Review Article

Approach to Respiratory Distress in the Newborn

Sai Sunil Kishore M, Siva Sankara Murty YV, Tarakeswara Rao P, Madhusudhan K, Pundareekaksha V, Pathrudu GB

Abstract: Respiratory distress is responsible for majority of neonatal admissions to neonatal intensive care unit. Various pulmonary and extra-pulmonary causes are responsible for respiratory distress. Prolonged and unattended distress leads to hypoxemia, hypercarbia and acidosis leading to pulmonary vasoconstriction and persistence of fetal circulation, thereby aggravating hypoxemia, worsening prognosis. A discussion on general approach to identify etiology, evaluation and management is made in this article.

Key Words : Respiratory distress, Meconium aspiration, Mechanical Ventilation, Continuous Positive airway pressure, Surfactant.

Introduction

Respiratory distress is the commonest morbidity requiring admission of a neonate in an intensive care unit. It is responsible for 30-40% of the admissions in the neonatal period [1,2]. Respiratory distress occurs in 11-14% of all live births [3]. The incidence of respiratory distress on first day of life increases with lower gestations : <30 wks- 60%, 30 to 34 wks- 43% and >34 wks: 5 to 6% [3]. Respiratory distress is defined by presence of at least 2 of the following three features- tachypnea (respiratory rate >60 per minute), retractions (intercostal, subcostal, sternal or suprasternal) and noisy respiration (grunt, stridor or wheeze)[1]. The distress may or may not be associated with cyanosis or desaturation on pulse oximetry. A working diagnosis should be made in the first few minutes of onset of respiratory distress and immediate resuscitative measures should be initiated till further management plans are drawn up. The objectives of this article include a discussion on the general approach to identify the etiology, initial evaluation and algorithm for initial management, interpretation of blood gases, and approach to initiation of ventilation, titration and weaning.

Etiology

The etiology of respiratory distress is the single most important determinant of the course and prognosis. The common etiologies based on the time of onset of respiratory distress and gestation are depicted in Fig1. Respiratory distress of the neonate can be attributed either to pulmonary or to extra-pulmonary disorders. 'Pulmonary' causes of respiratory distress are commoner than the 'extra pulmonary'. While conditions like structural anomalies and pneumonia are common to term and preterm infants, a condition like hyaline membrane disease (HMD) is almost always a disease of the premature infant. Meconium aspiration, on the other hand, is seen almost exclusively in term infants.

Among very low birth weight (VLBW) neonates, up to 60% may develop respiratory distress soon after birth [4]. Among them the contribution by various etiologies include: hyaline membrane disease- 36%, pneumonia- 28% and transient tachypnea of newborn (TTNB)- 27%[4].

A functionally normal lung sometimes needs to work at a capacity far exceeding normal levels, in order to compensate for abnormalities of other systems, e.g. in the presence of cardiac disease, shock, metabolic acidosis, or abdominal distension. The definitive management of such an infant would naturally be based on treating the primary 'extra-pulmonary' etiology.

Clinical/historical clues : Clinical clues and their possible disease associations are presented in table 1 and the initial examination that should be performed is shown in table 2.

Pathophysiology considerations unique to the newborn

Prolonged and unattended distress leads to hypoxemia, hypercarbia and acidosis. This leads to pulmonary vasoconstriction and persistence of fetal circulation with right to left shunting through the ductus and foramen ovale, thereby aggravating hypoxemia. An audible grunt is an important sign of pulmonary pathology in the newborn, indicating that the baby has a low lung volume or

Corresponding author
Sai Sunil Kishore M (Neonatologist)
Assistant Professor, Department of Paediatrics
Maharajah's Institute of Medical Sciences, Nellimarla, Vizianagaram - 535 217, Andhra Pradesh, India.

functional residual capacity (FRC). Breathing against a partially closed glottis increases the FRC of the baby and helps to keep the alveoli open. This is characteristically seen in a baby with respiratory distress syndrome (RDS) where surfactant deficiency tends to keep the alveoli collapsed during expiration. Indiscriminately inserting an endotracheal (ET) tube without giving positive end expiratory pressure (PEEP) to a neonate who is grunting will deprive the baby of this physiological effect and worsen the condition, instead of improving it. Hence any baby who is grunting, depending on the severity of respiratory distress, should be given either continuous positive airway pressure (CPAP) or intubated and put on ventilator support with PEEP, but never left to breathe spontaneously with a tube in situ.

Grading of severity of respiratory distress

The severity of respiratory distress can be assessed by Silverman-Anderson score (Fig2) and Downes' score (Table 3)[5,6]. While the Silverman Anderson retraction score is more suited for preterms with RDS, the Downes' score can be applied to any gestational age and condition. Serial monitoring of these scores with documentation will help in determining the progression.

Investigations

Essential investigations for all cases of respiratory distress include:

- **Arterial blood gas (ABG) analysis**: Blood gas is essential because with clinical assessment and pulse oximetry alone, one would not be able to assess PCO_2 and pH.

Normal & abnormal values:

PaO_2: Pre-ductal PaO_2 50–70 mmHg with an O_2 saturation of 87-93%. A PaO_2 up to 80 mmHg is acceptable in term infants

- Hypoxemia: PaO_2<50 mmHg
- Low normal oxygenation: PaO_2 - 50-60mmHg
- Hyperoxemia: >80 mmHg in preterm and >90 in term. Hyperoxia is associated with adverse effects like retinopathy of prematurity(ROP) and bronchopulmonary dysplasia(BPD) due to increase in the reactive oxygen species (ROS).

  $PaCO_2$: Normal $PaCO_2$ is 35-45 mmHg
- Acceptable upper limit: Acute stage – 45-50mmHg, Chronic (>72 hours of ventilation) – 55mmHg (with pH of e"7.2). There is increasing evidence that the strategy of permissive hypercapnia reduces the duration of ventilation and decreases the severity of bronchopulmonary dysplasia (BPD) [7].
- Hypocarbia: <35 mmHg. Hypocarbiawith $PaCO_2$<30 mmHg increases the risk of periventricular leucomalacia (PVL) in preterm neonates [8].

