

# **CPAP**

## **Bedside Application in Newborn**

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## FOREWORD

CPAP: Bedside application in the newborn by PK Rajiv covers an important aspect of ventilatory management in the newborn infant. The popularity of the application of Continuous Positive airway Pressure in the spontaneously breathing infant has gained momentum since its introduction in 1971. Most neonatal centres around the world use it as the first line of treatment in preterm Infants at risk of developing hyaline membrane disease. It is also used frequently when weaning infants from mechanical ventilation.

Under the circumstance, the advent of this book is timely. It is a step by step guide which takes the student and the clinician through the introduction of CPAP to the sick newborn infant to its weaning as the infant improves. Dr Rajiv has drawn from his extensive experience in India and overseas to develop his own strategies to achieve the desirable outcome in the infants. A wide range of radiographs and figures are used to illustrate his point.

A unique feature of this book is the description of the different types of devices available in India for the provision of CPAP to newborn infants. This is a useful book to have in all nurseries.

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## Applied physiology

A reasonable understanding of pulmonary physiology is elementary in the understanding the approach and bedside management of respiratory disorders. An attempt has been made to synchronize physiological or pathological deviations with ventilatory management.

### PHYSIOLOGICAL PRINCIPLES

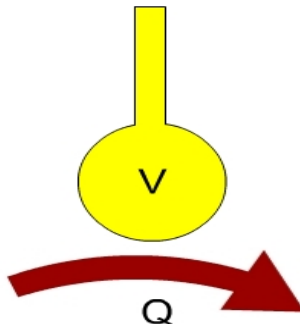
The physiological basis of treating any critical newborn lies in the optimization of ventilation and perfusion.

Under ideal conditions, Ventilation and Perfusion are evenly matched and the  $V/Q$  ratio = 1.

#### **FIGURE 2**

**V=VENTILATION**

**Q=PERFUSION**



- Blood Pressure
- O<sub>2</sub> content of blood
- Dilatation of air sac
- Nitric Oxide metabolism

## **VENTILATION (V):**

Ventilation applies to a physiological tidal volume, which reaches the alveoli and distends it optimally. If the minute ventilation ( tidal volume X rate ) is optimal , the ideal CO<sub>2</sub> will be 35 to 45 mm of Hg. Normal tidal volume in neonate is 4 to 6 ml / kg .

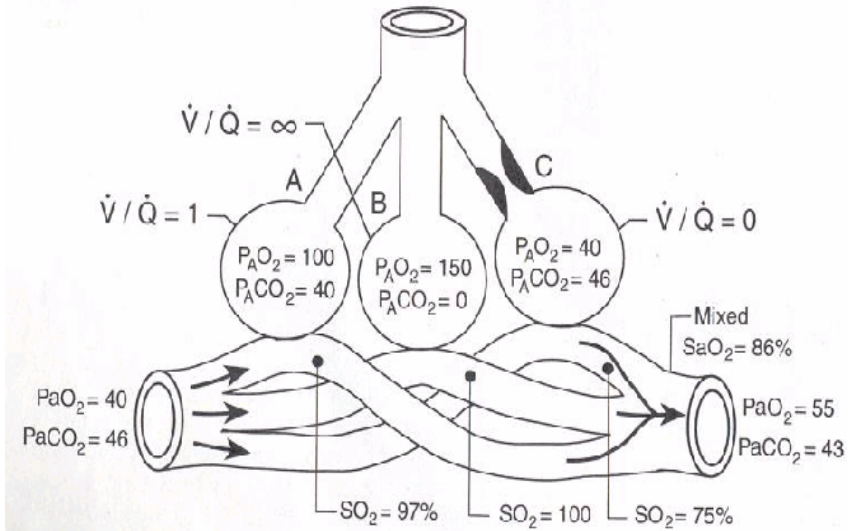
## **PERFUSION (Q):**

This is maintained by the state of dilatation of the pulmonary arterioles and capillaries supplying the air sac. It is controlled to a large extent by systemic blood pressure (Mean Arterial Blood Pressure). Myocardial pump function determines the systemic blood pressure directly. In addition certain physiological variables have to be taken into consideration.

- Hb content of the blood
- State of dilation of the air sac (FRC)
- Nitric Oxide Metabolism (maintains pulmonary arteriolar dilatation)

## Basic Understanding Of Various Lung Units In Disease States

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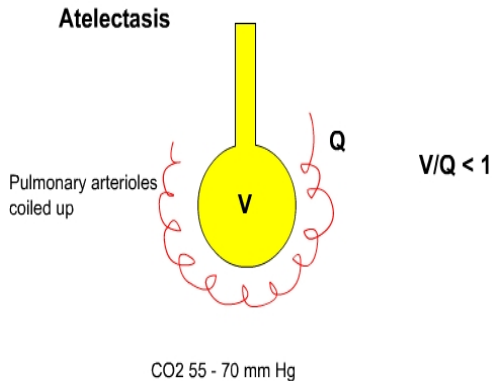


Three-lobed , three –compartment model of the lung with  $V/Q$  equal to 1 in A,  $V/Q$  equal to infinity in B, and  $V/Q$  equal to zero in C . Coming into the pulmonary circuit on the left is blood returning from the body and having a low  $P_{aO_2}$  (40mmHg) and a high  $P_{a CO_2}$  (46mm Hg) . Mixed blood returning from the lungs is not fully saturated with  $O_2$  because of venous admixture from the low

$V/Q$  unit C , where the lack of ventilation leaves the  $P_{a O_2}$  unchanged (40mmHg );  $SO_2$  of 75% at a  $P_{aO_2}$  of 40 mmHg, and a  $P_{a O_2}$  of 55 mmHg at a  $SaO_2$  of 86%

(Adapted from Goldsmith : Physiological principles pg 56)

## Physiological abnormalities in disease states

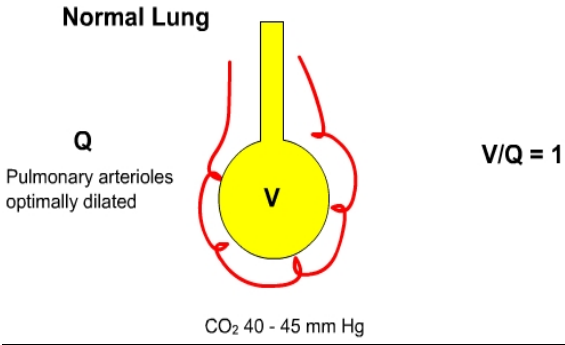


### ALVEOLI IS ATELECTATIC

The pulmonary arterioles are coiled up leading to less perfusion to those alveoli (Pulmonary hypertension) and if atelectasis is more profound perfusion proportionately is reduced leading to intrapulmonary shunting due to admixture of mixed venous blood into the systemic circulation. The density of the haziness on the X ray is a rough estimate of the blood CO<sub>2</sub> levels .

**$V/Q < 1$**   
**CO<sub>2</sub> 55-70 mmHg**

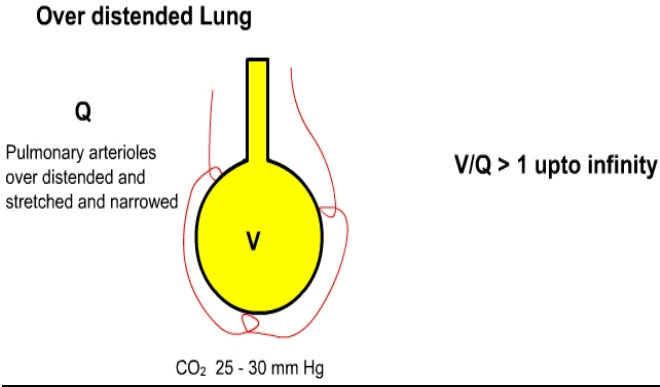
# ALVEOLI ARE OPTIMALLY DISTENDED 'NORMAL LUNG UNDER IDEAL CONDITIONS'



The pulmonary arterioles unfurl completely and perfuse the alveolar bed completely.

