Introduction
Respiratory distress is one of the commonest disorders encountered within the first 48 - 72 hours of life. It occurs in 0.96 to 12% of live births and is responsible for about 20% of neonatal mortality. Respiratory pathology is the commonest (32-54%) autopsy finding among early neonatal deaths. The spectrum of respiratory distress in neonates includes pneumonia, transient tachypnea of the newborn, hyaline membrane disease, meconium aspiration syndrome and other miscellaneous causes. The male: female ratio was found to be 2:1. The higher incidence in male is probably due to their bigger size and relative immaturity as compared to female infants of same weight group. In developing countries there is a paucity of studies on causes of respiratory distress in neonates and all respiratory distress in neonates are treated as pneumonia at the first referral unit.

The importance of respiratory distress in the neonates can be realized from the fact that the neonates with respiratory distress are 2-4 times more likely to die that those without respiratory distress. The knowledge of the causes of respiratory distress is important to plan facilities.
To prevent injury by microorganisms and foreign substances, a variety of defense mechanisms have evolved, both systemic and within the respiratory tract.

In the study by NB Mathur (2003), many of the defenses are compromised in the fetus and newborn infant. Newborn infants typically have sterile respiratory mucosa at birth. Access to distal respiratory structures and bypass of the ciliary escalator occurs in infants who require endotracheal intubation. In these infants, increased physical disruption of epithelial and mucous barriers also occurs. Exposure to high oxygen concentrations and airway pressures interferes with ciliary function and mucosal integrity. Secretory antibodies and mucosal lymphoid tissue are absent or minimally functional for the first month of life. Circulating complement components are present at approximately 50% of the concentration found in older children, although components of the alternative pathway are present in sufficient quantities to serve as effective opsonins. The neonatal granulocytes frequently decrease in response to early infection, while the phagocytes that are present often move much more sluggishly to the inflammatory focus. Interalveolar communications (pores of Kohn) are few in number in the neonate and become prominent only after the age of 7 years. The absence of
this ‘collateral ventilation’ probably increases the risk of pulmonary 
atelectasis in the neonate.

The diagnosis of clinical conditions producing respiratory 
distress is usually based mainly on careful scrutiny of the history, 
clinical and radiological findings.

According to Avery ME, Fletcher BD (1974), the infant with HMD 
is almost always premature and is cyanotic in room air. There is rapid 
or labored breathing beginning at or immediately after birth. The 
severity of respiratory distress can be represented by the Silverman 
score.

Infants usually have a characteristic grunt during expiration, 
caused by closure of the glottis, the effect of which is to maintain lung 
volume and gas exchange during exhalation. Frequently the 
unventilated infant requires 40% to 50% oxygen after birth for relief of 
central cyanosis but then develops an increasing oxygen requirement 
over 24 to 48 hours; this may reach as high as 100%.

Clements and Tooley (1977), described diffuse fine granular 
densities that develop during the first 6 hours of life are seen on the 
chest radiograph, these densities are influenced by size of the infant, 
severity of disease, and degree of ventilatory support. The 
appearance may be more marked at the lung bases than at the 
apices. The lung volume is decreased.
According to Avery ME, Fletcher BD (1974), HMD was a problem of insufficient lung maturity, the best way to prevent it is to prevent premature birth. 1). Prediction of the risk for HMD by antenatal testing of amniotic fluid samples, and 2). Antenatal treatment of women in preterm labor with glucocorticoid hormones to accelerate fetal lung maturation.

William H Tooley, found that the second-born twin is more likely to be affected, and a family history of HMD increases the risk for any premature infant.

Another more rapid test for lung maturity is the foam stability or shake test, which was described by Clements and associates (1975). This test is based on the ability of pulmonary surfactant to form highly stable surface films that can support the structure of a foam for relatively long periods.

Meconium is biochemically composed of a mucopolysaccharide of high blood group specificity, a small amount of lipid, and a small amount of protein that decreases throughout gestation. Its blackish green color is the result of bile pigments.

According to Nathan et al (1994), it is possible that the passage of meconium in utero is the result of transient parasympathetic stimulation from cord compression in a neurologically mature fetus.
MSAF in connection with fetal heart rate abnormalities is a marker for fetal distress and is associated with an increased perinatal morbidity.

As per Yeoman S et al (1989), if meconium is not removed from the trachea after delivery, with the onset of respiration it migrates from the central airways to the periphery of the lung. Initially, particles of meconium produce mechanical obstruction of the small airways that results in hyperinflation with patchy atelectasis. Later, small airway obstruction is the result of chemical pneumonitis and interstitial edema.

