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"LUNG ULTRASONOGRAPHY SCORE TO EVALUATE OXYGENATION AND SURFACTANT NEED IN NEONATES TREATED WITH RESPIRATORY SUPPORT"

BY

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Dissertation submitted to

BLDE (DEEMED TO BE UNIVERSITY) VIJAYAPURA KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR IN MEDICINE IN PEDIATRICS

UNDER THE GUIDANCE OF

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GLOSSARY OF ABBREVIATIONS

MSAF	Meconium stained amniotic fluid
IR	Ionizing Radiation
NICU	Neonatal Intensive Care Unit
BPD	Broncho pulmonary Dysplasia
CXR	Chest X Ray
LUS	Lung Ultrasound Score
TTN	Transient Tachypnoea of Newborn
FIO2	Fraction of Inspired Oxygen
CHD	Congenital Heart Disease
PPHN	Persistent Pulmonary Hypertension of the Newborn
RCT	Randomized Controlled Trials
EOS	Early onset sepsis
LOS	Late onset sepsis
CPAP	Continuous positive airway pressure
HIE	Hypoxic Ischemic Encephalopathy
IUGR	Intra Uterine Growth Restriction
NVD	Normal Vaginal Delivery
LSCS	Low Segment Cesarean Section
APGAR	Appearance, Pulse, Grimace, Activity and Respiration
RDS	Respiratory Distress Syndrome
ECMO	Extra corporeal Membrane Oxygenation
ROC	Receiver Operating Characteristic
MAS	Meconium Aspiration Syndrome

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PEEP	Positive End Expiratory Pressure
HFOV	High frequency oscillatory Ventilation
HFJV	High frequency jet ventilation
INO	Inhaled Nitric Oxide
PROM	Prolonged Rupture of Membranes
ELBW	Extremely low birth weight
VLBW	Very Low Birth weight
ABCA3	ATP Binding cassette Transporter A3
CI	Confidence Interval
AUC	Area Under Curve
GA	Gestational Age
IVH	Intra ventricular Hemorrhage
INSURE	Intubation Surfactant Extubatiion
SCR	Sub costal Retractions
ICR	Inter costal Retractions
MV	Mechanical Ventilation
EF	Extubation Failure
ESTHER	Echography Guided Surfactant Therapy
PIH	Pregnancy Induced Hypertension
APH	Antepartum Hemorrhage
ABG	Arterial Blood Gas
SIMV	Synchronized intermittent mandatory ventilation

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Abstract

Background:

Lung ultrasonography (LUS) has emerged as a radiation-free alternative to chest X-rays, showing promise in assessing lung aeration and guiding therapeutic decisions in neonates with respiratory distress. Despite its potential, widespread adoption of LUS requires standardized protocols and further validation against traditional imaging modalities like CXRs.

Aim

This study aimed to evaluate the effectiveness of Lung Ultrasonography Score (LUS) in assessing oxygenation levels and identifying the need for surfactant therapy in neonates receiving respiratory support at a Level IIB Neonatal Intensive Care Unit (NICU).

Methodology:

This prospective observational study was conducted from January 2021 to June 2022 at a Level IIB Neonatal Intensive Care Unit (NICU) in Karnataka, India. The study included neonates <34 weeks and >34 weeks gestational age admitted with respiratory distress, excluding those with chromosomal abnormalities or congenital lung diseases. Lung ultrasonography scores (LUS) were assessed upon admission and before surfactant administration, using a scoring system ranging from 0 to 3 across defined thoracic regions to evaluate lung aeration. Statistical analysis included correlation tests and ROC curve analysis to assess LUS's ability to predict oxygenation parameters and need for surfactant therapy.

Results:

A total of 75 neonates were included in the study, with mean LUS scores correlating significantly with arterial to alveolar ratios ($r = -0.74$, $p < 0.001$) and PaO2 values ($r = 0.68$, $p < 0.001$). LUS demonstrated good predictive accuracy for surfactant therapy need, with an area

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under the ROC curve of 0.85 (95% CI: 0.75-0.95). Neonates requiring surfactant therapy had significantly higher mean LUS scores compared to those managed conservatively ($p < 0.001$).

Conclusion:

These findings underscore the utility of LUS in assessing lung aeration and guiding clinical management decisions in neonates with respiratory distress, potentially enhancing early intervention strategies in NICU settings.

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Introduction:

One of the most frequent causes of an infant's admission to the neonatal critical care unit is respiratory distress.[1] Significant respiratory morbidity affects 15% of term babies and 29% of late preterm babies admitted to the neonatal intensive care unit; the percentage rises for babies delivered before 34 weeks of pregnancy.[2] Neonatal respiratory illness is more likely to occur when certain risk factors are present. Prematurity, meconium-stained amniotic fluid (MSAF), caesarian section birth, gestational diabetes, maternal chorioamnionitis, and abnormalities in the lungs or oligohydramnios shown on prenatal ultrasound are some of these risks.[3,4] It is not always feasible to anticipate which babies may exhibit symptoms prior to delivery, however. Whatever the reason, respiratory distress may progress to respiratory failure and cardiopulmonary arrest if it is not identified and treated very once.[5] Surfactant treatment lowers the risk of pneumothoraxes, increases the survival rate of preterm newborns, and is crucial in the care of RDS.[6] The risk of bronchopulmonary dysplasia (BPD) and mortality from RDS is decreased with early identification and treatment.

Preterm newborns are exposed to ionising radiation (IR) via repeated chest X-rays (CXRs). Epidemiological research findings have shown that early childhood exposure to IR may raise the risk of cancer. With early exogenous surfactant replacement treatment, the mortality from RDS has dropped by around 50% in recent decades.[7]

Clinical research on neonatal lung ultrasonography is active and expanding, with significant clinical consequences.[8,9] This non-invasive imaging method at the bedside may be used to forecast the requirement for mechanical ventilation or noninvasive respiratory support, as well as to characterise the postnatal adaption and differential diagnosis of newborn respiratory distress.[10] By proving a strong association between the lung ultrasound score (LUS) and a

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number of invasive oxygenation indices, Brat et al.[11] have validated the LUS. The same group is now testing LUS as a potential standard for surfactant delivery as a result of these results.[12]

Lung aeration evaluation aids in illness diagnosis and serves as a reference for tracking therapeutic outcomes.[13] In preterm newborns with RDS, the initial LUS classifications using a six-area assessment demonstrate an inverse connection with lung aeration.[14] In order to get around the limits of CXRs, LUS has become a viable alternative for doctors to supplement physical examination results. Additionally, LUS has been effectively used in NICUs for the detection of several illnesses, including RDS, transient tachypnea of the Newborn, pneumonia, meconium aspiration syndrome, and pneumothorax. In some severe circumstances, LUS even demonstrates superior diagnostic accuracy than CXR. Although there is a danger of radiation exposure, repeated CXRs are often required for the diagnosis and monitoring of these conditions.

It was hypothesised by Escourrou and De Luca that using LUS might lower the chance of being exposed to IR.[15] LUS is still not widely used, despite mounting evidence that it is a valid and dependable diagnostic method for RDS. The widespread use of LUS requires clear guidelines, standards, and instruction. In 2017, a systematic review and meta-analysis examined the diagnosis accuracy of LUS in newborns with RDS in comparison to reference standard clinical tests and CXRs. The results showed that LUS had a total sensitivity of 97% and specificity of 91%. This analysis did highlight the possibility that LUS might overlook air leak syndromes, hence CXRs are still required for suspected newborn RDSs.[16]

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Aim and Objective:

Aim:

To evaluate the effectiveness of Lung Ultrasonography Score (LUS) in assessing oxygenation levels and identifying the need for surfactant therapy in neonates receiving respiratory support.

Objective:

1. To evaluate the effectiveness of Lung Ultrasonography Score (LUS) in assessing oxygenation levels in neonates receiving respiratory support.
2. To determine the effectiveness of LUS in identifying the need for surfactant therapy in neonates under respiratory support.

Review of literature:

Respiratory distress in neonates:

When a newborn has to be admitted, respiratory distress is often a symptom.Its relevance spans from potentially fatal diseases to a self-limiting process brought on by a delayed adaptation to the postnatal environment. Respiratory issues or non-respiratory pathologies such as neurologic, cardiovascular, metabolic, hematologic, or neuromuscular diseases, as well as sepsis, drug withdrawal, and other illnesses such severe anaemia, might be the underlying cause of distress. Tailoring the care according to the aetiology is made feasible by a good history to identify potential risk factors and a clinical examination of the infant with supporting data from investigations. While all babies with respiratory distress need standard supportive measures, such as the administration of oxygen, thermal support, and enough hydration and caloric. A precise diagnosis is necessary for a particular action. Most babies will recover fully from their

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respiratory infection on their own, although treatment and prognosis greatly depend on the underlying diagnosis.

Transient Tachypnoea of newborn

The most frequent cause of respiratory distress in term newborns is transient tachypnoea of the newborn (TTN). Usually innocuous, it goes away in 24 to 72 hours. The majority of afflicted infants are delivered either late preterm or term. Delays in the removal of foetal lung fluid are the cause of it. The afflicted baby often appears with limited or no retractions, tachypnoea, and sometimes even cyanosis. FiO2 <40% is administered to alleviate respiratory distress and enhance oxygen saturation.[17]

Risk factors:

Risk factors	Cesarean section
	Precipitous labour
	Preterm birth (including late preterm), at earlier gestations other comorbidities like Respiratory distress syndrome (RDS) also occur.
	Male gender
	Macrosomia
	Maternal diabetes
	Family history of asthma
	Multiple gestations

Clinical presentation:

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Symptoms typically manifests six hours postpartum, presenting as tachypnoea. little to no retractions, grunting, nasal flaring, and sometimes cyanosis. Supplemental oxygen at Fio2<40% generally alleviates mild to moderate discomfort. A chest shaped like a barrel is the consequence of hyperinflation brought on by airway blockage and trapped air. When bilateral air ingress is sufficient during auscultation, crackles might be audible.In moderate situations, the distress begins to get better and ends entirely in 12 to 24 hours; in severe cases, it may persist up to 72 hours.[18]

Chest x ray:

Chest radiography in TTN reveals hyperinflation with widening intercostal spaces, fluid collections at the interlobar fissure, dilated lymphatics, coarse, fluffy densities indicating alveolar edema, and occasionally mild cardiomegaly and mild pleural effusions. These factors contribute to the retention of lung fluid, particularly in the perihilar region (sunburst pattern). In TTN, radiological clearing begins 12 to 18 hours in advance, and full resolution occurs 48 to 72 hours later. This sets TTN apart from pneumonia and meconium aspiration. Pneumothorax, lung abnormalities, and respiratory distress syndrome are a few other aetiologies that may be ruled out by CXR.[17,18]

Treatment:

Management basically acts as a support system by supplying more oxygen as needed. In severe circumstances, continuous positive airway pressure (CPAP) may cause a reaction. In case of continued distress, nasogastric feedings or intravenous fluids can be required. Diuretics should not be used. While TTN is generally benign, problems may arise with further oxygen treatment. A higher risk of air leak is linked to CPAP. TTN has a good prognosis with reduction in the chance of recurrence.[19]

Birth Asphyxia

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One of the most frequent reasons for neonatal hospitalisation is perinatal asphyxia, which also raises the risk of serious illness and death in infants. It is a worldwide issue that mostly affects underdeveloped nations. Only around 1% of infants need significant resuscitation attempts, out of the approximately 10% of live births that require them.[20] After hypoxia, there are two stages to the neuronal injury: the initial phase, which is followed by a temporary recovery phase during which potential therapeutic treatments may be used, and the secondary phase. Reperfusion damage from free radical production also occurs after reperfusion after a hypoxic phase.As resuscitation techniques have improved, the percentage of asphyxia resulting in hypoxic ischemic encephalopathy is decreasing. Nevertheless, 5 to 10 per 1000 live births have severe HIE, and roughly 1/4 of these newborns have long-term neurological consequences.[21]

Clinical manifestations:

The initial sign of foetal hypoxia is antenatally IUGR accompanied by increased vascular resistance and either no end diastolic flow or a reversal of it on Doppler.Foetal bradycardia and a decrease of beat-to-beat variability are seen in the foetal cardiotocograph during the intrapartum period. Variable or late decelerations are also observed in continuous foetal heart monitoring. These are indications for oxygen supplementation to the mother to lessen foetal hypoxia and for pregnancy termination to lessen foetal brain damage and mortality, particularly in near-term newborns. Amniotic fluid that has meconium stained in indicates that the foetus is in distress. The afflicted infant is apneic after birth, with a low tone, no or faint scream, and no attempt to breathe. This hypotonia persists throughout the first several hours before transitioning to hypertonia or normal tone. Additional symptoms of HIE include pallor, cyanosis, apnea, bradycardia, and lack of response to stimulus. Within the following 24 hours, cerebral edema develops, which causes significant depression of the central nervous system. Seizures ensue, which may be challenging to manage with standard anticonvulsants. Even while the primary cause of the encephalopathy linked to hypoxia in these infants is

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hypoglycemia, hypocalcemia, and infection, these factors should also be taken into account. Hypoxia not only damages the central nervous system but affects other essential organs, causing multiorgan dysfunction. Acute kidney damage is most often caused by kidney involvement, which is followed by heart failure and cardiogenic shock, chronic pulmonary hypertension, and gastrointestinal perforations.[22]

Diagnosis:

The main studies are electroencephalography and imaging. MRI is the better option than CT for determining the amount of damage. When forecasting the long-term result, amplitude-integrated electroencephalography, or EEG, might be helpful. This process helps to guide treatment and prognosis since it gives valuable data throughout the therapeutic window.[23]

Treatment:

In term newborns with HIE, whole body (systemic) or selective cerebral therapeutic hypothermia lowers death or significant neurological damage. Systemic hypothermia may lessen neurological damage by causing a more even cooling of the brain and deeper CNS components. However, there are a number of difficulties linked to it. The first-line antiepileptic for seizures is phenobarbitone. For refractory seizures, phenytoin or lorazepam are used. A poor prognosis is linked to status epilepticus, multifocal seizures, and the use of numerous anticonvulsant drugs during therapeutic hypothermia. It's critical to manage the multiorgan dysfunction linked to the illness. Avoiding hyperthermia is advised since it might have a negative impact on the nervous system. It's important to guarantee other supporting measures including proper ventilation, blood pressure, and pH. It's also necessary to use antibiotics to manage infections.

Prognosis:

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In milder situations, the prognosis of HIE might range from full recovery to serious brain impairment and death. The outcomes of tests such as EEG and MRI may also be used to predict prognosis. Neurodevelopmental issues should be monitored in all survivors of moderate to severe HIE.[23]

Meconium Aspiration Syndrome

Infection and/or acute or chronic hypoxia are conditions that facilitate the passage of meconium during pregnancy. This may cause the gasping foetus or infant to aspirate meconium during the intrapartum or postpartum phase, respectively. Meconium-stained amniotic fluid often occurs in term or post term newborns and is detected in 10-15% of deliveries. In 5% of these babies, meconium aspiration syndrome (MAS) develops; 30% need mechanical breathing, and 3-5% of them pass away. Meconium stimulates cytokines and releases inflammatory mediators, which results in lung damage. The action of surfactants is likewise inhibited by aspirated meconium. At delivery, the meconium-stained newborns may be depressed and need resuscitation.[24]

Clinical manifestations:

Respiratory distress in the first hours after delivery, characterised in seriously afflicted babies by increased breathing rate, retractions, grunting, and cyanosis. A ball valve mechanism traps air when there is a partial blockage of the airways, which may lead to air leak syndromes. A chest that is overextended and shaped like a barrel may result from hyperinflation in certain lung regions. A substantial risk of death exists when the sickness is severe enough to need mechanical breathing, although mild to moderate cases usually recover in 72 hours or less. A tachypnea attack might last for a few days or even weeks.[25]

Chest radiograph:

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The characteristic roentgenographic findings include regions of consolidation, frequently worse on the right, diffuse, asymmetric patchy infiltrates, hyperinflation, and diaphragmatic flattening. When a baby with severe hypoxia and no heart abnormalities has a normal chest radiograph, pulmonary hypertension should be suspected.

Prevention:

It is crucial to monitor the health of the foetus in all high-risk moms in order to detect meconium passage early on.Timely delivery and timely detection of foetal distress may reduce the risk of meconium aspiration.

Treatment:[25]

For respiratory distress, oxygen therapy and supportive care are part of the treatment

Oxygen Supplementation:

In mild situations, free flow oxygen is provided.

CPAP:

When Fi02 requirements rise beyond 40%, CPAP should be taken into account. While CPAP may help ease discomfort, each baby's needs must be catered to since it may exacerbate air trapping and raise the possibility of air leak syndromes.

Mechanical ventilation:

Severe diseased babies often need mechanical ventilation. When a newborn with MAS, significant peak inspiratory pressures are required during ventilation. To reduce air trapping, PEEP should be kept to a minimum and expiratory duration should be sufficient.Babies who

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have respiratory insufficiency that is not responding well may need extracorporeal membrane oxygenation (ECMO).

Surfactant:

Meconium prevents the action of surfactants. Instilling surfactants may enhance oxygenation, lessen pulmonary problems, and eliminate the need for ECMO. However, it is not recommended to use surfactant regularly to treat MAS. Even so, it could be beneficial for newborns who are not responding to conventional care.

Complications:

About 15% to 30% of infants with meconium aspiration syndrome have complications due to air leak syndromes, such as pneumothorax or pneumomediastinum. Mechanical ventilation and CPAP increase the risk of an air leak. About 30% of cases are complicated by persistent pulmonary hypertension in newborns, which further raises the mortality rate related to MAS.[26]

Persistent Pulmonary Hypertension of Newborn

A malfunction in the transition from foetal to neonatal circulation causes persistent pulmonary hypertension of the newborn (PPHN), a disorder marked by increased pulmonary resistance and a right-to-left shunt across the ductus arteriosus. Systemic hypoxia is the consequence of the foetal circulation's persistence, which lowers blood flow to the lungs and, in turn, oxygenation at the pulmonary vascular bed.[27]

Epidemiology:

Babies delivered on time or after may have persistent pulmonary hypertension of the newborn (PPHN). Risk factors include hypoglycemia, polycythemia, respiratory distress syndrome, perinatal asphyxia, meconium aspiration, EOS, NSAIDS, SSRI use in the third trimester that causes ductus arteriosus constriction, and pulmonary hypoplasia linked to defects like CDH,

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Potters sequence, etc.Most PPHN cases are idiopathic. However, a small number may have decreased arginine and nitric oxide levels, with a probable cause of a malfunction in nitric oxide production.[28]

Clinical manifestations:

Distress in PPHN often begins during the first 12 hours of life or just after delivery. In cases of polycythemia, hypothermia, hypoglycemia, or hypoplasia of the lung, the newborn may exhibit severe cyanosis, increased respiratory rate, and minimal retractions. In cases of MAS, CDH, or hypoplasia of the lung, the newborn may exhibit marked distress, including grunting, nasal flaring, retractions, and shock. Myocardial dysfunction as a consequence might lead to cardiogenic shock. Usually, hypoxia is more severe than anticipated.[28]

Diagnosis:

For every newborn who is cyanotic, a differential diagnosis of PPHN has to be made.[29]

A. Cyanotic baby may also have congenital heart disease, parenchymal lung disease, or sepsis as further differential diagnoses.
B. The afflicted infant often exhibits cyanosis and respiratory distress.Certain individuals may exhibit varying degrees of cyanosis in the preductal and postductal areas. Other observations in PPHN include exaggerated S2, systolic murmur of TR, and precordial pulsations.
C. A right to left shunt is indicated by a PaO2 or SpO2 gradient of 10% or more between the preductal (right upper limb) and postductal (lower extremities). In this case, ruling out structural lesions increases the likelihood of PPHN.
D. CXR either shows the accompanying abnormalities, such as pulmonary hypoplasia, or is normal.

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E. In order to prove right-to-left shunting and rule out structural anomalies, an echo is required.

Treatment

PPHN is an emergency requiring quick action to enhance lung perfusion and rectify hypoxia. Failure to do so leads to malfunction of several organs. In order to facilitate the shift from foetal to neonatal circulation, supporting measures are provided.[28]

Supplemental oxygen:

The cornerstone of treatment to lower pulmonary vascular resistance is oxygen supplementation. Sustaining saturation levels between 90 and 98% ensures sufficient oxygenation and prevents the negative consequences of hyperoxia.

Intubation and mechanical ventilation:

Mechanical ventilation is indicated in PPHN in the following cases: respiratory failure as seen by elevated PaCO2 and reduced pH; persistent hypoxia despite maximal oxygen delivery. The illness course is unstable, and the final result is unpredictable, thus it's crucial to carefully reduce oxygen support.The underlying disease causing PPHN is taken into consideration while adjusting the ventilator settings. High-frequency oscillatory ventilation (HFOV) or high-frequency jet ventilation (HFJV) is recommended in disorders affecting the lung parenchyma, particularly in multiple sclerosis (MAS). In these situations, HFO is also helpful when administering nitric oxide treatment.

Inhaled nitric oxide:

A component of the vascular endothelium is nitric oxide.Nitic oxide that is inhaled causes the pulmonary vasculature to dilate with little to no systemic impact, which reduces pulmonary vascular resistance specifically. It helps lessen the need for extracorporeal membrane

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oxygenation as well. At greater levels, methemoglobinemia poses a major toxicity.Rebound hypoxia, which happens when nitric oxide is abruptly stopped rather than tapered and stopped after making sure there is enough oxygenation with a minimum dosage of iNO and Fio2 < 50%, is another major consequence that has been seen.ECMO: Even in situations when inhaled nitic oxide therapy failed to improve, this life-saving procedure is still associated with a positive prognosis in around 75 to 85% of patients.

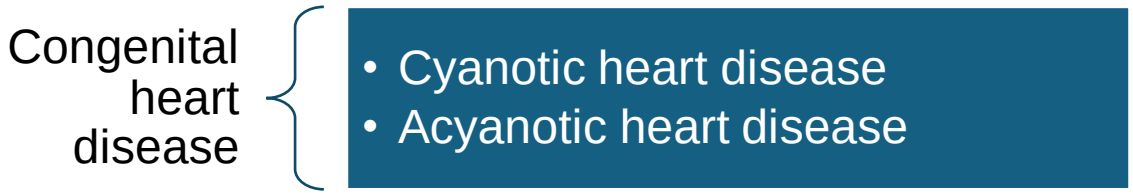
Prognosis:

The pathophysiology underlying the development of persistent pulmonary hypertension in neonates, related comorbidities, and the need for ECMO all affect survival.

Congenital Heart Disease

With a frequency of up to 10 per 1,000 live births, congenital heart disease (CHD) is one of the most frequent causes of neonatal death and morbidity.[30] In the birth period alone, 15% of these lesions are fatal.[31]

Congenital cardiac conditions are divided into:



Diseases with normal to enhanced blood flow to the lung with intracardiac mixing (complete or partial) and decreased pulmonary blood flow with an intracardiac right-to-left shunt are further categorised into congenital cardiac heart disease (CCHD).

Clinical evaluation:

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Various heart abnormalities have various onset times for symptoms, which are determined by changes in the circulation that take place after birth. Certain lesions may not be noticeable prior to release. Thus, it is challenging to diagnose these illnesses at an early stage. Numerous heart disease signs, such as hypotension and hypoxia, may also arise as a result of other infant diseases, which further complicates the diagnosis, particularly in preterm newborns.Lethargy, perspiration in the forehead, retractions, trouble feeding in the form of a suck rest suck cycle, and respiratory discomfort are a few signs that indicate a heart ailment.[32]

In cyanotic cardiac disorders, cyanosis is obvious, and pallor may also be present in certain circumstances. These infants have inadequate weight growth. These symptoms are nonspecific, not indicative of heart abnormalities on their own, and they don't show up in three quarters of neonates.Although cyanosis is a significant discovery in cyanotic heart disease, it may be challenging to understand in the first two days of life and in neonates with dark pigmentation. When this occurs, pulse oximetry might be helpful. A post-ductal oxygen saturation of less than 95% suggests that congestive heart failure may be present.