**V/Q = 1**  
**CO<sub>2</sub> 40-45 mm Hg**

## ALVEOLAR OVERDISTENTION:



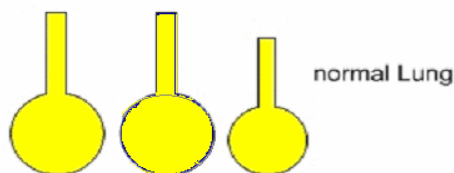


On the other hand if the alveoli is overdistended due to inappropriate pressures on the ventilator (CPAP), the pulmonary arterioles got stretched to a point where narrowing of their lumen causing pulmonary hypertension leading, to decreased perfusion in the lung and intrapulmonary shunt. The X Ray shows hyperlucent lung fields .

**V/Q >1 (upto infinity)**  
**CO2 =25-30mmHg**

## CONCEPTUAL MODELS OF APPLIED PHYSIOLOGY

conceptual model of lung disease in RDS

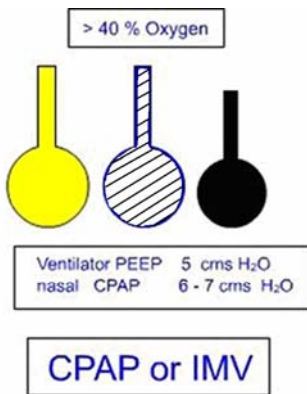


- Over expanded
- normal

It would be over simplification of the lung units and the surface area for gas exchange by presenting a model of lung units with 3 generations of alveoli, the collapse of which present with increasing oxygen requirements in atelectatic lung disease.

## OXYGEN REQUIREMENTS > 40% (FiO2 >0.4)

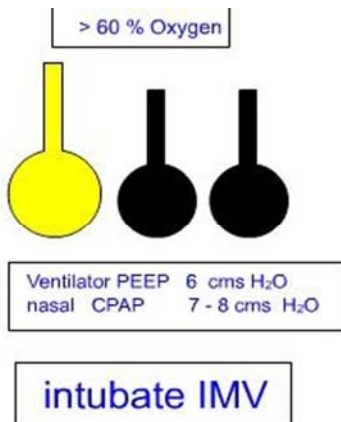
One generation of alveoli maybe collapsing and it would be appropriate to institute assisted ventilation ideally CPAP or IMV at this stage. This would recruit lung volume and optimize gas exchange and prevent morbidity and air leaks.



- atelectasis
- ▨ Over expanded
- normal

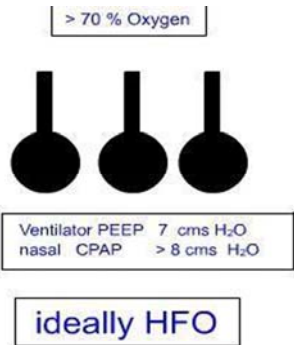
## **OXYGEN REQUIREMENTS > 60% (FiO2 >0.6)**

Two generations of alveoli have collapsed and unless interfered, actively with intubation and ventilation (IMV), air leaks, pulmonary hypertension and morbidity could occur.



## **OXYGEN REQUIREMENTS > 70-80% (FiO2 >0.7-0.8)**

Most generations of alveoli have collapsed and only approximately 1/3 alveolar surface area is available for gas exchange. It would be inadvisable to allow a neonate with RDS to progress to this stage. When ventilation (IMV) is resorted to at this stage air leaks, severe pulmonary hypertension and pulmonary hemorrhage could occur.



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From the above discussion it is apparent that EARLY RECRUITMENT OF LUNG VOLUME is essential for optimum ventilation and prevention of morbidity. The early and optimal addition of PEEP or CPAP in lung disease is the sine qua non of ventilatory care and reflects the great variability in morbidity and mortality of various centers dealing in newborn care.

A thumb rule in this lung volume recruitment strategy is highlighted below which is basically intended for atelectatic lung disease.

<b>FiO<sub>2</sub></b>	<b>PEEP</b>	<b>CPAP</b>
Fi O <sub>2</sub> > 0.4	5cms H <sub>2</sub> O	6cms
Fi O <sub>2</sub> > 0.5-0.6	6cms H <sub>2</sub> O	7-8cms can be tried
<b>Fi O<sub>2</sub> &gt; 0.7</b>	<b>8 cms H<sub>2</sub>O</b>	<b>Not recommended</b>

## **Anatomical considerations**

The closing volume of alveoli is larger than the FRC until 6-8 years of age. This causes an increased tendency for airway closure at end expiration. Thus, neonates and infants generally benefit from use of continuous positive airway pressure ( CPAP ) in suitable conditions . For the most part, neonates are preferential nose breathers, which easily facilitates the application of nasal CPAP. CPAP is accomplished by inserting nasopharyngeal tubes, affixing nasal prongs, or fitting a nasal mask to the patient.

## **Physiological considerations**

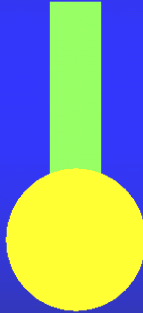
The application of assisted ventilation requires the basic understanding of a few physiological parameters which are to be assessed in order to optimally ventilate a baby with CPAP

**Airway resistance**

**Lung compliance**

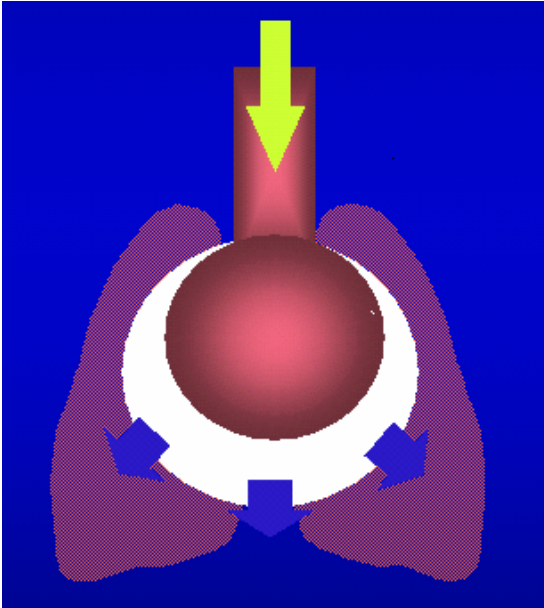
# Respiratory Tract

- Tube + Balloon model
- Airways  
(Tube)
- Alveoli  
(Balloon)



The respiratory tract can be simplified into a tube and balloon model to understand the basic concepts of application of CPAP. The baby's ventilation is influenced by the caliber of the air ways (airway resistance) and the stiffness of the lung which is reflected by alveolar collapse (atelectasis) this indicates the lung compliance.

