

Respiratory support in babies with Respiratory distress syndrome

SUSHMA NANGIA

Department of Pediatrics, Kalawati Saran Children's Hospital and Lady Hardinge Medical College, New Delhi

Abstract: Respiratory distress is the commonest cause of admission in neonatal intensive care units. Respiratory support in form of oxygen therapy, CPAP, and ventilation has improved the survival in these patients. CPAP has become more popular in such cases and more emphasis is on early, nasal CPAP. Mechanical ventilation also has become less invasive with concepts of minimal handling, permissive hypercapnia, patient triggered ventilation taking edge over the old concepts. Weaning from ventilation is a difficult but most important art to learn. Careful monitoring is very important for a baby on mechanical ventilation. The survival depends on the disease entity for which baby is ventilated rather than ventilation per se.

Introduction

Respiratory distress syndrome is an acute illness usually seen in preterm infants characterized by respiratory rate > 60/min, dyspnea (intercostals, subcostal indrawing, sternal retraction) with a predominantly diaphragmatic breathing pattern and characteristic expiratory grunt or moan, all presenting within four to six hours of delivery. Pathophysiologically the condition is characterized by noncompliant (stiff) lungs, which contain less surfactant than normal and become atelectatic at end-expiration. RDS is a developmental disorder rather than disease per se and it is usually associated with premature birth. Incidence and severity of RDS generally increases with decreasing gestational age and is worse in male infants i.e. 50% infants 26-28 weeks and 20-30% of premature infants 30-31 weeks, develop RDS.

Pathophysiology

The principle mechanism of hypoxemia in HMD is venous admixture resulting from both intrapulmonary shunting and right to left shunting across the foramen ovale and PDA. *Venous admixture* (VA) an expression of the percent of this mixture made up of venous blood, can be derived from the standard shunt equation :-

$$VA(\%) = \frac{Cc'O_2 - CaO_2}{Cco_2 - CvO_2} \times 100$$

Where $Cc'O_2$ is the end pulmonary capillary blood oxygen content, CaO_2 the arterial blood oxygen content, and CvO_2 the mixed venous blood oxygen content. When the venous admixture is less than 40% an adequate arterial PO_2 can be attained by increasing FIO_2 . When venous admixture is 50% or more, increasing FIO_2 from room air to 1 changes arterial PO_2 a few mmHg, but adequate oxygenation is not achieved. There is a range of venous admixture for which oxygen therapy alone will result in adequate arterial oxygenation but with severe venous admixture arterial hypoxemia cannot be overcome even with 100% oxygen and adequate alveolar ventilation.

Intrapulmonary shunting

Within the lung some venous admixture occurs because of pulmonary blood flow through pulmonary arteriovenous anastomoses (bronchial vein-pulmonary vein communication) and

due to pulmonary capillaries perfusing areas of the immature lung where terminal airway formation is incomplete. However the contribution of these anatomic shunts to total intrapulmonary venous admixture is small.

In *hyaline membrane disease* (HMD), intrapulmonary venous admixture occurs largely as a result of the perfusion of terminal airways that are partially collapsed, collapsed, and fluid filled. For the most part, these terminal airways are either extremely under ventilated or non ventilated at all as a result of epithelial slough, exudation, and hyaline membrane formation at the junction of the respiratory bronchioles and alveolar ducts. The effect of this pathological process on terminal airway collapse is compounded by the associated surfactant deficiency, whether primary or secondary in origin. Based on this, HMD can be divided into three compartments. First compartment is made up of terminal gas-exchange units that are intact and adequately ventilated and perfused. The *second* compartment is characterized by terminal airways that are open but markedly under ventilated. These units may be perfused, but only if local alveolar hypoxia can be relieved by breathing 100% oxygen. The *third* compartment is totally unventilated but perfused. It includes those units with terminal conducting airways that are severely affected by epithelial damage, exudates and slough. In addition, this compartment also represents any anatomic right to left shunt that is present.

Oxygenation of arterial blood in HMD depends up on proportion of terminal airways that are available for gas exchange. As long as 60% or more of the terminal airways units remains open and oxygenated and adequately perfused, acceptable arterial oxygen tension greater than 50mm Hg can be maintained by increasing the concentration of inspired gas.

Extrapulmonary shunting

During the first 12 hours after birth, predominant shunt is right to left from the inferior vena cava across the foramen ovale into the left atrium. Even though the ducts are widely patent there is little flow through it in either direction until 12 to 24 hours of age when a left to right shunt of increasing magnitude occurs as a result of falling pulmonary vascular resistance. Significant right to left shunting at the ductus does not occur unless pulmonary vascular resistance is markedly elevated in the presence of severe hypoxemia, hypercarbia, or acidosis or systemic vascular pressure is extremely low.

