"A PROSPECTIVE OBSERVATIONAL STUDY OF PREDICTORS OF MORTALITY IN MECHANICALLY VENTILATED NEONATESAT A TERTIARY CARE CENTRE'"

By

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In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE IN PEDIATRICS

Under the Guidance of

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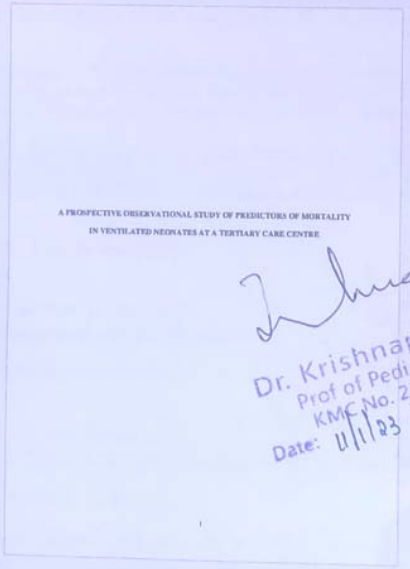
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ABBREVATIONS

GLOSSARY	ABBREVIATIONS
NICU	Neonatal Intensive Care Unit
RLJH	R L Jalappa Hospital
RLJH&RC	R L Jalappa Hospital and Research centre
SPSS	Statistical Package for Social Sciences
BMI	Body Mass Index
DIC	Disseminated Intravascular Coagulation
IVH	Intraventricular Haemorrhage
HMD	Hyaline Membrane Disease
MAS	Meconium Aspiration Syndrome
LBW	Low Birth Weight
FRC	Functional Residual Capacity
RBF	Renal Blood Flow
GFR	Glomerular Filtration Rate
NRDS	Neonatal Respiratory Distress Syndrome
AOP	Apnoea of Prematurity
GA	Gestational Age
VG	Volume Guarantee
PRVC	Pressure-Regulated Volume Control
TTV	Targeted Tidal Volume

HFOV	High Frequency Oscillatory Ventilation
FVC	Forced Vital Capacity
PEF	Peak Expiratory Flow
SIMV	Synchronised intermittent mandatory ventilation
ACV	Assist Control Ventilation
PSV	Pressure Support Ventilation
PSV-VG	Pressure Support with Volume Guarantee
PIP	Peak Inspiratory Pressure
ROP	Rates of Retinopathy of Prematurity
NEC	Necrotising Enterocolitis
MAP	Mean Airway Pressure
NICHD- **NRN**	National Institute of Child Health and Human Development Neonatal Research Network
VLBW	Very Low Birth Weight
ETT	Endotracheal Tube
ADHD	Attention Deficit Hyperactivity Disorder
ELBW	Extremely Low Birth Weight
PAV	Proportional Assist Ventilation
RDS	Respiratory Distress Syndrome

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ABSTRACT

BACKGROUND

Mechanical ventilation is one of the significant developments that has helped reduceinfant mortality in many parts of the world. Many unwell new-born's who are hospitalised to the neonatal intensive units (NICU) for a variety of clinical disordersrequire mechanical ventilation, however this has a number of difficulties and the outcome of these neonates is often unpredictable.

OBJECTIVES

To determine the predictors of mortality in newborns who are mechanically ventilated and to study association between duration of ventilation and mortality.

METHODS

This prospective observational study was done at Tamaka, Karnataka, in the Neonatal Intensive Care Unit (NICU), Paediatrics Department, RL Jalappa Hospital and Research Centre, from February 2021 to January 2022. The infants who needed to beput on mechanical ventilation were enrolled in the study. All the new borns'clinical profile, outcome, and complications was studied. Neonatal participants were split intotwo groups. Neonates who were successfully extubated and did not need to be re- intubated were classified as survivors, whereas those who died while on mechanical ventilation were classified as non-survivors. Clinical, demographic, biochemical data,ventilator parameters and complications were analysed to determine the factors leading to mortality of ventilated new-born's.

RESULTS

95 (100%) of the total new-borns admitted to the NICU throughout the research period received mechanical ventilation. Among 95 of these neonates, 48.4% were males and 51.6% females. There were 64.2%inborn babies and 35.8% out-born babies. The mean gestational age of ventilated neonates was 33.6+/-2.8. The mean gestational age of survivors group was 36.7 ± 2.9 weeks and non survivors group mean gestational age was 33.8 ± 3.2 weeks. The gestational age and birth weight of non-survivors were significantly different from survivors. Hyaline membrane disease(HMD) was the most common indication (47.8%), followed by birth asphyxia (28.4%), and meconium aspiration syndrome (MAS) (16.4%). We observed lesser incidence of shock in survival groups (1.5 %) compared to non-survivors' group (21.4%). Instances of pulmonary haemorrhage were noticeably more among non-survivors.($P > 0.05$). When compared to the non-survivors' group, DIC was considerably higher among non-survivors ($P > 0.05$).

CONCLUSION

Hyaline membrane disease (HMD), meconium aspiration syndrome (MAS), birth asphyxia, and congenital pneumonia were commonest indications for mechanical ventilation. Significantly higher mortality was seen amongst low birth weight (LBW), preterm neonates. And complications like persistent pulmonary artery hypertension,DIC, shock, pulmonary haemorrhage and IVH lead to adverse outcomes.

KEYWORDS

Mechanical Ventilation, Neonates, Complications, Mortality, Predictors.

A PROSPECTIVE OBSERVATIONAL STUDY OF PREDICTORS OF MORTALITY IN VENTILATED NEONATES AT A TERTIARY CARE CENTRE

INTRODUCTION

The primary goal in all severely ill patients in intensive care is to maintain life while minimising harm to crucial organs like the kidneys and brain. The management of patients with immediate life-threatening illnesses is the main focus of intensive care medicine. [1] More critically ill neonates are now being managed with great success because of the improvements in neonatal care units, but many of them still face a very critical outcome, and their fatality rate is still quite high.[2]

The survival of sick babies increased as mechanical ventilation came into use in neonatal intensive care units in the 1960s and 1970s. [3] Assisted ventilation has completely changed the new-born's fate in NICUs, one significant development in neonatal medicine that lowers new-born mortality. [4] The neonatal critical care unit has a considerable proportion of new-born's who require mechanical ventilation. The mortality rate for these infants on mechanical ventilation is significant. [4,5] The mortality rates of ventilated newborns in industrialised and poor nations may be affected by a variety of parameters, such as the readily available surfactant, qualified medical personnel, parenteral feeding, etc. Limited technical proficiency and technology development in less developed countries might also be factors. Although intensive care, including ventilation, has many benefits, it is labour- and financially intense to administer these operations. Additionally, infants were put on mechanical ventilators so far have significant rates of morbidity and mortality. Identification of risk factors is crucial for lowering mortality in this group of neonates. According to recent research, disease severity of the neonate at admission and birth weight are the main factors influencing infant death, along with gestational age.[6] In ventilated neonates, pH, PaCO4, base overload, and FiO2 have long been recognised as major risk factors.[4] The frequency of complications from ventilatory methods and strategies also affects the results.[6] Data on ventilated ill neonates was more abundant in developed countries. The decreased new-born mortality in the modern era is

largely due to the easier access to neonatal mechanical ventilation. However, a sizeable portion of ventilated infants do not survive negative consequences as a result of their stressful NICU stay and the numerous economic and infrastructure challenges faced in a low-income nation like India. A survival rate of 40% to 60% has been reported in several Indian studies.[7] A treatment protocol for use of mechanical ventilator implementation is required that help lower neonatal mortality and morbidity in a country like India where financial constraints limit technological developments. The current study aims to identify the predictors of death in these populations so we can adopt sufficient resource management and planning to increase the survival among mechanically ventilated neonates in critical care units in tertiary care centres.