It might be difficult to distinguish between central cyanosis and respiratory illnesses since they both cause it. When determining the presence of an underlying heart condition and ruling out pulmonary illnesses, the hyperoxia test is useful. Cyanosis from hypoxemia may also arise from decreased respiratory drive from the central nervous system (CNS) in conditions such as hypoxic ischemic encephalopathy.

Management:

Early diagnosis is essential for the administration of preventative therapies like PGE1, which raises the probability of survival. Clinical suspicion, a chest radiograph, an electrocardiogram, and an echocardiogram are used to make the diagnosis. The underlying heart pathology

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basically directs the course of treatment. Heart disease may be treated with supportive measures, supplemental oxygen, antifailure treatments if necessary, and surgical preparation.[33]

Neonatal Sepsis

Severe disease exacerbates almost every illness that affects a baby and is a leading cause of morbidity and death in this age group.[34]

Types:

It is classified according to when it first appears as follows:

I. Early onset Sepsis (EOS): starts within 72 hours (some studies even up to 7 days) of life.

II. Sepsis with Late Onset.(LOS): in the fourth or eighth day of life.

This assists in selecting the right antibiotic regimen and raises suspicions about potential risk factors and the organisms that contribute to sepsis.Whereas LOS is generally caused by environmental factors that result in infection by hospital- or community-acquired organisms, EOS is mostly caused by maternal vaginal flora and perinatal risk factors. As a result, the infections that cause sepsis change over time. The most frequent bacteria causing sepsis up until a few years ago were Group A streptococcus and Streptobacterium pneumoniae. This is no longer the case, with E. coli and Group B streptococcus now being the most prevalent infections. These are the most prevalent organisms in industrialised nations; information about poor countries is scarce, however Group B streptococcus is less common there, whereas Staphylococcus aureus and Klebsiella species are more common pathogens.[34]

Clinical manifestations:

Sepsis does not have a characteristic trait. The symptoms are not particular, fever or hypothermia, Insufficient nourishment / Lethargic behaviour, Irritable cry. breathing difficulties and sleep apnea, nausea and jaundice. These characteristics may also be present in

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encephalopathy and inborn metabolic abnormalities, which further complicates the precise identification of sepsis in areas with inadequate laboratory resources. The presence of bacteremia in a blood culture is what defines sepsis. There might also be other infectious foci, such as meningitis, pneumonia, or UTI.A diagnosis is made when risk factors and sepsis-like symptoms are present. EOS is suggested by maternal fever, chorioamnionitis, PROM, and maternal UTI. Sepsis kills more premature and low birth weight newborns.[35]

Diagnosis:

The gold standard for diagnosing bacteremia is a blood culture. However, it is insensitive. Additional laboratory tests for sepsis include measuring the band ratio, leucocyte count, ANC, C reactive protein, and ESR.[36]

Treatment:

Following the identification of risk factors, empirical therapy is started, sepsis screen studies are sent, and antibiotics are administered until a negative result is obtained. Ampicillin and an aminoglycoside, which provide efficient coverage against both Gram-positive and Gram-negative organisms and hence demonstrate strong response for the major etiological agents, are the most often used antibiotic regimen for empirical therapy. The common agents that are frequent in the area should be taken into consideration while choosing an empirical antibiotic regimen. The antibiotic regimen is adjusted based on the sensitivity pattern as soon as the culture findings are received. Effective infection management requires the use of strategies throughout the antepartum, intrapartum, and postnatal stages of pregnancy.[37]

Neonatal Pneumonia

Pneumonia is a deadly illness that accounts for most newborn deaths worldwide. Another possible outcome is stillbirth. The primary difference in the causal agent is the timing of infection acquisition. The most frequent cause of pneumonia in the early newborn era is

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Grampositive organisms, mostly Group B streptococci; in the late neonatal period, pneumonia is caused by Gramnegative species, such as Escherichia coli and Klebsiella pneumoniae. Pneumonia severity is correlated with both birth weight and time of acquisition. Pneumonia in low birthweight newborns and those with an early neonatal start are more severe and have higher fatality rates.[38]

Epidemiology

Two categories of pneumonia exist based on when symptoms first appear:

I. Early onset pneumonia (<7 days) and

II. Late onset pneumonia (>7 days).

When a foetus aspirates amniotic fluid during delivery, microorganisms from the fluid enter the lungs and transmit maternal illness to the developing foetus. Listeria, tuberculosis, retrovirus, and TORCH infections may also be transferred from the mother to the foetus. As a result, the microorganisms that entered the lungs colonise, multiply, and create an infection that eventually led to pneumonia.

Aetiology:

Early-onset neonatal pneumonia: Pneumonia that develops within the first week of life is often caused by a mother's illness, such as chorioamnionitis, or by aspirating amniotic fluid contaminated with the infectious agent, which allows the infectious organisms to invade the lungs and cause pneumonia. Thus, the most frequent microorganisms causing early-onset pneumonia are those found in the mother genitourinary tract, such as Group B Streptococcus, Staph aureus, and S. pneumoniae.[39]

Diagnosis:

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Early-perinatal respiratory distress characterised by elevated respiratory rate, SCR, ICR, expiratory grunt, chest indrawing, and dyspnea, along with sepsis-like features such as reduced DTR, refusal of feeds, sluggish newborn reflexes, and temperature instability, should raise the possibility of an infectious cause for the respiratory distress, potentially pneumonia. Consolidation of a lung segment or lobe, patchy infiltrates, an air bronchogram, and lung haziness are CXR features indicative of pneumonia. Certain abnormalities may also be present in TTN, RSD, etc., but pneumonia is suggested by the persistence of these findings and the lack of radiological clearance within 48 hours.[40]

Treatment:

It is necessary to start using empirical antibiotics until the findings on culture sensitivity are available. a mix of aminoglycoside (gentamicin; 5 mg/kg od) and penicillin (Ampicillin; 50 mg/kg bd). When hypoxemia occurs, oxygen supplementation is crucial. Fio2 should be titrated according to saturation in order to prevent both hyperoxic and hypoxic harm. Gavage feeding and intravenous fluids are required until stabilisation since the afflicted neonates may be unwell and may find it difficult to feed themselves directly. Severe instances may not improve with more oxygen, in which case mechanical ventilation or continuous positive airway pressure are needed.[40]

Respiratory Distress Syndrome

Preterm newborns are the majority of those affected by respiratory distress syndrome.The primary risk factor is gestational age; the lower the gestational age, the higher the risk, and vice versa. The next significant factor is birth weight, which likewise has an inverse relationship and is associated with increased susceptibility in ELBW and VLBW newborns.The following is the risk percentage for gestational age:[41]

I. <28 weeks: 60–80%

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II. 15–30% for 32–36 weeks

III. >37 weeks - uncommon.

Additional risk factors include perinatal asphyxia, gestational diabetes, multiple pregnancies, LSCS, cold stress, and a history of afflicted siblings. A shortage of surfactant in the lungs causes respiratory distress syndrome.The chemical that reduces surface tension at alveoli and stops the lung from collapsing is called a surfactant. B (SP-B) and, to a lesser degree, C (SP-C) of the four surfactant-associated proteins (A to D) are essential for proper surfactant activity. In rare cases, deadly forms of respiratory distress syndrome in term newborns are caused by mutations affecting the ATP-binding cassette transporter A3 (ABCA3) gene and these surfactant proteins, B and C.

Clinical manifestations:

In preterm neonates, symptoms include increased respiratory rate, SCR and/or ICR, nasal flaring, grunting, and cyanosis. Onset occurs within six hours after birth, occasionally later. The first 24 hours see the worsening of the anguish, which is followed by stabilisation and recuperation in the next 72 to 96 hours, signalled by spontaneous diuresis.This path is altered by therapeutic intervention.[42]

Diagnosis:

Clinical symptoms, a characteristic chest radiograph image, and an ABG are used to establish the diagnosis. The following are typical observations on chest radiographs:

1. Reticulo nodular pattern.
2. Bronchogram of the air
3. Ground glass or white out lung.

Variable metabolic acidosis and hypercarbia ensue after the initial hypoxia.[43]

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Prevention:

1. Avoid the early and close term elective sections.
2. Suspecting immaturity of the lungs and treating the immaturity with treatments.
3. To reduce the risk of RDS and the related mortality, pregnant women with a gestational age of less than 34 weeks should receive antenatal corticosteroids.

Treatment:

The severity of the condition determines how it will be treated.

Mild cases: oxygen supplementation as well as supportive treatment.

Moderate to severe cases: Surfactant treatment with CPAP.

Constant pressure is provided by continuous positive airway pressure (CPAP), which preserves the lung's functional residual capacity. This reverses microatelectasis and lowers the likelihood that atelectasis may arise.Surfactant treatment is administered using the INSURE approach to very preterm infants (GA< 28 weeks) and newborns in acute distress who need mechanical ventilation. Term newborns may also need surfactant, like in the case of a diabetic mother's child and meconium aspiration syndrome, if there is insufficient surfactant function. For the treatment of RDS, recent research supports the use of CPAP alone without the addition of surfactant. The outcomes are similar to those who were given surfactant and mechanical ventilation. Thus, CPAP's function in settings with limited resources is encouraging.[43]

Prognosis:

The prognosis is influenced by many things.[44]

I. Gestational age: A higher gestational age is associated with better results.

II. The occurrence of other comorbid illnesses such as gestational diabetes, prenatal hypoxia, MAS, etc. that cause RDS.

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III. Additional issues related to preterm babies, such as sepsis, hypoglycemia, and hypocalcemia.

IV. Respiratory Distress Syndrome does not usually end in mortality; instead, sepsis, IVH, or pulmonary haemorrhage are the most common causes of death in premature infants.

Congenital Diaphragmatic Hernia

The symptoms of CDH include hypoplasia of the lung and protrusion of the abdominal contents into the chest due to a diaphragm abnormality.This is caused by an embryonic anomaly in which the structures that make up the diaphragm are either inadequately developed or do not fuse together, leaving the diaphragm defective. The primary cause of the morbidity and death linked to the syndrome is hypoplasia of the lung, which results from the mechanical action of the intestines disrupting lung development since the abdominal viscera herniated into the chest.[45]

Clinical manifestations:

Usually, the first signs of distress appear just after delivery. Upon auscultation, the afflicted side exhibits reduced air entry, while the opposite side's mediastinal contents move. On the side that is impacted, there may sometimes be a bowel sound that is unique to the illness. The abdomen seems scaphoid because the contents have ruptured into the thorax.

Diagnosis:

The results of the x-ray and the clinical findings determine the diagnosis. Bowel is seen in the chest on a chest x-ray, however if air hasn't reached the bowel, a water-density mass with a mediastinal shift is visible.[46]

Treatment:

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It is crucial to provide oxygen by mechanical breathing and endotracheal intubation. Avoiding CPAP and bag and mask ventilation is necessary since air entering the intestines during the treatment exacerbates the illness. To facilitate stomach decompression, a naso-or oro-gastric tube is immediately inserted. Since lung hypoplasia, which cannot be corrected surgically, is the primary cause of morbidity and death in CDH, the condition is no longer regarded as a surgical emergency.There is a higher risk of mortality if surgery is attempted right after delivery when the baby is not yet hemodynamically stable. Therefore, in order to achieve better results, surgery is now advised three to seven days after the newborn's overall state has stabilised.[46]

Extrapulmonary Air Leaks

Pneumothorax, pneumopericardium, pneumoperitoneum, and pneumomediastinum are examples of air leak syndromes. With a frequency of 1 to 2%, unilateral pneumothorax is often more prevalent among these symptomatic cases; this incidence rises with the use of mechanical ventilation, RDS, and MAS.[47]

Clinical Manifestations

Clinical signs include reduced breath sounds and hyperresonance on the afflicted side, along with abrupt onset respiratory distress. The heart is moved to the unaffected side, which causes the apical impulse to change. One helpful bedside test for identifying the illness is transillumination.

Diagnosis

A chest X-ray is used to confirm the diagnosis in suspected infants. Pneumomediastinum is indicated by hyperlucency surrounding the heart border and between the sternum and the heart border; the edge of the collapsed lung stands out in relief against the pneumothorax.

Treatment

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Conservative care may be used to mild, asymptomatic instances. A soft, tiny catheter inserted with a needle is recommended for emergency aspiration in situations of acute respiratory or circulatory impairment. Intercostal tube insertion or air drained into the underwater seal should be done either right after or following catheter aspiration.Air trapped in a pneumopericardium has to be evacuated immediately. Selective bronchial intubation may be helpful for severe localised interstitial emphysema.When a baby resists using a ventilator, prudent sedative usage may lower the chance of pneumothorax.[48]

Lung Ultrasound:

In the past, lung ultrasound was limited in its usefulness due to the inability of ultrasound waves to penetrate air, with its main recognized application being the assessment of pleural effusion. However, recent years have seen extensive demonstrations of its efficacy in managing various diseases clinically. Point-of-care ultrasonography has the potential to reduce reliance on chest x-rays, computed tomography (CT) scans, and other radiation-based imaging techniques in both Intensive Care Units (ICUs) and Emergency Departments (EDs). Integrating bedside ultrasonography into daily clinical practice among intensivists could mitigate risks associated with radiation exposure, patient transportation needs, and hospital costs, thereby optimizing patient management. Qualitative Lung ultrasound involves interpreting artifacts like A-lines and B-lines, along with real images, to distinguish between normal and pathological lung conditions. While qualitative assessment provides crucial insights into lung morphology for diagnosis, a quantitative approach further enhances the utility of lung ultrasound by enabling continuous lung monitoring.[49]

Lung Ultrasonography score:

A lung ultrasound (LUS) guideline has been put forward to help standardise the use of this instrument, which has become more and more common for evaluating newborn disease.

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Because of the tiny chest size, lack of fat, and light muscle mass, LUS can identify the newborn lung with ease and speed. Quick and easy LUS at the patient's bedside allows for timely diagnosis and treatment, as well as real-time information on pulmonary conditions. Studies have shown that doctors trained in LUS have a high level of interobserver agreement. Furthermore, LUS is well regarded for being simple to learn and having a high learning curve. The diagnosis and differential diagnosis of a number of newborn respiratory disorders are greatly enhanced by LUS. Notably, there was a significant decrease in the number of chest X-rays (CXR) performed in the NICU. Furthermore, compared to CXR, LUS is recognised to have a greater diagnostic accuracy.[50]

It is well established that many infant pulmonary disorders may be diagnosed with qualitative LUS. Semi-quantitative scoring methods have been developed since the emergence of LUS to guide clinical treatment and dynamically measure lung aeration. Nowadays, LUS has progressed from qualitative diagnosis to semi-quantitative assessment of lung disease. The anterior and posterior axillary lines were used as boundaries to partition the chest surface into three zones in order to standardise the scanning process and compute a pulmonary ultrasonography score:

The three regions are the anterior (from parasternal to anterior axillary line), posterior (from posterior axillary to paravertebral line), and lateral (from anterior to posterior axillary line).

In this method, each lung area is assigned a numerical score based on ultrasound findings, primarily focusing on the number of B-lines and/or sub-pleural consolidations. The specific score varies depending on the scoring system employed. By summing the scores from all areas, a comprehensive score is obtained, which facilitates the evaluation of the severity of neonatal lung disease in an individual infant. This also enables objective comparisons with other infants. The neonatal scoring system was initially introduced by Brat et al.[11]

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Each lung is divided into three regions: upper anterior, lower anterior, and lateral. Each region is scored from 0 to 3 as follows:

- 0: Presence of only A-lines.
- 1: Presence of three or more well-spaced B-lines.
- 2: Presence of crowded and coalescent B-lines, with or without subpleural space consolidations.
- 3: Presence of extensive consolidations.

The total score, ranging from 0 (completely normal) to 18, represents the infant's overall lung condition.

Fig 1: Division of chest wall

LUS in neonatal respiratory distress syndrome:

Respiratory distress (RD) that becomes worse gradually after delivery is a hallmark of neonatal respiratory distress syndrome (RDS). This prevalent illness, which mostly affects preterm infants, is brought on by a lack or malfunction of surfactants, which causes alveolar collapse and reduced lung aeration. When a newborn has severe or persistent transient tachypnea, there may be some surfactant degradation (TTN). Because they are more likely to have long-term respiratory consequences and may need recurrent surfactant therapy, very preterm newborns

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stand to gain the most from prompt and optimised surfactant administration. Early surfactant injection within the first two hours of birth lowers the risk of bronchopulmonary dysplasia (BPD), air leakage, and mortality.[49]

Since CXR only offers a static evaluation of lung aeration, it cannot be used to track lung recruitment during interventions. However, it may typically be used to measure lung aeration. The harmful effects of ionising radiation, to which newborns are believed to be particularly vulnerable, are the main obstacle to repeated CXR tests.[52] On the other hand, infant lung disorders may already be diagnosed using the LUS guideline. Compared to CXR, LUS performs better in the diagnosis of RDS because it is more sensitive and specific. LUS outperformed the clinical diagnosis or CXR findings in terms of sensitivity and specificity, with values of 97 and 91%, respectively, according to a systematic review.[16]

LUSs is a non-invasive technique that has a significant relationship to oxygenation status and prediction of non-invasive ventilation failure; in addition to being used as a diagnostic tool, it has also been proposed to monitor the progression of disease, to determine whether to perform a specific treatment, to assess the severity of RDS, and to evaluate the prognosis. Evidence from a multicenter research indicates that throughout the infant's NICU stay, there is a substantial link between LUSs and the inspired oxygen ratio/saturation.[10]

European recommendations state that when oxygen needs rise and FiO2 > 0.30 on CPAP pressure of at least 6 cm H2O, surfactant replacement should be carried out.[53] Arbitrary FiO2 thresholds, however, could not reliably indicate the state of oxygenation, and FiO2 needs might rise slowly, postponing the delivery of surfactants beyond the ideal window for maximum effectiveness. When comparing observable clinical signs to the earlier existence of ultrasound abnormalities indicative of RDS, the predictive usefulness of the LUSs about the requirement for surfactant treatment is related. This would allow exogenous surfactant therapy to be

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administered to neonates who may eventually have the clinical characteristics needed to comply with existing treatment standards.

The first research evaluating LUSs' capacity to forecast newborns with RDS's need for exogenous surfactant treatment was reported in 2015 by Brat et al.[11] Each lung was separated into three sections for the 130 newborns who were included in this research (upper anterior, lower anterior, and lateral). A score ranging from 0 to 3 was assigned to each lung region. Before surfactant was given, LUS was carried out as soon as the patient was admitted to the NICU. European principles formed the basis of the surfactant protocol. LUSs and oxygenation indices were shown to have significant relationships, suggesting lung aeration.

LUSs fared better in preterm newborns, according to the authors' findings. The area under the curve (AUC) was 0.93 with a 95% confidence interval (CI) of 0.86–0.99 in newborns whose gestational age (GA) was less than 34 weeks, while it was 0.71 (95% CI: 0.54–0.90) in infants whose GA was more than 34 weeks. When it came to preterm newborns with GA <34 weeks who were given continuous positive airway pressure (CPAP) treatment from birth, the LUSs demonstrated high reliability in predicting the delivery of surfactants. In order to provide exogenous surfactant treatment to kids in need as soon as possible—without waiting for their oxygenation to worsen—the LUSs assist in accurately identifying these babies. In a similar vein, a prospective research on the prognostic power of LUS for exogenous surfactant treatment required in babies with GA ≤30 weeks was reported by De Martino et al.[12] Both the lateral and anterior chest walls were scanned. Even after accounting for GA, the results indicated a substantial correlation between LUSs and the oxygenation index. With values of 0.98 and 0.93, respectively, the AUC for the worldwide population was 0.94 (95% CI: 0.90–0.98), demonstrating outstanding performance in both subgroups of babies with GA > or ≤28 weeks. With an AUC of 0.80 (95% CI: 0.72–0.89), the need for a second dosage of surfactant was predicted, but less accurately.

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In a prospective research, Perri et al.[54] examined LUSs and CXR scores to predict the early administration of surfactant in babies with RDS. The study included 56 newborns with a mean GA of 31 weeks. When it came to the early identification of babies with RDS who needed surfactant therapy, LUSs had a higher AUC than X-ray scores. In keeping with this, the author also investigated the shift in LUSs two hours after surfactant delivery. They discovered that LUSs obtained two hours after surfactant delivery may be helpful in determining whether individuals need further therapy. Specifically, a score of ≥7 indicated a 60% specificity and 94% sensitivity.

The LUSs increased with the severity of RDS, according to Pang et al.'s[55] scans of 12 locations (upper and lower parts of anterior, posterior, and lateral sections) of infants with RDS. Using a cut-off of 21.5, the LUSs for RDS vs. non-RDS demonstrated 80.2% sensitivity and 100% specificity (Area under the ROC curve, AUC = 0.938; $P < 0.001$). Using a cut-off of 25.5 (AUC = 0.944; $P < 0.001$), the LUSs for severe vs. mild/moderate RDS demonstrated 73.1% sensitivity and 95.7% specificity.

Based on these findings, Raschetti et al.[56] recently completed a quality improvement initiative on echography-guided surfactant THERapy (ESTHER). The ESTHER approach, they discovered, lowered the peak FiO2 prior to surfactant replenishment, increased the number of newborns getting surfactant during the first three hours of life, shortened the time of invasive ventilation, and enhanced the number of ventilator-free days. The requirement for surfactants throughout the world has not changed all that much. In a randomised experiment, Rodriguez-Fanjul et al.[57] discovered that the ultrasound group had reduced CO2 (48 vs. 54, $p = 0.011$) and FiO2 (25 vs. 30%, $p < 0.001$) as well as surfactant given sooner (1 h of life vs. 6 h, $p < 0.001$). Newborns in the ultrasonography group showed higher SpO2 and SpO2/FiO2 ratios after surfactant therapy. Early life exposure to oxygen is decreased due to LUSs, and post-treatment

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oxygenation is improved. In a prospective research, Gregorio-Hernández et al. looked at neonates who required noninvasive ventilation at delivery and were younger than 35 weeks.[58]

Compared to the no surfactant group, the LUSs in the surfactant group were substantially greater. The surfactant treatment's ROC curve showed an AUC of 0.97 (95% CI 0.92–1). In a prospective double-blind trial, Vardar et al.[14] In the first two hours of life, newborns under 34 weeks old who had clinical and radiological indications of RDS were assessed for the requirement for surfactant treatment using a six-area LUS examination. There was a strong link between the need for total surfactant dosages and LUSs. With 100% specificity and 96% sensitivity, a threshold LUS of 4 indicated the requirement for surfactant (AUC: 1.00; 95% CI: 0.97–1.00). The effectiveness of LUSs in guiding surfactant replacement was further supported by a meta-analysis, which also showed that newborns with LUSs >5–6 had a considerably higher chance of receiving surfactant therapy than infants with LUSs <5–6.[59] To determine how well LUSs on the first day of life might predict the need for surfactant therapy in preterm newborns, another systematic study is now being conducted.[60]

New uses of LUS as a functional tool and as an imaging technology are being made possible by these investigations. Based on the aforementioned study, most studies followed Brat et al.'s[11] technique when it came to zoning. The 12-area approach, which includes the posterior parts, is only used in one research. Further benefits were not shown by the outcome. When it comes to scoring, most research still use the approach of Brat et al. Just three of them deviate from Brat et al. in their scoring methodology. Thus, the usefulness of this scoring method for newborn RDS has been shown.

LUS in neonatal respiratory support

High LUSs are predictive of the requirement for respiratory assistance in both term and preterm newborns, and they have been linked to many indicators of lung damage and oxygenation.

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Despite being extensively utilised in the past, CXR has a weak connection with lung function, and when a direct comparison was made, it was often shown that CXR performed worse than LUS.[12]

Raimondi F et al.'s[10] initial research suggested that LUSs may be used to predict whether or not infants will need respiratory assistance. However, they did so using a postulated LUS pattern grade, which they defined as not being semi-quantitative. 54 newborns participated in a research that was later carried out by the author. Following a 2-hour nasal breathing experiment, bilateral type 1 lung profile sensitivity was 88.9%, particularly 100%, and LUS significantly outperformed conventional radiography in predicting the need for intubation.