- **Chest radiograph:** Radiological findings in various causes of respiratory distress are shown table 4.
- Electrolytes, blood glucose, hematocrit
- Sepsis work up which includes C-reactive protein (CRP), micro ESR, total leukocyte count (TLC), absolute neutrophil count (ANC) and Immature to total leukocyte ratio (ITR). The parameters like TLC and ANC should be interpreted according to the age and gestation of the baby [9,10].
- Assessment of gas exchange: Alveolar-arterial oxygen gradient ($AaDO_2$), Oxygenation index (OI)

Special investigations like echocardiography and neuro-sonogram may be required on case to case basis depending on the clinical presentation.

Bedside investigations:

1. **Gastric aspirate shake test:** This is useful in assessing the risk of RDS. This is useful in all neonates with respiratory distress who are <34 wks of gestation or have risk factors for RDS. In a glass test tube with dimensions of 82 mm × 10.25 mm and 4 ml capacity, 0.5 ml of gastric aspirate is taken and mixed with 0.5 ml of absolute alcohol. The tube is shaken vigorously for 15 seconds and allowed to stand for 15 min. If at least one complete rim of bubbles are present all the way round the meniscus the risk of RDS is <1% where as complete absence of bubbles is associated with a risk of 50-60%.
2. **Gastric aspirate for polymorphs:** The gastric fluid should be aspirated preferably within one hour after birth and mixed with one drop of heparin. A drop of this is placed on a glass slide and a thick smear is made and stained with Leishman's stain. More than 5 polymorphonuclear leucocytes/HPF is suggestive of infected amniotic fluid or chorioamniotis. The test is not useful if the aspirate is contaminated with blood or meconium.

Assessment of gas exchange[11]

Though the blood gas parameters indicate oxygenation and ventilation at a single point of time, these parameters alone would not be sufficient to evaluate gas exchange. Interpretation of PaO_2 without FiO_2 is misleading. Hence the gas exchange should be assessed using various parameters like

1) Alveolar-arterial Oxygen gradient (A-aDO_2)
2) a/A ratio
3) Oxygenation Index (OI)

Calculation and interpretation:

1) A-aDO_2 (alveolar arterial oxygen diffusion gradient): This is to be calculated as shown below. A-a D_{O2} = $PAO_2 - PaO_2$ (PAlveolar – Parterial oxygen)

   $= [PiO_2 - PACO_2] - PaO_2$

   $= [(P_B - P_W) \times F_iO_2 - PaCO_2] - PaO_2$

   $= [(760-47) \times FiO_2 - PaCO_2] - PaO_2$

[$PiO_{2=}$ Partial inspired oxygen pressure P_B= Baromatric pressure, P_W= water vapor pressure, $FiO_{2\ =}$ Fractional inspired oxygen concentration]

Normally it ranges between 5-15, if breathing room air. A-aDO2 is considered to be abnormal if more than 40.

2) a /A ratio: Ratio of PaO_2 to PAO_2. It is considered to be a better indicator of gas exchange as the ratio is usually not affected by changes in FiO_2

Interpretation:

a) Greater than 0.8: Normal
b) Less than 0.6: indicates need for O_2 therapy
c) Less than 0.15: severe hypoxemia

3) Oxygenation Index (OI): Recommended in babies who are mechanically ventilated as this index includes mean airway pressure (MAP).

   $OI = (MAP \times FiO_2) / PaO_2$

Interpretation:

a) OI 25 – 40: severe respiratory failure; mortality risk is 50 – 60%
b) OI > 40: Mortality risk is >80%

Prevention: role of antenatal steroids

Antenatal steroids should be administered to pregnant women of 24 to 34 weeks of gestation with intact membranes or premature rupture of membranes without chorioamnionitis, who are at high risk of preterm delivery within next 7 days[12]. This strategy induces surfactant production and accelerates maturation of lungs and other fetal tissues. Treatment with antenatal corticosteroids is associated with an overall reduction in neonatal mortality (RR: 0.69, 95% CI: 0.58 to 0.81), RDS (RR 0.66, 95%CI: 0.59 to 0.73), IVH (RR 0.54, 95% CI 0.43 to 0.69) and NEC (RR 0.46, 95% CI 0.29 to 0.74)[13]. Obstetricians should be made aware of these benefits so that this preventive strategy can be utilized optimally.

Treatment

While managing a neonate with respiratory distress, the initial treatment is aimed at resuscitation of the neonate, optimizing tissue oxygenation, decreasing the work of breathing, preventing hypoxia, hypercapnia and acidosis. The supportive measures could vary from oxygen supplementation to various strategies of mechanical ventilation. An algorithm for initial management of a neonate with respiratory distress is presented in Fig 3. Subsequent management should focus on further evaluation to identify the etiology, definitive management and follow-up.Details of oxygen therapy and other modalities of respiratory support are described below.

Supportive therapy: Apart from providing respiratory support, supportive care and proper nursing care are very crucial for success of management.

- Thermo-neutral environment : The neonate should be nursed in a thermoneutral environment. Hypothermia will initiate the cascade of PPHN and aggravate hypoxemia. Baby's temperature should be maintained between 36.5^0-37.5^0C. VLBW neonates need incubator for their temperature maintenance and for providing adequate humidity
- Electrolyte balance, fluids and normal acid-base balance should be maintained. Preterm babies have higher insensible water loss (40-100 ml/kg). Fluid intake should be titrated accurately by recording serial weight, intake/ output, serum sodium and urine specific gravity.
- Antibiotics should be started in all cases of suspected sepsis or pneumonia.
- Calcium and glucose homeostasis should be ensured.
- Maintain normal mean arterial pressure. Hypotension should be corrected by using appropriate fluid volumes and inotropes if necessary.

