Management of Respiratory Distress in the Newborn

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Introduction

Respiratory distress is a common emergency responsible for 30-40% of admissions in the neonatal period [1]. A working diagnosis should be made in the first few minutes of seeing the baby and immediate life-saving measures should be undertaken till further management plans are drawn up.

Respiratory distress in the neonate is diagnosed when one or more of the following is present; tachypnoea or respiratory rate of more than 60/minute, retractions or increased chest in drawings on respirations (subcostal, intercostal, sternal, suprasternal) and noisy respiration in the form of a grunt, stridor or wheeze[1]. The distress may or may not be associated with cyanosis and desaturation on pulse oximetry. The common causes of respiratory distress in the neonate are:

1. Hyaline Membrane Disease (HMD)
2. Meconium Aspiration Syndrome (MAS)
3. Transient Tachypnoea of the Newborn (TTNB)
4. Congenital or acquired pneumonia
5. Persistent Pulmonary Hypertension of the Newborn (PPHN)
6. Air leaks
7. Congenital anomalies of upper airway (choanal atresia), gut (tracheoesophageal fistula, congenital diaphragmatic hernia) or lungs (lobar emphysema, congenital cystic adenomatoid malformation, cysts)
8. Cardiac shock or Congenital Heart Disease (CHD).
9. Haematological causes (severe anaemia, polycythaemia)
10. Neurological causes leading to hyperventilation like seizures
11. Metabolic causes- Inborn Errors of Metabolism (IEM)

Pathophysiology Unique to Newborn

Prolonged and unattended distress leads to hypoxaemia, hypercarbia and acidosis. These causes lead to pulmonary vasoconstriction and persistence of foetal circulation with right to left shunting through the ductus and foramen ovale, thereby aggravating hypoxaemia which leads to multi system organ dysfunction.

An audible grunt (forced expiratory sound) is an important sign of pulmonary pathology in the newborn indicating that the baby has a low lung volume or functional residual capacity (FRC). Breathing against a partially closed glottis increases the FRC of the baby and helps keep the alveoli patent. This is characteristically seen in a baby with HMD 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 rather than improve his condition. Hence any baby who is grunting should either be given continuous positive airway pressure (CPAP) or intubated and put on ventilator support, but never left to breathe spontaneously with a tube in situ.

Grading of Distress Severity

The severity of respiratory distress is assessed by Silverman- Anderson Score and Downes' Score. While the Silverman Anderson Retraction Score is more suited for preterms with HMD, the Downes' Score is more comprehensive and can be applied to any gestational age and condition. Scoring should be done at half hourly intervals and a chart maintained to determine progress (Tables 1,2). A progressively increasing FiO₂ requirement to maintain a saturation of 90-92% in a preterm and 94-96% in a term baby is also a sensitive indicator of the severity and progress of distress.

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Table 1**Silverman Anderson retraction score [2]**

Score	Upper chest retraction	Lower chest retraction	Xiphoid retraction	Nasal dilatation	Grunt
0	Synch	None	None	None	None
1	Lag on inspiration	Just visible	Just visible	Minimal	Stethoscope only
2	See-Saw	Marked	Marked	Marked	Naked ear

A score of >6 is indicative of impending respiratory failure.

Table 2**Downes' score [3]**

Score	Respiratory rate	Cyanosis	Air entry	Grunt	Retraction
0	<60/min	Nil	Normal	None	Nil
1	60-80/min	In room air	Mild ?	Ausc with stethoscope	Mild
2	>80/min	In $\geq 40\%$	Marked ?	Audible with naked ear	Moderate

A score of >6 is indicative of impending respiratory failure.

Besides assessing the severity of the distress it is essential to determine the underlying pathology for further management. For a new born baby (within few hours of birth) with respiratory distress, a quick review of the following antenatal and peripartum events including the condition at birth is a must :-

- Were there any risk factors in the antepartum period or evidence of foetal distress prior to delivery? (Birth asphyxia or PPHN)
- Did the mother receive antenatal steroids if it was a preterm delivery? (Antenatal steroids decrease the incidence of HMD by 50%)
- Was there a history of premature rupture of membranes and fever? (congenital pneumonia or sepsis)
- Was there meconium stained amniotic fluid? (MAS is a possibility)
- A look at the antenatal ultrasonography (USG) for the amount of amniotic fluid would tell us the status of the foetal lung. (congenital anomalies of lung)
- Was resuscitation required at birth? (resuscitation trauma/PPHN/ acidosis)
- Did the distress appear immediately or a few hours after birth? (HMD appears earlier than pneumonia)
- Was it related to feeding or frothing at the mouth? (tracheo-esophageal fistula or aspiration)
- Does the distress decrease with crying? (choanal atresia).

For babies presenting later with distress we have to ask a few other questions :-

- Is the distress associated with feed refusal and lethargy? (sepsis, pneumonia)
- Did the distress appear slowly after starting feeds? (IEM).
- Is there a family history of early neonatal deaths? (CHD, IEM).

The algorithm shown in Fig.1, helps in reaching a working aetiological diagnosis [4].

Clinical Examination

Clues to the likely aetiology can be picked up on examination of the neonate

- A preterm baby weighing <1500 gms with retractions and grunt is likely to have HMD.
- A term baby born through meconium stained amniotic fluid with an increase in the anterior-posterior diameter of the chest (full chest) is likely to be suffering from MAS.
- A depressed baby with poor circulation is likely to have neonatal sepsis with or without congenital pneumonia.
- A near term baby with no risk factors and mild distress may have TTNB.
- An asphyxiated baby may have PPHN.
- A growth retarded baby with a plethoric look may have polycythaemia.
- A baby with respiratory distress should be checked for an air leak by placing a cold light source over the chest wall in a darkened room.
- A baby presenting with tachypnoea and a cardiac murmur may have a congenital heart disease.
- Inability to pass an 5F catheter through the nostril of a term baby is suggestive of choanal atresia.