As a result, Pang et al.[54] separated the front, posterior, and lateral regions of each lung into six areas, including the upper and lower areas. A 0–3 point score was assigned to each lung area (13). Using a threshold of 25.5 (AUC = 0.912; $P < 0.001$), the author discovered that LUSs for predicting mechanical ventilation (MV) demonstrated 81.3% sensitivity and 88.8% specificity. Later, Szymański et al.[60] suggested modified LUSs in newborns, which include a 5-grade rating scale in place of a 4-grade rating scale and posterior lung fields instead of lateral lung fields. Thirty preterm babies weighing less than 1,500 g at birth and less than 32 weeks GA were scanned. Evaluations were conducted on Days 2, 3, 5, 7, 10, 14, 21, and 28 as well as within 24 hours after delivery (LUS 0). According to the findings, there was a strong correlation between LUSs and SpO2/FiO2 (Spearman rho = - 0.635; $p < 0.0001$). The DOL 3 breathing needs were significantly predicted by LUS 0 ($p < 0.016$) and birth weight ($p < 0.001$). When predicting invasive ventilation on DOL 3, LUS 0 demonstrated a high degree of reliability (AUC = 0.845; 95% CI: 0.738–0.951; $p < 0.001$).

Additionally, patients who performed the LUSs guided recruitment manoeuvre exhibited early lowest FiO2, shorter O2 dependence, shorter invasive ventilation durations, and a significant

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reduction in lung inflammation (Abushady et al.).[62] A prospective, double-blind research using LUS and CXR assessment at admission was carried out on babies with RDS and a GA <34 weeks.[14] High LUSs soon after delivery were shown to be significantly correlated with PEEP levels. Patients with CPAP failure showed a considerably greater LUS. CPAP failure was correctly predicted by LUSs (AUC = 0.804; 95% CI: 0.673–0.935; p = 0.001). But no relationship between LUSs and CPAP days was seen.

For severely unwell infants, invasive ventilation is a lifesaver. Extended mechanical ventilation is linked to higher rates of pulmonary problems, neonatal death, morbidity, and neurodevelopmental impairment. Minimising these consequences requires early weaning and limiting the length of time that invasive ventilation is used. Premature baby weaning is linked to extubation failure (EF), which might lead to unfavourable consequences. According to earlier research, EF affects 24–42% of newborns in the NICU.[63]

Long-term MV was also identified as a risk factor for EF. The process of weaning off of MV has remained difficult and imprecise up to this point. As a result, determining the ideal weaning window and forecasting EF have critical therapeutic implications. Several adult research have indicated that LUS predicts both post-extubation failure and successful weaning.[64,65] Furthermore, not much research have been done on newborns. Since loss of lung capacity for gas exchange is represented by the loss of pulmonary aeration after extubation, it may be used to predict EF.

LUSs are capable of assessing lung aeration loss. Unlike CXR, LUS allows a dynamic evaluation of changes in lung aeration. In a prospective trial, 40 newborns, regardless of GA, with various causes of RD required MV. At the time of admission, prior to switching to MV mode, and before to weaning, LUS was carried out three times minimum. Each hemithorax has six parts that were scanned: the anterior, lateral, and posterior (each area was split into superior

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and inferior sections). Patients who were able to effectively wean off of SIMV scored far lower than those who were unable to do so. When predicting weaning success at a score of six, LUS had a sensitivity of 87.5% and a specificity of 100%, according to ROC analysis.[65]

El Amrousy et al.[67] evaluated three chest regions—the upper anterior, lower anterior, and lateral—for each lung in a recent prospective experiment, for a total of six locations in both lungs. Eighty consecutive infants on MV with various pulmonary disorders were enrolled in this research. Just before to extubation and six hours after extubation, all patients received LUS. In this research, infants with EF had substantially greater LUSs both before and after extubation compared to those who had a successful weaning process. At a cutoff point of six, post-extubation LUS exhibited an 89% sensitivity and 90% specificity in predicting neonatal weaning success. The LUS findings were consistent with adult results that had been previously published. In addition, EF newborns had far lower weight and GA than did extubation-successful neonates. This was in line with previous researchers' findings.

Summary of past literatures on Lung Ultrasound score:

Brat R et al. examined the diagnostic accuracy of a neonatal-adapted Lung Ultrasound (LUS) score to evaluate oxygenation and predict the necessity for surfactant therapy in a study involving 130 newborns. The study found a significant correlation between the LUS score and various oxygenation indices, regardless of gestational age (GA). For neonates with a GA of 34 weeks or more, the correlation coefficients were: Ptco2 to Fio2 ratio ($\rho = -0.57$), alveolar-arterial gradient ($\rho = 0.62$), oxygenation index ($\rho = 0.63$), and arterial to alveolar ratio ($\rho = -0.60$), all with P-values <.001. For those with a GA less than 34 weeks, the correlations were: Ptco2 to Fio2 ratio ($\rho = -0.62$), alveolar-arterial gradient ($\rho = 0.59$), oxygenation index ($\rho = 0.69$), and arterial to alveolar ratio ($\rho = -0.56$), also with P-values <.001. When evaluating the ability to predict the need for surfactant, the LUS score had an area under the curve (AUC)

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of 0.71 (95% CI, 0.54-0.90; P =.02) for neonates with GA of 34 weeks or more. In contrast, for those with GA less than 34 weeks, the AUC was 0.93 (95% CI, 0.86-0.99; P <.001), indicating significantly better predictive power (P =.02). For infants with a GA less than 34 weeks, a LUS score threshold of 4 could predict the need for surfactant with 100% sensitivity and 61% specificity, resulting in a 72% posttest likelihood.[11]

In order to ascertain the diagnostic accuracy of LUS in anticipating the need of surfactant therapy and re-treatment in newborns, De Martino L et al. conducted a research. Even after accounting for gestational age (P <.0001), there is a substantial correlation between a LUS and oxygenation index (ρ = 0.6; P <.0001). Both the requirement for surfactant redosing (area under the curve = 0.803; 95% confidence interval: 0.72–0.89; P <.0001) and the need for the first surfactant dosage (area under the curve = 0.94; 95% confidence interval: 0.90–0.98; P <.0001) may be correctly predicted using a LUS. The prediction of surfactant therapy and re-treatment has a worldwide accuracy of 89% and 72%, respectively.[12]

In order to assess the reliability of LUS in identifying the requirement for mechanical ventilation or surfactant therapy in babies experiencing respiratory distress who are receiving nasal continuous positive airway pressure (NCPAP), Razak A et al. conducted a comprehensive study. Six trials totaling 485 babies were reviewed. To assess the outcome, three studies employed the LUS score, two used the type 1 lung profile, and one used high-risk LUS. At the LUS score cut-off of >5–6, the pooled sensitivity and specificity were 88% (95% CI 80% to 93%) and 82% (95% CI 74% to 89%), respectively. Compared to babies with a LUS score of less than five or six, those with a score of five or more had a substantially higher chance of requiring surfactant therapy (relative risk=7.51; 95% CI 4.16 to 13.58; two trials; participants=189; I2=0%). Compared to late preterm and term children (sensitivity 100%, specificity 28%), the type 1 lung profile had higher diagnostic accuracy in younger preterm newborns (sensitivity 88.9%, specificity 100%).[58]

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Gregario Hernandez R et al. conducted a study to examine the correlation between early ultrasound scores and the necessity for endotracheal surfactant administration within the first 72 hours of life in preterm infants. They identified a cut-off score of 12 points, which offered the best balance of sensitivity and specificity for predicting the need for the first dose of surfactant. This cut-off score demonstrated a sensitivity of 92.9% and a specificity of 92.5%. The receiver operating characteristic (ROC) curve analysis produced an area under the curve (AUC) of 0.97 (95% CI, 0.92–1, $p < 0.00001$).[57]

Vardar G et al. conducted a study aimed at evaluating the accuracy of neonatal lung ultrasound (LUS) in predicting the necessity for surfactant therapy in preterm infants, compared to chest X-ray (CXR). The study included 45 preterm infants and found that the median LUS score (interquartile range [IQR]) was 4 (2–8) in the group with mild respiratory distress syndrome (RDS), while it was 10 (IQR: 9–12) in the group with severe RDS ($p < 0.01$). There was a significant correlation between the LUS score and the total number of surfactant doses required. A LUS score cut-off of four was found to predict the need for surfactant with 96% sensitivity and 100% specificity (area under the curve [AUC]: 1.00. Additionally, LUS scores accurately predicted continuous positive airway pressure (CPAP) failure (AUC: 0.804) and showed a significant correlation with positive end-expiratory pressure levels. During the study, the number of CXRs per infant with RDS significantly decreased compared to previous months. However, the LUS score on the first day of life did not predict the development of bronchopulmonary dysplasia (AUC: 0.274).[14]

Raimondi F et al. conducted a multicenter, pragmatic study involving preterm neonates who underwent lung ultrasound (LUS) at birth and those administered surfactant by masked physicians. These infants were also scanned within 24 hours post-administration. The study included 240 infants, of whom 108 received at least one dose of surfactant. LUS predicted the first surfactant administration with an area under the receiver operating characteristic curve

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(AUC) of 0.86, using a cut-off score of 9. This yielded a sensitivity of 0.79, specificity of 0.83, positive predictive value of 0.79, negative predictive value of 0.82, positive likelihood ratio of 4.65, and negative likelihood ratio of 0.26. There were no significant differences observed among different gestational age groups: 25 to 27 weeks (AUC, 0.91), 28 to 30 weeks (AUC, 0.81), and 31 to 33 weeks (AUC, 0.88). LUS scores significantly decreased within 24 hours in infants who received a single dose of surfactant. When comparing the criteria for surfactant administration—FiO2, oxygen saturation to FiO2 ratio, LUS, and Silverman scores—only the Silverman score showed significantly poorer performance. Combining the oxygen saturation to FiO2 ratio with LUS demonstrated the highest predictive power, with an AUC of 0.93, regardless of gestational age.[10]

Perri A et al. conducted a study to evaluate the diagnostic accuracy of a lung ultrasonography (LUS) score in early predicting the need for surfactant therapy in newborns with respiratory distress syndrome (RDS), and to compare it with a chest X-ray score. The study found that the LUS score had a higher area under the curve (AUC) than the X-ray score for early recognition of infants requiring surfactant treatment (0.94 vs 0.80). Additionally, the LUS score demonstrated higher sensitivity (86% vs 82%), higher specificity (88% vs 76%), better positive predictive value (83% vs 69%), and better negative predictive value (91% vs 87%).[54]

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Material and Methods:

Study Design: The study was designed as a prospective observational study.

Study Setting: The study was conducted at the LEVEL IIB Neonatal Intensive Care Unit (NICU), BLDE (Deemed to be University), Shri B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR, KARNATAKA.

Study Duration: Data collection spanned from January 2021 to June 2022.

Study Participants: All newborns admitted to the NICU with respiratory distress were included in the study. They were categorized into two groups based on gestational age, namely <34 weeks and >34 weeks.

Inclusion Criteria: Inclusion criteria comprised all newborns admitted to the NICU with respiratory distress.

Exclusion Criteria: Newborns with known chromosomal abnormalities, congenital malformations, and congenital lung diseases were excluded from the study.

Sample Size: The sample size was determined based on anticipated correlation between Arterial to alveolar ratio and LUS Score, which was -0.492.[11] With a 95% confidence level and 99% power, the calculated sample size was 70.

Sampling Method: The study employed a purposive sampling.

Data Collection Procedures:

In the study, all inborn neonates admitted to the Neonatal Intensive Care Unit (NICU) with respiratory distress were categorized into two groups based on gestational age (<34 and >34 weeks). Lung ultrasonography was conducted promptly upon NICU admission and before surfactant administration. Each lung was systematically divided into six areas: upper anterior,

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lower anterior, upper lateral (axillary), lower lateral, upper posterior, and lower posterior. Linear microprobes were utilized for both transverse and longitudinal scans of each area. A scoring system ranging from 0 to 3 points was applied to assess the condition of each lung area, resulting in a total score ranging from 0 to 18. The Lung Ultrasonography (LUS) score was designed to describe the complete spectrum of possible lung conditions, encompassing normal aeration, interstitial patterns, alveolar patterns, and consolidations. Specifically, a score of 0 indicated an A pattern characterized by the presence of only A lines, while a score of 1 denoted a B pattern with the presence of more than three well-spaced B lines. A score of 2 indicated a severe B pattern, characterized by crowded and coalescent B lines with or without consolidations limited to the subpleural space, while a score of 3 indicated extended consolidations. A-lines represented the reflection of pleura due to ultrasound diffusing through an air-filled lung, whereas B-lines were attributed to fluid filling the interstitium, including the alveolar space if they became confluent.

Statistical Analysis: Data obtained were entered into a Microsoft Excel sheet and analyzed using Statistical Package for the Social Sciences (Version 20). Results were presented as Mean (Median) ±SD, counts and percentages, and diagrams. Categorical variables between groups were compared using Chi-square test. Pearson's/Spearman's correlation analysis was used to determine correlations between quantitative variables. Receiver operating characteristic (ROC) curve analysis was performed to assess the predictive ability of the LUS score. Statistical significance was set at $p<0.05$ for all analyses, which were conducted as two-tailed tests.

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Results

Table 1: Mode of Delivery (Vaginal Delivery/Cesarean Section)

Mode of delivery	Frequency	Percent
Cesarean section (C-section)	39	52.00
Normal Vaginal Delivery (NVD)	36	48.00
Total	**75**	**100.00**

Out of 75 deliveries, 52% were Cesarean sections (C-sections) and 48% were normal vaginal deliveries (NVD).

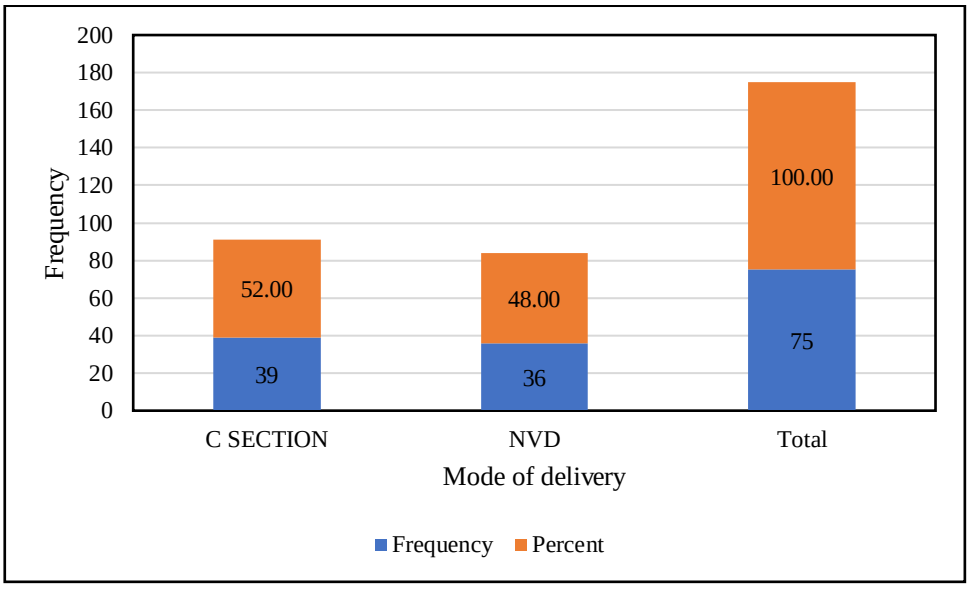

Figure 2: Mode of delivery

Table 2. Gestational Age (Full-term/Preterm)

Variable	N	Mean	Std. Deviation	Minimum	Maximum
Gestational Age	75	35.52	3.244	27	41

The sample included N = 75 neonates with gestational ages ranging from a minimum of 27 weeks to a maximum of 41 weeks. The mean gestational age was 35.52 weeks (SD = 3.24).

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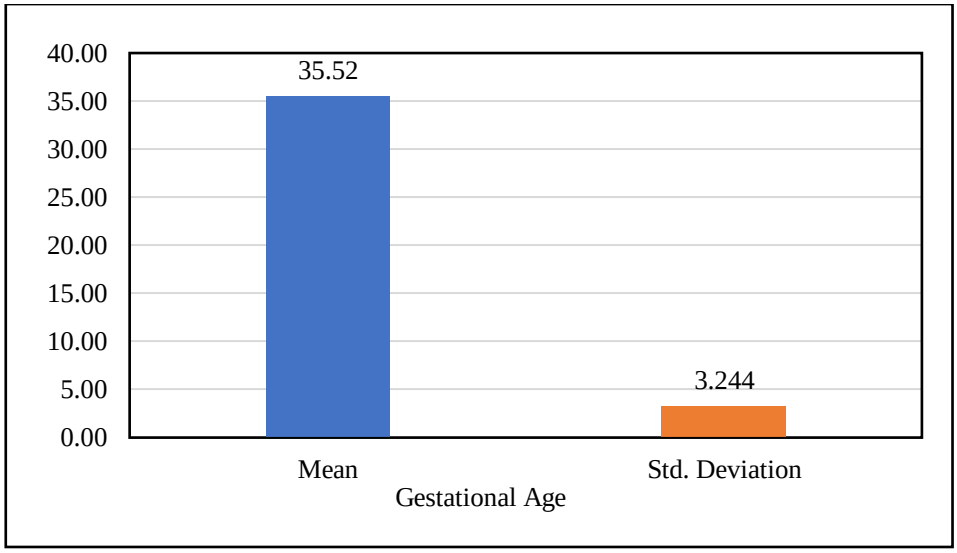

Fig 3: Gestational Age

Table 3. Gender (Male/Female) (delivery)

Gender	Frequency	Percent
Female	29	38.67
Male	46	61.33
Total	**75**	**100.00**

In the sample of 75, there was a slight male predominance, with males constituting 61.33% (n=46) and females 38.67% (n=29).

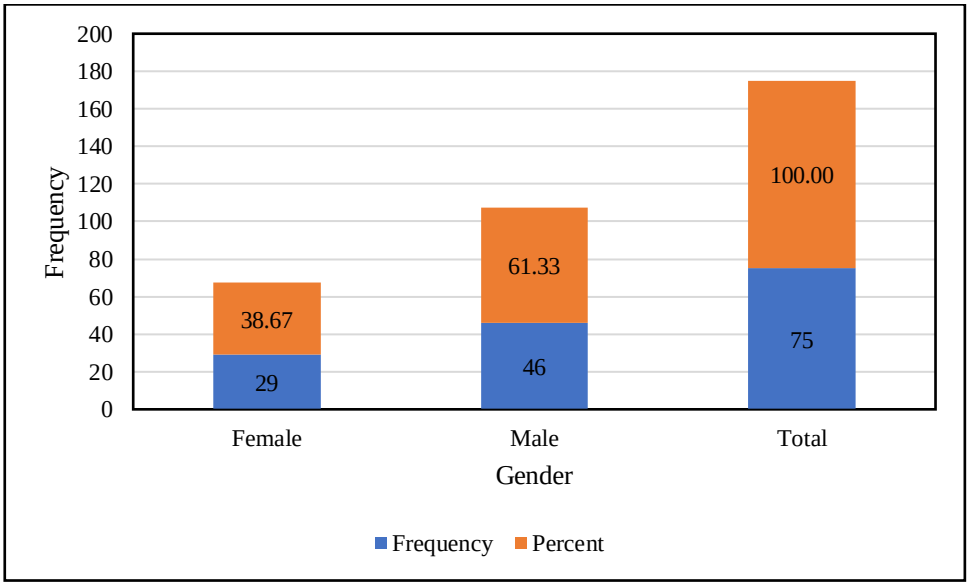

Fig 4: Gender

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Table 4. Birth Weight (Low Birth Weight/Normal Birth Weight)

Variable	N	Mean	Std. Error of Mean	Median	Std. Deviation	Minimum	Maximum
Birth weight	75	2.1453	.06926	2.2000	.59984	1.20	3.40

The birth weight of neonates in this investigation spanned from 1.20 to 3.40 kilograms, with an average weight (M) of 2.1453 kilograms and a standard deviation (SD) of 0.59984. The median birth weight was marginally higher than the mean at 2.2000 kilograms, indicating a slight skew in the distribution.

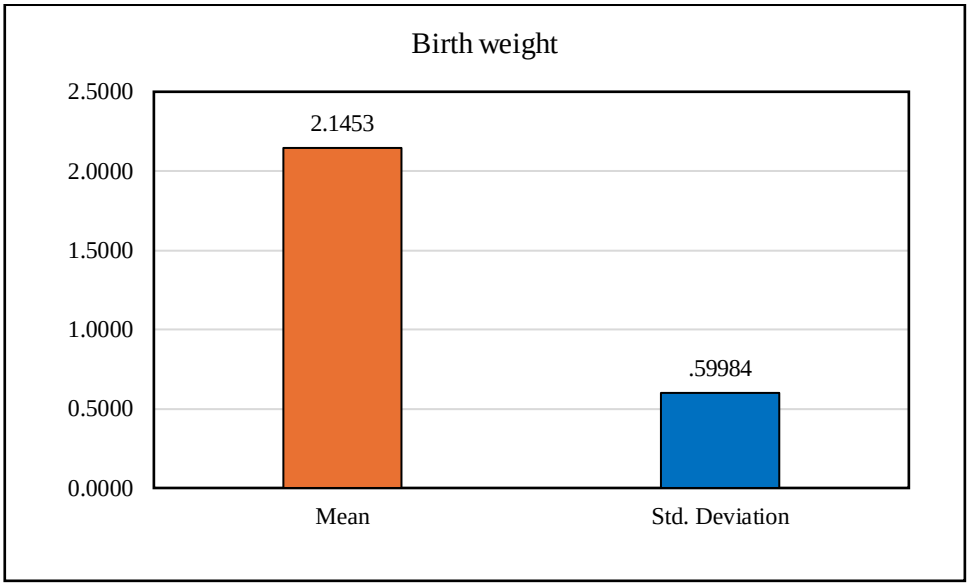

Fig 5: Birth weight of neonates

Table 5: Gestational Age and Birth Weight

Variable	N	Mean	Std. Deviation
Gestational Age	75	35.52	3.244
Birth Weight	75	2.1453	.59984

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The mean gestational age of the neonates in this study was 35.52 weeks (SD = 3.244, N = 75), indicating that on average, the neonates were born slightly before the full term of 40 weeks. The mean birth weight was 2.1453 kilograms (SD = .59984, N = 75), which is within the normal range for newborns but suggests that a significant proportion of the neonates were of low birth weight (i.e., less than 2.5 kilograms).

Table 6: Classification of Gestational Age (Small for Gestational Age/Appropriate for Gestational Age/Large for Gestational Age)

Gestational Age	Frequency	Percent
Appropriate for Gestational Age (AGA)	52	69.33
Large for Gestational Age (LGA)	2	2.67
Small for Gestational Age (SGA)	21	28.00
Total	**75**	**100.00**

In the given sample of 75 individuals, the classification of gestational age was as follows: 69.33% (n = 52) were deemed appropriate for gestational age (AGA), 2.67% (n = 2) were large for gestational age (LGA), and 28.00% (n = 21) were small for gestational age (SGA).