- Hypovolemia and anemia are to be treated adequately as necessary. Hematocrit should be maintained above 40% in the acute phase of the disease [14].
- Oral feeding is withheld initially. Once the baby stabilizes on the respiratory support and respiratory rates are less than 70/min, gavage feeding should be started.

Definitive management

Despite a relatively uniform approach to the initial management, one must realize that procrastination and delay in instituting definitive therapy may result in adverse outcomes. For example, an infant with tension pneumothorax could deteriorate rapidly despite the transient improvement seen with initial therapy with oxygen and increased ventilator support, if the pneumothorax is not drained. RDS will progress with time if surfactant is not administered in time. Similarly, repeated aspiration pneumonia would contribute to poor surgical outcome in patients with delayed diagnosis of tracheoesophageal fistula (TEF). Therefore a definite diagnosis and therapy is mandatory for successfully managing infants with respiratory distress.

Surfactant replacement

Surfactant replacement therapy is the standard of care for a baby with RDS. Surfactant should be administered early in the course of respiratory distress, preferably within first two hours of onset of symptoms in neonates at risk for RDS (Early rescue therapy). Delayed rescue therapy is less effective since the inflammatory processes and exudation already sets in. Prophylactic administration of surfactant may be used in neonates who are at a very high risk of RDS and its complications. Typically, this may be in neonates <28-30 wks of gestation depending on the local survival rates. This approach although very effective, would increase the costs. The dose of surfactant for RDS is according to the phospholipid and varies between 100 to 200mg/kg depending on the manufacturer. Two or more doses may be required especially in extremely low birth weight babies.

Surfactant activity is altered in other respiratory disorders also apart from RDS. Surfactant has been tried in various conditions such as meconium aspiration syndrome, pneumonia and pulmonary hemorrhage with variable benefits [16]. Further evidence is needed to establish the value and limitations of surfactant therapy for these conditions.

Oxygen therapy

Indications:

1) Clinical central cyanosis
2) Hypoxemia (O_2 saturation <87% and / or PaO_2<50 mmHg in room air)
3) Presence of respiratory distress irrespective of O_2 saturation

Saturation targets [17] : Preterm: 87-93%, Term: 90-95%, Bronhopulmonary dysplasia: 90-95%, PPHN: >95%

Commonly used oxygen delivery systems in neonates: Characteristics of common oxygen delivery systems are provided in table 5.

Note:

1) Short binasal prongs are currently available for O_2 administration, which can be attached to the humidification system to provide heated and humidified O_2
2) While using oxygen hood, change in the flow rate will not alter the FiO_2 much. If one wants to decrease the FiO_2, more mixing with air has to be allowed through port holes. Occlusion of these holes increases the O_2 concentration by preventing air entrainment. Approximate FiO_2 delivered with a flow rate of 5-8 Lpm:

- With all port holes closed: 60-70%
- With one port hole opened – 40-50%
- With both the port holes opened – 30-40%

Precautions while administering oxygen:

1. Prewarm and humidify the oxygen especially at flow rates > 2Lpm

2. Oxygen saturation should not cross 93% as hyperoxia leads to wide spread free radical injury

3. Flow rate should never be crossed as excessive flow can cause turbulence in the airflow and inadvertent uncontrolled PEEP delivery apart from causing local airway injury

4. Oxygen analyzer is to be used always to check the FiO_2. Desired FiO_2 can be administered by using a blender or by using varying flow rates of oxygen and air using a 'Y' piece.

Non-invasive ventilation

Continuous Positive Airway Pressure (CPAP)

Continuous distending pressure is applied throughout the respiratory cycle in a spontaneously breathing infant.

CPAP can be administered by applying different nasal or nasopharyngeal interfaces. Endotracheal CPAP should not be given. It works by allowing alveolar recruitment, preventing atelectasis thereby improving FRC. It also dilates the upper airways and decreases the airway resistance. In a preterm neonate who is at risk of respiratory distress syndrome, CPAP should be used early, to reduce the need for mechanical ventilation unless there is a contraindication to use CPAP. Early CPAP conserves the neonate's own surfactant stores and minimizes the stimulation of inflammatory cascade. A comparison of early versus delayed initiation of CPAP in RDS concluded that early CPAP reduces the subsequent use of intermittent mandatory ventilation (IMV) [typical RR 0.55, 95% CI: 0.32-0.96][18]. If used early, many lives can be saved and even upward referrals to a tertiary care unit can be reduced [19]. Considering the benefits of CPAP and problems associated with mechanical ventilation, all efforts should be made to manage babies on CPAP.

The level of CPAP should be individualized to the baby's disease. As a general guideline start CPAP of 5cmH_2O and $FiO_2$of 0.5 to maintain normal SpO_2. Subsequent titration can be done based on the clinical improvement, blood gases and lung volume on chest radiograph. The orogastric tube (OGT) should be inserted to decompress the stomach. The signs of over inflation should be monitored - inadequate cardiac output (prolonged CFT, reduced urine output, metabolic acidosis) and hyperinflated chest. Periodic inspection of the local area is important because patient interface can lead to septal and mucosal injury.

Nasal intermittent positive pressure ventilation (NIPPV)

NIPPV combines nasal CPAP and intermittent positive pressure ventilation (IPPV) via a timed cycled, pressure limited ventilator. NIPPV has been found to be more effective than NCPAP in weaning preterm infants from mechanical ventilation [20] and in apnea of prematurity [21]. There is evidence to suggest that NIPPV as a primary mode of respiratory support may be more effective than CPAP in reducing the need for intubation in RDS[22].