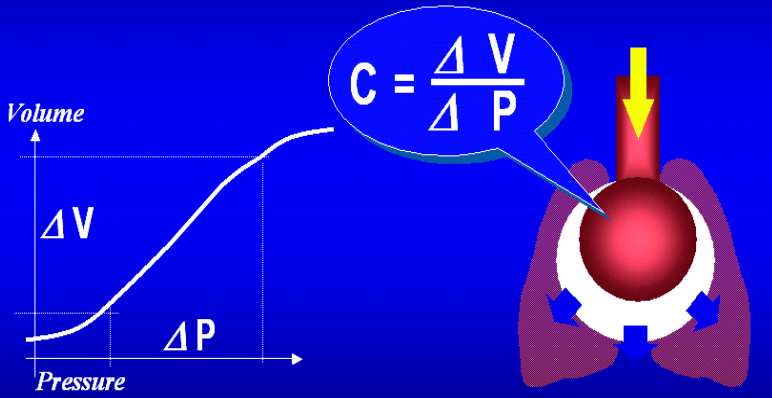
## **Airway resistance**



**Lung compliance**

# Compliance

Property of distensibility of the lungs and chest wall



Compliance is defined as the unit change in volume for a given unit change in pressure . In HMD, compliance is extremely low and at the lowest point of the pressure- volume curve .



## Resistance

Length of the airway  
Flow rate of Gas  
Density and viscosity of gas

$$R = \frac{\Delta P}{\Delta F}$$

Pressure Difference = Flow Rate x Resistance of the Tube

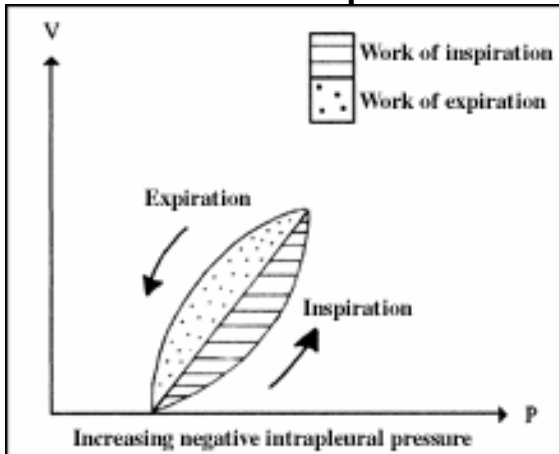
R – Resistance of airways

Delta P is pressure difference between upper airway and alveoli

Delta F is the difference in flow between upper airway and alveoli

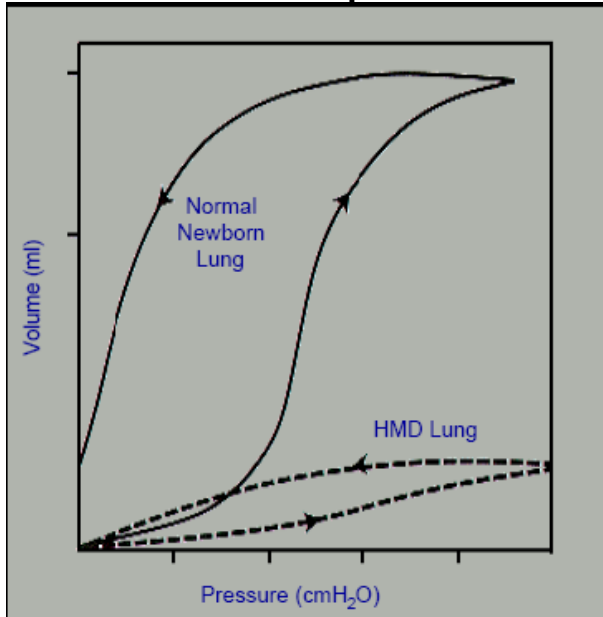
Airway resistance is directly correlated with the tube length, and divided by the fourth power of tube radius. Inspiratory flow resistance is always less than expiratory flow resistance as the airways dilate during inspiration. In meconium aspiration, the airway resistance is very much increased due to airway debris and bronchospasm, which reduce the cross-sectional diameter of the airways significantly.

## Pressure volume loop



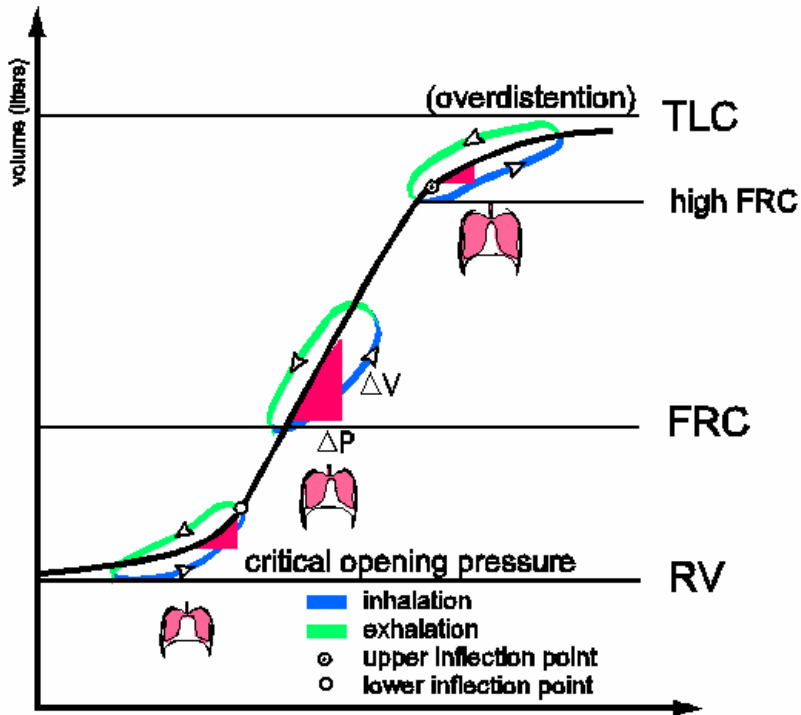
The work of breathing is best displayed on a pressure-volume curve of one respiratory cycle. The diagram shows the different pathways for inspiration and expiration, known as ***hysteresis***. The total work of breathing of the cycle is the area contained in the loop.

## Pressure volume loop in HMD



### Relationship of lung condition with pressure – volume loop

The normal lung exhibits good change in volume for a given unit change in pressure. This is in sharp contrast to the atelectatic ( HMD ) lung which shows minimal change in volume per unit pressure change. The direction of arrows show inspiration and expiration respectively.



The pressure – volume curve as it moves towards residual volume (RV) will indicate the severity of atelectasis. The degree of improvement in functional residual capacity (FRC) moves the point towards the middle of the pressure volume curve indicating good change in volume for unit change in pressure . It is evident that as the FRC gets over expanded with excessive CPAP, the change in volume is again reduced for unit change in pressure. TLC denotes total lung capacity

CPAP is the major factor determining **lung volume**. At low CPAP low lung volumes are evidenced as in RDS and compliance is low .At optimum lung

volumes compliance increases. At still higher volumes (overdistension) compliance again decreases . Optimum FRC results in optimum compliance and the lowest work of breathing . Optimum CPAP is indicative of optimum FRC .

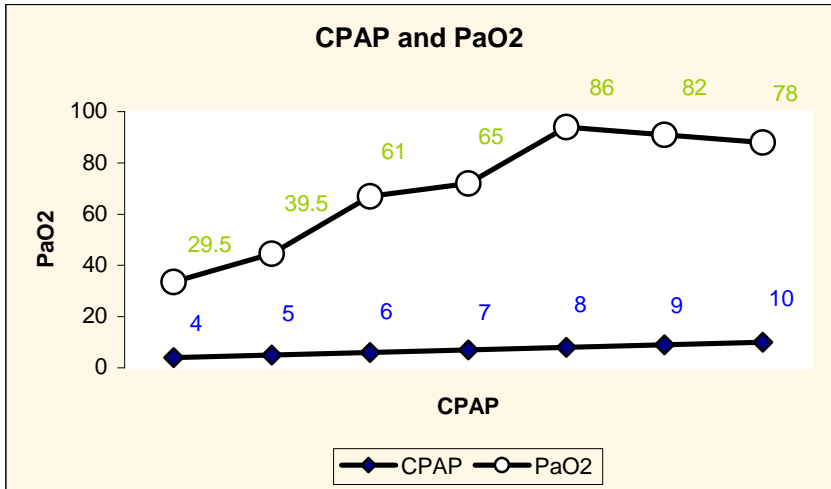
Lung volume is also related to **airway resistance**. At low lung volumes (insufficient CPAP) airway resistance is high and since atelectasis is not resolved, work of breathing is high . At optimum lung volumes airway resistance is low. Thus CPAP can improve distribution of ventilation to optimize FRC and therefore optimize both lung compliance and airway resistance.