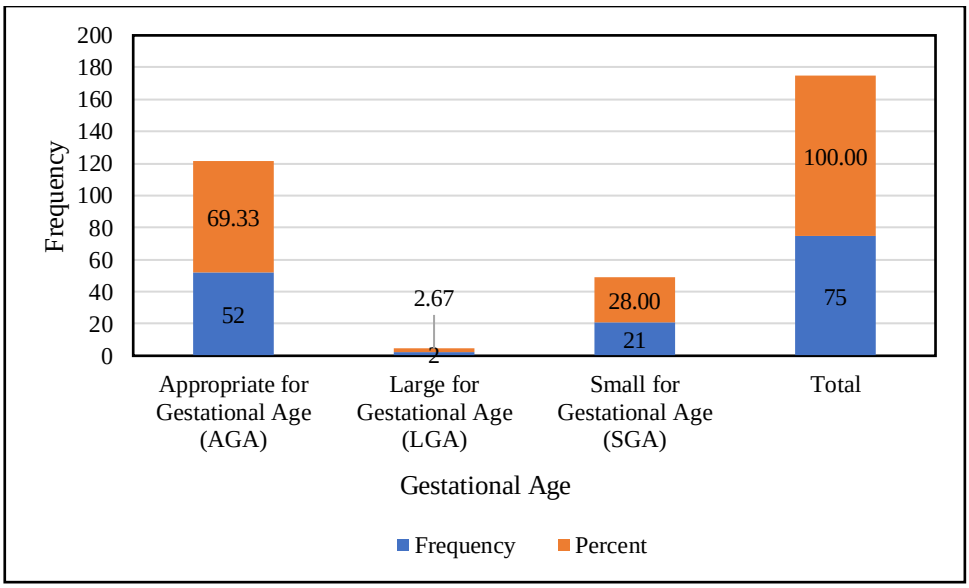

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Fig 6: Gestational age classification

Table 7. Parity (Primiparous/Multiparous)

Prenatal Parity	Frequency	Percent
Multigravida	23	30.67
Primigravida	52	69.33
Total	**75**	**100.00**

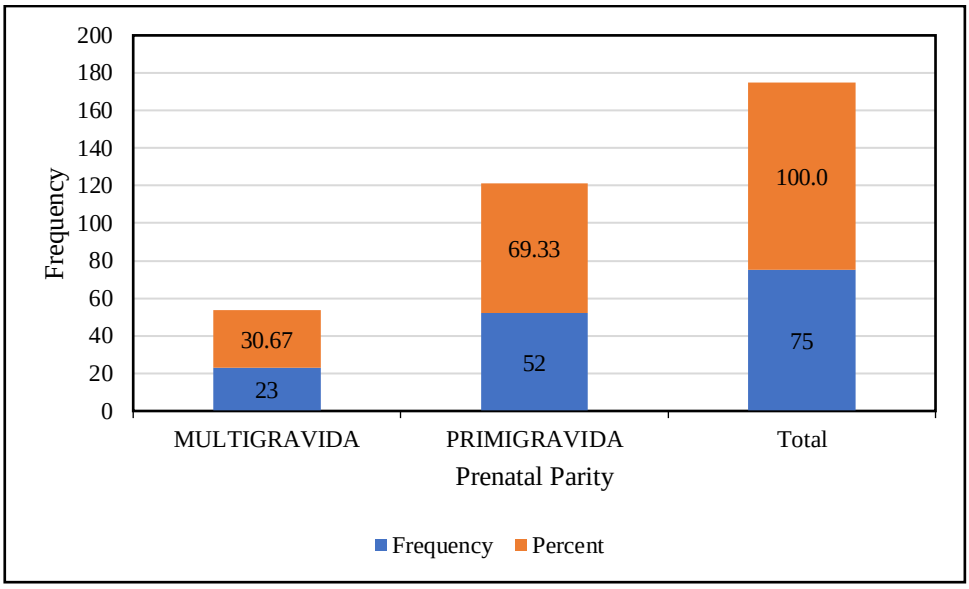

Fig 7: Prenatal Parity

Table 8: Socioeconomic Status (Lower-middle/Upper-middle)

Socioeconomic status	Frequency	Percent
Lower-middle	23	30.67
Upper-middle	52	69.33
Total	**75**	**100.00**

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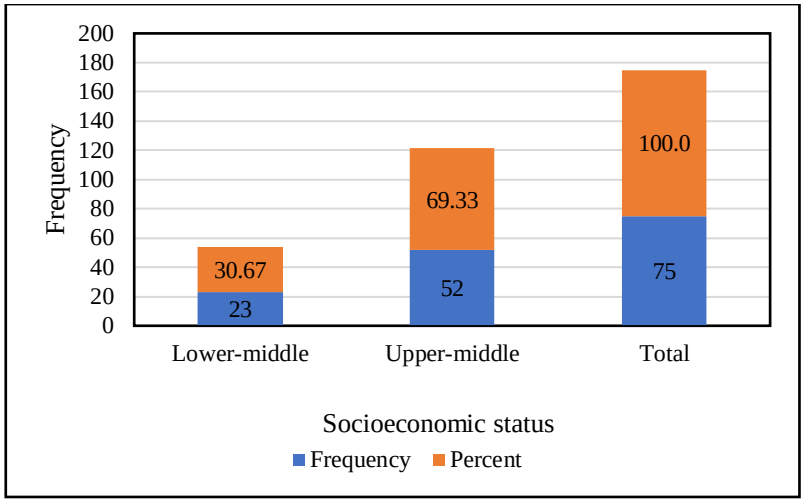

Fig 8: Socioeconomic status

Table 9: Residential Area (Urban/Rural)

Residential Area	Frequency	Percent
Rural	8	10.67
Urban	67	89.33
Total	**75**	**100.00**

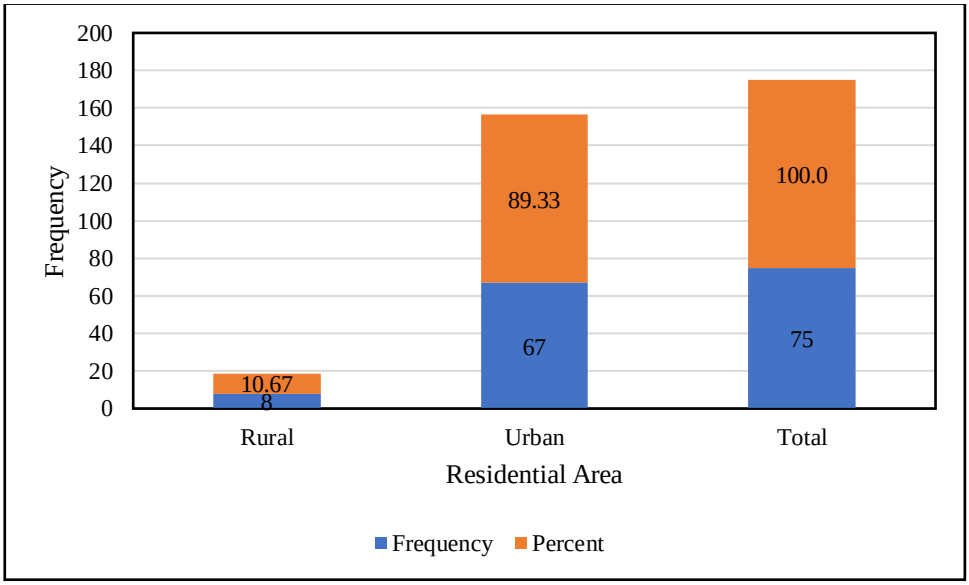

Fig 9: Residential Area

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Table 10: Pregnancy-Induced Hypertension (PIH)

Pregnancy-induced hypertension (PIH)	Frequency	Percent
Yes	12	16.00
No	61	81.33
Don't know	2	2.67
Total	**75**	**100.00**

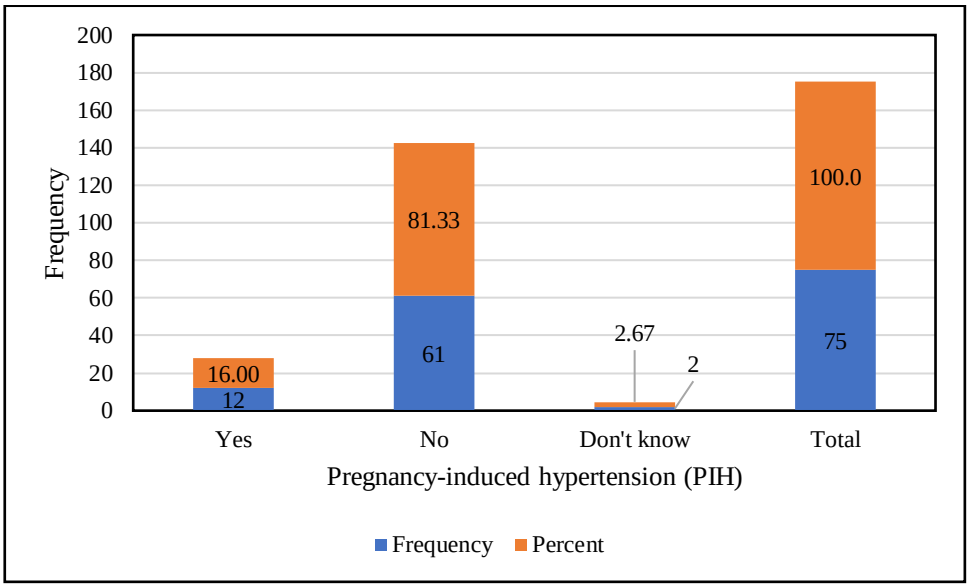

Fig 10: Pregnancy-induced hypertension (PIH)

Table 11: Gestational Diabetes

Gestational Diabetes	Frequency	Percent
Yes	2	2.67
No	71	94.67
Don't know	2	2.67
Total	**75**	**100.00**

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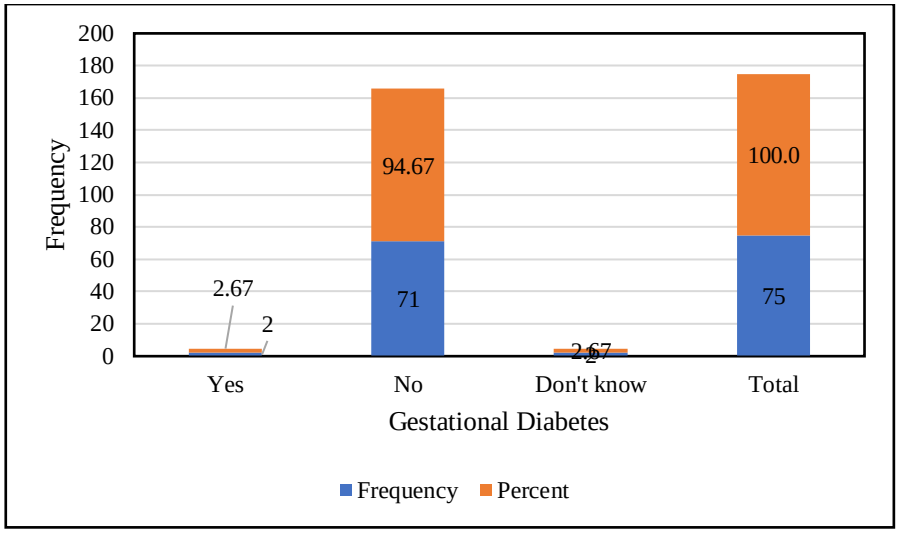

Figure 11: Gestational Diabetes

Table 12: Premature Rupture of Membranes (PROM)/Preterm Premature Rupture of Membranes (PPROM)

PPROM/PROM	Frequency	Percent
Yes	12	16.00
No	63	84.00
Total	**75**	**100.00**

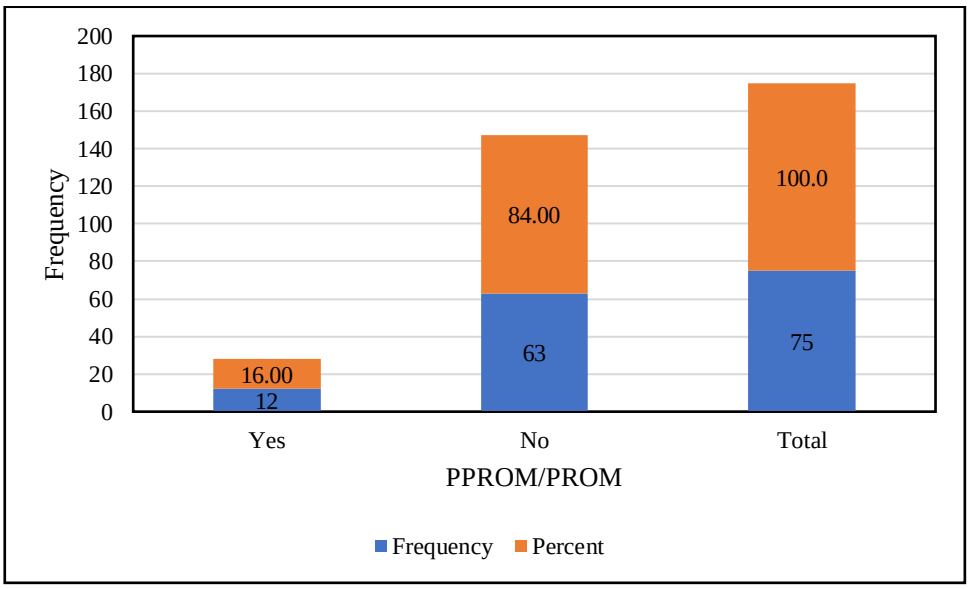

Fig 12: PPROM/PROM

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Table 13: Antepartum Haemorrhage

Antepartum Haemorrhage	Frequency	Percent
Yes	2	2.67
No	71	94.67
Don't know	2	2.67
Total	**75**	**100.00**

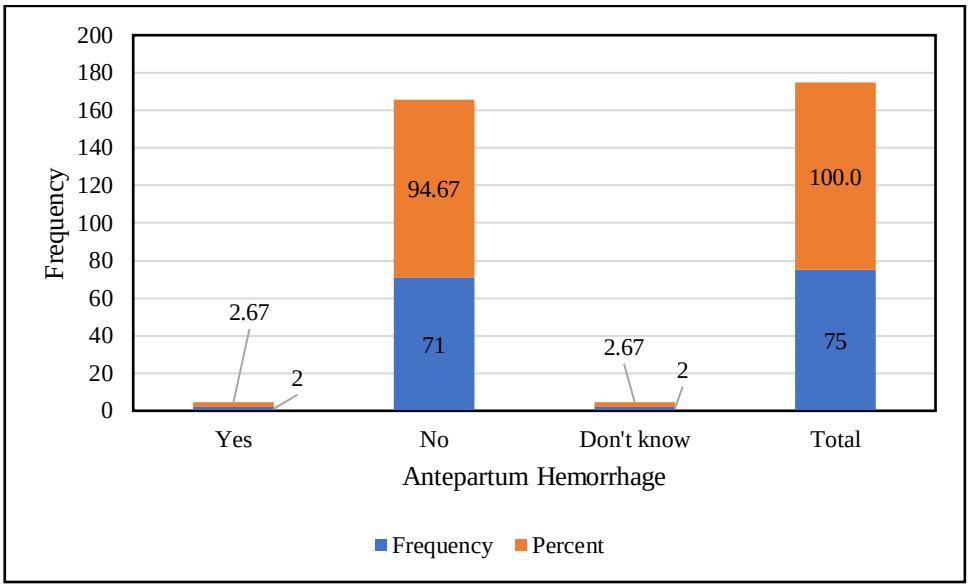

Fig 13: Antepartum Haemorrhage

Table 14: Multiple Gestation (Single/Twins/Multiples)

Multiple Gestation	Frequency	Percent
No	74	98.67
Twins	1	1.33
Total	**75**	**100.00**

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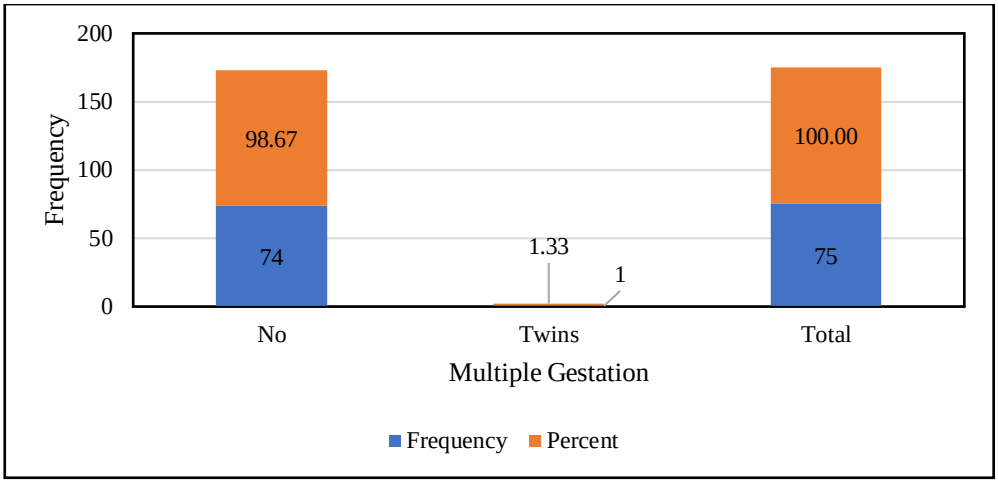

Figure 14: Multiple Gestation

Table 15. APGAR Scores (At 1 minute and 5 minutes)

APGAR	N	Mean	Std. Deviation	Std. Error Mean
APGAR at 1 min	75	7.09	.961	.111
APGAR at 5 min	75	7.99	.951	.110

It shows that the mean APGAR score at 1 minute (M = 7.09, SD = 0.961) was lower than at 5 minutes (M = 7.99, SD = 0.951).

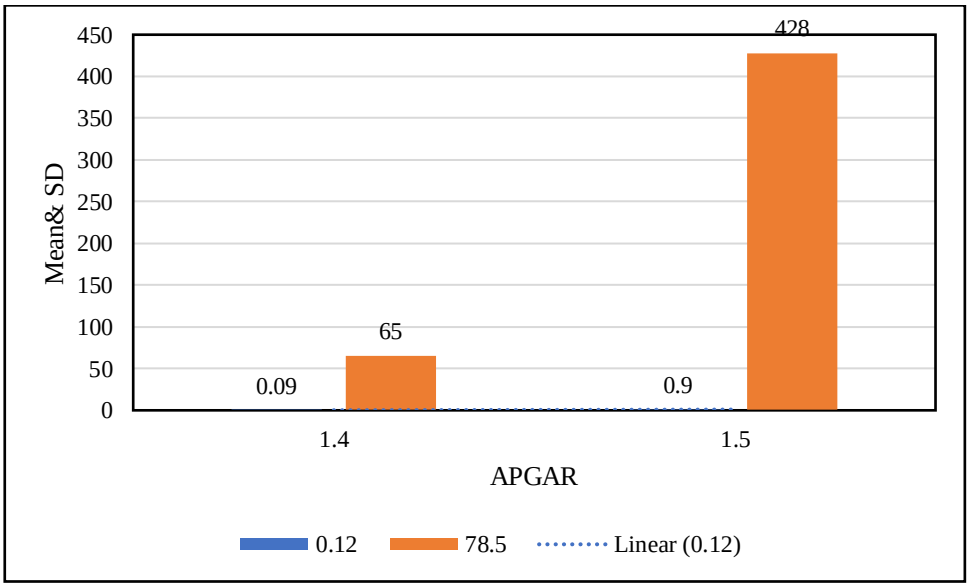

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Fig 15: APGAR Scores (At 1 minute and 5 minutes)

Table 16. NICU Admission in Hours

Variable	N	Mean	Std. Deviation	Minimum	Maximum
NICU Admission in Hours	75	2.05	.804	1	5

The average time for NICU admission in this study was 2.05 hours (SD = .804, N = 75), with a range from 1 to 5 hours.

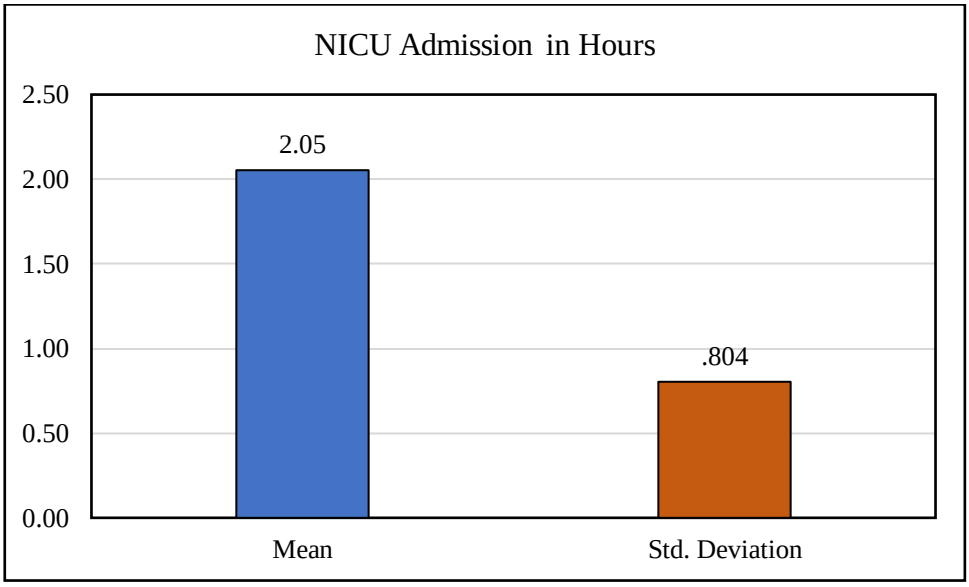

Fig 16: NICU Admission in Hours

Table 17. Meconium-Stained Liquor

Meconium-stained liquor	Frequency	Percent
Yes	3	4.00
No	72	96.00
Total	75	100.00

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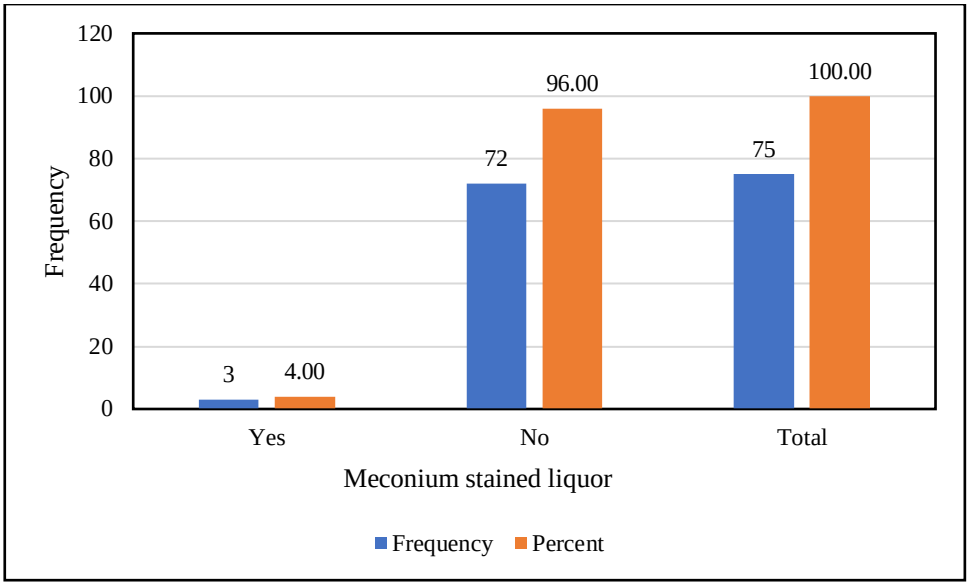

Fig 17: Meconium-stained liquor

Table 18: Gestational Age (Reiteration)

Table 0.0: Gestational Age

Gestational Age	Frequency	Percent
Early Preterm (<30 Weeks)	5	6.67
Mid Preterm (30 -33)	16	21.33
Late Preterm (34 - 37)	20	26.67
Term	34	45.33
Total	75	100.00

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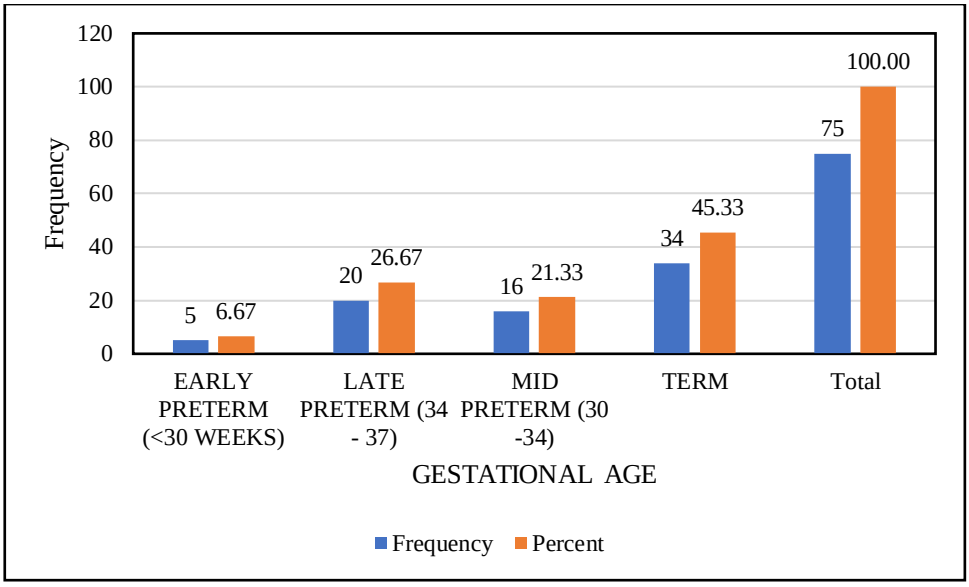

Fig 18: Gestational Age

Table 19: Silverman-Anderson Scoring at Admission

Silverman Anderson Scoring	N	Mean	Std. Deviation	Minimum	Maximum
Silverman Anderson Scoring At Admission	75	4.40	1.952	1	7

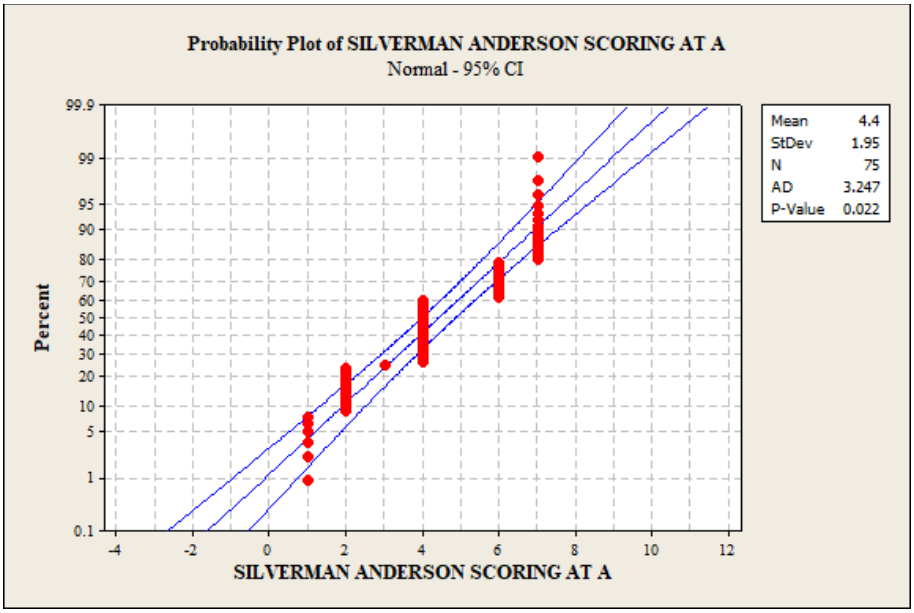

Fig 19: Silverman Anderson Scoring at Admission

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The Probability Plot for the Silverman Anderson Scoring at Admission follows a normal distribution with a 95% confidence interval. The mean score is 4.4, with a standard deviation of 1.95, based on a sample size of 75 (n = 75).