Mechanical ventilation

Mechanical ventilation needs to be started whenever there is failure of noninvasive ventilation (see fig 2) or if it is contraindicated in a neonate with respiratory distress. However the decision to initiate mechanical ventilation should be individualized for each baby and depends on several factors. The severity of distress, severity of blood gas abnormalities, natural history of disease and degree of cardiovascular and other physiologic instabilities must be taken into account. Because invasive ventilation is associated with serious pulmonary morbidities like subglottic stenosis, respiratory infections, ventilator associated lung injury and BPD[23,24], the decision to intubate and ventilate should not be taken lightly. For best results with minimum damage, it is useful to follow the laws of ventilator efficiency (LOVE). One needs to know the baby, the disease, the machine and have an EXIT strategy[25].

Initiation of mechanical ventilation

Initial steps in starting positive pressure ventilation include endotracheal intubation, selection of appropriate ventilator settings and evaluation to check adequacy of ventilatory support. Time cycled pressure limited ventilation is the commonest type of ventilation used in neonates. Mean airway pressure (MAP) and the delivered volumes are dependent on the ventilator settings. Establishing the correct ventilator settings is the key to successful respiratory support.

Parameters in a pressure limited ventilator [26]

Rate: Ventilator rate determines the minute ventilation in combination with tidal volume (V_T). This usually varies from 20-60 depending on the disease pathology and course of the baby on ventilator.

Flow: Initial flow rate should be 5-7 Lpm to drive the respiratory gases and for adequate delivery of the pressures. Flow rate above 10 Lpm can cause lot of turbulence and increase the resistance.

PEEP: PEEP is the distending pressure in the expiratory phase of the respiratory cycle that recruits the alveoli and maintains FRC. Settings for PEEP range from 3-6 cm H_2O depending on the disease pathology.

PIP: Delivered V_T is dependent on the driving pressure which is the difference between PIP and PEEP. Initial choice of PIP is dependent on observation of chest wall movement during hand-bag ventilation, manometer readings during hand-bag ventilation and auscultation of breath sounds. Subsequently PIP should be adjusted to achieve optimal MAP and V_T and effective gas exchange based on the blood gas parameters.

Ti: The inspiratory time should be in the range of 0.35-0.45sec depending on the inspiratory time constant.

FiO2: Normal tissue oxygenation can be accomplished by achieving a PaO_2 of 50-70 mm Hg. FiO_2 should be titrated according to the target saturation (see section on oxygen therapy). Increasing the inspired oxygen is the simplest and most direct means of improving the oxygenation.

The usual initial settings for common diseases are provided in table 6.

Adjustment of ventilatory settings according to arterial blood gases (Table 7):

a) Oxygenation depends on mean airway pressure (MAP) and oxygen concentration (FiO_2).

b) Mean airway pressure (MAP) is given by the formula:

$$MAP = [k^* (PIP \times Ti) + (PEEP \times Te)] / Ti + Te$$

*'k' is a constant that depends on the flow rate and the type of waveform generated. Generally, 'k' is '1' for a square wave and '0.8' for a sine wave

c) PEEP has the maximum influence on MAP. But before increasing PEEP, the underlying disease process for which ventilation has been initiated has to be considered.

d) Ventilation depends on the driving pressure (²%P), which is the difference between PIP and PEEP. Tidal volume (TV) is dependent on ²%P).

e) $PaCO_2$ is inversely proportional to the minute ventilation, which is given by the product of TV and ventilatory rate.

Note:

a) Always compare the blood gas parameters with the previous gases to identify the trend.

b) Changes in FiO_2 (especially while weaning) may be done by monitoring the SPO_2 alone provided the ventilation is adequate

e) Hypocarbia of less than 30 mmHg has been shown to be associated with periventricular leukomalacia (PVL) and a poor long term neurodevelopmental outcome (8). Hence, target a low normal TV (~ 4 – 5ml/kg) to avoid hyperventilation.

Weaning of a baby from mechanical ventilation

Weaning is the process of gradual, measured reduction of ventilatory settings to a minimum point at which risk of ventilator induced lung injury (VILI) is the least and from where the infant can be safely taken off ventilatory support. This depends on the underlying disease for which the infant was ventilated and the mode of ventilation from which the infant has to be weaned off.

Prerequisites for weaning from acute ventilation (up to 7 days of ventilation) include

a) Clinically & hemodynamically stable

b) Effective respiratory efforts

c) Basic lung pathology / disease improving

d) No significant co-morbidity

e) Optimum blood gases

Prerequisites for weaning from chronic ventilation (> 7 days of ventilation):

a) All the above features for acute ventilation

b) Adequate and consistent weight gain (over the last 5-7 days)

c) Normal serum electrolytes (especially potassium)

d) Acceptable hematocrit

General principles of weaning:

a) Decrease the most potentially harmful parameters first (PIP & FiO_2).

b) Limit changes to one parameter at a time.

c) Avoid changes of a large magnitude.

d) Document the infant's response to each change in the ventilation parameter.

Summary

Respiratory distress could be a clinical presentation of both pulmonary and non-pulmonary causes. The etiology may vary according to the age of onset and gestation of the baby. After initial resuscitation and respiratory support, targeted history and examination will guide in instituting definitive therapy. A systematic approach is mandatory to confirm the diagnosis of respiratory distress. Antenatal steroids have a definitive role in preventing pulmonary morbidity and overall mortality in preterm neonates and must be promoted to our obstetric colleagues. Respiratory therapy will be successful only if essential supportive care in the form of effective thermal support, maintenance of metabolic parameters, fluid and electrolyte balance , cardiac output, perfusion, nutrition and asepsis is provided. CPAP and noninvasive ventilation have considerable benefits over intubation. If intubation is still required, the goal should be to extubate at the earliest opportunity.