Infants with MAP are often postmature and have visible meconium staining of the nails, the skin and the umbilical cord. Many infants with MAP have been asphyxiated, and much of the early distress may relate more to asphyxia and retained fetal lung fluid complicated by elevated pulmonary vascular resistance than to the presence of meconium in the airway. Infants with MAP have clinical evidence of lung overinflation, with a barrel chest. Auscultation of the chest reveals diffuse rales and ronchi. The chest radiograph shows patchy areas of atelectasis and areas of overinflation.

Hyperaeration is evident by hyper translucence, horizontal ribs and depressed domes of diaphragm (with more than 7 intercostal spaces being visible). Findings are characteristically non-uniform and asymmetric.
Avery and associates (1966), found that persistent postnatal pulmonary edema is more common in boys. The disorder typically begins soon after birth with a rapid respiratory rate, ranging from 60 to 160 per minute, and sometimes with sternal and subcostal retractions of the chest wall, grunting, during expiration, and occasionally, mild cyanosis that disappears with the delivery of supplemental oxygen.

The radiographic picture of TTNB is characterized by prominent hilum and symmetrical streaky opacities emanating from the hila because of excess interstitial fluid. The pleural fluid tracks into the inter-lobar fissure which appears prominent. At times, there may be a small pleural effusion, minimal cardiac enlargement and mild hyperaeration.

Lower incidence of Respiratory Distress Syndrome in India and also in postmortem finding of Indian authors as compared to that of Western countries is possibly due to failure of many premature infants to survive for a certain minimum period to have developed pulmonary hyaline membrane and relative maturity of pulmonary alveolar epithelial cells in response to more corticosteroid secretion by our mothers in response to stress and higher rate of infection.

Naeye and Peter (1978), “In assessing the risk of neonatal infection to premature in premature infants, the important thing to
consider is not the length of membrane rupture but whether or not preterm labor occurred and whether or not amnionitis was present”.

In transplacental pneumonia, bacteria cross the placenta and invade the fetal lungs by the hematogenous route, as in congenital syphilis and in some cases of listeriosis with maternal septicemia. In transnatal pneumonia, there is no evidence of either preceding amnionitis or maternal infection, although the infant is believed to aspirate vaginal bacteria during the birth process. The onset of clinical signs of pneumonia is delayed for a few hours or days or even longer. A true inflammatory process in the lung is always present.

S.P. Khatua et al (1979) stated that, pneumonia is diagnosed from the history of early rupture of membranes with prolonged labour, recent febrile illness of the mother or operation in a dirty place, respiratory distress, fever in a full term infant.

Thomas Hansen and Anthony Corbet (1988), observed that the usual clinical picture is that of respiratory distress with onset at or soon after birth. Sometimes, however, the onset of respiratory distress is delayed, preceded by increasing tachypnea during the 1st day of life. Infants with infection often have poor peripheral perfusion and tachycardia.
Other signs are abdominal distention, temperature instability, unexplained metabolic acidosis, or excessive jaundice. Some infants progress to a state of septic shock.

The positive gastric aspirate is not diagnostic of pneumonia: it indicates only increased risk.

The gastric aspirate can also be evaluated with the foam stability (shake) test. If the test is positive, pneumonia is more likely (and RDS is very unlikely). A negative test may not distinguish between RDS and pneumonia.

Sherman et al (1980), stressed that, the blood culture should be done in all cases of suspected pneumonia as well as cerebrospinal fluid analysis because meningitis may also be present.

Sometimes the radiograph initially is normal, but later films show abnormalities developing over the first few days; this course is suggestive of a transnatal pneumonia.

NB Mathur et al (2003), found that the organisms responsible for pneumonia mirror those responsible for early-onset neonatal sepsis. Group B Streptococcus has been the most common bacterial isolate in the west. However, Group B Streptococcus is not commonly seen in India. Commonly implicated bacterial organisms in India include Klebsiella pneumoniae, Escherichia coli and Staphylococcus aureus. Transplacental pneumonia usually occurs in association with
congenital syphilis, cytomegalovirus, herpes virus, rubella, toxoplasma, listeria monocytogenes and mycoplasma infections. These infants show involvement of many organ systems and manifestations of pneumonitis may be obscured. Chlamydia is presumably transmitted at birth during passage through an infected birth canal, although most infants are asymptomatic during the first 24 hours and develop pneumonia only after the first 2 weeks of life.