Table 20: Primary Respiratory Support

Primary Respiratory Support	Frequency	Percent
HHHFNC	20	26.67
HOOD	19	25.33
NASAL OXYGEN	1	1.33
NCPAP	6	8.00
SIMV	29	38.67
Total	**75**	**100.00**

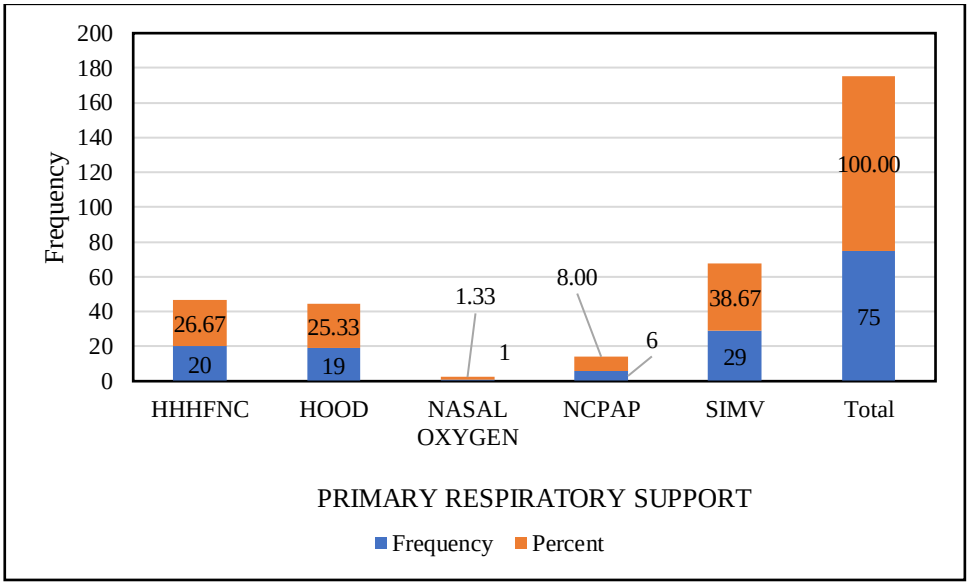

Fig 20: Primary Respiratory Support

Table 21: FIO2 at Admission -Fraction of Inspired Oxygen (FiO2)

FIO2	Frequency	Percent
30	21	28.00
40	25	33.33
60	2	2.67

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70	7	9.33
80	6	8.00
90	1	1.33
100	13	17.33
Total	**75**	**100.00**

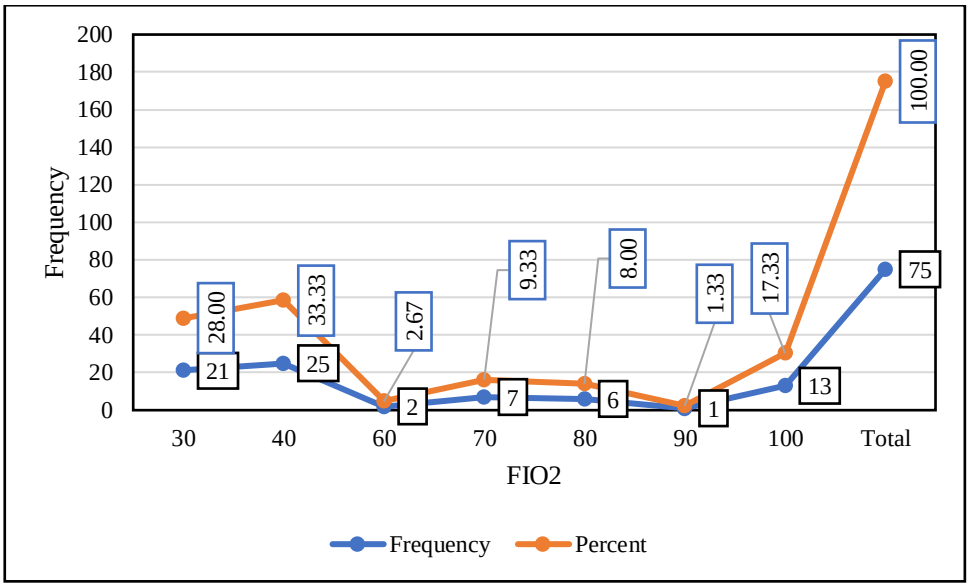

Fig 21: Fraction of Inspired Oxygen (FiO2)

Table 22: Descriptive Statistics for Fraction of Inspired Oxygen (FiO2)

FIO2	N	Mean	Std. Deviation	Minimum	Maximum
FIO2 AT ADMISSION	75	54.80	26.425	30	100

Table 23. Sepsis Evaluation (Categories and Timing)

Sepsis Onset	Frequency	Percent
Early Onset Sepsis	22	29.33
Late Onset Sepsis	16	21.33
Total	38 out of 75	50.7

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Table 0.0: SEPSIS

Sepsis	Frequency	Percent
Yes	38	50.67
No	37	49.33
Total	75	100.00

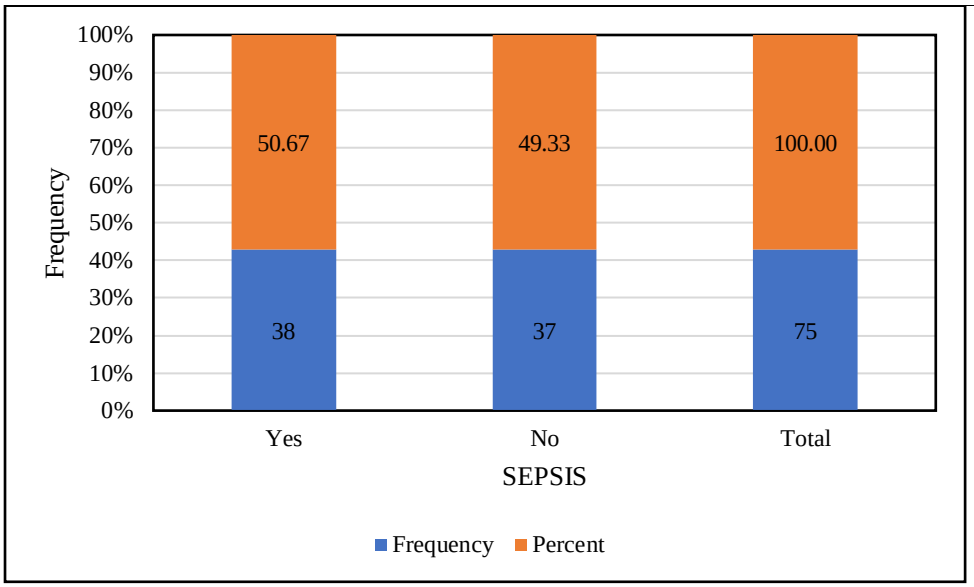

Fig 22: SEPSIS

Table 24: Sepsis Classification

Table 0.0: Early Onset Sepsis

Early Onset Sepsis	Frequency	Percent
Yes	22	29.33
No	53	70.67
Total	75	100.00

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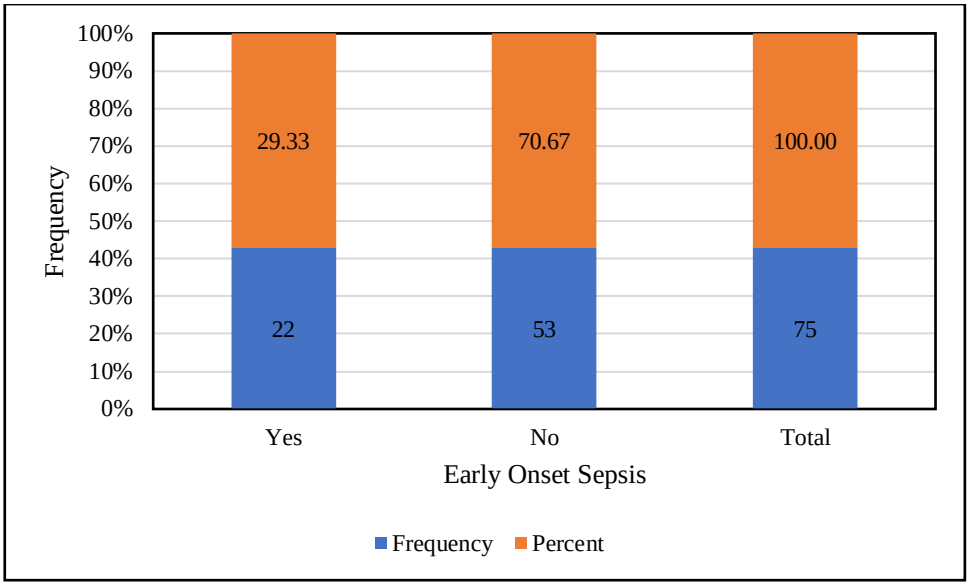

Table 0.0: Late Onset Sepsis

Late Onset Sepsis	Frequency	Percent
Yes	16	21.33
No	59	78.67
Total	75	100.00

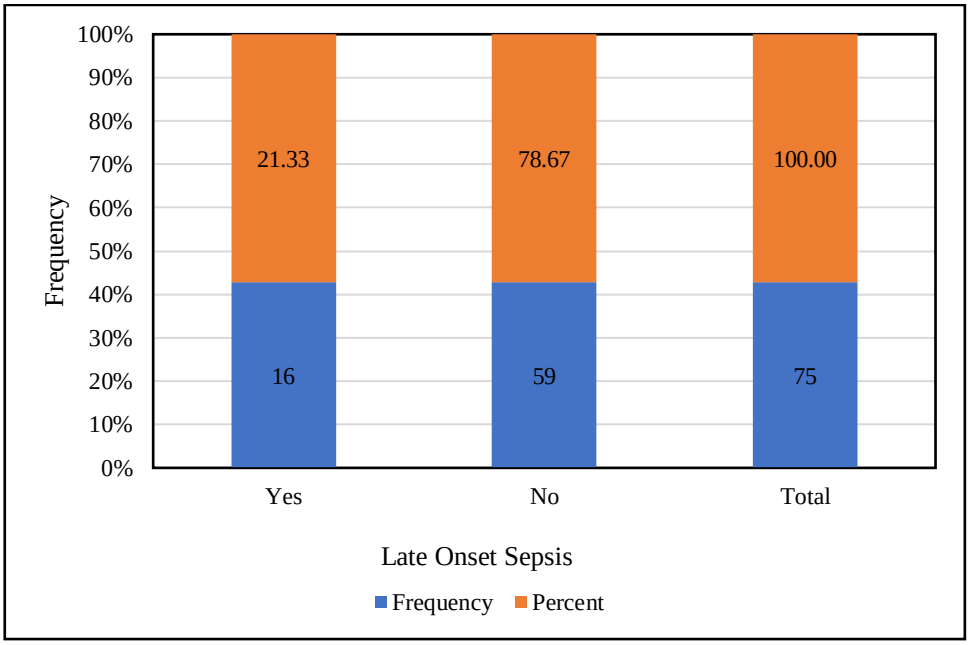

Fig 23: Classification of Sepsis

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Table 25. Respiratory Concerns

Respiratory Concerns	Frequency	Percent
Congenital Pneumonia	2	2.70
Hypoxic-Ischemic Encephalopathy (HIE)	12	16.00
Meconium Aspiration Syndrome (MAS)	3	4.00
Respiratory Distress Syndrome (RDS)	54	72.00
Transient Tachypnea of the Newborn (TTNB)	4	5.30
Total	**75**	**100.00**

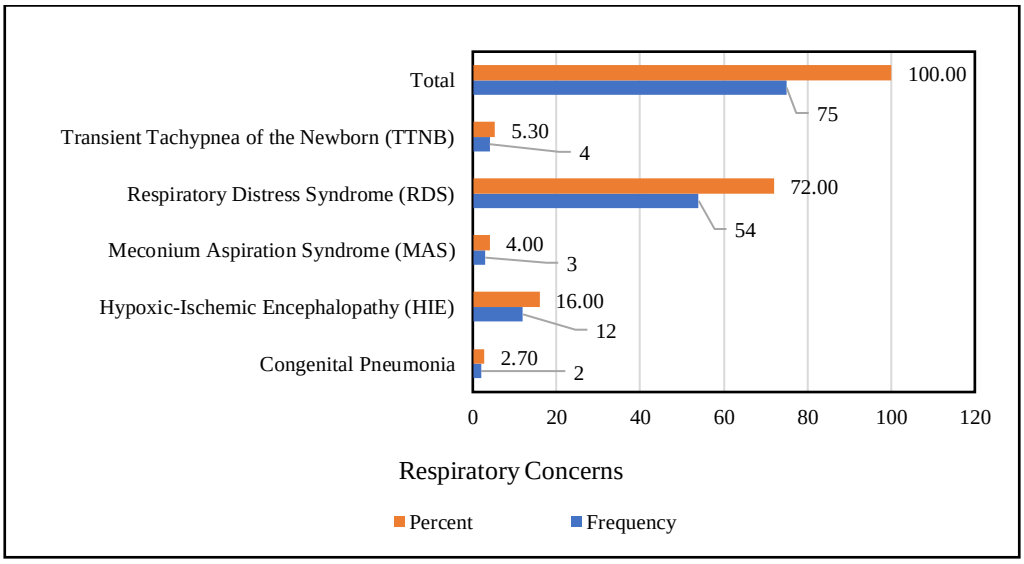

Fig 24: Distribution of Respiratory Concerns

Table 26: Cardiac Concerns

Cardiac Concerns	Frequency	Percent
Atrial Septal Defect (ASD)	8	10.70
Congenital Heart Disease	1	1.30
Good Biventricular Function	30	40.00
Not Done	5	6.70
Patent Ductus Arteriosus (PDA)	29	38.70
Persistent Pulmonary Hypertension of the Newborn (PPHN)	2	2.70
Total	**75**	**100.00**

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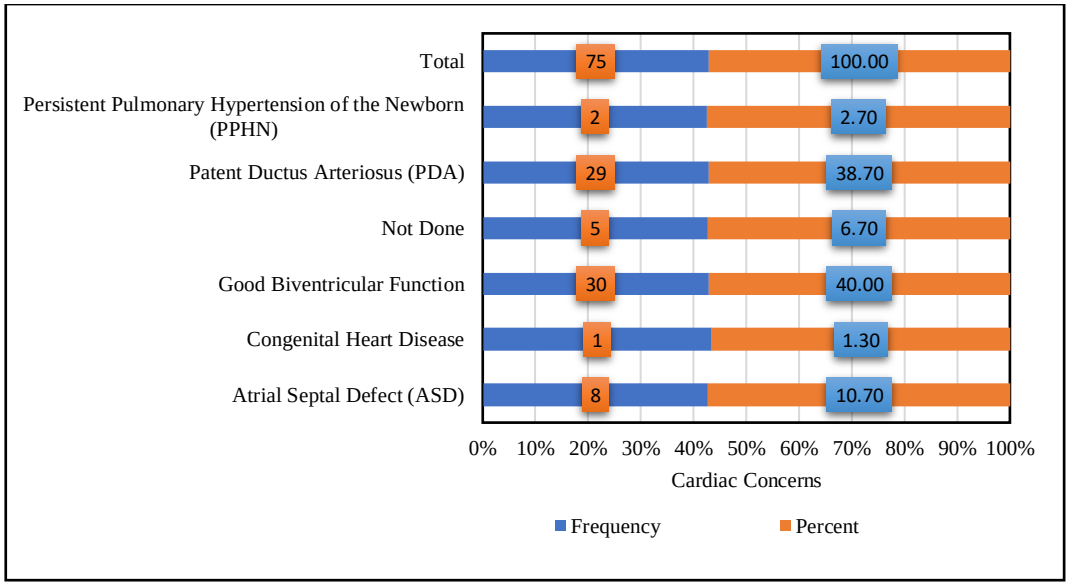

Fig 25: Distribution of Cardiac Concerns

Table 27. Central Nervous System (CNS) Concerns (Normal/Abnormal)

CNS Concerns	Frequency	Percent
Normal	75	100.00

Table 28: Hypoxic-Ischemic Encephalopathy (HIE)

HIE	Frequency	Percent
Yes	15	20.00
No	60	80.00
Total	**75**	**100.00**

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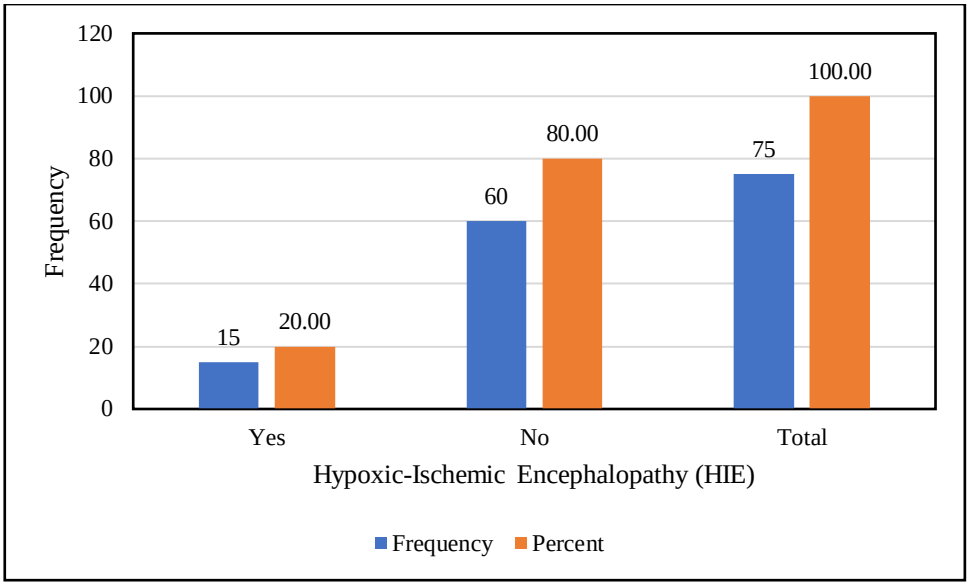

Fig 26: Distribution of Hypoxic-Ischemic Encephalopathy (HIE)

Table 29: Support in/at 4 hours

Support Type	Frequency	Percent
Continuous Positive Airway Pressure (CPAP)	4	5.3
High-Flow Nasal Cannula (HFNC)	18	24
High-Frequency Oscillatory Ventilation (HFOV)	1	1.3
Hood	17	22.7
Nasal Prongs	2	2.7
Pressure-Support Intermittent Mandatory Ventilation (PSIMV)	13	17.3
Synchronized Intermittent Mandatory Ventilation (SIMV)	20	26.7
Total	**75**	**100**

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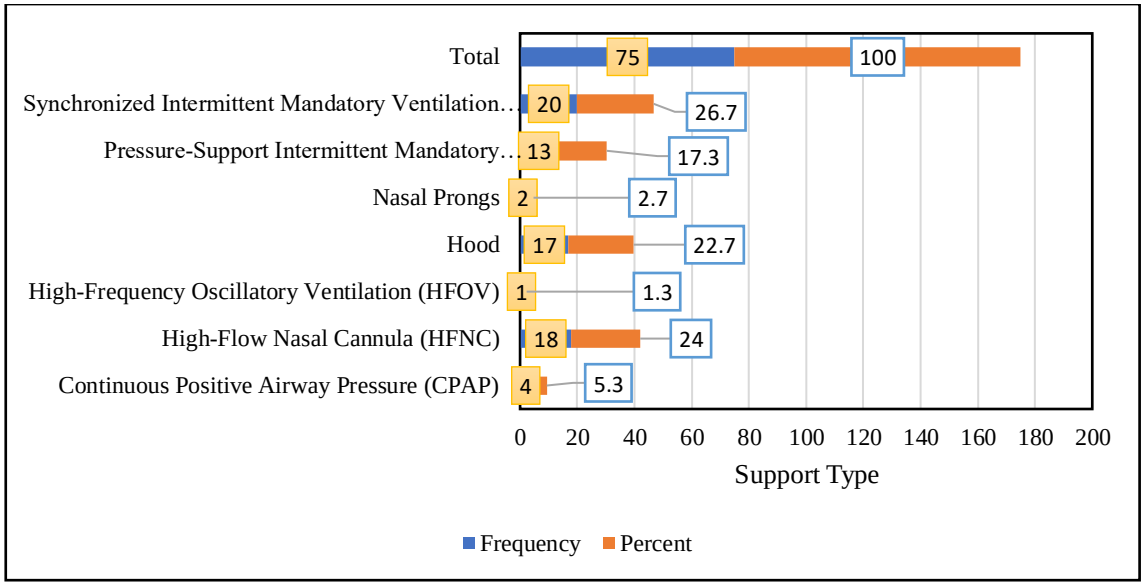

Fig 27: Support in/at 4 Hours

Table 28: Arterial Blood Gas (ABG) Measurements

Arterial Blood Gas (ABG) Measurements	N	Mean	Std. Deviation	Minimum	Maximum
ABG PH	75	7.00	0.000	7	7
ABG PaO2	75	79.47	18.998	44	100
ABG PaCO2	75	50.60	6.384	34	69
ABG HCO3	75	17.35	3.667	8	23
Lactate	75	1.75	.438	1	2

The mean ABG PH was 7.00 (SD = 0.000, N = 75), indicating a normal blood pH level in the neonates. The mean partial pressure of oxygen (PaO2) was 79.47 mmHg (SD = 18.998, N = 75), suggesting adequate oxygenation. The mean partial pressure of carbon dioxide (PaCO2) was 50.60 mmHg (SD = 6.384, N = 75), which is slightly elevated and may indicate respiratory issues. The mean bicarbonate (HCO3) level was 17.35 mEq/L (SD = 3.667, N = 75), which is within the normal range and suggests no significant metabolic disturbances. The mean lactate level was 1.75 mmol/L (SD = .438, N = 75), which is within the normal range and indicates no significant tissue hypoxia.