## Effects of CPAP

The effect of CPAP works on lung mechanics **to improve oxygenation** (PaO<sub>2</sub>). The effect on CO<sub>2</sub> is only secondary to the primary process of improvement in lung volume and minute ventilation. CPAP maintains inspiratory and expiratory pressures above ambient pressure, which should result in an increase in **functional residual capacity** (FRC) and improvement in **static lung compliance** and **decreased airway resistance** in an infant with unstable lung mechanics. This allows a greater volume change per unit of pressure change (i.e., greater tidal volume for a given pressure change) with subsequent reduction in the work of breathing and stabilization of minute ventilation ( $V_E$ ). The beneficial physiologic effects of CPAP are created by an

- Increased transpulmonary pressure resulting in an increased functional residual capacity and potentially reduce oxygen demands
- Stabilization of an unstable chest wall
- Improves lung compliance ( causes redistribution of fluid in the lungs )
- Reduces airway resistance
- Reduces work of breathing
- Improvement in ventilation – perfusion ratios
- May expand, or stent, upper airway structures preventing collapse and upper airway obstruction

- Preserves endogenous surfactant. Maintenance of optimal functional residual capacity improves surfactant synthesis and release



**When CPAP used in RDS (Clinical study from AIMS 2005 – 2007 , Kochi), relationship with PaO<sub>2</sub> is evident and the optimum level of CPAP in preterm appears to be between 7-8cm of H<sub>2</sub>O**

CPAP improves oxygenation by an increase in functional residual capacity by recruiting atelectatic alveoli thereby increasing the surface area for gas exchange . The locally vasoconstricted vessels due to underventilation opens up with an increase in functional residual capacity achieved with an appropriate CPAP . These effects decreases

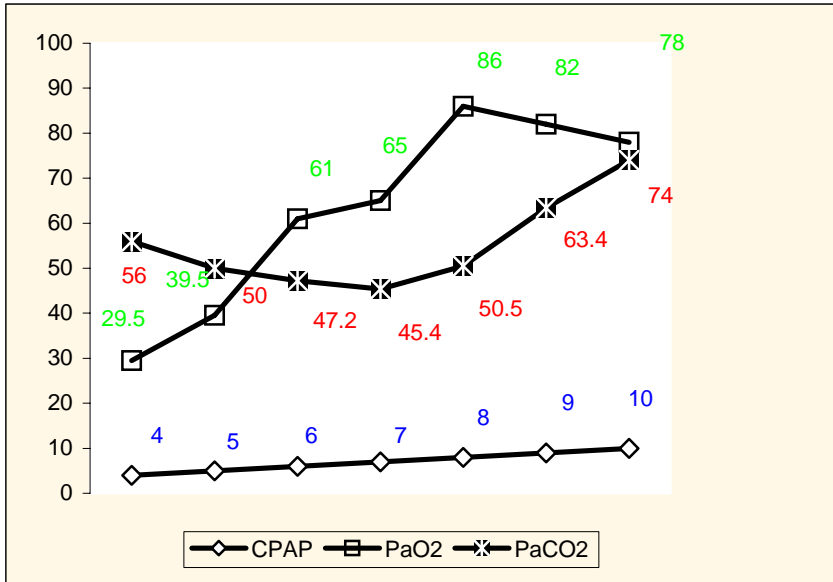
intrapulmonary shunt and causes a reduction of PaCO<sub>2</sub> levels .

It stabilizes the chest wall thereby reducing **airway resistance** . This reduces the work of breathing and improves ventilation – perfusion mismatch .The intrapulmonary shunt is reduced . All this add up to cause an increase in PaO<sub>2</sub> levels

It splints open the upper airway and thus reduces the possibility of obstructive apnea . The application of CPAP increases airway caliber according to their individual compliances lowering the airway resistance .This improves the ventilation of regions of the lung where narrowing of the airway has occurred

Application of CPAP stretches the pleura and lungs resulting in stimulation of stretch receptors . This has beneficial effect on mixed and central apnea  
Alveolar collapse results in a higher consumption of surfactant owing to reduced surface area , and CPAP could conserve surfactant by prevention of collapse or enhancement of surfactant release through a cholinergic mechanism . This may explain why CPAP is more effective when used **early in the course of the disease** while most alveoli are open . Early application of CPAP is seen to reduce the need for mechanical ventilation





### CPAP in RDS and relationship with PaO<sub>2</sub> and PaCO<sub>2</sub> (Clinical study from AIMS, Kochi )

It appears from this study that CPAP levels beyond 8 cm of H<sub>2</sub>O should be used cautiously in preterm babies. With high CPAP levels ,a reduction in tidal volume occurs resulting in hypercarbia and hypoxemia . A reduction in CPAP level at this point would result in an improvement of ventilation

### Long term implication

Maintaining lung volumes within the optimal range between atelectasis and over distension have been found to decrease the incidence and severity of later chronic lung disease

## Cardiovascular stability

High CPAP can have a detrimental effect on the cardiovascular system . The effects include

- Compression of right sided vessels thereby decreasing cardiac return resulting in decreased cardiac output .
- Decreased peripheral and regional blood flow
- Decrease in oxygen available to tissue
- Increase in pulmonary vascular resistance and thereby increase extrapulmonary shunts

Decrease in cardiac output can lead to acidosis , tachycardia and reduced arterial blood pressure . The amount of CPAP that is excessive will produce this effect depends on the lung compliance . If the lung compliance is low ( HMD ) , less intra airway pressure will be transmitted to the pleural space and cardiac compromise will be less. Hypovolemia will exacerbate the negative effect of high CPAP . Excessive CPAP may be detected by the development of acidosis , decreased dynamic lung compliance and increased carbon dioxide retention . A trial of lower CPAP or increased intravenous fluids will resolve the problem . However it should be recalled that too low a CPAP will also cause acidosis ( respiratory ) due to atelectasis .. A precise level based on the operators experience would be instrumental before conclusions could be drawn on the level . **It would be grave mistake to experiment with CPAP levels in a critically ill baby**  
Sequence of events in excessive CPAP

Tachycardia → increased capillary filling time  
→fall in blood pressure →diminished urine output →  
acidosis ( metabolic ) . **Blood PCO<sub>2</sub> may initially fall  
to rise subsequently over a period due to gas  
trapping .**

## **Renal system**

CPAP can result in decrease in glomerular filtration rate and thus the urine output. Renal effects are directly proportional to compliance of the chest wall. Decreased renal blood flow results in increased aldosterone and ADH secretion