Right to left shunting through persistent fetal pathways is promoted by increased pulmonary vascular resistance which in turn may be further increased by any hypoxemia resulting from shunts. A

large shunt present due to arterial hypoxemia can be diminished by increasing the inspired oxygen concentration. This sequence which is probably triggered by a reduction in pulmonary vascular resistance, helps explain why total venous admixture in HMD decreases as inspired oxygen concentration is increased. Shunting through a PDA: The ductus is patent most cases of RDS during the first 48-72 hours.

If PAP exceeds aortic pressure there will be a significant right-left shunt. Right-left shunts at ductal level are common in persistent pulmonary hypertension of the newborn, but in uncomplicated RDS these are small and constitute less than 10% of the total right to left shunt. One clinically important fact of right to left ductal shunting is the blood drawn from an umbilical artery catheter can have a much lower PaO₂ than blood passing up the carotid arteries to the eyes. Colour Doppler studies of the vascular channels in babies with RDS have demonstrated that intra vascular shunting at ductal or foramen ovale level is relatively unusual in uncomplicated RDS (as opposed to PPHN), even though both channels stay potentially patent in the early neonatal period. In fact, the shunt through the channel is predominantly bidirectional or left to right in the first few days of life. This will have little effect on blood gas values, but will increase the cardiac output and the load of the right ventricle. Obligatory shunts caused by drainage of veins of the myocardium directly in to the left side of the heart and also by anastomoses between the bronchial and pulmonary circulation are present in every individual, are of no hemodynamic or clinical significance.

Lung Function

Functional Residual Capacity (FRC) Functional residual capacity is decreased in HMD. In newborn infants without lung disease, FRC is approximately 30ml/kg. Loss of FRC occurs in HMD as a result of surfactant deficiency and displacement of gas volume by pulmonary vascular congestion, interstitial edema, and airway flooding with proteinaceous fluid. In HMD, FRC can be as low as 3ml/kg. FRC can be recovered and protected by the addition of distending airway pressure in the form of continuous positive pressure or intermittent mandatory ventilation. Change in FRC mirrors the improvement in oxygenation that occurs with the addition of distending airway pressure or without spontaneous recovery beginning on the second or third day after birth. During recovery, there is a striking correlation between improving oxygenation, increasing FRC and diuresis as excess lung water reabsorbs into the circulation and is cleared by the kidneys.

Mechanical Properties of the Lung

The marked decrease in lung compliance, one of the hallmarks of HMD, occurs as a consequence of two factors, these are 1) A decrease in the number of ventilated terminal air spaces and 2) An increase in the recoil pressure of ventilated terminal air spaces. In addition, dynamic but not static, compliance is further reduced in HMD owing to change in the viscoelastic properties of lung tissue and the presence of inhomogeneity of ventilation. Like FRC, compliance also mirrors the course of pulmonary insufficiency in HMD. Intercostal and sternal retractions in the infants capable of the spontaneous ventilatory efforts provide a visual assessment of compliance at bed side. As compliance improves either as a consequence of manipulation of ventilation, or surfactant replacement therapy, or with evolution of natural course of disease, chest wall retraction diminishes, indicating that less transpulmonary

pressure is required to expand lung.

Lung resistance, which is the sum of airway resistance and lung tissue resistance, is three to six times greater in infants with HMD than in normal newborn infants. Pressure volume loops on lungs excised at post mortem from babies dying of HMD have a characteristic pattern. During inflation the volume change for a given increase in pressure is very small and during deflation the change in volume follows a tract almost similar to that seen during inflation, where as in the normal lung air is retained until low volume are reached. Furthermore, as the pressure drops to zero, very little or no air is retained within the surfactant less alveoli, corresponding to the very small FRC measured in vivo. An inevitable sequel of the abnormal lung mechanics is that the work of breathing is increased in neonates with RDS.

Effect of Alveolar Instability on Lung Volume and Compliance

Alveolar instability refers to the tendency for an alveolus to switch abruptly between the inflated state and the collapsed state. When pressure across the alveolus exceeds the critical opening pressure, an unstable alveolus inflates suddenly. During deflation, an unstable alveolus collapses abruptly when trans-alveolar pressure falls below the critical closing pressure. One compartment might remain collapsed throughout inspiration and expiration, another might be ventilated during inspiration but collapsed during expiration and yet another might be ventilated and stable to the extent that gas volume is retained at end expiration. The distribution of these compartments is affected by a variety of factors, especially treatment with surfactant replacement or with distending airway pressure with CPAP.