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Table 29: Alveolar-Arterial Gradient

Variable	N	Mean	Std. Deviation	Minimum	Maximum
Alveolar-arterial gradient	75	286.81	238.326	6	643

The Alveolar-arterial gradient, a critical measure in assessing respiratory efficiency, exhibits considerable variability in this study. With a sample size of 75 (N = 75), the mean value stands at 286.81, indicating an average substantial difference between alveolar and arterial oxygen levels. The standard deviation is 238.326, reflecting a wide dispersion in the data. The gradient values span from a minimum of 6 to a maximum of 643, suggesting diverse respiratory statuses among the neonates.

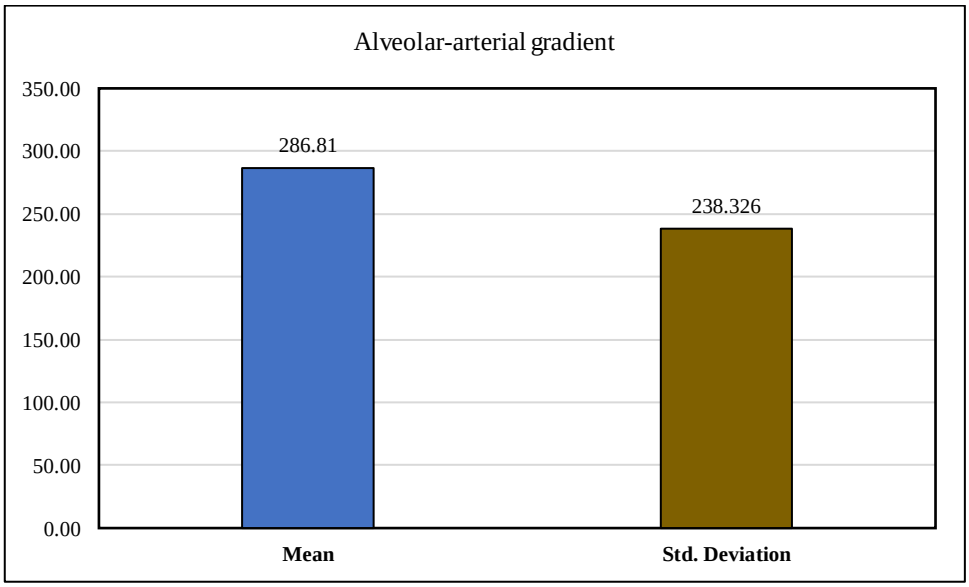

Fig 28: Alveolar-arterial gradient

Table 30: Arterial to Alveolar Ratio

Arterial to Alveolar Ratio	Frequency	Percent
0	52	69.33
1	23	30.67
Total	75	100.00

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The Arterial to Alveolar Ratio, a key indicator of respiratory efficiency, shows a distinct distribution in this study. Out of 75 neonates (N = 75), 52 (69.3%) had a ratio of 0, while 23 (30.7%) had a ratio of 1.

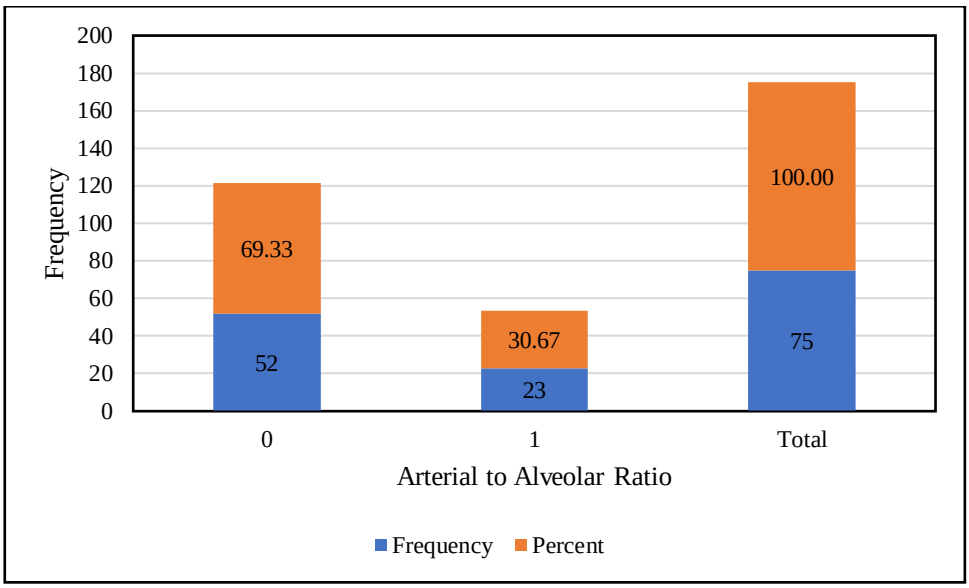

Fig 29: Arterial to Alveolar Ratio

Table 31: PtCO2 to FIO2 Ratio

Variable	N	Mean	Std. Error of Mean	Median	Std. Deviation	Minimum	Maximum
Ptco2 to Fio2 Ratio	75	214.68	15.594	192	135.044	45	480

The Ptco2 to Fio2 Ratio, a key indicator of respiratory efficiency, exhibits a mean of 214.68 in this study (N = 75). The standard deviation is 135.044, indicating substantial variability.

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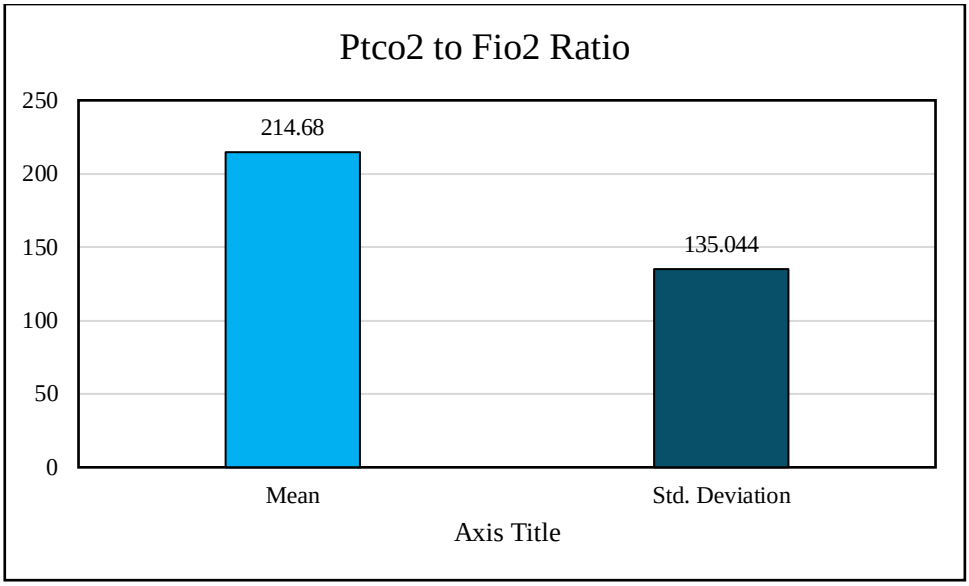

Fig 30: PtCO2 to FIO2 Ratio

Table 31: Oxygenation Index

Variable	N	Mean	Std. Error of Mean	Std. Deviation
Oxygenation Index	75	5.91	0.923	7.992

The Oxygenation Index, a critical measure of respiratory efficiency, has a mean value of 5.91 in this study (N = 75). The standard deviation is 7.992, indicating a substantial variability in the data.

Table 32: Chest X-ray Scoring

Variable	N	Mean	Median	Std. Deviation	Std. Error of Mean
Chest X-ray Scoring	75	6.23	5	5.835	0.674

The Chest X-ray Scoring, a crucial measure in assessing neonatal respiratory conditions, has a mean value of 6.23 in this study (N = 75). The median score is 5, indicating a skewed distribution. The standard deviation is 5.835, reflecting substantial variability in the data.

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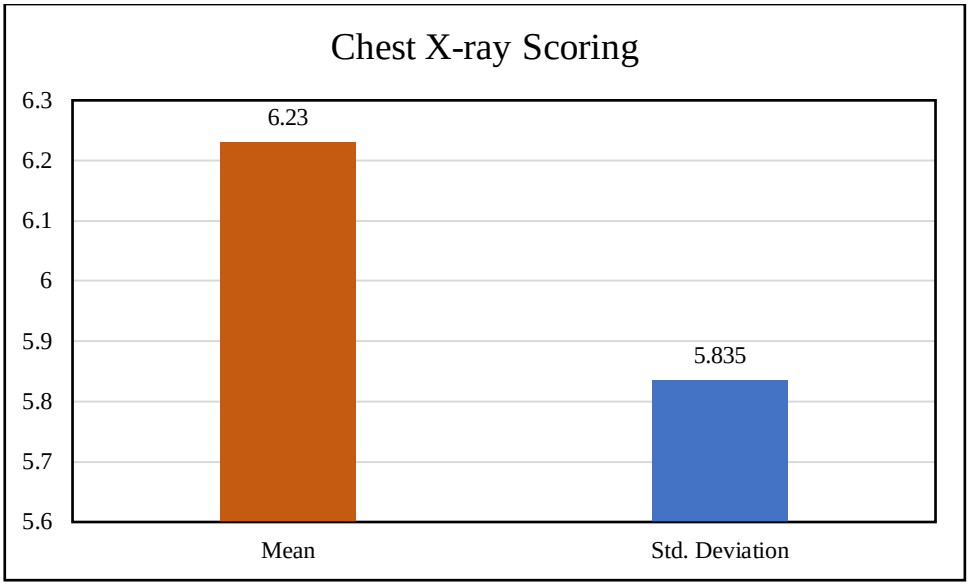

Fig 31: Chest X-ray Scoring

Table 33. Lung Ultrasonography (Lung USG) Scoring (At Admission/12 Hours)

Variable	N	Mean	Std. Deviation	Median	Minimum	Maximum
Lung USG Scoring at Admission	75	5.69	5.604	4	0	21
Lung USG Scoring at 12 hr	75	0.37	0.487	0	0	1

The mean Lung Ultrasonography (USG) score at admission was 5.69 (SD = 5.604), indicating a diverse range of respiratory conditions. At 12 hours post-admission, the mean score significantly decreased to 0.37 (SD = 0.487), suggesting an overall improvement in the neonates' respiratory status.

Table 34: correlation between the lung ultrasound (USG) scores at admission and at 12 hours

Pair 2	N	Correlation	Sig.
Lung USG scoring at admission & Lung USG scoring at 12 hr	75	0.816	0.00

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Table 35: Paired Samples Test Comparing lung ultrasound (USG) scores at admission and at 12 hours

Pair 1	Paired Differences					t	Df	Sig. (2-tailed)
	Mean	Std. Deviation	Std. Error Mean	95% CID*				
				Lower	Upper			
Lung USG scoring at admission – Lung USG scoring at 12 hr	.533	3.481	.402	-.268	1.334	1.327	74	.189

Note: *Confidence Interval of the Difference

A paired samples test was used to compare lung ultrasound scores at admission and 12 hours. The mean difference was 0.533, with a standard deviation of 3.481 and a standard error of 0.402. The 95% confidence interval for the mean difference ranged from -0.268 to 1.334.

Table 36: Surfactant

Surfactant	Frequency	Percent
Not Given	47	62.67
Given	28	37.33
Total	**75**	**100.00**

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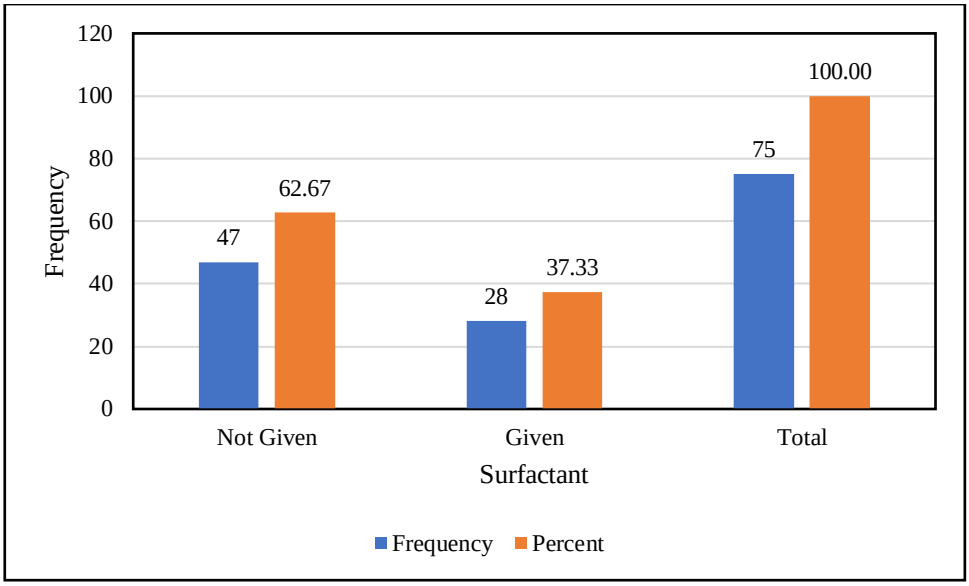

Fig 32: Surfactant

In the study, the administration of surfactant was recorded for a total of 75 patients. The data reveals that surfactant was not given to 47 patients, representing 62.7% of the total sample. Conversely, surfactant was administered to 28 patients, which constitutes 37.3% of the sample.

Table 37: Descriptive statistics of the scores

Variable	N	Mean	Std. Deviation
Chest X-ray Scoring	75	1.27	1.417
Lung USG Scoring at Admission	75	6.23	5.835
Lung USG Scoring at 12 hr	75	5.69	5.604

The variable 'Chest X-ray Scoring' had a mean score of 1.27 (SD = 1.417), suggesting a relatively low average score with a moderate degree of variability. In other way, 'Lung Ultrasound (USG) Scoring at Admission' variable had a higher mean score of 6.23 (SD = 5.835), indicating a higher average score upon admission with a substantial degree of variability in the scores. Further, 'Lung USG Scoring at 12 hours' variable showed a slight

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decrease in the average score from admission to 12 hours later, with a mean score of 5.69 (SD = 5.604) and a similar degree of variability in the scores.

Table 38: Correlation Matrix Between Lung USG Scoring at Admission and Clinical/Biochemical Parameters

Variable	Pearson's r	p-value	Lower 95% CI	Upper 95% CI
Silverman Anderson Scoring at Admission	***0.239***	***0.039****	***0.012***	***0.442***
ABG Ph	-0.177	0.129	-0.388	0.052
ABG PaO2	0.055	0.639	-0.174	0.278
ABG PaCO2	0.003	0.981	-0.224	0.230
ABG HCO3	-0.206	0.076	-0.413	0.022
Lactate	0.117	0.316	-0.113	0.335
ABG pH at 12 hr	***-0.233***	***0.045****	***-0.437***	***-0.006***
ABG PaO2 at 12 hr	-0.090	0.440	-0.311	0.139
ABG PaCO2 at 12 hr	0.115	0.326	-0.115	0.333
ABG HCO3 at 12 hr	***-0.230***	***0.047****	***-0.434***	***-0.003***
ABG Lactate	0.086	0.464	-0.144	0.307
Alveolar-arterial Gradient	0.128	0.272	-0.102	0.345
Arterial to Alveolar Ratio	-0.177	0.129	-0.388	0.052
Ptco2 to Fio2 Ratio	-0.124	0.289	-0.342	0.106
Oxygenation Index	0.065	0.579	-0.164	0.288
FiO2 at Admission	0.116	0.324	-0.114	0.334
APGAR at 1 min	-0.104	0.376	-0.323	0.126
APGAR at 10 min	-0.092	0.435	-0.312	0.138

Note: This table presents the Pearson correlation coefficients (r) and their corresponding p-values for the relationships between lung USG scoring at admission and various clinical/biochemical parameters. Significant correlations are indicated with asterisks based on their p-values: *p < .05, **p < .01, ***p < .001.

There was a statistically significant correlation between lung ultrasonography (USG) scoring at admission and Silverman Anderson scoring at admission. The Pearson's r value for this correlation was 0.239 with a p-value of 0.039, indicating a positive relationship. The 95%

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confidence interval for this correlation was [0.012, 0.442], suggesting that higher lung USG scores were associated with higher Silverman Anderson scores at admission. Additionally, a negative correlation was found between lung USG scoring at admission and arterial blood gas (ABG) pH at 12 hours. The Pearson's r value for this correlation was -0.233 with a p-value of 0.045, and the 95% confidence interval was [-0.437, -0.006]. This indicates that higher lung USG scores at admission were associated with lower pH levels at 12 hours.

Similarly, there was a negative correlation between lung USG scoring at admission and ABG bicarbonate (HCO3) levels at 12 hours. The Pearson's r value for this correlation was -0.230 with a p-value of 0.047, and the 95% confidence interval for this correlation was [-0.434, -0.003]. This suggests that higher lung USG scores were associated with lower bicarbonate levels at 12 hours. Several correlations between lung USG scoring at admission and other parameters were not statistically significant. These included the correlations with ABG pH (Pearson's r = -0.177, p = 0.129), ABG PaO2 (Pearson's r = 0.055, p = 0.639), ABG PaCO2 (Pearson's r = 0.003, p = 0.981), ABG HCO3 (Pearson's r = -0.206, p = 0.076), and lactate levels (Pearson's r = 0.117, p = 0.316).

Other non-significant correlations were observed with ABG PaO2 at 12 hours (Pearson's r = -0.090, p = 0.440), ABG PaCO2 at 12 hours (Pearson's r = 0.115, p = 0.326), and ABG lactate at 12 hours (Pearson's r = 0.086, p = 0.464). The alveolar-arterial gradient (Pearson's r = 0.128, p = 0.272), arterial to alveolar ratio (Pearson's r = -0.177, p = 0.129), and Ptco2 to Fio2 ratio (Pearson's r = -0.124, p = 0.289) also showed non-significant correlations. Additionally, the oxygenation index (Pearson's r = 0.065, p = 0.579), FiO2 at admission (Pearson's r = 0.116, p = 0.324), APGAR score at 1 minute (Pearson's r = -0.104, p = 0.376), and APGAR score at 10 minutes (Pearson's r = -0.092, p = 0.435) did not show significant correlations with lung USG scoring at admission.

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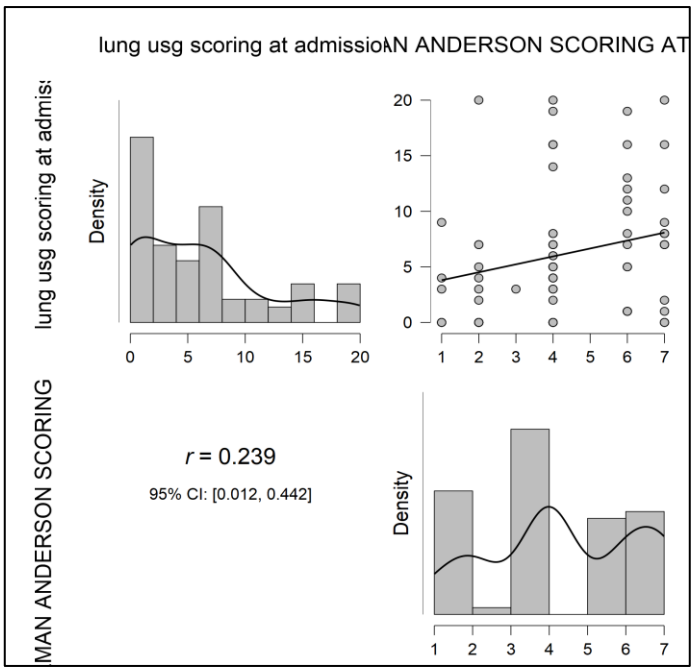

Fig 33: Correlation Plot of Lung USG scoring at admission and Silverman Anderson scoring at admission

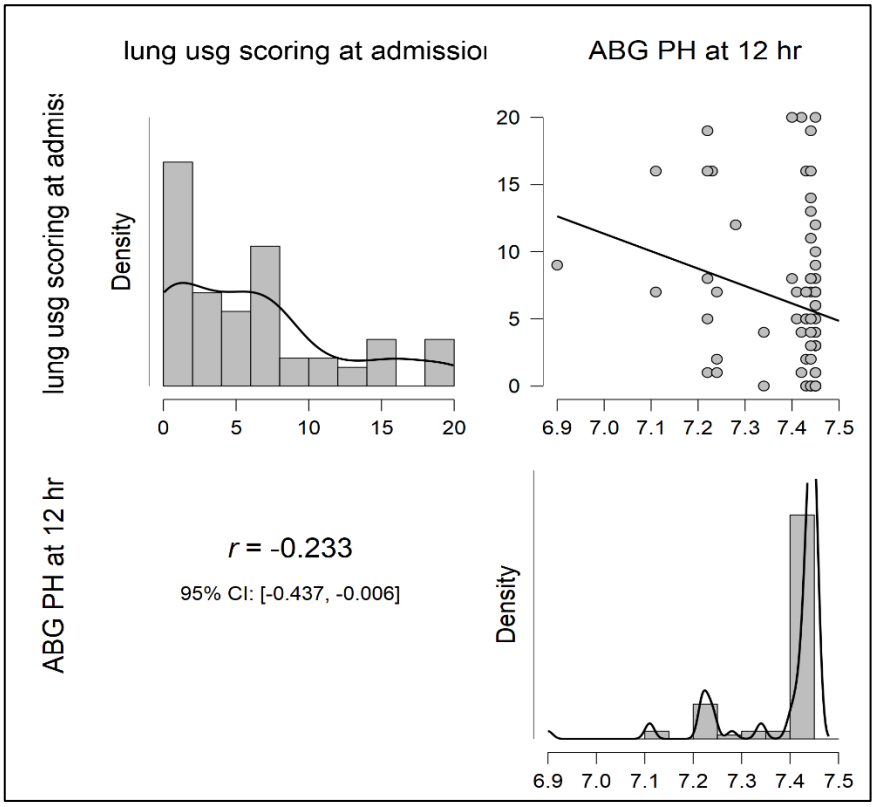

Fig 34: Correlation Plot of Lung USG scoring at admission and ABG pH at 12 hours

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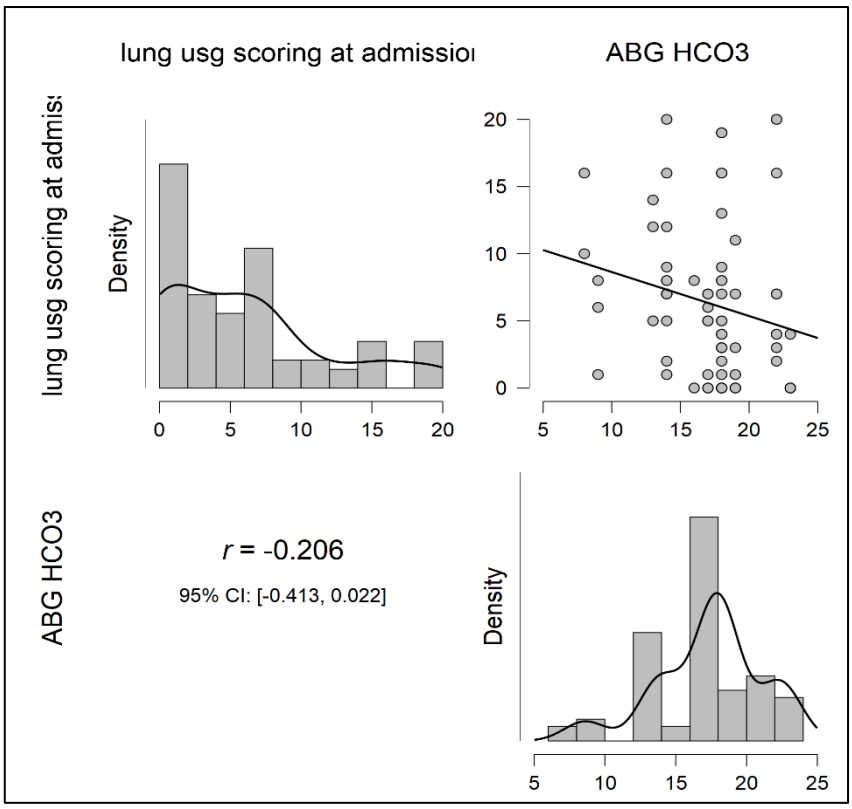

Fig 35: Correlation Plot of Lung USG scoring at admission and ABG HCO3 at 12 hours

Table 39: Correlation Between Lung USG Scoring at 12 Hours and Clinical/Biochemical Parameters

Variable	Pearson's r	p-value	Lower 95% CI	Upper 95% CI
Silverman Anderson Scoring at Admission	0.334	0.003 **	0.116	0.521
ABG pH	-0.163	0.163	-0.376	0.067
ABG PaO2	0.132	0.258	-0.098	0.349
ABG PaCO2	0.033	0.778	-0.195	0.258
ABG HCO3	-0.170	0.146	-0.382	0.060
Lactate	0.146	0.211	-0.084	0.361
ABG pH at 12 hr	-0.234	0.043 *	-0.438	-0.008
ABG PAO2 at 12 hr	-0.197	0.091	-0.406	0.032
ABG PACO2 at 12 hr	0.159	0.174	-0.071	0.372
ABG HCO3 at 12 hr	-0.187	0.108	-0.397	0.042

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ABG LACTATE	0.069	0.555	-0.160	0.292
Alveolar-arterial Gradient	0.209	0.072	-0.019	0.416
Arterial to Alveolar Ratio	-0.215	0.064	-0.422	0.012
Ptco2 to Fio2 Ratio	-0.179	0.124	-0.390	0.050
Oxygenation Index	0.126	0.282	-0.104	0.343
FiO2 at Admission	0.181	0.121	-0.048	0.392
APGAR at 1 min	-0.187	0.109	-0.397	0.042
APGAR at 10 min	-0.199	0.087	-0.408	0.029

Note: This table presents the Pearson correlation coefficients (r) and their corresponding p-values for the relationships between Lung USG Scoring at 12 Hours and various clinical/biochemical parameters. Significant correlations are indicated with asterisks based on their p-values: *p < .05, **p < .01, ***p < .001.