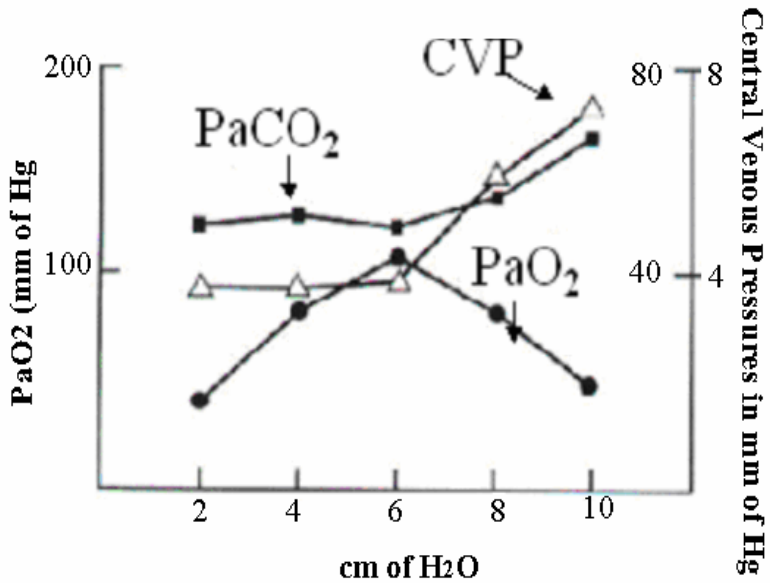
## **Gastrointestinal tract**

Abdominal distension can occur in babies on CPAP. It is compounded by presence of immature gut in preterms and decrease in blood flow to the gut. All these together lead to what is called as 'CPAP belly syndrome'. Clinically the baby develops increased abdominal girth and dilated bowel loops, which may cause upward pressure on diaphragm and respiratory compromise.

## **Central nervous system**

There is increase in intracranial pressure (ICP) with application of CPAP. This, in combination with decrease in arterial pressure, results in decrease in cerebral perfusion pressure (CPP). Increase in ICP is seen more with head box CPAP than with endotracheal CPAP or nasal prongs. High ICP is instrumental in pathogenesis of intraventricular hemorrhage in low birth

weight babies ventilated for RDS . Head box CPAP is not used currently . The significance of this finding in clinical practice is not evidenced with the current CPAP devices and protocols



(Gregory GA. Continuous positive airway pressure. Neonatal Pulmonary care .2<sup>nd</sup> ed. Norwalk, CT, Appleton and Lang, 1986. p 355)

It is evident that when CPAP pressure exceeds 6 cm of H<sub>2</sub>O and especially when it nears 8 cm of H<sub>2</sub>O , significant increase in central venous pressure coincides with decreases in PaO<sub>2</sub> levels and increased PaCO<sub>2</sub> levels .

## **OPTIMUM CPAP**

It is the level of CPAP that results in maximum PaO<sub>2</sub> on lowest FiO<sub>2</sub> without increase in PaCO<sub>2</sub> in the absence of any adverse hemodynamic effects

### **Optimum CPAP**

CPAP is considered optimum when the baby on CPAP is

- Comfortable and pink
- Has normal blood pressure and normal capillary filling time
- Has no respiratory distress
- No cyanosis
- Audible air entry on auscultation
- SpO<sub>2</sub> more than 90-93 %
- ABG : PaO<sub>2</sub> 60-80 / PaCO<sub>2</sub> 40-45 /p H 7.30 - 7.40
- Optimal chest expansion ( Chest X ray in supine : Post. Intercostal space 7 - 8
  - Improvement in air entry is also a good measure of improvement with CPAP especially in baby more than 1.5 kg .

## Road to Optimum CPAP

Optimising CPAP requirement is an art . The decision is based on the underlying lung condition, disease pattern, pressure requirement, opacity of chest X-ray , ABG and the clinical condition . Always increase pressure in increments of 1 cm and observe the effect.

In our unit , we start CPAP at 6 cm H<sub>2</sub>O in atelectatic disorders guided by the clinical status and the degree of opacification on chest X-ray .Clinically there should be an improvement in chest recessions and tachypnea, The degree of expansion of the lungs is assessed by an X-ray is taken within 4-6 hrs after initiation of CPAP. A decrease in FiO<sub>2</sub> requirement would suggest an improvement of the oxygenation status . Of all the parameters assessed , objectivity is best maintained with chest X ray and ABG

- a) **Based on Chest X-ray:** Presence of 8 posterior intercostals spaces above diaphragm indicates optimum CPAP.Less than 6 intercostal spaces indicates low volume lungs in an inspiratory film .A low lung volume as evidenced by chest X ray may require an increase in CPAP level .Presence of more than 8 spaces with flat dome of diaphragm indicates hyperinflation which could be one of the indication to decrease the level of CPAP.
- b) **ABG** – ABG is done within 20 mins of setting CPAP .ABG is preferable done after any subsequent change of pressure within 15 to 30 minutes. Increasing PaO<sub>2</sub> with decreasing FiO<sub>2</sub>

requirement indicates that the baby is on road to optimum CPAP. If PaO<sub>2</sub> falls with increase of CPAP, it could imply over distension of lungs. This may necessitate decrease of CPAP pressure. PaCO<sub>2</sub> generally increases with increasing pressure. Accept mild CO<sub>2</sub> retention if the need is to improve oxygenation with increasing pressures.

### **Measures to maintain optimum CPAP**

- Correctly set up and maintain low resistance delivery circuit
- Securely attach interface
- Assure minimal pressure leaks
- Maintain optimal airway
- Prevent nasal septal injury
- Provide meticulous attention to detail

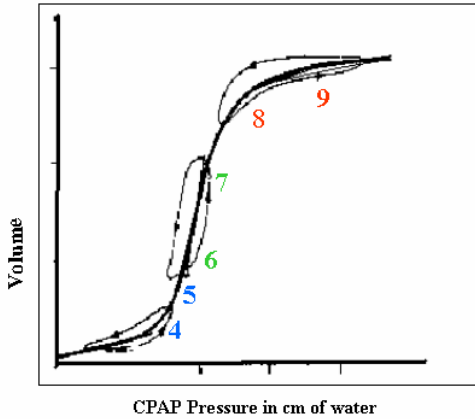
### **Success with CPAP**

Remaining without ventilatory support for more than 3 days

The author feels that experimenting with CPAP levels is dangerous. It should be based on the trend shown in oxygen saturations, the XRay and ABG.