General Management

Minimal Handling : When hypoxic babies are disturbed and handled their respiration may become very irregular or stop altogether, their right left shunts increase and their PaO₂ falls rapidly. Even listening to the chest with a stethoscope or palpating the abdomen, may have this effect. Major disturbances such as sucking out an endotracheal tube, performing a lumbar puncture or taking a chest x-ray can cause catastrophic falls in PaO₂.

Physiotherapy : In the non-intubated baby with RDS secretions are not a problem unless infection develops, and physiotherapy is contraindicated as it opposes the principle of minimal handling.

Posture : Babies with RDS should have their position changed every 4-6 hours. In general, prone position is preferable as the blood gases tend to be better maintained with much improved lung function.

Temperature : The baby's thermal environment should be controlled if the baby is exposed during a procedure, he should be under a radiant heat source. As far as possible the baby should be kept covered to minimize heat loss.

Blood Gas Management PaO₂ : Maintain it in the range 8-12kPa (60-90mmHg) The upper limit set to avoid hyperoxaemia which may predispose the baby to ROP. PaCO₂ in normal newborn babies the normal range is 4.6-5.4kPa (35-40mmHg). If a VLBW baby in the first 6-12 hours cannot ventilate himself sufficiently well to keep his PaCO₂ below 6kPa (45mmHg), he should be ventilated. **pH:** Acidemia is common in neonates with RDS which may be of respiratory or metabolic origin. Commonest cause of

metabolic acidemia is a raised lactate from anaerobic metabolism. This may be secondary to hypoxemia, hypotension, anemia, infection, sepsis or strenuous muscle activity. Acidemia inhibits surfactant synthesis and increases pulmonary resistance. Once pH falls below 7.15 other physiological functions such as myocardial contractility and diaphragmatic activity begin to deteriorate. In all VLBW infants, pH should be kept >7.25 with a base deficit $<10\text{mmol/L}$.

Blood Pressure : In all neonates with RDS, mean BP should be maintained at around gestational age in weeks + 5mmHg and the mean BP should not be allowed to fall below their gestational age in weeks (i.e. for a 33 weeks neonate mean BP should be maintained at around 38-40 mmHg and should not be lower than 33mmHg).

Maintenance of Haemoglobin : Preterm neonate can be anemic because of an intrapartum hemorrhage, defective placental transfusion, or a twin-twin, or fetal maternal hemorrhage. Blood loss after birth is mostly iatrogenic as a consequence of sampling, but a sudden drop in the hematocrit/hemoglobin level in a baby with RDS is highly suggestive of development of an IVH. III neonates, in particular those who are premature, tolerate hemoglobin levels $<13\text{gm/dl}$ (PCV $<40\%$) badly, because of the increase in cardiac output required to meet the oxygen demands of the tissues. All hemodynamically unstable neonates with hemoglobin $<13\text{gm/dl}$ (PCV $<40\%$) should be transfused in order to maintain their hemoglobin and prevent cardiopulmonary decompensation.

Fluid and Electrolyte : Infants with RDS should be started on 40-60ml/kg/24 hour of a 10% dextrose solution. Fluid intake should subsequently be guided by the baby's weight, serum sodium, urine specific gravity and serum osmolality. The ill neonate loses 1-3% of his body weight per day. If he is losing more than desired then he may be dehydrated due to excessive insensible losses, if his weight is static or he is gaining weight, he may have difficulty handling the excess fluid which must be curtailed. Characteristically, a diuresis occurs when the baby's lung function improves concomitant with an improvement in FRC and compliance. When this occurs it is a marker of improvement and now the previous constraints on the fluid balance need to be relaxed to prevent dehydration, hemoconcentration and jaundice.

Oxygen Therapy : The primary challenge in managing the pulmonary insufficiency of HMD is achieving an adequate PO_2 . If oxygenation can be achieved, carbon dioxide removal is only a matter of increasing minute ventilation and to make up for an inadequate alveolar portion of tidal volume. Oxygen can be administered by a head box with a starting FiO_2 of 0.4 which can be increased to 0.6 as judged by the sPO_2 . The sPO_2 should be maintained between 92 ± 3 percent. If the need for FiO_2 is increasing (in an attempt to maintain SpO_2), or if the baby is developing retractions, then the baby needs to be taken up for CPAP therapy. If the neonate is accumulating CO_2 as evidenced by a blood gas then the baby needs to be taken up after mechanical ventilation. A trial of CPAP may be justified before mechanical ventilation.