In terms of interpretation, the table shows that the Silverman Anderson Scoring at Admission has a significant positive correlation with the Lung USG Scoring at 12 hours (r = 0.334, p = 0.003). This suggests that as the Silverman Anderson Scoring at Admission increases, the Lung USG Scoring at 12 hours also tends to increase. On the other hand, the ABG pH at 12 hr shows a significant negative correlation with the Lung USG Scoring at 12 hours (r = -0.234, p = 0.043), indicating that as the ABG pH at 12 hr decreases, the Lung USG Scoring at 12 hours tends to increase. Other variables do not show a significant correlation with the Lung USG Scoring at 12 hours.

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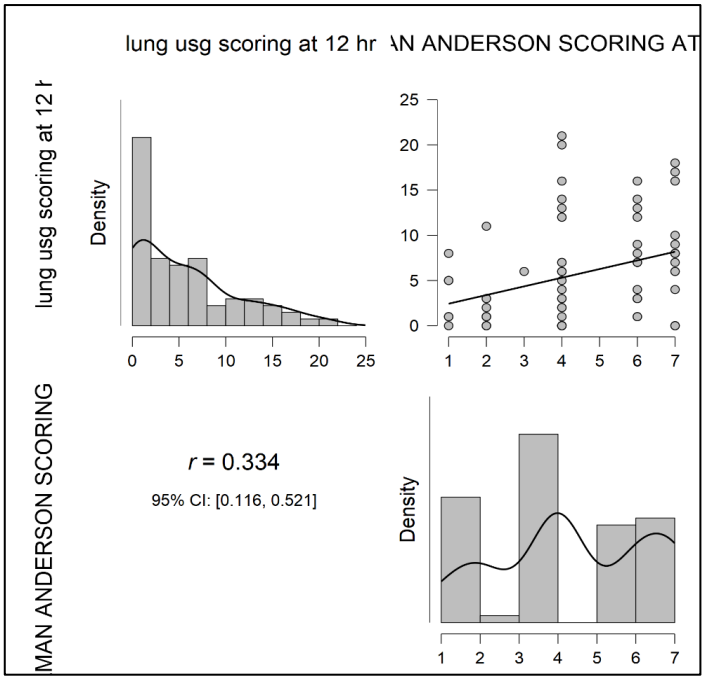

Fig 36: Correlation Plot of Lung USG Scoring at 12 hours and Silverman Anderson Scoring at Admission

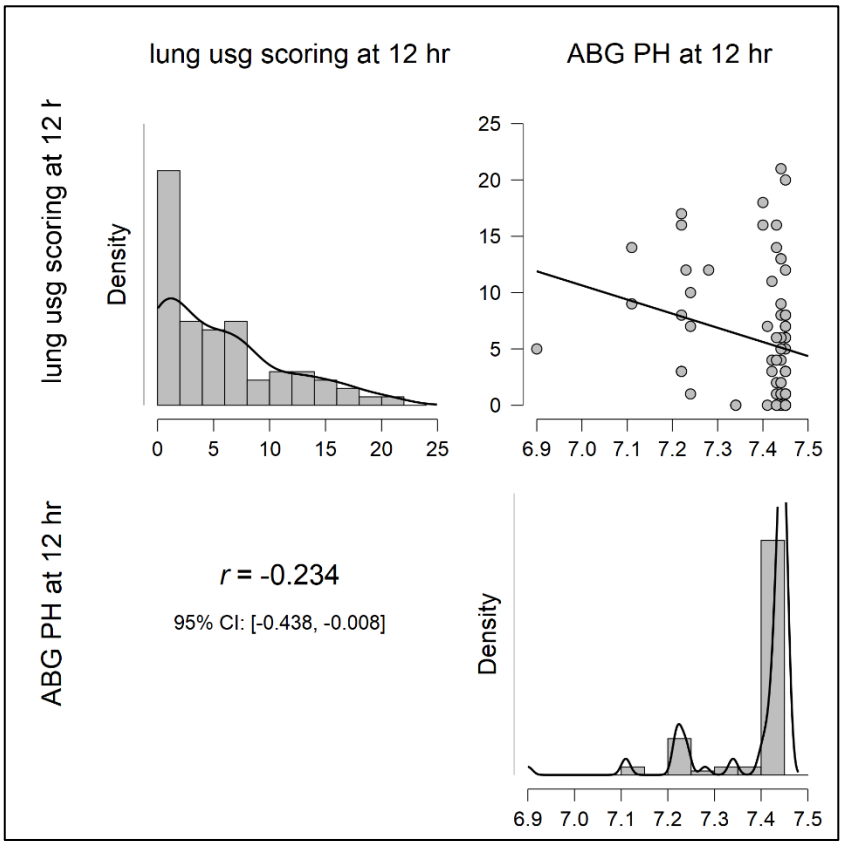

Fig 37: Correlation Plot of Lung USG Scoring at 12 hours and ABG pH at 12 hr

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Table 40: Correlation Between Chest X-ray Scoring and Clinical/Biochemical Parameters

Variable	Pearson's r	p-value	Lower 95% CI	Upper 95% CI
Silverman Anderson Scoring at Admission	***0.338***	***0.003 *****	***0.116***	***0.521***
ABG Ph	0.035	0.765	-0.194	0.263
ABG PaO2	-0.109	0.350	-0.327	0.122
ABG PaCO2	0.004	0.976	-0.218	0.225
ABG HCO3	-0.044	0.708	-0.263	0.176
Lactate	-0.045	0.704	-0.264	0.175
ABG pH at 12 hr	-0.135	0.247	-0.354	0.094
ABG PaO2 at 12 hr	-0.097	0.407	-0.317	0.133
ABG PaCO2 at 12 hr	0.204	0.080	-0.015	0.408
ABG HCO3 at 12 hr	-0.090	0.442	-0.310	0.140
ABG Lactate	-0.102	0.383	-0.321	0.128
Alveolar-arterial Gradient	***0.261***	***0.024 ****	***0.035***	***0.464***
Arterial to Alveolar Ratio	-0.164	0.160	-0.376	0.056
Ptco2 to Fio2 Ratio	-0.194	0.095	-0.404	0.026
Oxygenation Index	***0.235***	***0.043 ****	***0.011***	***0.443***
FiO2 at Admission	***0.296***	***0.010 *****	***0.079***	***0.492***
APGAR at 1 min	-0.177	0.129	-0.388	0.052
APGAR at 10 min	***-0.237***	***0.041 ****	***-0.444***	***-0.012***

Note: This table presents the Pearson correlation coefficients (r) and their corresponding p-values for the relationships between lung Chest X-ray Scoring and various clinical/biochemical parameters. Significant correlations are indicated with asterisks based on their p-values: *p < .05, **p < .01, ***p < .001.

There was a significant positive correlation between Silverman Anderson scoring at admission and Chest X-ray scoring, with a Pearson's r value of 0.338 and a p-value of 0.003. The 95% confidence interval ranged from 0.116 to 0.521, indicating that higher Chest X-ray scores are

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associated with higher Silverman Anderson scores at admission. A significant positive correlation was also found between Chest X-ray scoring and the alveolar-arterial gradient (r=0.261, p=0.024). The 95% confidence interval for this correlation ranged from 0.035 to 0.464, suggesting that higher Chest X-ray scores are linked to higher alveolar-arterial gradients.

The oxygenation index showed a significant positive correlation with Chest X-ray scoring (r=0.235, p=0.043), with a 95% confidence interval ranging from 0.011 to 0.443. This indicates that higher Chest X-ray scores correspond to higher oxygenation index values. Furthermore, a significant positive correlation was observed between Chest X-ray scoring and FiO2 at admission (r=0.296, p=0.010). The 95% confidence interval ranged from 0.079 to 0.492, suggesting that higher Chest X-ray scores are associated with higher FiO2 levels at admission.

A significant negative correlation was noted between Chest X-ray scoring and APGAR scores at 10 minutes (r=−0.237, p=0.041). The 95% confidence interval ranged from -0.444 to -0.012, indicating that higher Chest X-ray scores are associated with lower APGAR scores at 10 minutes. In contrast, several correlations between Chest X-ray scoring and other parameters were not statistically significant. These included ABG pH (r=0.035, p=0.765), ABG PaO2 (r=−0.109, p=0.350), ABG PaCO2 (r=0.004, p=0.976), ABG HCO3 (r=−0.044, p=0.708), and lactate levels (r=−0.045, p=0.704). Other non-significant correlations were observed with ABG pH at 12 hours (r=−0.135, p=0.247), ABG PaO2 at 12 hours (r=−0.097, p=0.407), and ABG PaCO2 at 12 hours (r=0.204, p=0.080). Additionally, there were no significant correlations with ABG HCO3 at 12 hours (r=−0.090, p=0.442), ABG lactate at 12 hours (r=−0.102, p=0.383), arterial to alveolar ratio (r=−0.164, p=0.160), and Ptco2 to Fio2 ratio (r=−0.194, p=0.095). Finally, the correlation between Chest X-ray scoring and APGAR scores at 1 minute was not significant (r=−0.177, p=0.129), with a 95% confidence interval ranging from -0.388 to 0.052.

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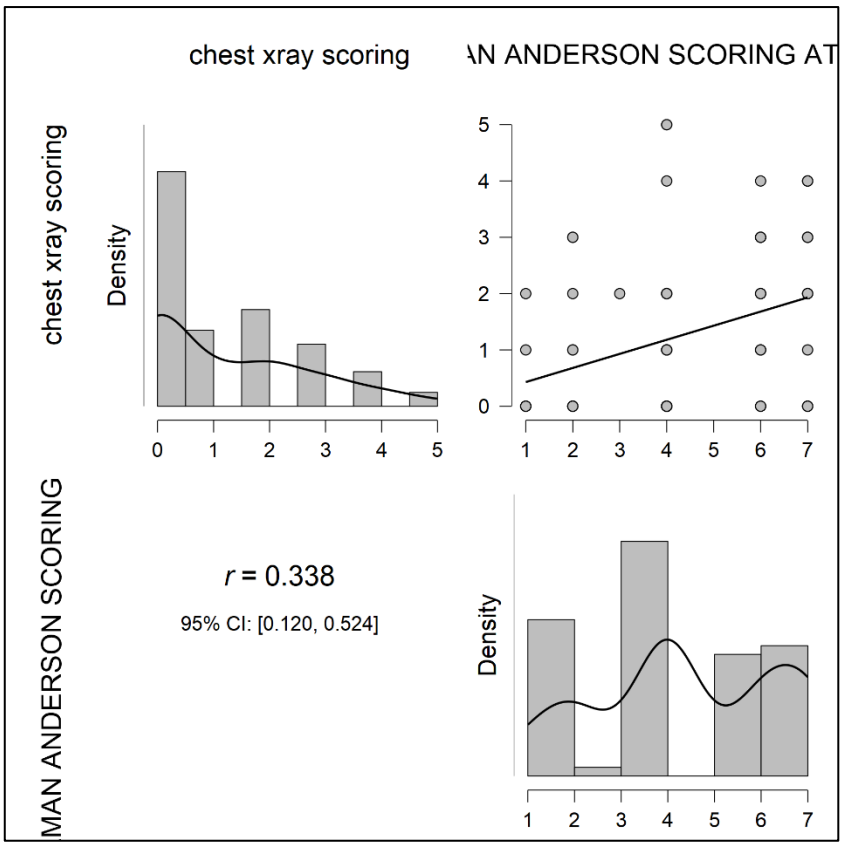

Fig 38: Correlation Plot of Chest X ray Scoring and Silverman Anderson Scoring

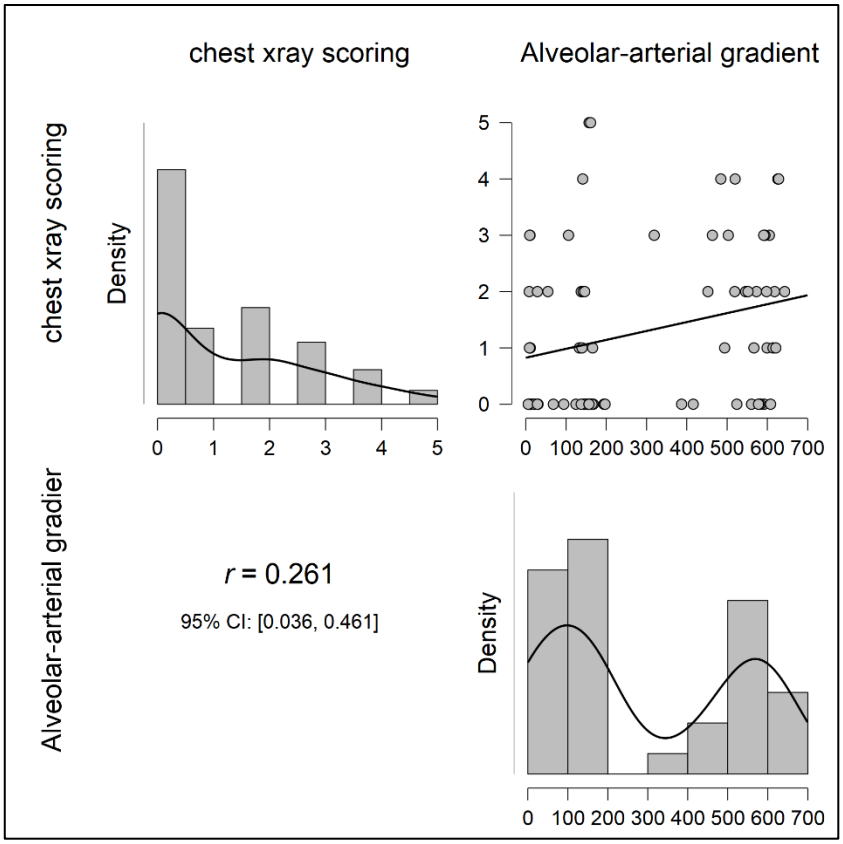

Fig 39: Correlation Plot of Chest X ray Scoring and Alveolar arterial gradient

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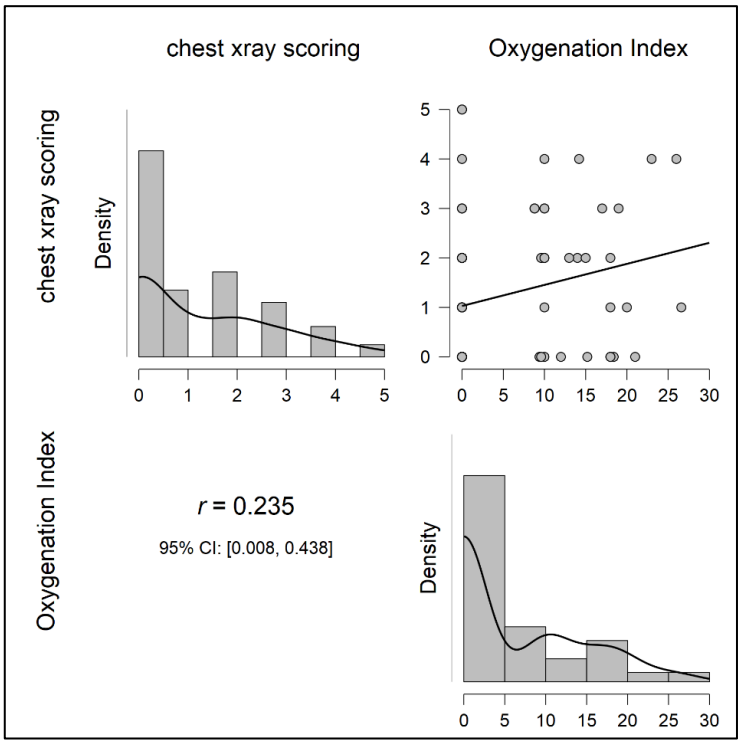

Fig 40: Correlation Plot of Chest X ray Scoring and Oxygenation Index

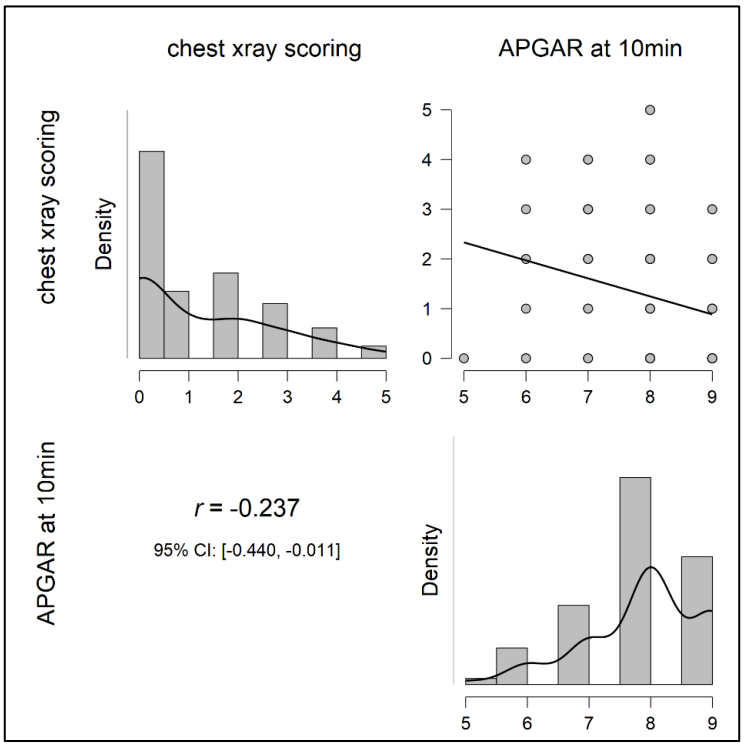

Fig 41: Correlation Plot of Chest X ray Scoring and APGAR at 10 min

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Table 41: Area under the curve

Test Result Variable(s)	Area	Std. Error [a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Chest Xray Scoring	.671	.066	.014	.542	.799
Lung USG Scoring at Admission	.725	.063	.001	.601	.849
Lung Usg Scoring At 12 Hr	.791	.059	.000	.675	.908

Table 41 observes the Area Under Curve (AUC) analysis provides insights into the diagnostic accuracy of the test variables. The AUC for chest X-ray scoring was 0.671 (SE = 0.066, p = 0.014, 95% CI [0.542, 0.799]), indicating moderate accuracy. Lung USG scoring at admission had a higher AUC of 0.725 (SE = 0.063, p = 0.001, 95% CI [0.601, 0.849]), suggesting good diagnostic accuracy. Lung USG scoring at 12 hours showed the highest AUC of 0.791 (SE = 0.059, p < 0.001, 95% CI [0.675, 0.908]), indicating strong diagnostic performance.

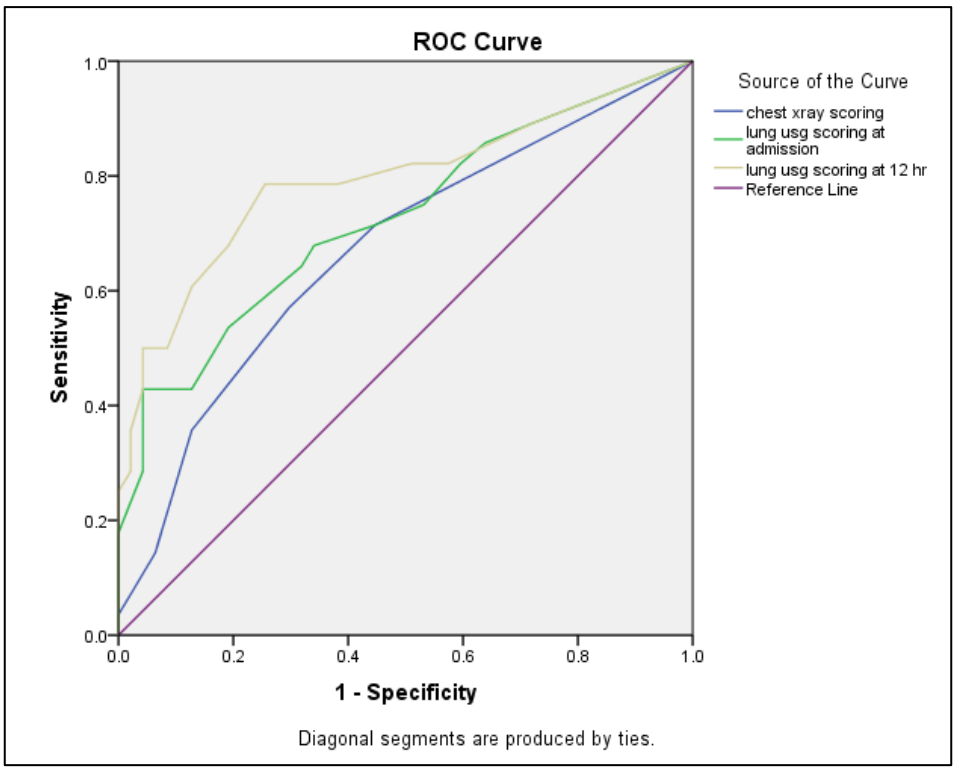

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Fig 42: ROC curves.