OBJECTIVES

Objectives:

1. To identify the risk factors for mortality in neonates on mechanical ventilation.
2. To study the association between duration of ventilation and mortality.

STUDY JUSTIFICATION

For respiratory and cardiac support, the majority of infants in the intensive neonatal care units require mechanical ventilator support. After neonatal care units were established, morbidity and mortality considerably decreased, and the survival rate for neonates using artificial ventilation is reported to be 64–67%. [8] Even with the availability of mechanical ventilators, neonatal mortality remains high. As a result, early detection of risk factors is essential for a better outcome and prompt action. Numerous studies have demonstrated that a wide range of variables were strongly related to neonatal mortality when supported by a mechanical

ventilator. Effective clinical decision-making and resource allocation depend on having awareness of the unfavourable effects of ventilator support. This study will be conducted to learn the risk variables and parameters that are significantly associated with mortality in ventilated neonates admitted to the RLJH hospital because there haven't been any studies done in and around Kolar.

REVIEW OF LITERATURE

PHYSIOLOGY OF NEWBORN:

The physiology of a new-born is very complex as it changes with every minute, hour, day of life of the new-born. The physical characteristics of the new-borns may make it difficult for them to breathe effectively. Their paradoxical chest movements are a result of their rig cages that are cartilaginous, horizontal alignment of the ribs, and compliance of the lung that is low. Due to functional residual capacity (FRC), higher minute ventilation to FRC ratios being low and requirement of twice the oxygen levels to that of adults, they are often prone to desaturations. Small airways may close during expiration and impede gaseous exchange because the closure volume in new-born's is greater than the FRC. When compared to an adult , neonatal respiratory system has more dead space , alveoli are comparatively lesser in number , but are thicker and their efficiency of gas exchange is low. Neonates are prone to complications like hypothermia due to their body surface area:weight ratio being high and requirement of brown fat for thermogenesis. Also neonates are susceptible to hypoglycaemia, hyperbilirubinemia, poor medication metabolism, and coagulopathy because of their immature liver function. As TBW rises in new-born's, several drugs may become less effective as a result of dilution. Neonatal kidneys struggle to handle large fluid quantities and effectively control

electrolytes because of their restricted renal blood flow (RBF) and glomerular filtration rate (GFR) being low . [9]

This article's objective is to draw attention to the vital physiology associated with the new-born period in order to increase understanding of the difficulties encountered when treating a baby with ventilator in a neonatal unit.

PHYSIOLOGY OF LUNGS IN NEONATES:

The release of surfactant, the beginning of regular breathing, and the elimination of foetal lung fluid must all occur in precisely timed succession for the lungs to acclimatise to the external environment. The airway epithelium secretes foetal lung fluid during intrauterine life, which is necessary for healthy lung development. The prompt and adequate evacuation of lung fluid is the most crucial step in the foetal shift to extra uterine air breathing. Osmotic gradients produced by alveolar epithelial cells', active solute transport are responsible for the majority of the fluid clearance. Both the hormonal changes related to labour and the developmental changes in sodium channel expression control this process. [10]

A new-born’s rapid onset of vigorous breathing is facilitated by a many of the following factors, including umbilical cord clamping (which halts the prostaglandin release that suppresses breathing), diffuse tactile and cold sensations, changes in the blood's CO and O2 levels during delivery, and others. Most term new-born’s will successfully start breathing on their own without any sever hypoxia. Specific endocrine adjustments start prior to delivery and are crucial for the removal of foetal lung fluid during delivery. Foetal lung fluid’s active chloride mediation secretion is inhibited by a rise in cortisol levels, thyroid hormone levels, and catecholamine levels. These hormones lead to Na-K-ATPase activation of the type II cells of the airway epithelium, which pushes sodium in foetal lung into the interstitial compartment

along with water and other electrolytes and clears the airways of fluid. At delivery, neonates that are physiologically healthy expand their lungs by taking deep breaths with high negative pressure, which forces lung fluid out of the airways and into the distal airspace. With each consecutive inflation, the baby continues to drain fluid from the lungs. [11] The "thoracic squeeze," which causes compression of fluid in the lungs and permits rapid fluid clearance, aids in clearance of foetal lung fluid in neonates who were born vaginally. In new-born's, breathing is often laboured, requiring the use of accessory muscles of respiration causing retractions of intercostal spaces and grunt in order to reduce the surface tension (30 to 60 breaths per minute). More significantly, the circulatory shunts make the baby's breathing more difficult. The effort required to breathe is diminished as the fluid exits the lungs' alveoli. It is usual for neonates to experience apnoeic episodes and new-born's may experience spells of apnoea lasting less than 5 seconds due to undeveloped central drive responses. [12]

MANAGEMENT OF NENOATAL RESPIRATORY SYSTEM:

The management of new-borns with signs of respiratory distress is the primary duty of neonatal intensivist. Even in the most preterm infants, acute respiratory distress is not a major cause of mortality nowadays, but mechanical ventilation still causes a significant amount of morbidity. The use of non-invasive type of ventilation is typically favoured, and current new-born ventilation techniques place a strong emphasis on minimising lung injury induced by ventilator. When mechanical ventilator is required, new ventilators give the patient the most control imaginable. The ideal course of treatment is still hard to pin down, and there is a lot of heterogeneity when comparing the risk-benefit ratios of different management techniques.

PATHOLOGIES OF NEONATES REQUIRING MECHANICAL VENTILATION:

We know that the lung development and secretion of the surfactant are essential adaptations during the birth for extra-uterine life. Around 22 - 28 weeks of gestation is the time where surfactant synthesis begins.This surfactant plays a major role in preventing alveolar lung collapse even at low lung volumes. [13] During the time of delivery, the volume of the foetal lung fluid decreases as the secretion of the fluid stops. A new-born's pulmonary pathophysiology can be divided into groups based on the gestational weeks at birth. Premature new-borns, for instance, have immature lungs that have difficulty breathing because of insufficient surfactant levels.

The pathologies include 1. "Neonatal respiratory distress syndrome (NRDS) with cause: Deficiency of pulmonary surfactant with Clinical features: cyanosis, hypoxia, tachypnoea, decreased breath sounds and Complications: bronchopulmonary dysplasia, pneumothorax" [14]

"Apnoea of prematurity (AOP) with causes being immature medullary respiratory centre and/or weak airway or breathing muscle and Clinical features: cessation of breathing for > 20 seconds and typically accompanied by bradycardia" [15]

These are some of the conditions of premature neonates that require mechanical ventilation. Most of the studies showed that the main causes requiring mechanical ventilation as birth asphyxia, meconium aspiration syndrome, congenital heart diseases, congenital pneumonia, sepsis and neonatal seizures.

The main aim of the current research is to predict the risk variables contributing to poor outcomes in ventilated neonates. In ventilated new-born's, a variety of clinical, biochemical, and haematological markers were examined in an effort to link them to the outcome.