CPAP : CPAP should be viewed as the vital link between oxygen therapy and positive pressure ventilation. Gregory et al used this modality in newborns with RDS in 1971 using head box, and later using endotracheal tube. It was in 1973 that Agostino et al reported the first small series of infants with RDS treated with Nasal CPAP. The superiority of nasal breathers and thus they spontaneously form a seal between the palate and the tongue. Any preterm with respiratory distress soon after birth should be put on

continuous positive airway pressure (CPAP). Numerous studies have shown the fact that early use of CPAP reduces the need for subsequent intubation and mechanical ventilation in RDS. In those who require it later, ventilation is successful at lower pressure. According to a Cochrane review, early use of CPAP (at onset of respiratory distress) was associated with decreased need for intermittent positive pressure ventilation (IPPV) by about 50% when compared to late initiation of CPAP i.e. when FIO_2 requirement of baby is more than 60%. This is clinically important, as IPPV is associated with considerable increase in complications and cost to the family. Most babies respond to oxygen therapy and CPAP. According to the Cochrane review, use of CPAP was associated with lower rates of failed treatment by about 30%, overall mortality by 50% and mortality in infants with birth weight above 1500 grams by as much as 75%. Early use of CPAP will be low-cost, simple and noninvasive option for a country like India, where most places cannot provide invasive ventilation.

Physiologic effects of CPAP

The physiologic effects of CPAP vary depending on the underlying condition of the lung. CPAP is believed to result in progressive alveolar recruitment, inflating collapsed alveoli and reducing intrapulmonary shunt. Some of the effects of CPAP like increase in gas volume in the lung and increased functional residual capacity have been measured. Initially as the FRC increases, gas exchange improves; PaO_2 increases and PaCO_2 decreases. Improved oxygenation relieves the hypoxic vasoconstriction in the pulmonary vascular bed and decreases pulmonary vascular resistance leading to increased pulmonary blood flow, decreased shunting and increased PaO_2 . In addition, CPAP has some non-specific beneficial effects on neonatal ventilation in terms of more regular breathing pattern in neonates. This is achieved by chest wall stabilization and reduction of thoracic distortion. CPAP also splints the airways and the diaphragm. It reduces obstructive apnea, increases both inspiratory and expiratory times and helps in enhanced surfactant release.

Optimal CPAP improves ventilation but excessive CPAP leads to over-distension of lung resulting in high PaO_2 along with a high PaCO_2 due to diminished tidal volume. If high CPAP continues for a considerable period of time, this can lead to serious consequences like air leaks. Excessive CPAP also leads to increased dead space ventilation leading to a rise in PaCO_2 .

Methods of applying CPAP include : (i) face mask (ii) face chamber (iii) nasal prongs (iv) nasopharyngeal tube and (v) gregory box.

Indications of CPAP : (i) oxygen concentration $>60\%$ to keep $\text{PaO}_2 > 8\text{kPa}$ (60mmHg). (ii) Recurrent apneic attacks. (iii) Weaning from IPPV.

Newer CPAP Systems : Bubble CPAP is CPAP delivered by CPAP system with underwater seal. It has been shown that CPAP delivered by underwater seal causes vibration of the chest due to gas flow under water, which is transmitted to infant's airway. These vibrations simulate waveforms produced by high frequency ventilation. Bubble CPAP has also been shown to reduce need for intubation and mechanical ventilation, as well as use of postnatal steroids and trend towards decreased incidence of chronic lung disease. Another new mode of CPAP delivery is use of dual flow CPAP, or Infant Flow Drive CPAP. Mazzella et al have shown superiority of IFD over nasal CPAP in terms of decreased oxygen requirement and respiratory rates and lesser need for mechanical ventilation. Babies who failed nasal CPAP could be rescued by

IFD and mechanical ventilation could be avoided.

There is evidence to prove that giving surfactant to the patient after brief intubation can enhance beneficial effect of early CPAP in preterms. In this approach, the preterm is started on CPAP as soon as he develops respiratory distress. When respiratory distress on CPAP progresses beyond a predetermined point (ratio of arterial to alveolar oxygen tension (a/A) of less than 0.36, the baby is intubated, given surfactant, and then extubated and put back on CPAP again. This minimizes the hazards of mechanical ventilation in the baby. Surfactant and CPAP act in conjunction in fulfilling the aim of increasing lung volume and the functional residual capacity.

Mechanical ventilation

The decision to start mechanical ventilation in a neonate should be individualized and based on clinical as well as blood gas parameters. Though there are guidelines for initiating mechanical ventilation, the best guide is the evolving clinical state of the neonate.