The coordinates of the curve were used to identify the sensitivity and specificity of different cutoff points for each test variable. For chest X-ray scoring, a cutoff of 0.50 yielded a sensitivity of 0.714 and a specificity of 0.553, while a cutoff of 2.50 resulted in a sensitivity of 0.357 and a specificity of 0.872. This demonstrates that higher cutoffs generally reduce sensitivity but increase specificity. In terms of lung USG scoring at admission, a cutoff of 0.50 provided a sensitivity of 0.893 and a specificity of 0.277, whereas a cutoff of 7.50 resulted in a sensitivity of 0.536 and a specificity of 0.809, showing that higher cutoffs lead to reduced sensitivity but improved specificity. Similarly, for lung USG scoring at 12 hours, a cutoff of 0.50 had a sensitivity of 0.893 and a specificity of 0.277, and a cutoff of 6.50 had a sensitivity of 0.679 and a specificity of 0.809, indicating that increased cutoffs decreased sensitivity while enhancing specificity. The findings indicate that lung USG scoring, both at admission and at 12 hours, provides better diagnostic accuracy compared to chest X-ray scoring for evaluating the need for surfactant in neonates receiving respiratory support. The AUC values for lung USG scoring are higher, suggesting that this method is more reliable for assessing neonatal respiratory conditions. Lung USG scoring at 12 hours demonstrated the highest AUC, suggesting that it is the most effective tool among those studied for making clinical decisions regarding surfactant administration. The high sensitivity at lower cutoffs and the increased specificity at higher cutoffs provide clinicians with flexibility in using lung USG scores to tailor interventions based on the severity of the neonate's condition.

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Table 42: Neonatal Respiratory Pre- and Post-Surfactant Administration Measurements

Pair	Variable	Mean	Std. Deviation	Std. Error Mean
1	FIO2 at Admission	54.80	26.425	3.051
	FIO2 after Surfactant	38.55	21.474	2.480
2	Chest X-ray Scoring	1.27	1.417	0.164
	Chest X-ray Scoring after Surfactant	0.59	0.856	0.099
3	LUS Scoring at Admission	6.23	5.835	0.674
	LUS Scoring after Surfactant	1.17	1.083	0.125

The mean FiO2 at admission was 54.80% (SD = 26.425%). Following surfactant administration, the mean FiO2 decreased to 38.55% (SD = 21.474%). The mean Chest X ray score at admission was 1.27 (SD = 1.417). This score improved after surfactant administration, with a mean of 0.59 (SD = 0.856). The mean LUS score at admission was 6.23 (SD = 5.835). Surfactant administration led to a significant decrease in the mean LUS score to 1.17 (SD = 1.083).

Table 43: Correlations Between Pre- and Post-Surfactant Measurements

Pair	Variables	Correlation	Sig.
1	FIO2 at Admission & FIO2 after Surfactant	0.630	.000
2	Chest X-ray Scoring & Chest X-ray Scoring after Surfactant	0.560	.000
3	LUS Scoring at Admission & LUS Scoring after Surfactant	0.272	.018

A strong positive correlation exists between FiO2 at admission and FiO2 after surfactant administration ($r = .630$, $p = .000$). A significant positive correlation is observed between chest X-ray scores at admission and chest X-ray scores after surfactant administration ($r = .560$, $p = .000$). A moderate positive correlation is evident between lung ultrasound scores (LUS) at admission and LUS scores after surfactant administration ($r = .272$, $p = .018$).

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Table 44: The effectiveness of surfactant administration on Respiratory Parameters in Neonates:

Pair	Variable	Mean Difference	Std. Deviation	Std. Error Mean	95% CI of the Difference	t	df	Sig. (2-tailed)
1	FIO2 at Admission – FIO2 after Surfactant	16.253	21.069	2.433	11.406 to 21.101	6.681	74	.000
2	Chest X-ray Scoring - Chest X-ray Scoring after Surfactant	0.680	1.176	0.136	0.409 to 0.951	5.008	74	.000
3	Lung Ultrasound Scoring at Admission – LUS Scoring after Surfactant	5.053	5.637	0.651	3.756 to 6.350	7.763	74	.000

A paired samples t-test comparing FIO2 at admission to FIO2 after surfactant administration revealed a statistically significant difference, $t(74) = 6.681$, $p < .001$. The mean difference in FIO2 levels between the two time points was 16.253% (SD = 21.069), with a 95% confidence interval of 11.406% to 21.101%. Chest x-ray scoring at admission to chest x-ray scoring after surfactant administration also revealed a statistically significant difference, $t(74) = 5.008$, $p < .001$. The mean difference in chest x-ray scores was 0.680 (SD = 1.176), with a 95% confidence interval of 0.409 to 0.951. lung ultrasound scoring at admission to lung ultrasound scoring after surfactant administration showed a statistically significant difference, $t(74) = 7.763$, $p < .001$. The mean difference in lung ultrasound scores was 5.053 (SD = 5.637), with a 95% confidence interval of 3.756 to 6.350.

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Discussion:

The present study evaluated the diagnostic accuracy of lung ultrasonography (LUS) scores in assessing oxygenation and the need for surfactant therapy in neonates with respiratory support, comparing it to chest X-ray (CXR) scores. The findings of our study align well with and expand upon the existing literature.

The results of this study highlight the utility of lung ultrasonography (USG) in evaluating oxygenation and determining the need for surfactant in neonates requiring respiratory support. Among the 75 neonates included, 52% were delivered by Cesarean section, and 48% were born via normal vaginal delivery. The sample had a slight male predominance, with 61.33% male neonates. The mean gestational age of 35.52 weeks and the mean birth weight of 2.1453 kilograms indicate that the sample consisted of preterm and low birth weight infants, which are common risk factors for respiratory distress.

The study found that the Silverman Anderson Scoring at admission showed a significant positive correlation with Chest X-ray scoring, with higher scores on chest X-rays associated with higher Silverman Anderson scores. This correlation reinforces the reliability of Silverman Anderson scoring as a clinical tool for assessing respiratory distress in neonates. The correlation between chest X-ray scoring and various parameters such as the alveolar-arterial gradient, oxygenation index, and FiO2 at admission further supports the use of chest X-ray as a diagnostic measure, although it is less sensitive than lung USG scoring.

In present study, the mean LUS score at admission was 5.69 (SD = 5.604), which significantly decreased to 0.37 (SD = 0.487) at 12 hours post-admission, suggesting an overall improvement in respiratory status. The area under the curve (AUC) for LUS scoring at admission was 0.725 (SE = 0.063, $p = 0.001$, 95% CI [0.601, 0.849]), and for LUS scoring at 12 hours, the AUC was 0.791 (SE = 0.059, $p < 0.001$, 95% CI [0.675, 0.908]). These findings indicate strong diagnostic

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performance of LUS, consistent with Brat et al.,[11] who reported an AUC of 0.71 for neonates with a gestational age (GA) of 34 weeks or more and 0.93 for those with a GA less than 34 weeks . Similarly, De Martino et al.[12] found that LUS scores could predict the need for surfactant with an AUC of 0.94 for the first dose and 0.803 for redosing .

Present study found a significant positive correlation between the LUS score at admission and the Silverman Anderson score at admission (Pearson's $r = 0.239$, $p = 0.039$) and a significant negative correlation with ABG pH at 12 hours (Pearson's $r = -0.233$, $p = 0.045$). These correlations indicate that higher LUS scores are associated with higher clinical severity and lower blood pH levels. This is comparable to findings by De Martino et al.,[12] who reported significant correlations between LUS and oxygenation index ($\rho = 0.6$; $P < .0001$), emphasizing the relevance of LUS in predicting oxygenation status and surfactant need. Brat et al.[11] also observed significant correlations between LUS scores and oxygenation indices such as the Ptco2 to Fio2 ratio, alveolar-arterial gradient, and oxygenation index in neonates of different gestational ages.

Present study identified that LUS scores provide better diagnostic accuracy for assessing the need for surfactant therapy compared to CXR scores. The AUC for LUS scoring at admission was higher than that for CXR scoring (0.725 vs. 0.671), supporting its use in clinical decision-making. Similar conclusions were drawn by Perri A et al.,[53] who found that LUS had a higher AUC (0.94) than CXR (0.80) for early recognition of infants requiring surfactant. Additionally, Vardar et al.[14] highlighted the superior predictive power of LUS scores (AUC: 1.00) compared to CXR, further corroborating our findings.

In present study, LUS scoring at admission demonstrated a sensitivity of 0.893 and a specificity of 0.277 at a cutoff of 0.50, and a sensitivity of 0.536 and a specificity of 0.809 at a cutoff of 7.50. These findings align with those of Raimondi F et al.,[10] who reported an AUC of 0.86 for

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LUS with a sensitivity of 0.79 and specificity of 0.83 at a cutoff score of 9. The high sensitivity at lower cutoffs and increased specificity at higher cutoffs observed in our study provide clinicians with flexibility in tailoring interventions based on the severity of the neonate's condition. Razak A et al.[58] also demonstrated similar findings with high sensitivity (88%) and specificity (82%) at a LUS score cut-off of >5-6.

Our study showed that the average LUS scores were higher at admission compared to CXR scores, and the decrease in LUS scores over 12 hours post-admission indicated a significant improvement in respiratory status. In this study, the mean FiO2 at admission was 54.80% (SD = 26.425%), which significantly decreased to 38.55% (SD = 21.474%) following surfactant administration. The mean Chest X-ray score at admission was 1.27 (SD = 1.417), improving to 0.59 (SD = 0.856) post-surfactant. Similarly, the mean LUS score dropped from 6.23 (SD = 5.835) to 1.17 (SD = 1.083). A strong positive correlation was found between FiO2 at admission and post-surfactant ($r = .630$, $p = .000$), and significant positive correlations were observed for chest X-ray ($r = .560$, $p = .000$) and LUS scores ($r = .272$, $p = .018$) at these time points. Paired samples t-tests indicated statistically significant improvements in FiO2 ($t(74) = 6.681$, $p < .001$), chest X-ray scores ($t(74) = 5.008$, $p < .001$), and LUS scores ($t(74) = 7.763$, $p < .001$) post-surfactant administration, with mean differences of 16.253%, 0.680, and 5.053 respectively.

Study results also observed that statistically significant decrease in both LUS scores (mean difference = 5.053) and chest X-ray scores (mean difference = 0.680) after surfactant administration. However, the magnitude of change is larger for LUS scores. This suggests that LUS might be more sensitive in detecting the initial improvement in lung function following surfactant therapy. This study provides compelling evidence for the efficacy of surfactant therapy in neonatal respiratory distress.

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This is in line with the study by Brat et al.,[11] where LUS scores effectively correlated with oxygenation indices and predicted the need for surfactant therapy more accurately than traditional methods. Furthermore, the significant positive correlation between Silverman Anderson scoring at admission and Chest X-ray scoring (Pearson's $r = 0.338$, $p = 0.003$) observed in our study aligns with findings from past research, highlighting the complementary role of both methods in assessing neonatal respiratory conditions.

The results of the present study reinforce the clinical utility of LUS in the neonatal intensive care setting. The significant correlations between LUS scores and other clinical and biochemical markers of respiratory distress underscore its value in providing real-time, bedside assessments of neonatal lung function. The higher diagnostic accuracy and predictive power of LUS compared to CXR, as evidenced by our study and corroborated by existing literature, suggest that LUS should be integrated into standard neonatal care protocols for early and accurate detection of surfactant need.

Our study adds to the growing body of evidence supporting the use of lung ultrasonography as a reliable and effective tool for evaluating oxygenation and predicting the need for surfactant therapy in neonates with respiratory support. The findings indicate that LUS scores, particularly when assessed at multiple time points, offer superior diagnostic accuracy and predictive capability compared to traditional CXR scores. Future research should focus on standardizing LUS protocols and training to ensure consistent and accurate application in clinical practice.

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Conclusion:

Our study confirms that lung ultrasonography (LUS) is a valuable tool for assessing oxygenation and predicting the need for surfactant therapy in neonates requiring respiratory support. The LUS scores demonstrated significant correlations with clinical severity indices and blood gas parameters, offering real-time, non-invasive assessments of neonatal lung function. Compared to chest X-ray (CXR) scores, LUS showed superior diagnostic accuracy and predictive power, underscoring its potential as a standard diagnostic method in neonatal intensive care units (NICUs), demonstrating its utility in detecting the need for surfactant with significant improvements in FiO2, chest X-ray, and LUS scores post-administration.

One major limitation of our study is the relatively small sample size, which may restrict the generalizability of the findings. Future research should involve larger, multicenter trials to validate our findings and ensure they are applicable across different settings and populations. Developing standardized protocols for performing and interpreting LUS in neonates would improve the consistency and reliability of results. Implementing comprehensive training programs for clinicians on the use of LUS could mitigate the operator dependency issue and enhance the accuracy of assessments. Additionally, future studies should include long-term follow-up to assess the impact of LUS-guided management on outcomes like bronchopulmonary dysplasia and other chronic lung conditions.

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BLDE

(DEEMED TO BE UNIVERSITY)

Declared as Deemed to be University u/s 3 of UGC Act, 1956

Accredited with 'A' Grade by NAAC (Cycle-2)

The Constituent College

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 650/2022-23 30/8/2022

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. In the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "LUNG ULTRASONOGRAPHY SCORE TO EVALUATE OXYGENATION AND SURFACTANT NEED IN NEONATES TREATED WITH RESPIRATORY SUPPORT".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR. APOORVA GAYATRI ABBADI

NAME OF THE GUIDE: Dr. M. M. Patil, Professor Dept. of Pediatrics.

Dr.Santoshkumar Jeevanagi
Chairperson
IEC-SBMPMC,
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikawadi
Member Secretary
IEC-SBMPMC,
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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ANNEXURE- II

RESEARCH INFORMED CONSENT FORM

BLDEU (DEEMED TO BE UNIVERSITY) Shri B.M. PATIL Medical College, Hospital & Research Centre,Vijayapura, Karnataka -586103.

TITLE OF THE PROJECT:

LUNG ULTRASONOGRAPHY SCORE TO EVALUATE OXYGENATION AND SURFACTANT NEED IN NEONATES TREATED WITH RESPIRATORY SUPPORT

GUIDE

DR.M M PATIL MD

PROFESSOR

DEPARTMENT OF PEDIATRICS

CO-GUIDE

DR.RAVIKUMAR

ASSISTANT PROFESSOR, DEPARTMENT OF RADIODIAGNOSIS

PG STUDENT

DR. APOORVA GAYATRI ABBADI

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PURPOSE OF RESEARCH:

LUNG ULTRASONOGRAPHY SCORE TO EVALUATE OXYGENATION AND SURFACTANT NEED IN NEONATES WITH RESPIRATORY SUPPORT

PROCEDURE:

I understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a final work up of the procedure and its outcome is planned.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the treatment.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record but will be stored in the investigations research file. If the data are used for publication in the

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medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time;
Dr. APOORVA GAYATRI ABBADI, at the department of pediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. APOORVA GAYATRI ABBADI may terminate my participation in the study after he/she has explained the reasons for doing so.

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INJURY STATEMENT:

I understand that in the unlikely event of injury to my child resulting directly from child's participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the child. But no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability

DR. APOORVA GAYATRI ABBADI Date

(Investigator)

DocuSign Envelope ID: BBFCDE7D-057E-49BE-930E-1BAC6291F161

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr. APOORVA GAYATRI ABBADI is doing a study on **LUNG ULTRASONOGRAPHY SCORE TO EVALUATE OXYGENATION AND SURFACTANT NEED IN NEONATES WITH RESPIRATORY SUPPORT** in Shri B. M. Patil Medical College Hospital Vijayapura, Karnataka.

Dr. APOORVA GAYATRI ABBADI has explained to us the purpose of research and the study procedure. We are willing to allow our child to get treated in Shri B.M. Patil Medical College Hospital, Vijayapura. We have been explained about the study, benefits and possible discomforts in detail in our native language and we understand the same. We are aware that child will get best treatment and no compensation like financial benefits will be given if our child's condition deteriorates and any untoward event happens, and we will not sue anyone regarding this. Therefore, we agree to give our full consent for child's participation as a subject in this research project.

_____ _____

(Parents / Guardian) (Witness to signature)

Date Date

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ANNEXURE- III

PROFORMA

1) B/O
2) IP NO:
3) Date of Birth :
4) Mode of delivery :
   a) NVD
   b) C-Section
   c) Ventouse
   d)Forceps
5) LSCS Indication:
6) EDD-
7) Gestational Age:
8) Gender -
   A) Female
   B) Male
9) Birth weight:

-<1000 gms
-1000-1500 gms
-1501-2000 gms
-2000-2500 gms
-2501-3000 gms
3000-3500 gms
->3500 gms

10) Classification: AGA/SGA/LGA

11parity a) Primi b) Multi

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12) Socio economic status -

a) Lower

b) Lower middle

c) Upper middle

D) upper

13) Residential area urban Rural

14) Contact no

15)PIH Yes No

16) Gestational Diabetes Mellitus Yes No

17) PPROM / PROM. Yes No.

18) Maternal Fever Yes. No.

19) Maternal UTI. Yes. No.

20) Maternal chorioamnioitis. Yes. No.

21)Antepartum Hemorrhage Yes No

22) H/0 Maternal Blood Transfusion yes No .

23) Medical Disorders

-Anemia

-Hypothyroidism

-Cardiac Disease

-Epilepsy

-Asthma

- Autoimmune Disorders

- Others

24) Maternal medical History

25) Maternal ultra-sonogram

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- Oligohydramnios
- Polyhydramnios
- Adequate
- Abnormal umbilical artery
- Normal
- Not Available

26) Doppler Umbilical artery

- Reduced
- Increased
- Reversed
- Normal
- Not Done

27) Antenatal steroids

- Yes
- No
- Don’t Know

28) CTG

- Reactive
- Non Reassuring
- Pathological

29) Multiple Gestation

- Twins
- Triplets
- No

30)APGAR at 1 Min

31) APGAR at 5min

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32)NICU Admission in 1 Hour

33) Meconium stained liquor

a)Yes

b) No

34) Gestational Age

- Term
- Late preterm (34-37)
- Mid preterm (30-24)
- Early preterm (<30weeks

35) Silverman Anderson scoring At Admission

- O-2
- 3-4
- >5

36) Primary Respiratory support

- Nasal prongs
- Hood
- NCPAP
- HHHFNC
- SIMV

37) FIO2 at Admission

-30%

-40%

-50%

-60%

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>60%

100%

Option 7

38) Sepsis

- Early onset sepsis
- Late onset sepsis

39) Sepsis

- Clinical
- Probable
- Proven

40) Respiratory Concerns

- RDS
- TTNB
- Congenital pneumonia
- Apnea

41) Cardiac concerns

- PDA
- PPHN
- Congenital Heart disease
- Other

42) CNS concerns

- Intra-ventricular Hemorrhage

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- Periventricular Hemorrhage
- Congenital Malformations

43) Hemoglobin

44) Total Counts

45) Platelet count

46) Absolute Neutrophil Count

47) Serum Creatinine

48) Antibiotics

- Piperacillin tazobactam
- Amikacin
- Meropenem
- Vancomycin
- Colistin
- Linezolid
- Fluconazole
- Amphotericin B
- Others

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49) Delayed Perinatal Adaptation

- Yes
- No
- Don't know

50) HIE

a) Yes

b) No

51) HIE Modified Sarnath and Saranth staging

a) Stage1

b) Stage 2

c) Stage 3

52) Therapeutic Hypothermia

A) Yes

b) No

53)Support In/At 4 Hours

Nasal prongs
Hood
CPAP
HFNC
SIMV
HFOV
Option 7

54) Duration of nasal prongs

55)CPAPInitiation in hours

56)Duration of Cpap or interval

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57)Cpap failure in hours

- <12

-12 TO 24

- 24 - 48

- ->3

58)Conventional ventilator if started HOL

59)HFOV if Started HOL

60)Surfactant

YES
NO

61) If Surfactant given

Course

-1
-2
-3

62)First surfactant timing

63)First surfactant FIO2

-30
-40
-50
-60
-80
-100

64)Second surfactant timing

65) MAP at first surfactant

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66)PPHN

YES
NO
Option 3

67) Oxygen saturation Index

68) Oxygenation Index

69)PPHN Severity

Based on OSI

-Mild<7.5
-Moderate 7.5 TO 12.5
-Severe >12.5

70)Pulmonary vasodilator

-Yes
-No

71) Air leaks

Yes
No

72) If air leak

-Right pneumothorax

-Left pneumothorax

- PIE

73) Additional findings

Support

NASAL PRONGS
HOOD
CPAP
HFNC
SIMV
HFOV

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Option 7

74) Time of ordering of chest x ray

75)Time of chest xray done

76) Time of LUS done

77)LUS Intervalin minutes

- 30
- 31 - 60
- 61-90
- 91 - 120
- >120

78) Chest x ray

79) Chest X ray scoring

-Decreased aeration / normal
-Reticulo granularity
-Air bronchogram, indistinct diaphragm and heart border
-Confluent opacification of lungs with loss of mediastinal and diaphragmatic Borders (white out

80)USG chest position

- Supine
- Prone

81)USG chest score

O 1 2 3 PNEUMOTHORAX PLEURALEFFUSION

UPPER ANTERIOR
LOWER ANTERIOR
UPPER LATERAL
LOWER LATERAL
UPPER POSTERIOR
LOWER POSTERIOR

LEFT LUS

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0 1 2 3 PNEUMOTHORAX PLEUREFFUSION

UPPER ANTERIOR
LOWER ANTERIOR
UPPER LATERAL
LOWER LATERAL
UPPER POSTERIOR
LOWER POSTERIOR

82)Surfactant

83)LUS scoring after surfactant

84)Chest xray scoring after surfactant

85)FIO2 after Surfactant

VARIABLES

-Alveolar-arterial gradient

-oxygenation index

-Arterial to alveolar ratio

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BIODATA OF THE GUIDE

NAME : Dr. M M PATIL

EDUCATION : M B B S – KIMS HUBLI

M D PEDIATRICS – KIMS HUBLI

KMC REGISTRATION NUMBER : 52830

WORK EXPERIENCE : UG TEACHER EXPERIENCE -14 YEARS

PG TEACHER EXPERIENCE – 8 YEARS

MEMBERSHIP : IAPL 2007p1100

PRESENTLY WORKING AS : PROFESSOR & HOD

DEPARMENT OF PAEDIATRICS

BLDE (DU), VIJAYAPURA

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BIODATA OF THE CO-GUIDE

NAME : Dr. RAVI KUMAR

EDUCATION : MBBS – BR AMC BANGLORE
DNBRADIODIAGNOSIS – MANIPAL HOSPITAL, NBE, NEWDELHI

KMC REGISTRATION NUMBER : 93103

WORK EXPERIENCE : UG TEACHER EXPERIENCE – 6YEARS
PG TEACHER EXPERIENCE – 4 YEARS

PRESENTLY WORKING AS : ASSISTANT PROFESSOR
DEPARTMENT OF RADIODIAGNOSIS
BLDE (DU), VIJAYAPURA, KARNATAKA.

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BIODATA OF THE INVESTIGATOR

NAME	DR. APOORVA GAYATRI ABBADI
DOB	01/11/1995
QUALIFICATION	
MBBS	MAMATA MEDICAL COLLEGE KHAMMAM, TELANGANA. PINCODE 507002. KMC REGISTRATION NUMBER 155845
PRESENTLY WORKING AS	POST GRADUATE STUDENT/ JUNIOR RESIDENT, DEPARTMENT OF PEDIATRICS, BLDE (DU), VIJAYAPURA , KARNATAKA.

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ANNEXURE IV

MASTER CHART

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