Investigations

Essential investigations for all cases of neonatal

Fig. 1 : Diagnostic approach to respiratory distress

respiratory distress include chest radiograph with an orogastric tube in situ, arterial blood gas (ABG) analysis (Table 3), sepsis screen including C-reactive protein, μ ESR, white blood cell count, peripheral smear for toxic granules, blood culture, surface swab culture (where indicated), maternal vaginal swab, blood glucose, serum calcium and central haematocrit assessment.

A score of 3 or more on the ABG indicates the need for CPAP or mechanical ventilation. A pH of <7.2 with hypercarbia ($p\text{CO}_2 > 60\text{mm}$) or a $p\text{O}_2 < 50\text{mm}$ Hg in FiO_2 of 0.8 is suggestive of frank respiratory failure.

Treatment

1. Clearing of airway, ensuring adequate breathing and circulation are the first line of management. A baby in obvious respiratory distress needs to be on continuous pulse oximeter monitoring to decide when intubation and ventilation is required.
2. Warm, humidified oxygen is given with a head box, preferably with a FiO_2 meter and pulse oximeter monitoring to determine the amount of oxygen required. Soft nasal cannulae may also be used to give oxygen. Small changes in FiO_2 are made and monitored on the pulse oximeter. Oxygen should be given in the correct dose, as it is toxic to preterm neonates and the suggested guidelines are given in Table 4. The "30-60-90" rule is a useful bedside indicator when using the pulse oximeter. This means that at a saturation of 90% the paO_2 is around 60mmHg and at 60% it is around 30 mmHg in a newborn. This is due to high HbF in neonatal blood which causes the left shift of oxygen dissociation curve. At saturation of 90-95%, the paO_2 may be between 60 to 98mm Hg and above 95% saturation, paO_2 is well above 100mmHg.
3. Maintenance of correct temperature is essential. HMD and PPHN are aggravated by hypothermia.

Table 3
ABG score

	Points			
	0	1	2	3
paO_2 mmHg	> 60	50-60	< 50	< 50
pH	> 7.3	7.20-7.29	7.1-7.19	< 7.1
paCO_2 mmHg	< 50	50-60	61-70	> 70

Score of > 3 suggestive of ventilatory support requirement

Table 4
Guidelines for monitoring oxygen saturation levels by pulse oximetry

$>95\%$	Term baby, pulmonary hypertension (PPHN)
88-94%	28-34 weeks preterm
85-92%	Below 28 weeks gestational age

4. Fluid and electrolyte management: Electrolyte balance, fluids, calcium and glucose homeostasis are all equally important. Fluids are usually started at a minimum of 60ml/kg/day of 10% D or three fourth of daily maintenance whichever is more. This will ensure a glucose infusion rate of about 4 mg/kg/min which is the minimum required for adequate glucose homeostasis. Calcium in the dose of 6-8 ml/kg/day of calcium gluconate should be added to the fluid in all preterms and term babies.
5. Maintenance of adequate haemoglobin: Any neonate with respiratory distress should have a packed cell volume (PCV) above 40% (but less than 75%).
6. All preterm babies with respiratory distress should be started on broad spectrum antibiotics. In term babies, decision to start antibiotics would depend on the clinical situation, but the threshold should be low.

Role of Surfactant

Surfactant is the drug of choice in a baby with HMD. This may be given either prophylactically if the baby is less than 28 weeks of gestation or within the first two hours of onset of symptoms in older babies [7,8]. Prophylactic surfactant is given in the labour room after the baby has been stabilized. Rescue therapy is most effective if given within the first two hours of birth. Presently both natural and synthetic surfactants are being marketed in India. Surfactant is given in a dose of 100 mg/kg through the endotracheal tube in small aliquots with intermittent bagging to prevent desaturation during administration and it should be followed by ventilatory support.

Respiratory Support

Respiratory support is given in the form of continuous positive airway pressure (CPAP) or intermittent mandatory ventilation (IMV). Short nasal or longer nasopharyngeal prongs are preferred to endotracheal CPAP as latter markedly increases the work of breathing and tires the infant. CPAP should be started early in a preterm with HMD. Indications for starting CPAP are a Downes' or Silvermann score of >6 at birth or a FiO_2 requirement of >0.4 to maintain an acceptable saturation on pulse oximeter. ABG score of more than 3 is also acceptable. CPAP is a gentler form of non-invasive ventilatory support as compared to IMV [6].

IMV: Time cycled pressure limited ventilation is the modality of choice for ventilation of a neonate in respiratory failure. If patient triggered ventilation is used it is given as synchronized intermittent mandatory ventilation (SIMV) or assist control mode ventilation (ACMV). For best outcomes this should be given to babies in impending respiratory failure or failed CPAP

rather than in complete respiratory failure [5]. Resistant apnoea is also an absolute indication. CPAP is said to have failed when the FiO_2 requirement is >0.6 or the pressure required to maintain oxygenation exceeds 7-8 cm of H_2O . Respiratory failure is defined a $\text{paCO}_2 > 60\text{mm}$ or $\text{paO}_2 < 50\text{ mm}$ or saturation $< 85\%$ in 100% O_2 with or without a pH of < 7.25 . A working algorithm for ventilatory support is given in Fig. 2.

Outcome

With good intensive care in a neonatal intensive care unit the outcome of neonates with respiratory distress has improved remarkably in the past decade with a survival rate of $> 60\%$ in babies weighing $> 1\text{ kg}$ [9].

Conflicts of Interest

None identified

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Fig. 2 : Algorithm for ventilatory support