Indications : (1) PaCO₂ greater than 50mmHg or rapidly rising. (2) PaO₂ less than 50mmHg or oxygen saturation less than 90% with FiO₂ above 0.6 with adequate trial of CPAP. (3) pH less than 7.25. (4) Intractable apneic spells (5) Impending or existing shock. (6) RR > 70 with moderate to severe retractions or cyanosis in FiO₂>0.4. (7) CPAP failure.

Ventilator : A continuous - flow, pressure limited, timed-cycled Ventilator is useful for ventilating newborn babies with HMD. High frequency oscillatory ventilation may be useful to minimize lung injury in very small and/or sick infants and to manage infants to whom air leak syndromes complicate RDS.

Initial Steps & Setting

The basic steps of mechanical ventilation initiation include :

- 1) **Endotracheal Intubation**
- 2) **Bag & tube ventilation using a manometer to access the initial pressure requirement.**
- 3) **Start ventilator, Ensure air oxygen mixture is warmed to 37°C & humidified 70-100%.**
- 4) **Selection of appropriate ventilator settings.**
- 5) **Evaluation to check adequacy of ventilatory support.**
- 6) **Initial Settings :**

a. FiO ₂	0.5
b. Rate	40-50/min
c. PIP	18-20 cm H ₂ O
d. PEEP	4-5 cm H ₂ O
e. Ti 0.4-0.5sec	
f. Flow	5-7 lit/min

The rationale for the initial settings is primarily physiologic, which means that the rate of 40-50 is chosen so as to make the baby breathe at a rate at which he would breathe, if he was breathing on his own. Similarly the inspiratory time of 0.5 seconds is also physiologic; a baby normally breathes at a rate of 40 breaths per minute, i.e. 40 breaths in 60 seconds with a normal I:E ratio of 1:2 and thus take some breath every one and a half second with inspiration lasting for 0.5 sec and exhalation lasting for 1.0 seconds. Derivation of PIP is based on the fact that a normal breath necessitates a drop of intrapleural pressure by 6 cm of water from -1 to -7 cm of water. In addition a pressure of 6 cm water is required to drive the gases through the ventilatory circuitry. Any baby on ppV has to be provided with a PIP greater than 12cm water so as to drive the gases into his lungs. This additional pressure would depend on the size of the baby and the status of the diseased lung. Pressures required would be higher for a large baby as well as a baby with severely diseased lungs.

Normal physiologic PEEP of approximately 3cm water is maintained by closure of glottis in a normal baby, this has been abolished as a consequence of intubation in a ventilated baby and hence one need to intermediate concentration as most babies who are being ventilated have been on an FiO₂ of 0.4-0.6 on hood/CPAP. This 0.5 can be adjusted subsequently based on Spo₂ and PaO₂ to achieve optimum oxygenation.

- 7) **Observe infant for absence of cyanosis & retractions and adequate chest wall movement and breath sounds**
- 8) **If chest wall movement is subnormal increase PIP by 1cm every few minutes till chest wall movement is adequate and SpO₂ is within normal range.**
- 9) **If oxygenation is inadequate as evidenced by presence of cyanosis or a subnormal SpO₂, increase FiO₂ by 0.05 every few minutes till saturation is between 92±3 percent**
- 10) **Draw an arterial blood gas sample and review the settings based on the report.**
- 11) **Subsequent clinical monitoring includes observing for color, chest wall motion along with respiratory effort, and adequacy breath sounds**

Monitoring adequacy of Ventilatory Therapy

The adequacy of ventilatory therapy is assessed by a combination of clinical parameters along with non-invasive adjuncts like pulse oximetry and capnography and a judicious use of invasive modality like the blood gases. The clinical parameters include absence of cyanosis, absence of retractions, a prompt capillary refill time of less than 3 seconds, a normal blood pressure, adequate chest expansion (easy chest rise) and an adequate air entry.

The pulse oximetry limits are set so as to avoid hypoxia as well as hyperoxia and the safe limit is 92±3% Capnography is a noninvasive estimate of the end tidal carbon dioxide which correlates well with the PaCO₂ under most situations. The blood gases should be maintained within normal range with the PaO₂ at 60-80mmHg, PaCO₂ at 35-45 mmHg and the PH at 7.35-7.45. The capillary refill time should be assessed at the sternum or the forehead with pressure by the index finger for a period of 5 seconds followed by removal of the finger. On removal the area under the finger appears blanched, the observer assesses the time taken for the colour to return, it should take less than 3 seconds.