Table 1: Clinical/historical clues to various causes

Diagnosis	Features
Respiratory distress syndrome	Prematurity, No/inadequate antenatal steroids, maternal diabetes mellitus, Rh isoimmunization, a suggestive gastric aspirate shake test
Congenital pneumonia	Feature suggestive of infection in the mother • Chorioamnionitis, diarrhea, urinary infection, unclean vaginal examination • Unexplained preterm onset of labor • Pre-labor prolonged rupture of membranes • Polymorphs in gastric aspirate (>5 per hpf)
Meconium aspiration syndrome & Asphyxial lung damage	Meconium stained liquor& cord, Evidence of fetal distress: Decreased fetal movements, abnormal fetal heart rate patterns, cord umbilical artery blood acidosis
Air leak syndromes (Pneumothorax, PIE, pneumopericardium)	Sudden deterioration on ventilator, underlying disease causing air trapping (MAS), chest hyperinflation, differential air entry
Tracheo-esophageal fistula	Polyhydramnios, excessive frothing from mouth, absent stomach bubble on abdominal x-ray, scaphoid abdomen
Cong. diaphragmatic hernia	Scaphoid abdomen, heart sounds over the right chest, bowel sounds over the thorax
Diaphragmatic paralysis	Abnormal presentation, difficult delivery (large baby, shoulder dystocia, forceps extraction), associated birth trauma, asymmetric Moro's reflex
Inborn errors of metabolism	Unexplained sibling death, unexplained metabolic acidosis and hypoglycemia, seizures
Aspiration pneumonia	• Risk factor for breathing-swallowing incoordination (asphyxia, CNS malformations) • Anatomical defects (cleft palate)
Upper airway pathologies (laryngomalacia, vascular malformations, subglottic stenosis)	Stridor, suprasternal recessions, minimal oxygen requirement
Persistent pulmonary hypertension of newborn (PPHN)	Antenatal ACE inhibitors, post-term, asphyxia, MAS, severe cyanosis (not responding to O_2), labile saturations, preductal & postductal SPO2 difference of >10%

Table 2: Initial examination of a neonate with respiratory distress

Respiratory

- Watch for respiratory rate, retractions, grunt, cyanosis and stridor
- Check the need for supplemental O_2 (use pulse oximeter)
- Observe- chest expansion and chest wall movement with respirations
- Check air entry/breath sounds (bilateral)
- Transillumination if air leak suspected
- Patency of nostrils (especially if cyanosis improves with crying)

Other systems

Abdomen:

- Contour (Scaphoid, distended)
- Palpate liver and spleen (hyperinflation, CCF, IEM)

Chest:

- Heart sounds (intensity, location)
- CVS examination for PPHN, CHD

Miscellaneous:

- Fontanel, sutures separation (IVH)
- Skin - color (pallor, plethora), mottling, meconium staining
- CNS-tone, pupils, alertness

Table 3: Downe's score (6)

Parameter	0	1	2
Respiratory rate (/min)	< 60	60-80	>80
Cyanosis	Absent	In room air	In 40% oxygen
Grunt	Absent	Audible with a stethoscope	Audible with a naked ear
Retractions	Absent	Mild	Moderate - severe
Air entry	Good	Diminished	Barely audible

Table 4: Chest X-ray findings of specific respiratory illnesses

Disease	CXR findings
RDS	• Low lung volume* • Reticulo-granular shadows, air bronchogram, ground glass opacities, whitened out lungs
TTNB	• Normal/hyperinflated# lungs • Prominent minor interlobar fissure • Mild cardiomegaly@ • Prominent hilar and pulmonary vascular markings
MAS	• Hyperinflation (localized or generalized) • Areas of patchy atelectasis
Congenital pneumonia	• Low to normal volume lungs • Patchy atelectasis
PPHN	• Pulmonary oligemia • Features of underlying lung disease

(*Low lung volume: Less than 6 posterior intercostals spaces
#Hyperinflation: Greater than 8 posterior intercostals spaces
@Cardiomegaly: Cardio thoracic ratio >65%)

Table 5. Characteristics of common oxygen delivery systems

Type	Landmarks for the depth of insertion	RFR* (Lpm)	FiO2 (%) (at an average RFR)	Complications	Remarks/ precautions
Nasal cannula	Nares to the inner margin of the eyebrow	1-2	25-45	Crusting, septal trauma, erosion, inadvertent PEEP	Alternate between nares every 12 hours
Nasopharyngeal cannula	Alanasi to the tragus	1-2	45-60	Crusting, septal trauma, septal erosion, inadvertent PEEP	Alternate between nares every 12 hours
Nasal prongs	0.5-1 cm	1-2	25-45	Crusting, erosion	Short, binasal prongs are recommended
Oxygen hood	--	2 - 4	30-70%	--	RFR should be at least 4 times that of the minute ventilation. Lesser flow rates carry risk of CO2 retention

(*RFR – recommended flow rate, PEEP – positive end-expiratory pressure)

Table 6.Typical initial settings for common diseases requiring ventilation

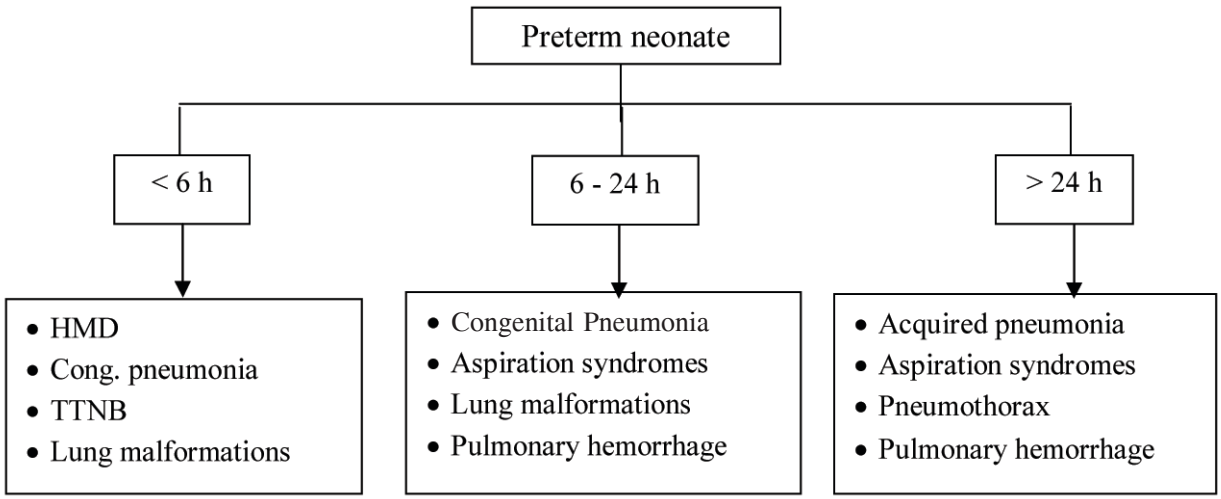
Disease	PIP (cmH2O)	PEEP (cmH2O)	Ti (Sec)	VR (per mt)
HMD	16 - 18	5 - 6	0.3-0.35	60
Pneumonia	14 - 16	3 - 4	0.35-0.4	50-60
MAS	14 - 16	3 - 4	0.35-0.4	40-50
Apnea	12 - 14	3	0.35-0.4	20-30
Air leak	14 - 16	3	0.3-0.35	60
CLD	12 - 14	3	0.3-0.35	30-40