Respiratory pathogens, such as respiratory syncytial virus, influenza, adenovirus, and others, may be transmitted by contact with infected family members or caregivers shortly after birth, but infection by these organisms rarely is manifested during the first 24 hours. Frequency of neonatal pneumonia due to chlamydia and viruses has not been evaluated in India.

Hallahakoon and Halliday (1995), observed that with transplacentally acquired infection there are diffuse, interstitial opacities giving a ground glass, reticular pattern which may be indistinguishable from RDS or there may be extensive consolidation. With ascending infection there may be alveolar involvement which produces bilateral coarse opacities which are much less uniform.

Pneumonia acquired following aspiration is usually less evenly distributed on chest radiograph and may show as segmental or lobar collapse.
Early contrast radiography studies show that 10-15% of newborn babies aspirate fluid into their lungs during the first few days after birth.

Aspiration can occur before birth and amniotic debris including squamous cells have been found in the lungs of stillborn babies. Aspiration of small amounts of fetal and maternal blood do not appear to cause major problems and are rapidly removed from the lungs. If purulent secretions are aspirated during birth there is an increased risk of subsequent bacterial pneumonia. Aspiration of milk may occur in the very preterm infant, those with swallowing disorders (Goodwin et al, 1985).

A number of factors may interfere with the ability to cultivate a likely pathogen from the sites noted, including the following: (i) pretreatment with antibiotics that limit in vitro but not in vivo growth, (ii) contaminants that overgrow the pathogen, (iii) pathogens that do not replicate in currently available culture systems, (iv) sampling of an inappropriate site, and (v) patients in whom the process is inflammatory but not infectious, such as with meconium aspiration.

Since the risk is increased, aspiration lung technique is usually reserved for circumstances in which empiric therapy fails after several days, less invasive cultures and detection tests are unrewarding, or the infant continues to deteriorate.
Mathur et al (2002) found that the presenting complaints in neonates with pneumonia include rapid breathing (83.4%), poor feeding (81%) and difficult breathing (79.1%). In primary neonatal care rapid breathing, poor feeding and difficult breathing are useful symptoms suggestive of respiratory distress. Presence of cough is significantly different in neonates with pneumonia, as compared to neonates with respiratory distress due to other causes. Cough, adventitious sounds and flaring of alae nasi had high specificity, while chest retractions, difficulty in feeding and RR > 60 have high sensitivity for diagnosis of pneumonia in neonates.

Earlier studies on neonatal pneumonia have included neonates with only radiological findings and have not considered blood culture positivity in diagnosis of neonatal pneumonia. Webber et al, however, had classified neonatal pneumonia as "definitive pneumonia" if respiratory pathogen was isolated from the blood and "probable pneumonia" if blood culture failed to show any pathogen, in presence of a positive chest X-ray. No earlier study has stated detailed diagnostic criteria for pneumonia in neonates utilizing blood culture or sepsis screen positivity. The National Neonatology Forum have included them in the diagnostic criteria but have not done any study evaluating utility of these criteria separately for diagnosis of pneumonia in neonates. Furthermore the diagnosis of pneumonia
based on a respiratory rate of more than 60 prescribed by the W.H.O. would also miss a significant number of cases.

A study done by Mathai et al (2004), regarding association between intrapartum risk factors for infection with CRP levels showed that several such risk factors can cause elevated CRP levels in the absence of infection. Since CRP does not cross placenta, the elevated levels are due to production of CRP in the neonate. Chorioamnionitis can result in elevation of IL-6 levels even in uninfected neonates. Stimuli other than infection, like hypoxia, trauma and metabolic changes can also induce production of proinflammatory mediators. This cytokine stimulates CRP production.

A prenatal risk score depending upon the foul smelling liquor, unclean vaginal examination done before delivery, duration of labour exceeding 24 hours, one minute apgar score of 0-6, duration of rupture of membrane before delivery exceeding 24 hrs., birth weight 2 kg or less and / or gestation less than 37 weeks can decide the amount of risk a neonate is exposed. Antibiotics can be safely withheld in the low risk group while antibiotics should be started in the high risk group infants. The moderate risk group must be investigated for the presence of infection and depending upon the circumstantial evidence may be given antibiotics.
Improved antenatal supervision, timely treatment of maternal diseases, improved obstetrical and neonatal management will go a long way in reducing the incidence of respiratory distress in newborn.