid, as this may cause hepatic injury.

Compression rate and coordination with PPV – Chest compressions are always accompanied by PPV. During neonatal resuscitation, the chest compression rate is 90 per minute accompanied by 30 breaths per minute in a 3 to 1 ratio (ie, one breath interposed after every third compression). Thus, the ventilation rate

is reduced from the 40 to 60 breaths per minute in the absence of chest compressions to 30 breaths in the presence of chest compressions.

Supplemental oxygen – Whenever chest compressions are provided, the oxygen concentration is increased to 100 percent, but it should be weaned rapidly when the HR recovers and chest compression is no longer needed based on targeted SpO2 levels, which are monitored by pulse oximetry.

Reassessment – After 60 seconds of chest compressions and PPV, the infant's HR, color, and respiratory effort should be reassessed to determine whether further interventions are required (eg, intubation or administration epinephrine).

IV. If the HR remains <60 bpm after 60 seconds of chest compressions and PPV:

- Secure the airway (if not already done)
- Obtain venous access (typically by inserting an umbilical venous catheter
- Administer epinephrine
- Address any other potential causes (eg, hypovolemic, pneumothorax)

Epinephrine can be administered intravenously (IV) or endotracheally; IV administration is preferred because it is more efficacious. However, the endotracheal route can be used while IV access is being obtained:

-IV administration – IV epinephrine is given at a dose of 0.01 to 0.03 mg/kg (0.1 to 0.3 mL/kg of a 1:10,000 solution [concentration 0.1 mg/mL]).

-Via ETT – If epinephrine is given through an ETT, the dose is 0.05 to 0.1 mg/kg (0.5 to 1 mL/kg of a 1:10,000 solution).

The dose may be repeated every three to five minutes if the HR remains <60 bpm. If the initial dose was given endotracheally, subsequent doses can be given IV once access is obtained.

Volume expansion — In the delivery room, neonatal hypovolemia requiring volume expansion is rarely encountered, and volume resuscitation should only be considered if the HR remains <60 bpm despite adequate ventilation and administration of epinephrine.

We administer a 10 mL/kg bolus of normal saline over 5 to 10 minutes to correct hypovolemia. This dose can be repeated if necessary, based upon the response to the initial bolus. Other acceptable solutions include Ringer's lactate or uncrossed O Rh-negative blood. The latter is preferred if available and severe blood loss and/or anemia is suspected or documented.

If the HR has increased to ≥100 bpm and neonate has effective spontaneous respirations:

- Discontinue PPV
- Administer supplemental oxygen as needed to maintain the target preductal SpO_2
- Monitor the neonate closely (including HR and SpO_2) to determine whether the spontaneous respiratory effort is adequate without need for further intervention

Monitoring

1. Pulse oximetry — Pulse oximetry is used to monitor oxygen saturation (SpO2) in the following settings because skin color (cyanosis) is a poor indicator of oxygenation immediately after birth:

- When resuscitation is anticipated
- When PPV is used for more than a few breaths
- When supplemental oxygen is administered
- When the neonate is persistently cyanotic

The oximeter probe should be placed in a preductal location (ie, on the right upper extremity, usually the wrist or medial surface of the palm). Targets for preductal SpO2 levels in the first few minutes after birth are as follows:

- 1 minute – 60 to 65 percent
- 2 minutes – 65 to 70 percent
- 3 minutes – 70 to 75 percent
- 4 minutes – 75 to 80 percent
- 5 minutes – 80 to 85 percent
- 10 minutes – 85 to 95 percent

2. HR monitoring is used to evaluate the effectiveness of the neonate's respiratory efforts and to assess the response to interventions. Auscultation of the precordium is the initial preferred physical assessment of HR. However, continuous electrocardiography (ECG) monitoring provides the most rapid and accurate estimation of neonatal HR in the delivery room and during resuscitation, and should be used to confirm HR prior to initiation of chest compressions for bradycardia . The ECG monitor should be used in conjunction with the pulse oximeter because it is faster and more accurate in detecting changes in HR compared with pulse oximetry alone.