# ATELECTASIS

## CPAP LEVELS INDICATIVE OF LUNG VOLUME

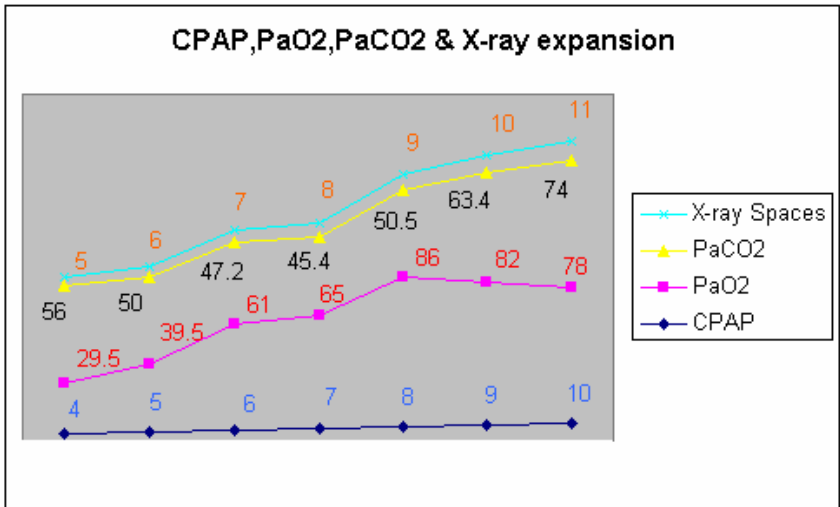
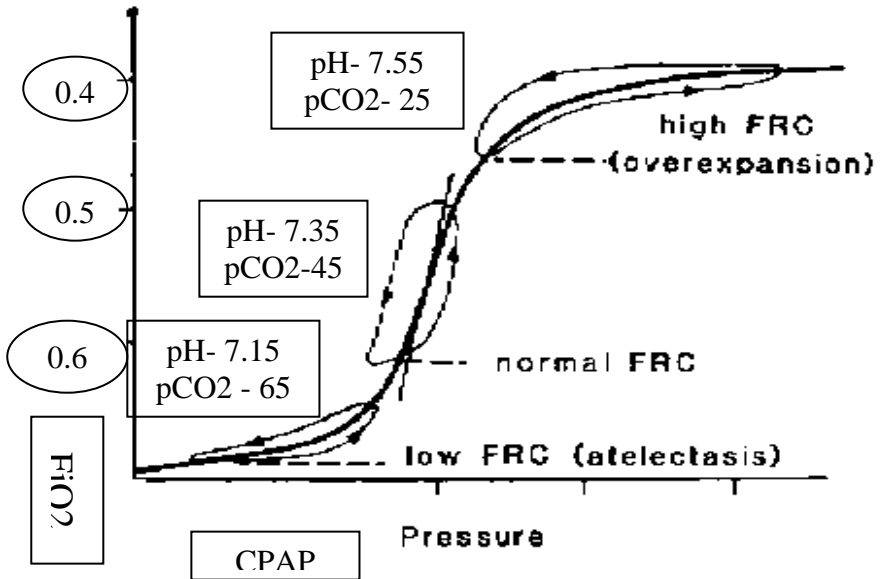


This is useful only as a guideline in mild to moderate respiratory distress syndrome . It is not useful for severe Respiratory distress syndrome .

The numerical projections superimposed on the pressure volume curve is based on the bedside application of CPAP levels in mild to moderate RDS , It is not an absolute value , but will serve as an important guideline to initiate management and titrate the CPAP level with X Rays and blood gas PaO<sub>2</sub> .

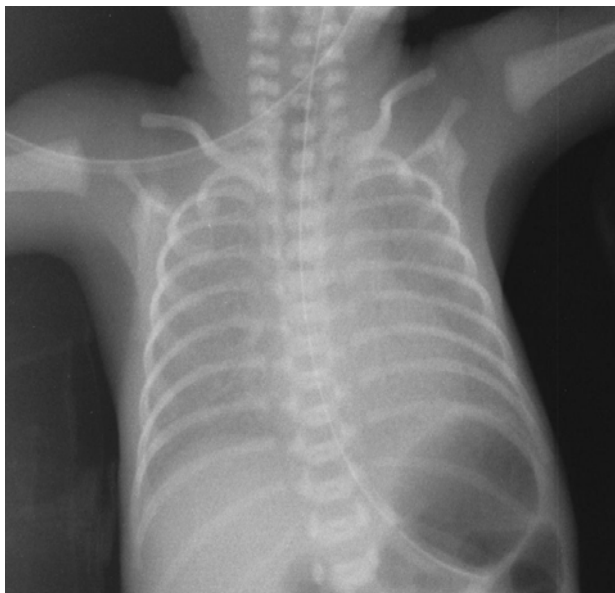


## FiO2 and appropriate blood gas Conceptual Model



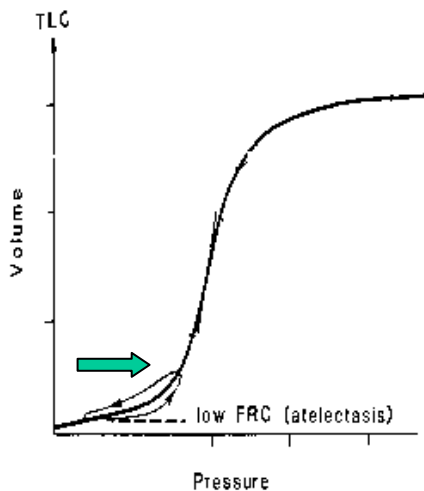
It is evident that once the CPAP reaches more than optimum for the disease process, an inverse relationship will start establishing between  $\text{PaO}_2$  and  $\text{PaCO}_2$  (Clinical study from AIMS, Kochi). This is seen when the X-ray expansion crosses 8 spaces.