HISTORY OF NEONATAL RESPIRATORY SUPPORT:

The Greek historian Plutarch claims that at Sparta, a group of elders assessed new-born children and decided which ones were healthy and had capability of survival and which one who should be let to perish. Many techniques have been advocated throughout history to assist in baby resuscitation upon delivery, including as warming the infant and spooning or blowing wine into their mouths. [16] There was another method , that seems to be similar to what we are practising today ,that was practised according to Leveret's writing from 1766. Using this technique, one places their mouth on the baby's and blows into it while being careful to squeeze the baby's nose tip. [17]

MECHANICAL VENTILATION AND IT'S HISTORY:

"In the 1770s, Hunter built a positive pressure ventilator with a bellows design, quickly followed by similar inventions by Chaussier and Gorcy, that used a thin ivory tube passed into the nostrils or mouth to 'restore respiration in apparent death." [17] "Positive pressure ventilation, however, subsequently fell out of favour due to concerns regarding pneumothoraces. Alexander Graham Bell invented a negative pressure ventilation system for neonates in 1889, in the hope that it would improve infant mortality due to inability to expand the lungs sufficiently when they take the first breath."[18] This was not in frequent practicse. "The use of positive pressure ventilation was consolidated into practice following widespread polio epidemics in the 1940s to 1950s, necessitating improvements in the delivery of prolonged mechanical ventilation."[19]

THE MODERN ERA:

Babies were often ventilated in the late 1950s, had increased number of deaths due to factors like lack of humidified circuits, pulmonary fibrosis and intraventricular haemorrhage. [20] Surfactant therapy and the use of neonatal-specific ventilators significantly increased the survival rates of new-born's with acute respiratory distress.[21] The optimization of new-born ventilation still faces difficulties in minimising chronic respiratory morbidity.

RESPIRATORY SUPPORT IN NEONATES:

Respiratory assistance is a crucial life-saving measure that has been linked to brain damage, particularly in premature new-borns. Preterm birth is giving birth before gestation of 37 weeks, is a significant contributor to perinatal death and morbidity. [22,23] Adverse neurodevelopmental outcomes were seen in around a million preterm new-borns who survive the neonatal period. [22] Prematurity has various difficulties because normal organ development, which would usually continue in utero to term, is halted. "For this reason, the distinction of babies by gestational age (GA) at birth-extremely preterm (< 28 weeks), very preterm (28 –< 32 weeks), and moderate to late preterm (32 –< 37 weeks)-helps to identify infant populations which are most at risk of complications related to preterm birth."[24]

Extremely preterm children frequently have lungs that lack the development necessary to sustain appropriate respiratory function outside of the womb. The infant's lungs are less developed and require more respiratory support the lower the GA is at birth. "An estimated 2.4 million babies who are extremely preterm are born worldwide each year [24] and ~60 – 95 % of these infants will require respiratory support during their neonatal period."[24]

MODES OF VENTILATION:

In 1960s main concern was that the adult ventilators would not suit neonates as they requires high respiratory rate and lesser tidal volume[25] "Although adaptations to adult ventilators were devised, limitations of available technology at the time meant that it was impossible to measure tidal volumes accurately, or to detect the small changes in flow or pressure generated by spontaneous effort in order to trigger breaths, and therefore time-cycled pressure-limited ventilation was the mainstay of neonatal ventilation."[26]

VOLUME-TARGETED VENTILATION:

"There has been increasing interest in volume-targeted ventilation in neonates. VTV aims to deliver a specified tidal volume at each breath. Different ventilators achieve VTV in different ways."[27] Volume control ventilation was utilized by early forms of volume ventilation. "A target inspiratory tidal volume is set during volume control ventilation and a constant inspiratory flow is delivered until this volume is reached, at which point the breath is terminated. There may be high airway pressure. Due to the frequent presence of leak around the endotracheal tube when ventilating neonates, and the limited ability of ventilators to deliver appropriately small tidal volumes at the point in time when volume control ventilation was one of the only options for volume ventilation, it has had limited popularity in neonates."[28]

VTV is now more practised, thanks to the development of more sophisticated processing technology. "Different manufacturers produce slightly different modes, such as volume guarantee (VG), pressure-regulated volume control (PRVC), and targeted tidal volume (TTV), but the basic principle between these modes is conserved, whereby the ventilator will attempt to deliver a clinician-set target tidal volume over a preset inspiratory time by adjusting

the pressure delivered based on feedback from previous breaths. This differs from volume control ventilation, whereby the inspiratory flow is terminated only once the target volume is achieved. During VTV, a pressure limit can be set to avoid delivery of very high inspiratory pressures. Compared to pressure-limited ventilation, VTV has been shown in meta-analyses to reduce episodes of hypocarbia, reduce rates of BPD and other complications in preterm babies, and to reduce the duration of ventilation. Since it is unclear as to what is the optimum target tidal volume to set, studies have been heterogeneous in terms of the ventilators used and also in the level of volume-targeting chosen." [29] "In preterm infants with acute respiratory distress, volume targeting at 4ml/kg significantly increased the work of breathing (as measured by the pressure-time product of the diaphragm) as compared to baseline pressure-limited ventilation (154 versus 112 cmH2O.s/min, $p < 0.001$) and 6 ml/kg (89cm H2O.s/min, $p < 0.001$)."[30] "In prematurely-born infants in the phase of weaning from respiratory support, targeted tidal volumes at the higher end of the physiological range (6ml/kg) reduced the work of breathing compared to lower tidal volumes of 4 and 5 ml/kg (141versus 220, 174 cmH2O.s/min, $p = 0.003$, < 0.001 respectively)."[31] It has not yet been determined what the ideal tidal volume objective should be for infants with developing or pre-existing BPD. "Additionally, there have been no trials of VTV in patients with CDH. CDH occurs in around 1 in 3000 pregnancies. Lung hypoplasia and pulmonary hypertension present significant challenges to the management of these patients."[32]

HIGH FREQUENCY OSCILLATORY VENTILATION:

In contrast to the rates observed in babies who breathe on their own, this mode of ventilation gives small tidal volumes and with high frequencies as required. Both creativity and exhaustion are in motion.

“Meta-analysis of trials in which preterm infants were randomised to either conventional ventilation or HFOV within the first 24 hours after delivery did not demonstrate any significant difference in mortality (mortality at 28 - 30 days RR 1.09, 95 % CI 0.88 to 1.34). There was some evidence of an improvement in rates of BPD with HFOV. The overall meta-analysis showed a potential signal towards less BPD in the HFOV group (RR 0.66, 95 % CI0.41 to 1.07), which became more convincing if those trials which did not use a high-volume strategy were excluded (RR 0.53, 95 % CI 0.36 to 0.76). The composite outcome of death or BPD was also reduced with HFOV (RR 0.89, 95 % CI 0.81 – 0.97).”[33] “The largest study included in this meta-analysis, enrolling 797 infants, was the UKOS study, which randomised infants born between 23- and 28-weeks’ gestation to either HFOV or conventional ventilation within one hour of birth. There was no significant difference in the primary outcome, death or BPD, between the two modes (RR 0.98, 95 % CI 0.89 – 1.08).”[34] “Three hundred and nineteen of the infants enrolled were subsequently followed up at 11 - 14 years of age, where they underwent comprehensive cardiopulmonary assessment. The primary outcome, FEF75 (a measure of small airway function) was significantly better in the HFOV group than in those who had received conventional ventilation (adjusted z-score difference 0.23 (95 % confidence interval 0.02 to 0.45).) Other measures of lung function were significantly better in the HFOV group (FEV1, forced vital capacity (FVC), peak expiratory flow (PEF), diffusing capacity, and impulse-oscillometry results), as were teacher ratings for art and design, information technology, and design technology.”[35] These findings show that early ventilation can affect loutcomes in the long run and that BPD might not be a reliable indicator of future respiratory morbidity.