3. Oxygen concentration — When providing respiratory support during neonatal resuscitation, the goal is to prevent hypoxemia while avoiding hyperoxemia, since both can have adverse effects in the neonate. Hyperoxemia can be harmful especially in preterm infants as it is associated with increased risk of bronchopulmonary dysplasia and retinopathy of prematurity. Suggested approach is as follows:

-For neonates born at >30 weeks gestation, initiate resuscitation with room air (ie, fraction of inspired oxygen [FiO2] of 0.21).

-For neonates ≤30 weeks gestation, initiate resuscitation with an FiO2 of 0.3 using a blender.

The FiO2 is subsequently adjusted as needed to maintain the target preductal SpO2 on pulse oximetry.

Discontinuing resuscitation.

Resuscitation efforts may be discontinued after 20 minutes of effective resuscitation including intubation and the use of epinephrine, if the neonate has demonstrated no signs of life (no heart beat or respiratory effort for >20 minutes).

RESPIRATORY DISTRESS SYNDROME.

Respiratory distress syndrome (RDS), formerly known as hyaline membrane disease, is a common problem in preterm infants. This disorder is caused primarily by deficiency of pulmonary surfactant in an immature lung.

The incidence of RDS increases with decreasing gestational age (GA). The risk is highest in extremely preterm infants, as illustrated by a study from the National Institute of Child Health and Human Development Neonatal Research Network that found a 93 percent incidence of RDS in a cohort of 9575 extremely preterm infants (GA 28 weeks or below) born between 2003 and 2007.

Etiology. The primary cause of RDS is deficiency of pulmonary surfactant, which is developmentally regulated. The fetal lung is filled with fluid and provides no respiratory function until birth. In preparation for air breathing, surfactant is expressed in the lung starting around the 20th week of gestation . Surfactant reduces the alveolar surface tension, thereby facilitating alveolar expansion and reducing the likelihood of alveolar collapse atelectasis.

In addition, mutations in the genes encoding surfactant proteins SP-B and SP-C and the adenosine triphosphate (ATP)-binding cassette (ABC) transporter A3 (ABCA3) [may cause surfactant deficiency and/or dysfunction, and hereditary respiratory failure in infants born at term.

Pulmonary surfactant is a complex mixture that is mostly composed of lipids (90 percent), primarily phospholipids, and approximately 10 percent proteins. Surfactant is synthesized within the alveolar type II cells. In the preterm infant, both a decrease in the quantity and quality of surfactant contributes to decreased surfactant activity, resulting in RDS. The administration of antenatal glucocorticoids reduces the risk of RDS in preterm infants because it improves neonatal lung function by enhancing maturational changes in lung architecture and by inducing enzymes that stimulate phospholipid synthesis and release of surfactant.

Pathogenesis. In the premature lung, inadequate surfactant activity results in high surface tension leading to instability of the lung at end-expiration, low lung volume, and decreased compliance. These changes in lung function cause hypoxemia due to a mismatch between ventilation and perfusion primarily due to collapse of large portions of the lung (atelectasis), with additional contributions of ventilation/perfusion mismatch.

Pulmonary function and gas exchange — The major negative effects of surfactant deficiency on pulmonary function are low compliance and low lung volume (functional residual capacity), and are primarily due to atelectasis, although both pulmonary edema and inflammation may be contributing factors. Total lung resistance is slightly increased, probably as a result of airway compression by interstitial edema and damage to the airways by the increased pressure needed to expand the poorly compliant alveoli

Hypoxemia — The hypoxemia that occurs in RDS is due primarily to mismatch of ventilation and perfusion with intrapulmonary right-to-left shunting of blood past substantial regions of the lung that are poorly ventilated. Extrapulmonary shunting also occurs typically across the foramen ovale and patent ductus arteriosus from intrapulmonary and extrapulmonary right-to-left shunts.

Clinical manifestation. The clinical manifestations of RDS result primarily from abnormal pulmonary function and hypoxemia. Because RDS is primarily a developmental disorder of deficient surfactant production, it presents within the first minutes or hours after birth. If untreated, RDS progressively worsens over the first 48 hours of life. In some cases, infants may not appear ill immediately after delivery, but develop respiratory distress and cyanosis within the first few hours of age. These infants may have a borderline amount of surfactant that is consumed or becomes inactivated.