**CXR showing deflation (Posterior ICS of <7spaces)**

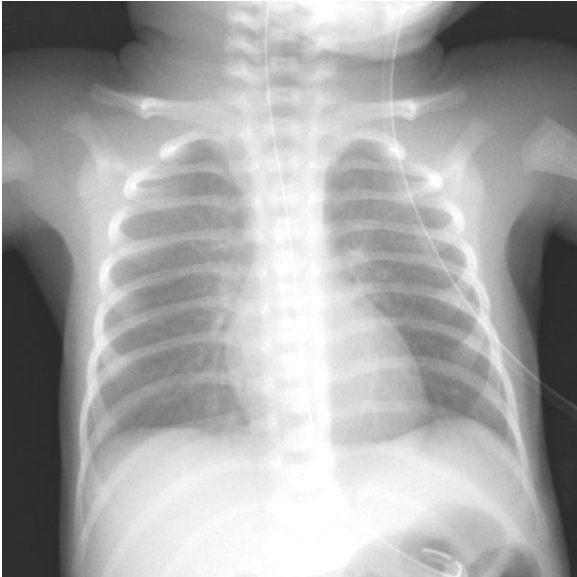


**Expected CO<sub>2</sub> –  
50 - 55 mm Hg**

**CPAP 5cmH<sub>2</sub>O**

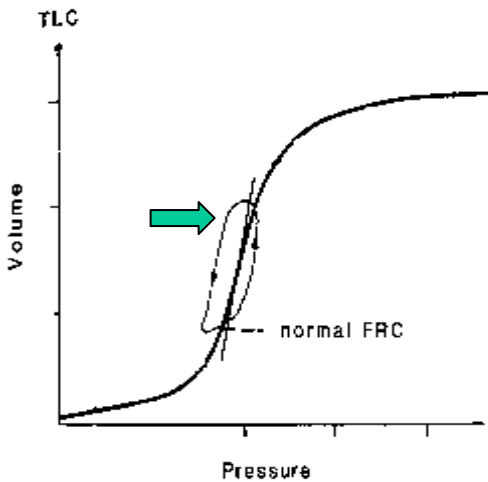


**CXR showing adequate chest expansion  
(Posterior Intercostal spaces of 8 nos)**



**Expected CO<sub>2</sub> –  
30- 35 mm Hg**

**CPAP 6-7 Cm H<sub>2</sub>O**

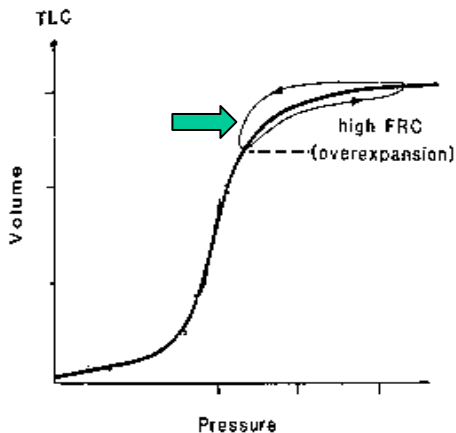


**CXR showing over expansion (Posterior ICS of >9 spaces Flattening of diaphragm)**

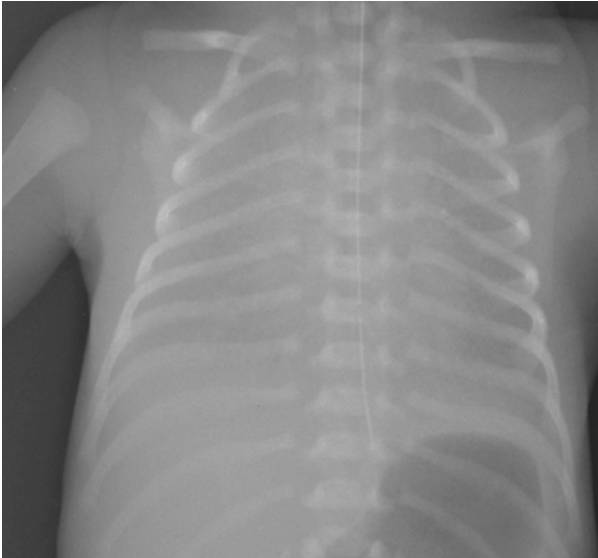


**Expected CO<sub>2</sub> –  
25- 28 mm Hg**

**CPAP 8-9 cm H<sub>2</sub>O**

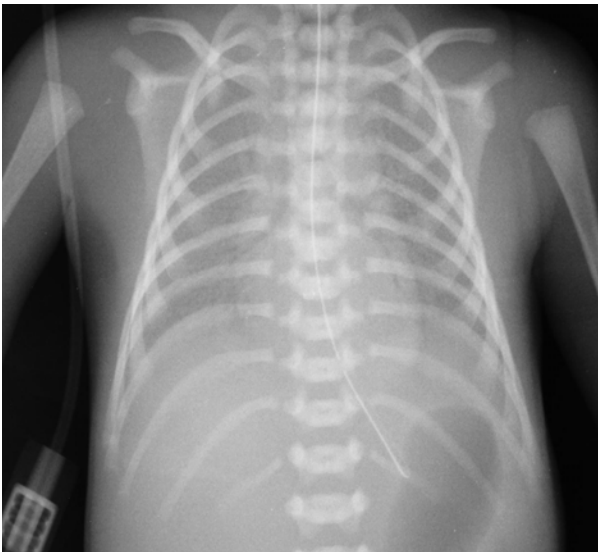


## Hyaline Membrane disease and CPAP



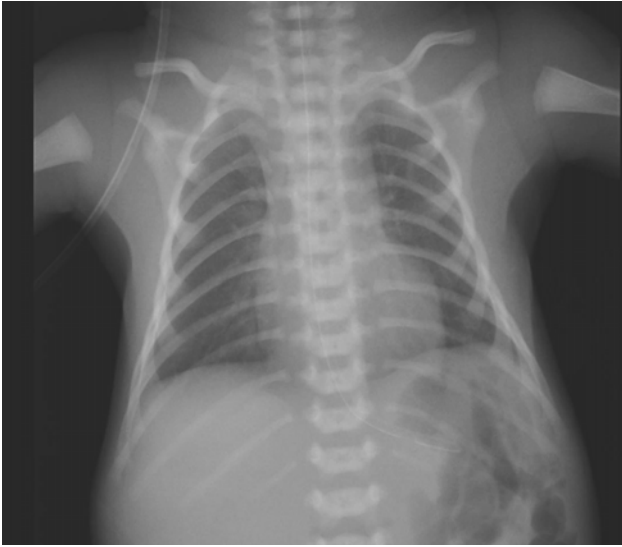
CPAP- 5cm H<sub>2</sub>O Fig 1

**Expected CO<sub>2</sub> –  
60- 65 mm Hg**



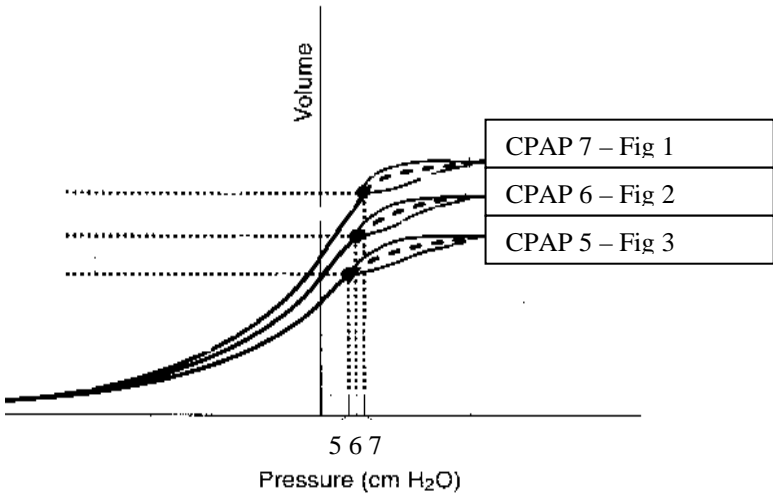
CPAP 6cm H<sub>2</sub>O Fig2

**Expected CO<sub>2</sub> –  
45- 50 mm Hg**

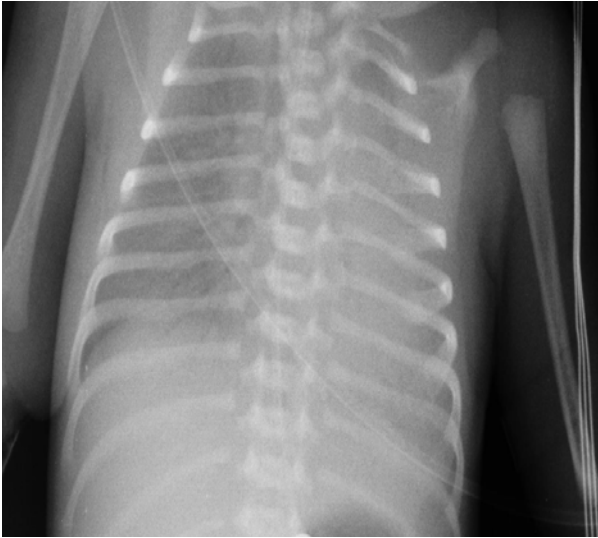


**Expected CO<sub>2</sub> –  
35- 40 mm Hg**

**CPAP- 7cmH<sub>2</sub>O Fig 3**

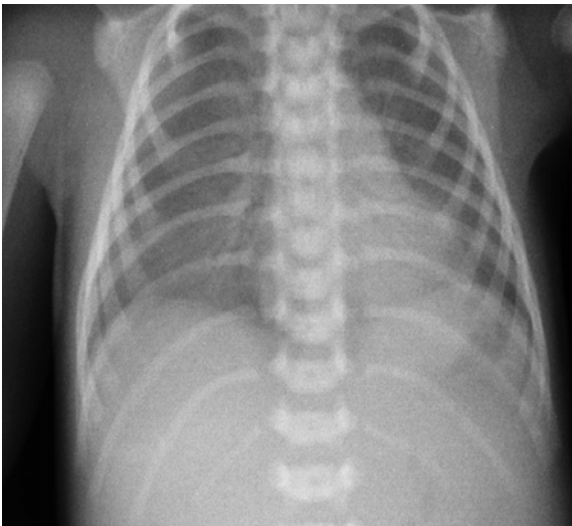


Pneumonia and CPAP



**Expected CO<sub>2</sub> –  
55- 60 mm Hg**

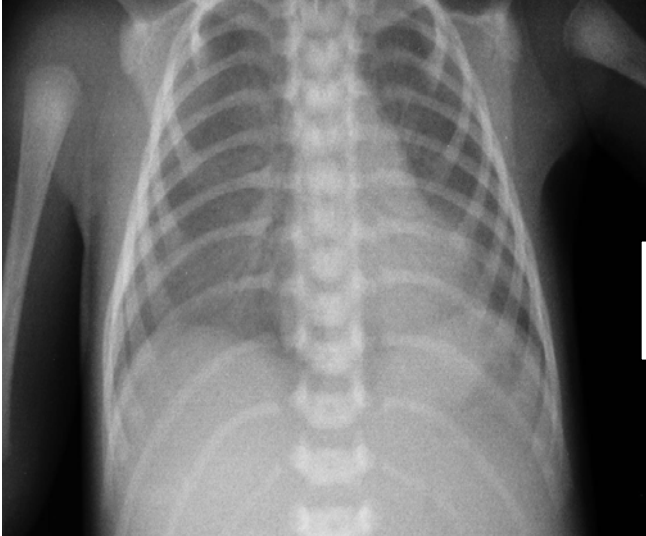
CPAP- 5cmH<sub>2</sub>O Fig 1



**Expected CO<sub>2</sub> –  
45- 50 mm Hg**

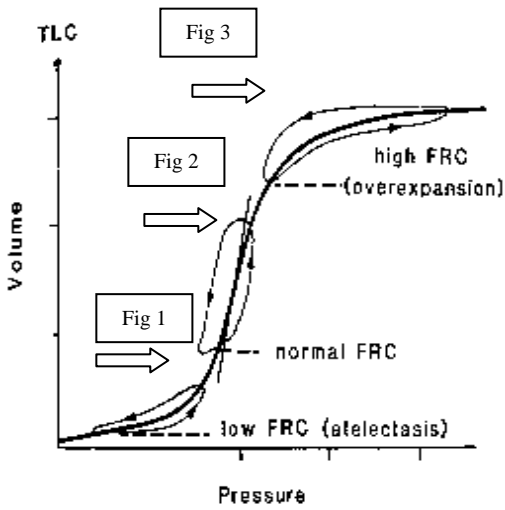
CPAP- 6cmH<sub>2</sub>O Fig 2





**Expected CO<sub>2</sub> –  
25- 30 mm Hg**

**CPAP- 7cmH<sub>2</sub>O Fig3**



I

## ALGORITHM OF APPROACH TO RDS

FiO <sub>2</sub>	CPAP	X-ray Spaces
<0.4	5 – 6	7 -- 8
0.4 to 0.6	6 –7	7 – 8
0.6 to 0.8	7 – 8*	8 – 9

\*It is vital to consider invasive ventilation once the FiO<sub>2</sub> crosses 0.6 as well as the CPAP requirement increases more than 8 cm of H<sub>2</sub>O.

- Upper limits of CPAP are still un-defined. But current evidence points on an upper limit of 8 cm of H<sub>2</sub>O in preterm infants and 9 cm of H<sub>2</sub>O in term babies .
- The author opines that the use of so called “higher CPAP” should not be based on numbers and their restrictions thereof .The clearance of haziness on X-ray should be the end point in the successful application of CPAP within the purview of haemodynamic stability.

In our unit, we may consider using CPAP within 30 minutes after birth for preterm babies below 28 weeks of gestational age, and for babies above that gestation we start once the clinical features of distress appears. ( refer protocol )