MODES OF PATIENT-TRIGGERED VENTILATION

INTERMITTENT SYNCHRONISED MANDATORY VENTILATION (SIMV):

SIMV delivers a predetermined volume or pressure at a predetermined rate of inflations per minute. These breaths may be required or initiated by the patient. Additional spontaneous breaths that occur after the predetermined number of inflations are not assisted.

ASSIST CONTROL MODE OF VENTILATION (ACV):

ACV provides an aided breath each moment the infant's breathing level goes above the trigger's essential threshold. If the patient can not breathe out enough, a "Back up" rate of breaths is established and made available. “In neonates with acute respiratory distress, volume-targeted assist control was associated more consistent tidal volumes at lower respiratory rates than SIMV or CMV. In stable preterm infants, ACV resulted in lower peak inspiratory pressures, lower heart rate, lower respiratory rate, and more stable oxygenation than SIMV, indicating that the work of breathing may be reduced with ACV compared to SIMV.”[36] “For infants with respiratory distress who are weaning from support, ACV has been shown to shorten the duration of weaning compared to SIMV.”[37] “This may be due to the reduced oxygen cost of breathing when infants are supported by ACV as compared to SIMV.”[38]

NEWER MODES OF TRIGGERED VENTILATION - PRESSURE SUPPORT VENTILATION (PSV):

This mode is a pressure-targeted, cycled flow form of ventilation that is patient-triggered. The ventilator is engaged to produce predetermined inspiratory pressure while the baby works to breathe. "When the inspiratory flow falls below a certain level, the inflation is terminated, thus the timing of both inspiration and expiration is under the control of the infant. A back up rate of mandatory inflations can be set that will be delivered in the case of apnoea. In a small study of nine full term neonates who had undergone cardiac surgery or cardiac investigation, and were stably ventilated on endotracheal CPAP, the effect of added pressure support on tidal volume, respiratory rate, and thoracoabdominal asynchrony was investigated. Addition of pressure support of 5 cmH_2O resulted in an increase in tidal volume of 23 %, and addition of pressure support of 10 cmH_2O resulted in an increase in tidal volume of 69 % above baseline. Despite a concurrent reduction in respiratory rate, the minute volume was increased with pressure support. There was a reduction in thoracoabdominal asynchrony with increasing levels of pressure support."[39] "PSV has been compared to pressure-limited ACV in a study of fifteen heterogeneous infants born at greater than 25 weeks' gestation, with weight at study of between 0.8 and 7.8kg. The level of pressure support was set to maintain the same end tidal carbon dioxide level as on baseline settings. The study showed a sixteen percent increase in cardiac output on pressure support ventilation. The difference in cardiac output was attributable to an increase in stroke volume on PSV, as the heart rate remained the same which the authors postulated may have been due to a decreased mean airway pressure, secondary at least in part to a reduction in the I:E ratio." [40]

VOLUME GAURANTEED PRESSURE SUPPORT (PSV-VG):

"PSV-VG is similar to PSV in that the infant determines both the onset and the termination of mechanical inflations, but the ventilator adapts the peak inspiratory pressure (PIP) to deliver a set tidal volume with the lowest possible pressure between the peak set pressure and the level of PEEP. Comparison of PSV-VG to SIMV showed that whilst the peak and mean airway pressures declined in both groups over the first 24 hours after surfactant administration, this decline in pressure requirements was faster in the PSV-VG group. As, however, the SIMV was not delivered in conjunction with VG, it is perhaps difficult to compare this directly, as the PSV-VG group will have 'auto weaned' the pressure, whilst the SIMV group will have been reliant on clinicians altering the settings in response to blood gases. There were no significant differences between the groups in terms of long-term outcomes such as BPD, death, or rates of retinopathy of prematurity (ROP) and necrotising enterocolitis (NEC)."[41] "PSV-VG compared to SIMV reduced the mean airway pressure (MAP) required to maintain oxygenation, and gave comparable ventilation in twenty-five preterm infants in a crossover study during weaning."[42] Therefore,currently there is low evidence to justify the regular use of PSV or PSV-VG.

RISK FACTORS AND ASSOCIATED COMPLICATIONS OF NEWBORN THAT IMAPACT THE MORTALITY AMONG MECHALINACLLY VENTILATED NEONATES:

Complications that can lead to a poor outcome include circulatory problems, hypothermia, pneumothorax, sepsis, DIC (disseminated intravascular coagulopathy), tube block, extremely low birth weight, early gestation, electrolyte problems, convulsions, and pulmonary haemorrhage.

In the case of shock, which is prevalent in very LBW infants, particularly in the early days, there is insufficient oxygen supply to the tissues. The shock is a "independent predictor of mortality" and survivors have an increased chance of developing neurologic impairment. Knowing the pathophysiology can help you identify shock early on in the compensated phase, classify it, and start the right kind of treatment. Hypovolemia is infrequently the main cause of death in new-born's. Myocardial dysfunction is generally prevalent in extremely preterm new-born's and term infants who have experienced prenatal hypoxia. In extremely low birth weight infants (60 – 100 % at 24 – 26 weeks) and very low birth weight infants (40 % at 27 – 29 weeks), low blood pressure is very common. Among these babies, hypotension is usually not due to hypovolemia and usually due to immature catecholamine responses, insufficiency of adrenocortical hormones, poor vascular tone, and transient left ventricular dysfunction. In ELBW infants, hypotension with evidence of improper functioning of the "end-organ" is associated with "intraventricular haemorrhage/periventricular leukomalacia (IVH/PVL)". An ELBW infant's blood pressure often improves on its own over the first 24 hours. When diagnosing low blood pressure in VLBW newborns in their first three days of life, a MAP that is less than the infant's gestational age in weeks or a MAP that is less than 30 mm Hg may be

helpful. For example, a mean BP of less than 27 for a neonate who is 27 weeks gestation. The mean blood pressure for babies weighing under 1000 g is shown in Appendix Figure C-1.

A "pneumothorax" is a buildup of gas or air between the pleura that can happen on its own or as a result of trauma. It could exist in a newborn, and compared to any other stage of life, the neonatal era is when it happens most frequently.

Neonatal sepsis is a "clinical syndrome" of "systemic disease" accompanied by bacteraemia usually occurring in a month of the new-born's life. The prevalence of primary sepsis is about 1–5 per 1000 live newborns and is more common in VLBW infants (birth weight 1500 grams), according to data from the "National Institute of Child Health and Human Development Neonatal Research Network (NICHD-NRN)," with an early-onset sepsis rate being 2% and with a late-onset sepsis (LOS) rate of 36%. Mortality is significant (13-25%) in premature newborns and people with early fulminant illness. Most of these newborns will improve with out any long term issues even though the mortality seems to be high. For VLBW newborns with early-onset illness, the number of neonates dying is higher ("16% based on latest study from NICHD NRN").

The newborn must be kept in a "neutral thermal environment," which is the range of ambient temperature where oxygen consumption and metabolic rate are at their lowest levels while the baby's temperature is within a normal range. The typical skin temperature of a newborn is 36.0–36.5°C (96.8–97.7°F), and the usual core temperature is 36.5–37.5°C (97.9–99.5°F). Normal body temperature is a sign of balance between heat generation and heat loss and is not the same as an ideal or minimal metabolic rate or oxygen consumption. Severe hypothermia may be accompanied by pulmonary haemorrhage and diffuse intravascular coagulation.