Adjustments

Common Problems and their solutions :

1) **Metabolic acidosis :** A neonate with RDS is likely to become acidotic during the course of the illness as a consequence of either (a) Hypovolemia &/or shock, (b) Excessive work of breathing, (c) Hypoxia and (d) Excessive PEEP. Metabolic acidosis must be avoided at all costs as the pulmonary vasculature is exquisitely sensitive to changes in PH; acidosis leads to pulmonary vasoconstriction causing hypoxia, further acidosis and right or left shunting. This vicious cycle once initiated perpetuates and thus ensuing hypoxia and acidosis affect the myocardial contractility as well as hamper many other vital cellular functions eventually leading to death. As soon as a low pH is detected, check the SpO₂, CFT and assess the work of breathing. If the baby is hypoxic improve his oxygenation, if work of breathing is increased support respiration and if PEEP is high reduce it. If non of these causes are apparent check CFT and if prolonged administer a bolus of normal saline at a dose of 10ml/kg and reassess perfusion. Metabolic acidosis may be the first indication of a poor perfusion and hence perfusion must be supported with bolus or if need be pressors.

2) *Excessive CO2 retention* : A baby with RDS may have accumulation of CO₂ as a result of a. severe RDS; b. tube block; c. increased dead space; d. Impending opening of the ductus arteriosus

Whenever the blood gas reveals an accumulation of CO₂, cut short the ET tube if it is more than 3-4 cms beyond the lips, suction the ET tube and check improvement on EtCO₂ if available. Next increase minute ventilation by increasing RR or PIP and check by repeating a gas after 20-30 minutes of ventilator setting change. If non of these problems are there, then Check the amount of fluid being infused, (Keep track of the boluses and IV pushes) if excessive, curtail the fluid as per the daily requirement.

3. *Sudden deterioration on IPPV* : a. pneumothorax b. IVH.

4. *Gradual deterioration on IPPV* : (a) Sepsis (b) Blocked tube (c) ICH (d) PDA (e) Anemia (f) Hypotension (g) Hypoglycemia etc.

Weaning

Weaning is a very delicate process and needs to be done with utmost care and precision. The process of weaning is begun once the lung condition has shown improvement i.e. the blood gases and the baby both have remained stable at a particular set of ventilatory settings for a period of 8-12 hours. In addition, as per the natural history of the disease under consideration, the phase of resolution or improvement should generally coincide with the timing of initiation of the weaning process.

The *first setting* to be reduced is the PIP (by 1 cm decrements) to 25cm H₂O. The next parameter of concern is the FiO₂ and hence this must be reduced next. PIP and FiO₂ (by 0.05 decrements) are reduced alternately till a comparatively safe level of 20cm H₂O and 0.6 respectively is attained.

Next FiO₂ and PEEP are reduced hand in hand (similar to their increments) and reduction of rate is begun (2 breaths at a time) along with successive decrements of PIP keeping a close vigil on the PaCO₂ values.

Rate is the last parameter to be weaned, as the rate is reduced one must watch for increased work of breathing, metabolic acidosis and fatigue which would be bothersome if the rate is decreased prematurely or at a fast pace. As the rate is reduced the Ti is also reduced in an attempt to provide prolonged expiratory phases wherein the baby can get ample opportunity to breathe on his own and takeover most of the function of breathing.

Once *minimal settings* have been reached which are defined as : (i) FiO₂ < 0.4, (ii) PIP 13-14, (iii) PEEP 3, (iv) Ti 0.3 and (v) Rate 10.

The baby can be extubated and provided oxygen by hood at a FiO₂ of 0.45. Aminophylline should be started 24 hours prior to expected extubation. A blood gas 30min after extubation must be done to assess the gas parameters. The baby should be carefully monitored clinically for signs of increased work of breathing, fatigue or exhaustion. An x-ray of the chest should be obtained about 6 hrs post extubation and at least once more thereafter to rule out post extubation collapse.

Though the newer modes of ventilation like synchronized intermittent mechanical ventilation (SIMV), patient triggered ventilation (PTV) and high frequency ventilation (HFV) are in vogue, the evidence does not show their superiority over CMV, except for shorter duration of ventilation on using SIMV. A recent multicentric trial by Johnston et al. did not reveal superiority of

Desired blood gas status and the possible changes in ventilator status

Ventilator	Settings				
Desired status	Rate	PIP	PEEP	Ti	FiO₂
Increase PaCO₂	Decrease	Decrease			
Decrease PaCO₂	Increase	Increase			
Increase PaO₂		Increase	Increase	Increase*	Increase
Decrease PaO₂		Decrease	Decrease	Decrease*	Decrease

Ventilator settings based on blood gas status

Gas status	Ventilator changes required
PaO₂ volume	PaCO₂ Increase PIP which will increase MAP and increase tidal volume
PaO₂	N PaCO₂ FiO ₂
NPaO₂	PaCO₂ Rate, PEEP and keep MAP constant
NPaO₂	PaCO₂ Rate and keep MAP constant
PaO₂	PaCO₂ PEEP, Ti and Rate
PaO₂	N PaCO₂ FiO ₂ and MAP
PaO₂	PaCO₂ PIP, Rate & FiO ₂
PaO₂	PaCO₂ Consider possibility of Over ventilation, Sepsis of PPHN. FiO ₂ , or MAP or use vasodilators

HFV over conventional ventilation in preterms with RDS (23).