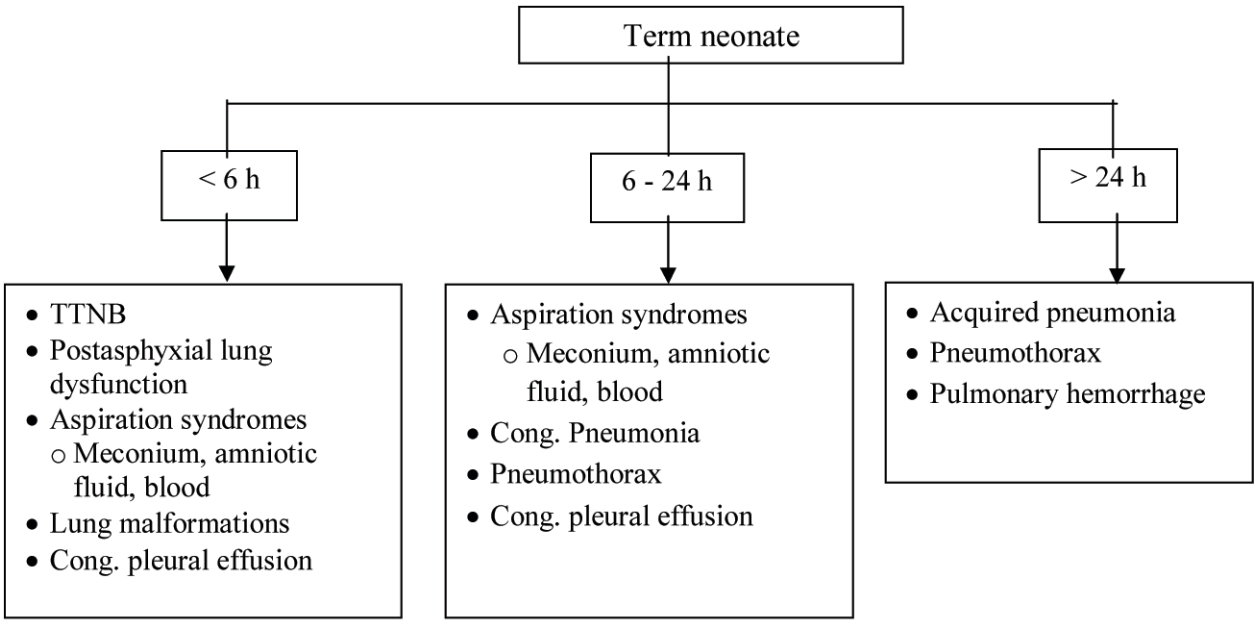
Table 7. Recommended adjustments in settings based on blood gases and clinical examination

PaO_2 (mmHg)	**$PaCO_2$ (mmHg)**	**Possible change**	**Remarks**
<50	normal	• ↑ FiO_2 in 3-5% increments • ↑ PEEP by 1 cmH20	PEEP change may be considered if associated chest retractions are observed
<50	>50	• ↑PIP (by 1-2 cmH2O)	Consider ↑FiO_2 appropriate for PEEP
<50	<40	• ↑ FiO_2 in 3-5% increments	PAH may present with purely oxygenation difficulty; do not hyperventilate
50 - 80	>50	• ↑ventilatory rates (by 5-10)	PIP change may be considered if PaO_2 is low normal
50 - 80	<40	• ↓PIP by 1 – 2 cmH_20 • ↓ventilatory rates (by 5-10)	Consider ↓rates if PaO_2 is low normal as change in PIP will ↑MAP
>80	<40	• ↓FiO_2 in 3-5% decrements • ↓PIP by 1 – 2 cmH_20	Consider weaning at this point

PAH – pulmonary artery hypertension (persistent or secondary)

Fig 1. Common pulmonary causes of respiratory distress based on gestation and time of onset