"Grossly bloody secretions are seen in the endotracheal tube (ETT). The incidence of pulmonary haemorrhage varies from 0.8 to 12 per 1000 live births. It can be as high as 50 per

1000 live births if high risk. The mortality rate can be as high as 50 %. Survivors of pulmonary haemorrhage require longer ventilator support, and many will develop bronchopulmonary dysplasia/chronic lung disease. Others survivors may have an increase in cerebral palsy, cognitive delay, seizures, and periventricular leukomalacia. Most cases of pulmonary haemorrhage are secondary to haemorrhagic pulmonary oedema and not a true bleed. Oscillatory ventilation [HFOV], and high-frequency flow interrupter were used in the review) after conventional ventilation failed. HFOV has been used as rescue therapy in some infants with massive pulmonary haemorrhage and showed dramatic improvements."[43]

COMPLICATIONS OF RESPIRATORY SUPPORT IN PREMATURELY BORNINFANTSNEUROLOGICAL SEQUELAE:

Intraventricular Haemorrhage(IVH):

IVH was first identified in 1826. The tiny veins of the germinal matrix, which are the source of the haemorrhage, are ruptured, causing bleeding that may be contained to this germinal layer or expand into the ventricle to varying degrees. While minor IVH may have little to no consequences, moderate or severe IVH may result in hydrocephalus due to haemorrhage or, even without it, frequently has a negative impact on neurodevelopment.[44] "Around six percent of babies born between 500 and 1500g in a large neonatal unit developed severe IVH 56 (haemorrhage that distends the ventricle, or periventricular haemorrhagic infarction),"[45] "despite improvements in neonatal care although rates are variable between different centres (5 - 14.5 % depending on centre)."[46] "It has long been known that both respiratory distress and mechanical ventilation of preterm neonates are factors for development of IVH, due to fluctuating cerebral blood flow."[47] "This maybe particularly an issue when babies 'fight' against the ventilator. Neuromuscular blockade, therefore eliminating 'fighting' the ventilator,

was shown to reduce the incidence and severity of IVH,[48] thus suggesting that modes of ventilation that improve patient-ventilator interaction may be beneficial in reducing the risk of IVH." "In addition, both hypercarbia and hypocarbia have been shown to increase the risk of IVH in preterm infants."[49]

Periventricular leukomalacia:

Periventricular leukomalacia (PVL) is a type of white matter damage in the brain's periventricular regions that is brought on by both localised ischemic processes and inflammation that can have either postnatal or prenatal origins. Following the occurrence of tissue necrosis, decreased myelination, and astrogliosis with glial scarring, cystic change and permanent white matter abnormalities ensue[50] "Affected infants frequently experience moderate to severe impairment including developmental delay, cerebral palsy, seizures, and visual impairment, with consequent impact upon quality of life."[51] "The incidence is variable, depending partly on patient population and diagnostic methods, but in babies born at less than 32 weeks' gestation or weighing less than 1500g may be between 3 and 15 percent."[52] PVL has been seen on MRI in up to 18% of CDH newborn survivors, and it has also been shown to have a positive link with motor delay in such neonates.[54] "Hypocarbia has been repeatedly shown to be a risk factor for PVL."[55] "Volume-targeted ventilation, as compared to pressure-limited ventilation, has been shown to reduce the incidence both of hypocarbia[56] and PVL."[57]

Neurodevelopmental outcomes:

Both IVH and PVL contribute to poor outcomes of neurodevelopment , also prolonged duration of ventilator requirement also causes poorer outcomes[58] "An increased risk of cerebral palsy and attention deficit hyperactivity disorder (ADHD) in extremely low birth weight (ELBW) infants occurred in those ventilated for more than two weeks, and persisted after adjustment for birth weight, postnatal steroid use, and other potentially confounding factors."[59] "Even a few days of mechanical ventilation, compared to CPAP, has been shown to cause diffuse cerebral injury in a preterm baboon model. To improve neurodevelopmental outcomes in babies born prematurely, minimizing ventilatory time through limitation of lung injury and diaphragm dysfunction is therefore an important contributory factor." [60] It's crucial to restrict the duration of the neonate on ventilator.

DIAPHRAGMATIC DYSFUNCTION SECONDARY TO VENTILATOR:

The effects of ventilator on diaphragm are atrophy and decreased contractile function of the diaphragm which is causing increased risk of extenuation failure [61]

"Length of mechanical ventilation in adults is associated with a logarithmic decline in diaphragmatic force."[62] "Diaphragm injury in those mechanically ventilated,[63] and remodelling of diaphragmatic fibres[64] are likely to contribute to ventilator-induced diaphragmatic dysfunction."

"Infants who remain ventilated for long periods of time may be given corticosteroids to facilitate weaning from ventilation and extubation, but animal studies have shown that newborn rats exposed to postnatal steroids have reduced diaphragm contractility and increased susceptibility to diaphragm fatigue. Intermittent spontaneous breathing, even for short periods,

and the use of modes of ventilation that allow for patient contribution to respiratory effort have been shown in animal models to reduce diaphragm atrophy and preserve diaphragm muscle strength. Preservation of diaphragmatic function may be improved by novel modes such as proportional assist ventilation (PAV) and neurally-adjusted ventilatory assist (NAVA), which allow partial redistribution of respiratory work from the patient to the ventilator. Careful evaluation of their use should be done in the neonatal population and the assessment of NAVA as compared to PAV." [65]

LUNG INJURY-VENTILATOR INDUCED:

Mechanical ventilation results in acute lung damage through a number of processes.

BAROTRAUMA:

During the period of 1700s it was noted that there was lung damage by using high ventilator pressures [66] "Application of high ventilatory pressures may cause air leak, as discussed above. A number of studies appeared to demonstrate that high airway pressures were also responsible for microvascular and epithelial injury. Ventilation of isolated rabbit lungs with higher pressures compared to lower pressures for only fifteen minutes significantly increased the capillary filtration coefficient, a marker of microvascular injury. Similarly, in rats, ventilation at 45 cmH_2O as compared to 7 cmH_2O resulted in markers of microvascular injury, including peribronchial oedema, and subsequently alveolar flooding with proteinaceous material, and epithelial lesions."[67] "Such damage was also shown to impair lung function in adult sheep. Those sheep ventilated with pressures of 50 cmH_2O, compared to 15-20 cmH_2O developed respiratory failure with reduced tidal volume, impaired compliance, and reduced FRC, with parenchymal consolidation on post mortem examination."[68] These researches indicated that lung injury during positive pressure breathing was due to barotrauma.

VOLUTRAUMA:

"Over expansion of lung parenchyma, rather than the absolute pressure, leads to lung injury whilst pressure and volume are inextricably linked. In animal models, the importance of so-called volutrauma has been demonstrated. In isolated lung preparations, young (4 – 6-week-old) rabbits were exposed to peak inspiratory pressures of 15, 30, and 45cmH_2O, and expansion was limited in unaltered rabbits, and rabbits in which the chest and abdomen were encased in plaster of Paris. The lungs appeared macroscopically normal, and measurements of capillary permeability, increased in lung injury, remained static at each of the pressure intervals of the restricted expansion rabbits. There was progressive macroscopic damage in the lungs from uncasted rabbits as the pressure to which they were exposed increased, with increasing capillary permeability. The worst affected were the isolated lung preparations, with no restriction to expansion. In provoking this aspect of lung injury, volutrauma was more important than the absolute pressure applied as demonstrated by this study. In premature neonates, whose compliant chest walls offer little protection against over-distension, this is particularly significant. There is disruption of the pulmonary capillary membrane and leakage of proteinaceous fluid and blood into the interstitium, airways, and alveoli as a result of over-extension. Inflammation is stimulated by this and the effect of surfactant is reduced, thus increasing damage.