Complications

Complications encountered in a baby with RDS are a combination of those due to Prematurity per se, sequelae of surfactant deficient RDS and those that are caused as a result of the treatment given. These are enumerated as : (1) Airleak (2) Patent Ductus arteriosus (3) Peri-ventricular hemorrhage-PVL (4) Infections (5) Bronchopulmonary dysplasia (6) Necrotising enterocolitis and (7) Renal failure (8) ROP.

Prognosis

The prognosis of RDS has improved tremendously in the western world with almost 90% survival and in most cases a neurologically intact survival. Most deaths occur as a consequence of either infection, IVH-PVH or BPD. Survival in our country is steadily improving but varies from centre to centre. There is considerable morbidity associated with RDS, nearly 50% seek readmission in the pediatric ward during the first year of life for surgery for hydrocephalus or inguinal hernia repair, failure to thrive or sequelae of NEC surgical repair. Most admissions are related to neurologic sequelae or respiratory problems.

References

1. Stahlman MT, BaHersby EJ, Shepard FM, Blankenship WJ. Prognosis in hyaline membrane disease: Use of a linear discriminant. *New Eng J Med* 1967;276:303.
2. Crowley P. Prophylactic corticosteroids for preterm birth. *Database Syst Rev* 2000.
3. Greenough Anne, Robertson NRC. Respiratory distress syndrome. In 'Neonatal Respiratory Disorders' Eds. Greenough Anne, Robertson-NRC Milner AD, Publ. Arnold London, 1999;238-279.
4. Gittermann MK, Fusch C, Gittermann AR, Regazzoni BM, Moessinger AC. Early nasal continuous positive airway pressure