The affected infant is almost always preterm and exhibits signs of respiratory distress that include:

1) Tachypnea.
2) Nasal flaring, which reflects the use of accessory respiratory muscles and lowers total respiratory system resistance.
3) Expiratory grunting, which results from exhalation through a partially closed glottis and slows the decrease in end-expiratory lung volume.
4) Intercostal, subxiphoid, and subcostal retractions, which occur because the highly compliant rib cage is drawn in during inspiration by the high intrathoracic pressures required to expand the poorly compliant lungs.
5) Cyanosis due to right-to-left intra- and extra-pulmonary shunting.

On physical examination, auscultated breath sounds are decreased, and infants may be pale with diminished peripheral pulses.

The urine output often is low in the first 24 to 48 hours and peripheral edema is common.

Prior to surfactant use, uncomplicated RDS typically progressed for 48 to 72 hours. This was associated with an improvement in respiratory function as endogenous surfactant production increased.

RDS typically resolves by one week of age. A marked diuresis typically preceded the improvement in lung function.

The natural history of RDS is greatly modified by treatment with exogenous surfactant, which dramatically improves pulmonary function, leading to the resolution of symptoms, and shortens the clinical course.

Laboratory findings. Chest radiography is generally obtained for all neonates with respiratory distress. The radiographic features of neonatal RDS (low lung volume and the classic diffuse reticulogranular ground glass appearance with air bronchograms) in a preterm infant with respiratory distress fulfill the clinical diagnosis criteria for RDS

Other laboratory findings associated with, but not diagnostic for, RDS include:

●Arterial blood gas measurements typically show hypoxemia that responds to administration of supplemental oxygen. The partial pressure of carbon dioxide (PCO2) initially is normal or slightly elevated, but usually increases as the disease worsens.

●As the disease progresses, infants may develop hyponatremia. This results from water retention, and usually improves with fluid restriction. Attentive fluid management prevents hyponatremia, and as a result, this finding is less commonly observed.

Differential diagnosis:

- Transient tachypnea of the newborn (TTN) in more mature infants (ie, term or late preterm infants) compared with RDS. Patients with TTN have milder respiratory distress and improve more quickly than those with RDS
- Bacterial pneumonia - blood cultures and, possibly, tracheal cultures should be obtained in all preterm infants who present with respiratory distress. Empirical antibiotics are given to infants at risk for infection pending culture results and clinical course.
- Air leak – Air leak (eg, pneumothorax) may be a complication of RDS, an isolated problem, or associated with another underlying disorder. It is detected by chest radiography.
- Cyanotic congenital heart disease – Most patients with cyanotic congenital heart disease (CCHD) have milder respiratory distress than that seen in patients with RDS. In addition, CCHD is usually differentiated from RDS by the absence of the characteristic diffuse reticulogranular ground glass appearance with air bronchograms on chest radiograph. If lung function and the chest radiograph do not improve with respiratory support and surfactant administration, an echocardiogram should be performed to rule out structural heart disease or persistent pulmonary hypertension of the newborn (PPHN) in infants with severe arterial hypoxemia. (See "Cardiac causes of cyanosis in the newborn".)
- Interstitial (diffuse) lung disease – A number of interstitial and diffuse lung diseases may present in the neonatal period, including genetic disorders of surfactant dysfunction, lung growth abnormalities, and pulmonary interstitial glycogenosis. (See "Classification of diffuse lung disease (interstitial lung disease) in infants and children".)
- Non-pulmonary systemic disorders, such as hypothermia, hypoglycemia, anemia, polycythemia, or metabolic acidosis, may present with respiratory distress. Differentiation from RDS is based on the history, physical findings and appropriate laboratory evaluation.

Management.

1. Prenatal care.

Interventions to improve outcome and prevent RDS begin before birth. Extremely preterm babies should, if possible, be transported in utero to tertiary centres where appropriate skills are available. Magnesium sulphate (MgSO4) given to women with imminent preterm delivery reduces cerebral palsy at 2 years of age by about 30%. Tocolytic drugs can be used in the short-term to delay birth, permit safe transfer to a perinatal centre and allow prenatal corticosteroids time to take effect, although tocolytics have no direct beneficial effect on the fetus.

Prenatal corticosteroid therapy is recommended in all pregnancies with threatened preterm birth before 34 weeks' gestation where active care of the newborn is anticipated.