Protecting infants from the effects of volutrauma from the first breath, including during positive pressure ventilation during resuscitation or stabilisation at delivery is very important. Even a small number of inflations with inappropriately large tidal volumes after delivery may be detrimental as demonstrated in preterm lambs. Inappropriately large tidal volumes at resuscitation can initiate lung damage and reduce the therapeutic effect of the following surfactant therapy as demonstrated by this experiment. Using preterm lambs, similar results

were demonstrated in a further experiment, this time randomised either to immediate surfactant therapy or surfactant at 30 minutes of life, and ventilation with either a low (5 – 6 ml/kg) medium (10 – 12 ml/kg), or high (20 ml/kg) tidal volume strategy. The proportion of protein was higher, compared to the other two groups and there was a smaller improvement in compliance than those from the other groups on transferring this surfactant to surfactant-deficient preterm rabbits. Ventilation at 20ml/kg before surfactant delivery causes initial lung injury that then interferes with the action of surfactant, reducing its effect as suggested by these results. What the optimum tidal volume to reduce work of breathing and optimise outcomes is in infants with evolving or established BPD, or in CDH is yet to be known." [68]

ATELECTRAUMA:

"Low lung volume, particularly at the end of expiration, is also associated with lung injury, probably due to increased shear stresses at terminal bronchioles due to recurrent collapsing and reopening of alveoli. The use of a high end expiratory lung volume ventilation strategy (HFOV with high volume) in rabbits resulted in less epithelial injury, less atelectasis, and improved compliance, compared to both HFOV at lower volume and conventional ventilation. Similar experiments using surfactant-depleted rabbits and administration of exogenous surfactant resulted in retrieval of higher levels of large aggregate phospholipid (a marker for functional surfactant) in the high volume group at the end of four hours, thus demonstrating that atelectrauma reduces the effectiveness of exogenous surfactant, resulting in less favourable pulmonary mechanics. Inflammatory markers in bronchoalveolar lavage fluid are increased by ventilation with zero PEEP in rat models, and a similar rise in inflammatory markers has been observed in human neonates ventilated with low tidal volumes of 3 ml/kg as compared to 5ml/kg."[69]

BIOTRAUMA:

"Mechanical ventilation provokes the release of cytokines, both pro-inflammatory (IL-1, IL-6, IL-8, TNF-α) and anti-inflammatory (IL-10), and other mediators from cells. In vitro studies suggest that this release is caused by mechanical stretch to the tissue, leading both to disruption of the contact between individual cells and also to transduction of the forces intracellularly via the cytoskeleton. The pro-inflammatory response, increased in preterm neonates due to reduced anti-inflammatory cytokine release, attracts neutrophils and other inflammatory cells to the airways, and also increases vascular permeability. Surfactant dysfunction, either due to a direct cytokine effect or due to increased alveolar capillary leak of proteins, increases the propensity for lung damage. It has been shown that preterm neonates who go on to develop BPD have higher levels of inflammatory cytokines and that these persist longer than those infants who do not develop BPD. This inflammatory cascade can induce apoptosis in pulmonary epithelial cells and is also implicated in the alteration of gene expression in injured lung tissue, both sustaining inflammatory responses and also modifying pathways involved in tissue repair and remodelling. Animal studies have demonstrated that lung injury induced by mechanical ventilation and associated with increased proinflammatory cytokine release leads to delayed alveolarization and saccular wall fibrosis, similar to the histological changes found in neonates with BPD."[69] The long-term respiratory outcomes in premature newborns may be improved by adjusting reducing the duration and oxygen rewuirement.

ASYNCHRONY:

Infants may exhibit spontaneous breathing while undergoing mechanical ventilation. Inadequate respiratory support may be given, such as unsupported respiratory efforts or difficulties brought on by unfavourable interactions between the patient and ventilator. Premature newborns have more variable cerebral blood flow, which could raise the risk of intraventricular haemorrhage in the event of asynchrony.

OXYGEN TOXICITY:

The increased duration of oxygen administration and the high partial pressures lead to BPD. "Premature neonatal lung is more vulnerable to reactive oxygen species, by the reduced antioxidant enzyme activity and some of the deleterious effects that oxygen has on the premature lung, leading to increased inflammation, increased apoptosis, dysplastic lung cell growth, abnormal lung tissue remodelling, and altered postnatal lung growth as demonstrated by multiple animal studies. Many markers of oxidative stress in tracheal aspirates and urine, such as uric acid, allantoin, and oxidized ascorbic acid have been exhibited in the first few days of life by the prematurely-born infants who go on to develop BPD. The impact of antioxidant therapy on the subsequent development of BPD and of chronic respiratory morbidity has been evaluated by several studies. In the control group of thirteen infants, six developed radiological appearances consistent with classical BPD, in an early study of vitamin E supplementation in prematurely-born infants whereas none of the group given vitamin E supplementation developed such changes ($p = 0.046$). In the rates of BPD, there were no significant differences between prematurely-born infants randomised to receive vitamin E and those who made up the control group by the subsequent studies. The incidence of BPD was not reduced by the administration of recombinant human CuZn Superoxide Dismutase, an antioxidant enzyme, to mechanically ventilated premature neonates, but respiratory morbidity (episodes of wheezing,

emergency department attendances, and hospital admissions) was improved at one year of age. Chronic respiratory morbidity is contributed by oxygen toxicity, and in those born prematurely, optimising ventilation to reduce the amount of oxygen required and its duration may help to improve long term respiratory outcomes." [69]

Clinical and biochemical criteria to ventilate a neonate:

a) Clinical criteria:

1. Central cyanosis with SPO2 < 85 %
2. Nasal flaring, grunting, severe chest in drawing
3. Respiratory distress like tachypnoea (> 60 breathing/minute),
4. CPAP or O2 through a hood with FiO2 > 0.6

b) Laboratory criteria:

1. pO2 < 40-50 mmHg on O2 indicating hypoxemia even with
    a. CPAP at FiO2 > 0.6 or through hood
    b. pCO2 (mmHg) < 50 50-60 61-70 > 70

2. Severe hypercapnia :

- pCO2 > 60 mmHg and with pH < 7.2,

B. Criteria for extubating a neonate:

a) Subjective criteria:

1. Clinically
2. Improving respiratory mechanics
3. Underlying disease process is improving as judged
4. Absence of other underlying respiratory pathology
5. Adequate gas exchange
6. There is spontaneous breathing

b) Objective criteria:

1. Stable vitals on ventilator
2. Neonate is not on any sedation
3. Breathing without distress
4. Secretions less than 1 ml sixth hourly
5. Haemoglobin > 13 g/dl
6. Alertness
7. Normal electrolytes
8. Gases:
9. pO2 > 60 mmHg and SPO2 > 90% with FiO2 < 0.4 & PEEP < 5
10. pCO2 < 50 mmHg
11. pO2 / FiO2 > 150
12. pH more than 7.2

C. Criteria for successful extubation

a) Subjective criteria:

1. No signs of respiratory distress
2. No onset / worsening of dyspnoea
3. No change in mental status
4. No diaphoresis

b) Objective criteria:

1. SPO2 > 90 %
2. pO2 > 50 mmHg
3. pCO2 rise < 10 mmHg
4. pH > 7.32
5. Respiratory rate rise < 50 %

Clinical Studies:

To learn more about the clinical circumstances and immediate results of newborns needing mechanical ventilation, Sultana et al. undertook a prospective observational study in 2019. The study was done at Dhaka, in a medical university called Sheikh Mujid, from August 2015 to July 2016. The newborns who needed to be put on mechanical ventilation were enrolled one after the other. "All babies were monitored for clinical profile and outcome as well as complications. The enrolled neonates were divided into two groups. Neonates who remained successfully extubated for > 48 hours and did not require re-intubation were grouped as survivors and who died during mechanical ventilation or within 48 hours of extubation were grouped as non-survivors. Clinical, biochemical, ventilator parameters and occurrence of complications were analysed to find out the factors associated with mortality of ventilated neonates. Results of the study showed that during the study period 53 (8.6 %) of admitted neonates in NICU received mechanical ventilation. Out of these, 53 neonates 69.8 % were male with male to female ratio 2.3 : 1. Inborn babies were more (58.5 %) than out born (41.5 %). Mean age, gestational age and birth weight were 3.58 ± 5.45days 33.34 ± 3.40 weeks and 1852.55 ± 513.48 g respectively. Commonest condition for initiating mechanical ventilation was refractory apnoea (35.8 %) followed by severe respiratory distress with Downe score > 6 (20.8 %) and SpO2 < accepted level (17.0 %). Disease pattern were sepsis (35.8 %), RDS (20.8 %), congenital pneumonia (18.9 %), perinatal asphyxia (15.1 %), meconium aspiration syndrome (3.8 %), TTN (1.9 %) and meningitis (3.8 %). The survival rate was 35.8 %. Factors significantly different in non-survivors were mean gestational age, mean birth weight, initial arterial pH, age at admission and age at initiation of ventilation ($p < 0.05$). The mean maximum PIP requirement was significantly higher in non-survivors ($p < 0.05$). Hospital acquired sepsis (67.9 %) was the most common complication during mechanical ventilation followed by tube

block (52.8 %) and ventilator associated pneumonia (26.4 %). Shock (64.2 %) was the commonest co-morbidity followed by dyselectrolytemia (52.8 %), sepsis (35.8 %) and DIC (28.3 %). Hospital acquired sepsis, shock and DIC were associated with mortality ($p < 0.05$). Shock was found independent predictor of mortality ($p = 0.01$). The authors concluded that the most common condition for initiating mechanical ventilation was refractory apnoea. Sepsis was the commonest disease for which ventilation required. The survival rate of ventilated neonates was 35.8 % and percentage of survival was more in babies with RDS. Hospital acquired sepsis was the major complication of ventilated neonates. Presence of hospital acquired sepsis, shock and DIC was significantly high in non-survivors. Shock was found as independent predictor of mortality."[70]

Trivedi et al. investigated the risk variables for mortality in neonates on mechanical ventilation in 2009 using multiple regression analysis. A 6-month study was carried out by Trivedi et al. Study design was prospective, at the Neonatal ICU of the New Civil Hospital in Surat, from December 2007 to May 2008. "Fifty neonates in NICU consecutively put on mechanical ventilator during study period were enrolled in the study. The pressure limited time cycled ventilator was used. All admitted neonates were subjected to an arterial blood gas analysis along with a set of investigations to look for pulmonary maturity, infections, renal function, hyperbilirubinaemia, intraventricular hemorrhage and congenital anomalies. Different investigation facilities were used as and when required during ventilation of neonates. Multiple logistic regression analysis was done to find out the predictors of fatality among these neonates. The study results indicated that various factors suspected as predictors of fatality of mechanically ventilated neonates were assessed. Hypothermia, prolonged capillary refill time (CRT), initial requirement of oxygen fraction (FiO_2) > 0.6, alveolar to arterial PO_2 difference ($AaDO_2$) > 250, alveolar to arterial PO_2 ratio (a/A) 10 were found statistically highly significant predictors of mortality among mechanically ventilated neonates. The study

concludes that hypothermia and prolonged capillary refill time were independent predictors of fatality in neonatal mechanical ventilation. Risk of fatality can be identified in mechanically ventilated neonates."[71]

Maiya et al. in 1995 performed "a retrospective study in NICU of a teaching hospital to analyse the indications, clinical profile, complications and outcome of the babies requiring mechanical ventilation. One hundred and twenty-one neonates requiring assisted ventilation during three years were included in the study. The results observed that of 121 babies, 59(48.76 %) survived. Hyaline membrane disease (HMD) was the commonest indication for ventilation followed by birth asphyxia, apnoea of prematurity, meconium aspiration syndrome (MAS) and septicaemia. Infants with HMD whose birth weight was more than 1.5 kg and those who required ventilation after 24 hours of birth had better outcome. Survival rates increased with increasing birth weight and gestational age. Prolonged ventilatory support was needed for HMD (mean 117.3 hr) and MAS (mean 82.6 hr). Pneumonia was the commonest complication, followed by sepsis, air leak syndromes and intracranial and pulmonary hemorrhage. Based on the study results it was concluded that ventilatory facilities must be focussed for neonates weighing > 1000 g. assisted ventilation may not be cost-effective in patients weighing."[72]

Thakkar et al. studied the clinical profile, the results of mechanical ventilation, and the risk factors for death and its complications brought on by ventilation at the NICU of a tertiary care hospital between May 2015 and April 2016 as part of a prospective study scheduled to be published in 2021. "Neonates who underwent mechanical ventilation and met the inclusion criteria were enrolled in the study. Their demographic profile, outcomes and risk factors were documented and analysed using appropriate statistical methods. The results found that 285 neonates required mechanical ventilation during the study period. Among them, 190 were included in the study. Overall mortality was 99 out of the 190 enrolled (52 %). The most common indications for mechanical ventilation were respiratory distress syndrome (RDS),

meconium aspiration syndrome (MAS) and apnoea. Risk factors contributing significantly to higher mortality of ventilated neonates were very low birth weight (VLBW), gestation of less than 32 weeks, shock, ventilator associated complications like pneumothorax and pulmonary haemorrhage. In multiple regression analysis, very low birth weight, circulatory disturbances, pneumothorax, pulmonary haemorrhage, and higher initial FiO2 requirement were found to be independent risk factors of mortality. Thakkar et al. concluded that the commonest indications for mechanical ventilation were RDS and MAS. Significantly higher mortality was seen amongst VLBW, preterm neonates. Co-morbidities like circulatory disturbance, and complications like pneumothorax and pulmonary haemorrhage contributed to adverse outcomes. Keywords: mechanical ventilation; neonates; outcome."[73]

A case study was reported by Yadav et al. in 2018 in a tertiary centre to review the prevalent signs of ventilation and also evaluated the final outcome as determined . Yadav et al. "found that the clinical and etiological pattern of ventilated newborns, their outcome in relation to morbidity and mortality was studied with 50 ventilated newborns, including outborns. M : f ratio was 2.1 : 1. The most common gestational age 28 – 36 weeks (60 %) and mostly were appropriate for gestational age (66 %). Survival rate 40 % (20/50) being directly proportional to the gestational age and intrauterine growth pattern ($P < 0.01$). Babies by LSCS survived more than born by normal vaginal delivery (46.7 % vs. 37.1 %). More outborn survival could be related to their advanced gestational age on presentation. The initial assessment of APGAR score of > 7 had a better outcome (56.3 %; $P < 0.03$). The most common indication of ventilation was hyaline membrane disease (19/50) but the survival rate best in babies with meconium aspiration syndrome (54.5 %). The most prevalent complication was sepsis (survival rate 60 %) while conditions such as shock, intraventricular hemorrhage, disseminated intravascular coagulation, air leak syndrome, and pulmonary haemorrhage had 100 %

mortality. Thus, the concludes that the outcome as survival is constrained by many factors; new-born's profile, conditions at birth, and postnatal resuscitation."[74]

