INTRODUCTION

The effect of CPAP works on lung mechanics to improve oxygenation ($\text{PaO}_2$). The effect on CO$_2$ 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.

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$_2$ levels (Fig. 2.1).

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$_2$ 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

**Fig. 2.1:** CPAP in RDS and relationship with PaO$_2$ and PaCO$_2$. When CPAP used in RDS (Clinical study from AIMS 2005-2007, Kochi), relationship with PaO$_2$ and PaCO$_2$ is evident, and the optimum level of CPAP in preterm appears to be between 7-8 cm of H$_2$O.
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.

It appears from this study that CPAP levels beyond 8 cm of H2O 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 overdistension have been found to decrease the incidence and severity of later chronic lung disease.

**PHYSIOLOGICAL SIGNIFICANCE OF CPAP LEVELS**

<table>
<thead>
<tr>
<th>Low (2-3 cm H2O)</th>
<th>Use</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Maintenance of lung volume in VLBW infants</td>
<td>• May be too low to maintain adequate lung volume or adequate oxygenation</td>
<td></td>
</tr>
<tr>
<td>• During weaning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• During hyperventilation in PPHN</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medium (4-7 cm H2O)</th>
<th>Use</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increasing lung volume in surfactant deficiency</td>
<td>If lungs have normal compliance</td>
<td></td>
</tr>
<tr>
<td>• Stabilizing areas of atelectasis</td>
<td>• Overdistention</td>
<td></td>
</tr>
<tr>
<td>• Stabilizing obstructed airway</td>
<td>• Impede venous return</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Airleak</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>High (8-10 cm H2O)</th>
<th>Use</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Preventing alveolar collapse with poor CL and poor lung volume distended</td>
<td>• Airleak</td>
<td></td>
</tr>
<tr>
<td>• Improving distribution of ventilation</td>
<td>• Decreased CL if over</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• May impede venous return</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• May increase PVR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• CO2 retention</td>
<td></td>
</tr>
</tbody>
</table>

CL – Compliance
PVR – Pulmonary vascular resistance
Effects of CPAP

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 oxygen available to tissue
- Increase 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 PCO₂ may initially fall, but may 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 distention 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 ‘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 headbox 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. Headbox CPAP is not used currently. The significance of

---

**Fig. 2.2:** Effect of CPAP on central venous pressure. (Gregory GA. Continuous positive airway pressure. Neonatal Pulmonary care. 2nd ed. Norwalk, CT, Appleton and Lang, 1986. p 355)
this finding in clinical practice is not evidenced with the current CPAP devices and protocols.

It is evident that when CPAP pressure exceeds 6 cm of H$_2$O and especially when it nears 8 cm of H$_2$O, significant increase in central venous pressure (Fig. 2.2) coincides with decreases in PaO$_2$ levels and increased PaCO$_2$ levels.