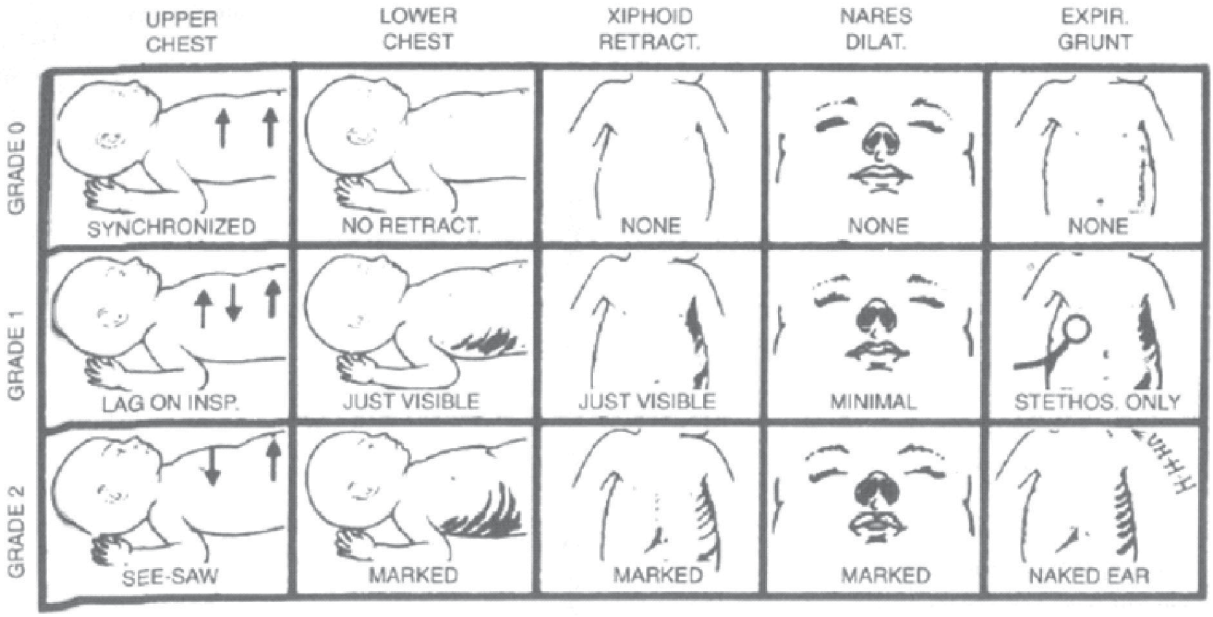




Extrapulmonary causes

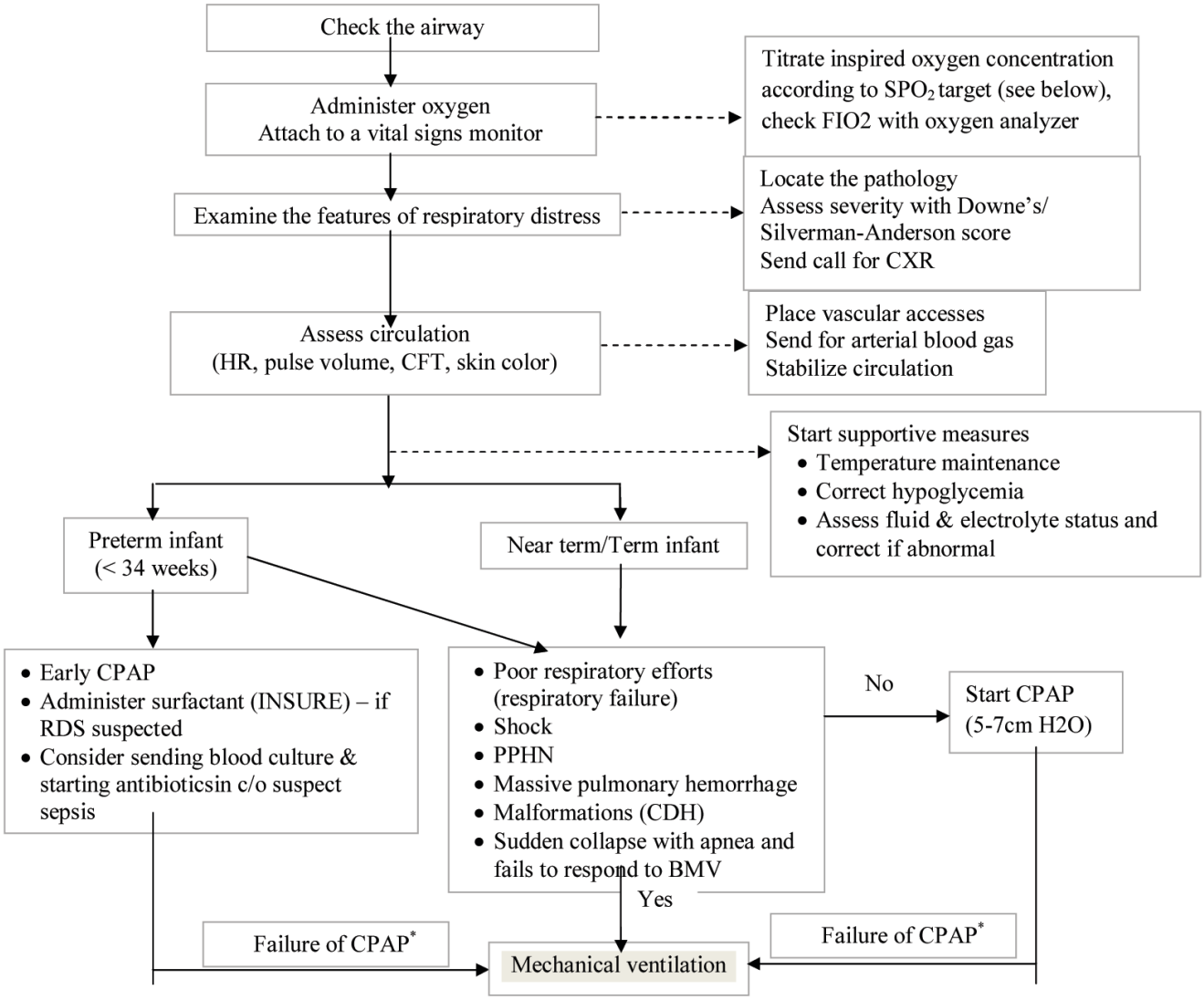
- Cardiac: Congenital heart disease, Ductusdependant lesions, PDA of prematurity, persistent pulmonary hypertension (PPHN)
- Shock due to any cause
- Surgical causes: Choanal atresia, tracheoesophageal fistula, diaphragmatic hernia
- Metabolic: Acidosis, hypoglycemia, polycythemia, anemia

Figure 2: Silverman-Anderson score



*A score of >6 indicates impending respiratory failure and warrants immediate respiratory support.