### **BABY-A: Moderate RDS with Failure of CPAP**

Preterm 940 gm born to primigravida mother with PIH on nicardia and methyl dopa, No h/o DM , leaking or bleeding PV , delivered by LSCS ( Indication: decrease fetal movements ) with apgar 5 & 7 at 1 and 5 min .Baby had tachypnea, Grunt, Inter Costal retractions, sub costal retractions. Baby was put on CPAP 6 (fig 1) and gradually increased to 7 along with FiO<sub>2</sub> increased from 0.4 to 0.6 as per protocol. (fig 2) The baby had clinically more retractions with loss of lung volume and hence was electively ventilated, despite bloodgas ph of 7.26 and CO<sub>2</sub> of 56mmHg.(fig 3)

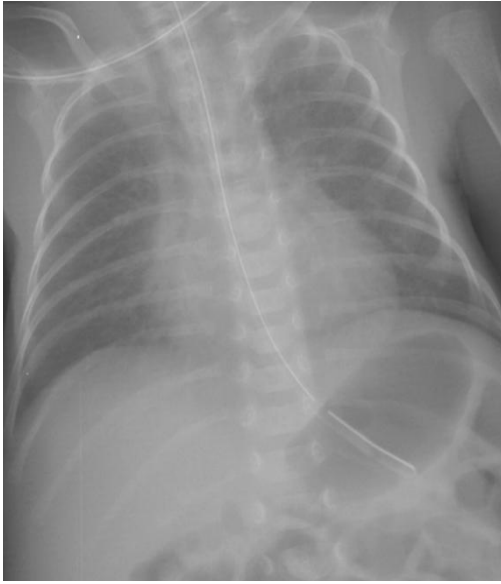


Fig 1

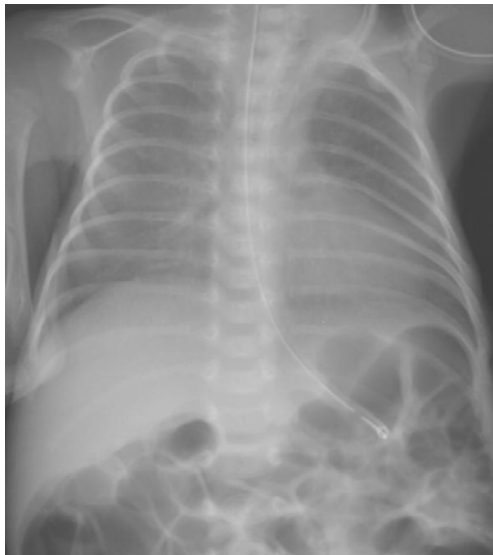


Fig 2



Fig 3

## **APNEA OF PREMATURITY:**

Studies have shown that CPAP has reduced the incidence and severity of Mixed and Obstructive apnea by preventing the collapse of upper airways and pharynx and by splinting the diaphragm.

Since the lung in this condition is usually normal, use low to moderate levels of CPAP. Pressures between 4 – 5 cms of H<sub>2</sub>O can be used with FiO<sub>2</sub> as low as possible to maintain O<sub>2</sub> saturation between 88-90% along with medical management. The initial CPAP would be 5 cm of H<sub>2</sub>O , but if the saturations exceed 95% or the X ray spaces exceed 8 , it is worthwhile to reduce the CPAP to 4 cm of H<sub>2</sub>O . Try to wean off the baby from CPAP once baby is free of apneic episodes for more than 24 hours

### **CPAP – 4 to 5 cm of H<sub>2</sub>O**

In our unit ,we do not use CPAP less than 4 cm of H<sub>2</sub>O . A CPAP below this level can actually increase the work of breathing since the unphysiological CPAP delivered to the distal airways can lead to collapse

### **Baby-B: Apnea of Prematurity**

Born by preterm 29wks, Primigravida mother-41yrs of age ,

Severe PIH on methyl dopa & Nicardia, and Hypothyroidism on eltoxin .

27wks USG and Doppler study showing Utero-Placental insufficiency

2 doses of Betnesol received, Delivered by LSCS .  
B.wt – 856gms, baby developed apnea and was put on CPAP 5 at FiO2 0.25 which was reduced to CPAP of 4 due to the lung over expansion and bloodgas PCO2 25mmHg. Aminophylline was started after birth. There after baby improved without apnea.