- treatment reduces need for intubation in very low birth infants. Eur J Pediatr 1997;156:384-388.
5. Poets CF, Sens B. Changes in intubation rates and outcome of VLBW - A populationbased study. Pediatrics 1996;98:24-24.
 6. 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. Oxford: Update Software Ltd.
 7. Allen LP, Reynolds ER, Rivers RPA, LeSouef PN, Wimberley PD. Controlled trial of continuous positive airway pressure given by face mask for hyaline membrane disease. Arch Dis Child 1977;52:373-378.
 8. Ho JJ, Subramaniam P, Herderson-Smart DJ, Davis PG. Continuous distending pressure for respiratory distress syndrome in preterm infants. Cochrane Database Syst Rev. 2002(2):CD002271. Oxford: Update Software Ltd.
 9. Lee US, Dunn MS, Fenwick M, Shennan AT. A comprison of underwater bubble continuous positive airway pressure (CPAP) with ventilator derived CPAP in preterm neonates ready for extubation. Biol neonate 1989;73:69-75.
 10. Klerk AMD, Klerk RKD. Nasal CPAP and outcomes of preterm infants. J Pediatr Child Health 2001;37:161-7.
 11. Narendran V, Donovan EF, Hoath SB, Warner BB, Streichen JJ, Jobe Aj. comparison between early bubble CPAP and conventional CPAP in reducing the incidence of chronic lung disease. Society Pediatric Research, Annual Meeting, Baltimore 2002; Abstract No.1960.
 12. Mazzella M, Bellinic, Calevo MG, Canpone F Massocco D, Mezzano P, et al. A randomized control study comparing Infant Flow Driver with nasal CPAP in preterm infants. Arch Dis Child Fetal Neonatal Ed 2001;85:F86-90.
 13. Verder H, Albertsen P, Ebbesen F, Greisen G, Robertson B, Bertelsen A, et al. Nasal continuous positive airway pressure and early surfactant therapy for respiratory distress syndrome in newborns of less than 30 weeks' gestation. Pediatrics 1999;103:e125.
 14. Stevens TP, Blennow M, Soll RF. Early surfactant administration with brief ventilation vs selective surfactant and continued mechanical ventilation for preterm infants with or at risk for RDS. Cochrane Database Syst Rev.2002(2):CD003063. Oxford: Update Software Ltd.
 15. Dambeau JM, Parmigiani S, Marinescu B, Bevilacqua G. Use of surfactant for prevention of respiratory distress syndrome in newborn infants with spontaneous breathing. A randomized multicentre clinical pilot study. Acta Biomed Atenwo parmense 1997;68 Supp 139-45.
 16. Soll RF, Blanco F. Natural surfactant extract versus synthetic surfactant for neonatal respiratory distress syndrome. Cochrane Database Syst Rev. 2001;(2):CD000144. Oxford : Update Software Ltd.
 17. Yost CC, Soll RF. Early versus delayed selective surfactant treatment for neonatal respiratory distress syndrome. Cochrane Database Syst Rev. 2000;(2):CD000144. Oxford: Update Software Ltd.
 18. Yost CC, Soll RF. Early versus delayed selective surfactant treatment for neonatal respiratory distress syndrome. Cochrane Database Syst Rev. 2000;(2):CD0001456. Oxford: Update Software Ltd.
 19. Soll RF. Multiple versus single dose natural surfactant extract for severe neonatal respiratory distress syndrome. Cochrane Database Syst Rev. 2000;(2):CD000144. Oxford: Update Software Ltd.
 20. Johnson AH, Peacock JL, Greenough A, Marlow N, Limb ES, Marston L, Calvert SA; United Kingdom Oscillation Study Group. High-frequency oscillatory ventilation for a the prevention of chronic lung disease of prematurity. N Engl J Med 2002;347:633-42.
 21. Silverman WA, Anderson DH. A controlled trial of effect of water mist on obstructive respiratory signs, death rate and necropsy findings among premature infants. Pediatrics 1961;17:1.
 22. Downes JJ, Vidyasagar D, Morrow GM, Boggs JR. Respiratory distress syndrome of newborn infants. J New Clinical Scoring system with Acid-base and blood gas correlations. Clin Pediatr 1970;9:325.
 23. Cloherty (1991) Neonatal Care, Little Brown, 188-95.
 24. Paul VK. Assisted ventilation in Neonates. In 'Challenged in Neonatology' A compendium of management protocols. Eds. Saily A. Publ. Jaypee bros. 1997:134-151.
 25. Kamper J, Ringsted C. The treatment of idiopathic RDS with binasal CPAP. Acta Pediatr Scand. 1990;79:581-6.
 26. Kamper J, Wolff K, Larsen C, Lindequist S. Early treatment with nasal CPAP in VLBW infants. 1993;82:193-7.
 27. So KW. (1997) Randomised controlled trial of colloid or crystalloid in hypotensive preterm infants. Archives of Disease in Childhood;76:f43-f46.
 28. Greenough A, Emery EF (1993) Randomized trial comparing dopamine and dobutamine in preterm infants. European journal of Pediatrics; 152:925-7.
 29. Roze JC, Tohier C, Maingueneua C, Lefevre M, Mouzard A (1993) Response to dobutamine and dopamine in the hypotensive very preterm infant. Archives of Disease in Childhood; 69:59-63.
 30. Klarr JM, Faix RG, Pryce CJE, Bhatt-Mehta V (1994) Randomized blind trial of dopamine versus dobutamine for treatment of hypotension in preterm infants with respiratory distress syndrome. Journal of Pediatrics; 125:117-22.

Future Special Issues / Symposia

Special Issues :

- Recent Advances in Radiation Oncology.
- Environmental pollution and Human Health.
- Breast Cancer - Management update
- Interventional Cardiology: New Trends
- Organ Transplantation: Current Scienario

Symposia :

- MDRT : Problems & Challenges
- Pathological Fractures : Management Dilemma
- Gastroesophageal Reflux Disease (GERD)
- Diabetic Heart Disease.
- Minimal Access Spinal Surgery
- Cosmetic Surgery: New Horizons

Next Issue - Highlights

Special Issue : Recent Advances in Radiation Oncology : Guest Editor: Dr. Tejinder Kataria

- Radiation Therapy: What is New
- The Emerging Role of Functional Imaging in Cancer Management
- Radiation Oncology and Molecular Biology - The Frontiers Ahead
- High Dose Rate Brachytherapy (HDR) - Its Role in Malignant Tumours
- An Overview of Prostate Brachytherapy
- Emerging Role of Endovascular Brachytherapy in Arterial Restenosis
- Evolution and Techniques of 3DCRT/SRT/SRS for Brain Tumours and Their Impact on Management of Intracranial Lesions
- Intensity Modulated radiation Therapy and its Scope in Radiation Oncology
- Telemedicine in Radiation Oncology : Challenges and Opportunities
- Heavy Particle Beams: Clinical Application