Figure 3: ***Algorithm for initial management of a neonate with respiratory distress***



[*Failure of CPAP (): Even on a CPAP of 7cmH_2O and 70% FiO_2if the neonate has excessive work of breathing (or) PCO_2>60mmHg with pH <7.2 (or) recurrent apnea or hypoxemia (PaO2 <50 mmHg), this should be considered as failure of CPAP].

Details of oxygen therapy and other modalities of respiratory support are described below.

References

1. NNF recommended basic perinatal-neonatal nomenclature. In: DK Guha, editors. Neonatology-Principles and Practice. 1sted. New Delhi: Jaypee Brothers, 1998:131-132.
2. National Neonatal Perinatal Data 2002-2003. http:// www.nnfi.org/images/NNPD_2002-03.pdf. (16 October 2009, date last accessed)
3. Kumar P, Kumar R, Narang A. Spectrum of neonatal respiratory distress at PGI. Bulletin NNF 2000; 14: 8-12.
4. Bhakoo O N, Narang A, Karthikeyan G, Kumar P. Spectrum of respiratory distress in very low birth weight neonates. Indian J Pediatr 2000; 67:803-804.
5. Silverman WC, Anderson DH. Controlled clinical trial on effects of water mist on obstructive respiratory signs, death rate andnecropsy findings among premature infants. Pediatrics 1956; 17: 1-4.
6. Wood DW, Downes' JJ, Locks HI. A clinical score for the diagnosis of respiratory failure. Amer J Dis Child 1972; 123: 227-9.

7. Miller JD, Carlo WA. Permissive hypercapnia in neonates. Neoreviews2007;8: e345-353
8. Okumura A, Hayakawa F, Kato T, Itomi K, Maruyama K, Ishihara N, et al. Hypocarbia in preterm infants with periventricular leukomalacia: the relation between hypocarbia and mechanical ventilation. Pediatrics 2001; 107:469-475.
9. Manroe BL, Weinberg AG, Rosenfeld CR et al. The neonatal blood count in health and disease. I. Reference values for neutrophilic cells. J Pediatr 1979; 95: 89-93.
10. Mouzinho A, Rosenfeld CR, Sanchez PJ, Risser R. Revised reference ranges for circulating neutrophils in very low birth weight neonates. Pediatrics 1994; 94:76.
11. Wood BR. Physiological principles. In: Goldsmith JP, Karotkin EH, editors. Assisted ventilation of the neonate. 4th ed. Saunders. Philadelphia, PA 2008: 15-40.
12. Bhakta KY. Respiratory distress syndrome. In: Cloherty JP, Eichenwald EC, Stark AR., editors. Manual of neonatal care. 6th ed. Lippincott Williams & Wilkins. Philadelphia, PA 2008: 323-330.
13. Roberts D, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. Cochrane Database of Systematic Reviews 2006, Issue 3. Art. No.: CD004454. DOI: 10.1002/14651858.CD004454.pub2.
14. Mathai SS, Raju CU, Kanitkar CM. Management of Respiratory Distress in the Newborn. MJAFI 2007; 63: 269-272.
15. Soll RF, Morley CJ. Prophylactic versus selective use of surfactant in preventing morbidity and mortality in preterm infants. Cochrane Database of Systematic Reviews 2001, Issue 2. Art. No.: CD000510. DOI: 10.1002/14651858.CD000510.
16. Lacaze-Masmonteil T. Expanded use of surfactant therapy in newborns. ClinPerinatol 2007; 34: 179–189
17. Ambalavanan N, Carlo WA. Ventilatory strategies in the prevention and management of Bronchopulmonary Dysplasia. SeminPerinatol 2006; 30: 192-199.
18. Ho JJ, Henderson-Smart DJ, Davis PG. Early versus delayed initiation of continuous distending pressure for respiratory distress syndrome in preterm infants. Cochrane Database Syst Rev. 2002;(2):CD002975.
19. Buckmaster AG, Arnolda G, Wright IM, Foster JP, Henderson-Smart DJ. Continuous positive airway pressure therapy for infants with respiratory distress in non tertiary care centers: a randomized, controlled trial. Pediatrics 2007;120:509-18
20. Lemyre B, Davis PG, De Paoli AG. Nasal intermittent positive pressure ventilation (NIPPV) versus nasal continuous positive airway pressure (CPAP) for preterm neonates after extubation (Cochrane review).Cochrane Database Syst Rev. 2001; (3):CD003212.
21. Lemyre B, Davis PG, De Paoli AG. Nasal intermittent positive pressure ventilation (NIPPV) versus nasal continuous positive airway pressure (CPAP) for apnea of prematurity (Cochrane review). Cochrane Database Syst Rev. 2002;(1):CD002272. Review.
22. Kishore MSS, Dutta S, Kumar P. Early nasal intermittent positive pressure ventilation versus continuous positive airway pressure for respiratory distress syndrome. ActaPaediatr 2009; 98: 1412-1415.
23. Greenspan JS, Shaffer TH. Ventilator-induced airway injury: a critical consideration during mechanical ventilation of the infant. Neonatal Netw 2006; 25: 159–166.
24. Hutchison AA, Bignall S. Non-invasive positive pressure ventilation in the preterm neonate: reducing endotrauma and the incidence of bronchopulmonary dysplasia. Arch Dis Child Fetal Neonatal Ed 2008; 93: F64 – 68.
25. Goldsmith JP, Karotkin EH. Preface. In: Goldsmith JP, Karotkin EH, editors. Assisted ventilation of the neonate. 4th ed. Saunders. Philadelphia, PA 2008: 149-170.
26. Spitzer AR,Greenspan JS, Fox WW. Positive-pressure ventilation: pressure-limited and time cycled ventilation. In: Goldsmith JP, Karotkin EH, editors. Assisted ventilation of the neonate. 4th ed. Saunders. Philadelphia, PA 2008: 149-170.