

Webinar : CPAP SCIENCE

Slide No.	Text matter to be spoken	Remarks
1.	In this Webinar we will learn about the science behind and physiology of antenatal steroids, continuous positive airway pressure or CPAP & Surfactant	
2.	Let us understand and learn How antenatal steroids work, how does CPAP work, and what is the mechanism of working of surfactant. In addition, we will try and learn the synergism between ANS, CPAP & surfactant in management of a preterm neonate with RDS.	
3.	<p>Let us begin by realising what is our quantum of preterm births.</p> <p>India has the dubious distinction of having the highest number of preterm births and neonatal deaths due to prematurity.</p> <p>Out of an estimated 2.6 crore live births in India each year, 35 lakh babies are born preterm, and out of these, 3 lakh babies (10% approximately) die due to complications of preterm births</p>	
4.	<ul style="list-style-type: none"> • Preterm neonates are vulnerable and Fetal lung immaturity is the principal contributor for neonatal mortality • Therefore lung has been the primary focus of strategies to improve the survival of newborn infants <p>Strategies for the prevention and treatment of RDS have been directed towards the acceleration of fetal lung maturation in utero mainly by administering ANS to the mother and CPAP with or without surfactant to neonate</p>	
5.	<p>Out of these three potentially useful strategies for the preterm neonate let us learn the science and understand how antenatal steroids work.</p> <ul style="list-style-type: none"> • Antenatal steroids accelerate development of pneumocytes, thus improving lung mechanics (maximizing lung volume and 	

	<p>compliance) and thus gas exchange.</p> <ul style="list-style-type: none"> • It increases surfactant production. <p>There is not just induction of surfactant release but also absorption of alveolar fluid and increase of lung antioxidant enzymes; resulting in reduction in RDS, reduction in Intraventricular hemorrhage, Necrotising enterocolitis, systemic sepsis and mortality.</p>	
6.	<p>After having learnt about Antenatal steroids Let us now talk about CPAP.</p> <p>First thing in CPAP is to know What is CPAP. Well CPAP as it is abbreviated is continuous positive airway pressure --- which really refers to application of continuous pressure to the airways during both inspiration and expiration in a spontaneously breathing baby</p>	
7.	<p>Now we will try and understand what does CPAP do? Well CPAP keeps the alveoli open by providing constant airway pressure, as also splints the airways so that they do not collapse. This thus increases the functional residual capacity (FRC) of the lungs resulting in better breathing and better gas exchange culminating in lesser lung injury.</p>	
8 & 9.	<p>Let us understand how CPAP helps –</p> <p>In a baby with RDS the Functional Residual Capacity of Lung i.e. FRC is reduced. FRC is the volume of air present in the lungs at the end of normal expiration. Reduced FRC allows alveoli to collapse at end of expiration.</p> <p>What CPAP does is - it increases the FRC which helps to keep the alveoli open and decreases the Ventilation Perfusion mismatch thus improving oxygenation, washing out carbon dioxide and improving the pH.</p> <p>It splints the upper airways preventing obstructive apnea and dilates the lower airways thus reducing the airway resistance.</p> <p>Overall the Tidal volume improves and the work of</p>	

	<p>breathing decreases. With improved physiology the pneumocyte 2 which produces surfactant functions better with better recycling and production of surfactant.</p> <p>Overall this culminates in improved lung compliance and better gas exchange.</p>	
10.	<p>Let us understand why conventional mechanical ventilation is not the best modality for managing RDS. I would like to illustrate this by sharing with you the animal experiments which will convince that CPAP is better than conventional ventilation.</p> <p>Here is preterm Lamb lung in which alveolar septa are damaged following 24 hours of ventilation. Though the FRC is increased following ventilation but the effective surface area for gas exchange has decreased. In preterm, the alveolar wall is thin with less supporting tissue , hence more prone to damage.</p>	
11.	<p>Notice in this preterm lamb, following 72 hours of conventional ventilation the inter alveolar wall thickness is 3 to 4 times when compared with another in which nasal CPAP is given. With thickened alveolar wall the gas exchange will become difficult in preterm.</p>	

12.	<p>The greatest hazard to preterm baby lung is due to endotracheal tube causing Biotrauma.</p> <p>In CV Many alveoli at end of expiration collapse due to low peak end expiratory pressure; – the sudden opening and closing of alveoli leads to atelecto trauma; while in CPAP due to constant continuous pressure this injury is minimal.</p> <p>CV causes Barotrauma due to high mean airway pressure and variable Peak Inspiratory Pressure while in CPAP pressure delivered is much less . Unregulated Tidal volume in CV causes Volutrauma.</p> <p>So CPAP is like a Magic. It opens the lung at FRC for better gas exchange and keeps the lung open with minimal pressure in stark difference to CV.</p>	
	<p>Thus there is negligible barotrauma, Baby is breathing spontaneously deciding its Tidal Volume , so no volutrauma but most important as there is no endotracheal tube there is no bio-trauma . Pulmonary arterial pressures are least with improved blood flow, hence less VQ mismatch.</p>	
13.	<p>Having understood the magic of CPAP, Let us understand the relationship between lung volume depicted on the X axis and pulmonary vascular resistance shown on the y axis. Pulmonary vascular resistance (PVR) is least once the lung is open at the FRC, at this the blood flow is maximum with best ventilation perfusion matching and gas exchange.</p>	
14.	<p>You can see on Right- baby with meconium aspiration syndrome has higher lung volume while on left a baby with hyaline membrane disease has lower lung volume but both result in high PVR. In clinical practice, for a baby on CPAP with higher pressure the lung will over distend while low CPAP will cause reduced FRC – in both PVR will increase causing Rt to left shunt impairing pulmonary blood</p>	

	<p>flow. Thus we must ensure lung is opened at FRC for best blood flow and better gas exchange.</p>	
15.	<p>Alveoli are lined with water molecules which tend to pull alveoli towards centre – this can be nullified by CPAP pressure or by reducing the surface tension by giving surfactant and if both are done simultaneously synergistic action can be obtained. This has gone into clinical practice and is called as INSURE.</p>	
16.	<p>Going further from this let us see how surfactant works. Pulmonary surfactant is a surface-active lipoprotein complex (phospholipoprotein) formed by <u>type II alveolar cells</u>.</p> <p>The proteins and lipids that make up the <u>surfactant</u> have both <u>hydrophilic</u> and <u>hydrophobic</u> regions. By <u>adsorbing</u> to the air-water <u>interface</u> of <u>alveoli</u>, with hydrophilic head groups in the water and the hydrophobic tails facing towards the air, the main lipid component of surfactant, <u>dipalmitoyl phosphatidyl choline</u> (DPPC), reduces <u>surface tension</u>.</p> <p>They thus increase <u>pulmonary compliance</u>, prevent <u>atelectasis</u> (collapse of the lung) at the end of expiration and also facilitate recruitment of collapsed airways.</p>	
17.	<p>Hence, we learnt CPAP is safe as it causes less lung injury. One should give optimal CPAP to open the lung at FRC as pulmonary vascular resistance is least with maximum blood flow. Use of surfactant and CPAP together is beneficial in RDS . It is also very important to understand that CPAP will give maximum dividends if you use antenatal steroids for preterm labor, early surfactant when required and have good delivery room care.</p>	