The optimal treatment to delivery interval is more than 24 h and less than 7 days after the start of steroid treatment; beyond 14 days, benefits are diminished.WHO recommends that a single repeat course of steroids may be considered if preterm birth does not occur within 7 days after the initial course and there is a high risk of preterm birth in the next 7 days. The Royal College of Obstetrics and Gynaecology guidelines recommend Betamethasone 12 mg intramuscularly for two doses, or dexamethasone 6 mg for four doses.

2. *Delivery room stabilization strategies.*

-Delay clamping the umbilical cord for at least 60 s to promote placento-fetal transfusion;

-Once the umbilical cord is cut, babies should be placed in plastic bags or wrapped in plastic under radiant warmers as this is effective in preventing heat loss in infants less than 28 weeks' gestation;

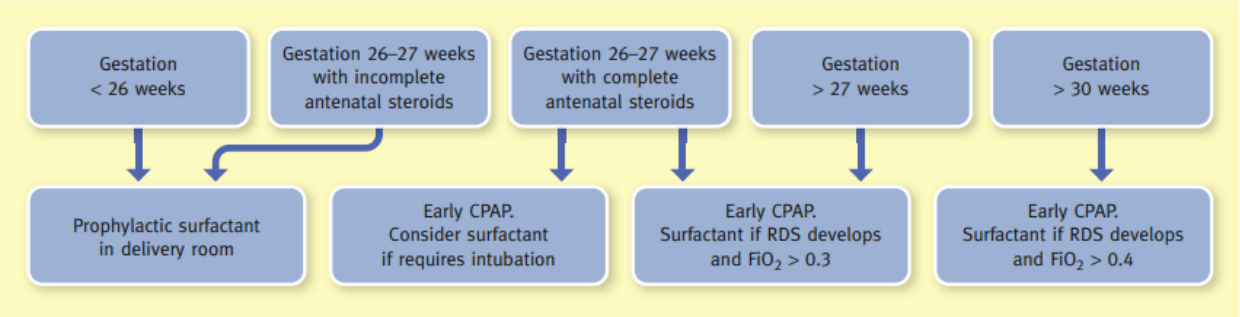
- Oxygen for resuscitation should be controlled using a blender. Use an initial FiO2 of 0.30 for babies less 28 weeks of gestation and 0,21-0,3 for those 28-31 weeks, 0,21- for 32 weeks and above.

- Normative data for pulse oximetry saturations during transition after birth are now available and clinicians should not intervene for low saturations immediately during this phase provided there is an adequate heart rate. During the transitional phase after birth, saturations should rise gradually from about 60% to 80% over 5 minutes, reaching above 85% by about 10 minutes of age and oxygen should only be needed if saturation falls outside this normal transitional pattern. In preterm babies receiving oxygen, the saturation target should be between 90 and 94%. Alarm limits should be set to 89 and 95%.

- CPAP(Continuos Positive Air Pressure) should be started from birth in all babies at risk of RDS, such as those <30 weeks' gestation who do not need intubation for stabilization.

-Intubation should be reserved for babies not responding to positive pressure ventilation via face mask or nasal prongs .

-Babies who require intubation for stabilisation should be given surfactant.



3. *Supportive care.*

- maintenance of a normal body temperature,
- proper fluid management,
- good nutritional support,
- appropriate management of the ductus arteriosus,
- support of the circulation to maintain adequate blood pressure.

Core temperature should be maintained between 36.5 and 37.5 °C at all times.

Most babies should be started on intravenous fluids of 70–80 mL/kg/day in a humidified incubator, although some very immature babies may need more.

Fluids must be tailored individually according to serum sodium levels, urine output and weight loss.

Parenteral nutrition should be started from birth.

-Amino acids 1–2 g/kg/day should be started from day one and quickly built up to 2.5–3.5 g/kg/day.

-Lipids should be started from day one and built up to a maximum of 4.0 g/kg/day if tolerated.

Enteral feeding with mother's milk should be started from the first day if the baby is haemodynamically stable.

Antibiotics should be used judiciously and stopped early when sepsis is ruled out.

Blood pressure should be monitored regularly aiming to maintain normal tissue perfusion, if necessary using inotropes. Haemoglobin should be maintained at acceptable levels.