In 2017, Sharma et al. conducted a prospective, cross-sectional study from the period first of July 2012 to 30 June 2013 to determine the outcome of neonatal ventilation. Included were all new-born's brought to the NICU who required mechanical ventilation. "The results indicate that out of 72 neonates studied, majority of preterm were ventilated for RDS - 34 (89.5 %) and majority of full term were ventilated for MAS - 16 (100 %) followed by HIE - 8 (88.89 %). Out of 38 RDS cases, 30 (79 %) were ventilated till 4 - 7 days duration and 3 (7.9 %) required ventilation for > 10 days. Out of 16 MAS cases, 10 (62.5 %) were ventilated for 4 - 7 days duration and none required prolonged ventilation. Duration of ventilation is not statistically associated with indication of mechanical ventilation with $p = 0.301$. The study inferred that mechanical and pulmonary complications of mechanical ventilation are not statistically significant for outcome of mechanical ventilation but it increases length of NICU stay. Hypotension on ventilator, requirement of more than 3 inotropes were associated with high mortality."[75]

The studies related to mortality in ventilated neonates conducted in and around Kolar are scarce, hence, this study was undertaken to know the risk factors and parameters that are significantly associated with mortality in ventilated neonates admitted in RLJH hospital.

METHODLOGY

Source of Data:

Our study is a prospective observational study done in the Department of Paediatrics, RL Jalappa Hospital and Research Center, Tamaka, Karnataka. Ethical clearance was approved from Institutional Ethical Committee of RL Jalappa Hospital and Research Center. The study was conducted for a period of 1 year from Feb 2021 to Jan 2022. The study included neonates under mechanical ventilator support admitted in the neonatal intensive care unit (NICU) at RL Jalappa Hospital (RLJH), who fulfilled the inclusion criteria.

Method of collection of data (including sampling procedure if any):

All parents of newborns meeting the inclusion criteria had to sign a pre-written consent form after the study's aim, protocol, and anticipated results had been thoroughly explained to them.

Selection Criteria:

Ninety-three patients/neonates under mechanical ventilator support admitted to the Department of Paediatrics admitted to RLJH were included in the study.

Methodology:

The study was carried out at the RLJH, which is a part of the “Sri Devraj Urs Medical College and the Sri Devraj Urs Higher Education and Research Academy”.

Inclusion Criteria:

1. Neonates who were on ventilator in the NICU at RJH.

Exclusion Criteria

1. Neonates with major congenital anomalies
2. Preterm babies less than 28 weeks
3. Babies with birth weight les than 750 gms

Sample Size Calculation

Based on the published literature by Sharma R et al. sample size was estimated by using the proportion of adverse outcome in subjects who were ventilated was 13.88 %. The sample size was calculated using the formula as follows:

Sample Size = $\frac{Z_{1-\alpha/2}^{2}\, P\,(1-P)}{d^2}$

$Z_{1-\alpha/2}$ = is standard normal variate (at 5% type 1 error ($P<0.05$) it is 1.96 and at 1% type1 error ($P<0.01$) it is 2.58). In majority of studies P values are considered significant below 0.05 hence 1.96 is used in formula.

Where, P = 13.8 or 0.138

q = 86.12 or 0.8612

d = 7.5 % or 0.075

Based on the calculation, assuming 95 % confidence level, the required sample was 84. Considering 10 % non-response, a sample size of 84 + 8.4 ≈ 93 patients was included in the study. Maternal data and neonatal date were collected and studied, after taking informed consent during the study period.

- Parameters / Data studied:

1. Maternal risk factors
2. Gestational Age
3. Birth weight
4. APGAR at birth
5. Out born / Inborn
6. Temperature
7. Oxygen index
8. Ventilator parameters and duration of ventilation
9. Indications/causes for requirement of mechanical ventilation.

   -MAS

   -Congenital pneumonia

   - Respiratory distress syndrome

   - Perinatal asphyxia

   - Hyaline membrane disease

   - Sepsis

   - Hypothermia

   - Others

All newborns who were admitted had "arterial blood gas analysis" as well as a series of tests to check for lung maturity, infections, renal function, and other associated complications like AKI , DIC , sepsis , pulmonary haemorrhage . We employed "pressure limited time cycled ventilators." In the NICU, use (Drager Babylog 8000 Plus) , SLE 500 and Hamilton. The associations between each of the characteristics and mortality were examined using a sample size of 95 newborns.

Statistical Analysis:

Data was gathered and entered into the MS Excel spreadsheet that was already developed. For the statistical analysis, SPSS Ver. 22 was employed. The categorical data was represented using frequencies and proportions. Using the “chi square test” , the significance was evaluated. Continuous data were represented using the mean and standard deviation. The significance test employed was the independent t test. Independent t test was employed as a test of significance to determine the mean difference. Statistics were considered to be statistically significant if the P value was < 0.05.

RESULTS

Gender	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Male	29	43.3	17	60.7	46	48.4	$\chi^2 = 2.402$ df = 1P = 0.120
Female	38	56.7	11	39.3	49	51.6	
Total	67	100.0	28	100.0	95	100.0	

Table 1:Gender Wise Distribution Among Survivors and Non Survivors

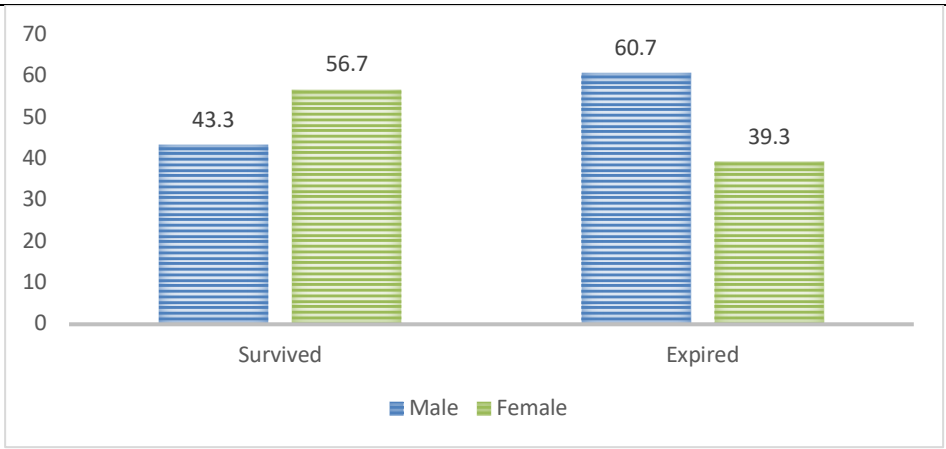

Figure 1:Gender Wise Distribution of Neonates Among the Survivors and Non Survivors

In the present study, about 38 (56.7 %) out of the 67 (100 %) survivors, 38 (56.7 %) were females and 29 (43.3 %) were males. Out of 28 non survivors, 17 (60.7 %) were males and 11 (39.3) were females. The outcome with gender (P > 0.05) did not have any statistically significant association (Table 1 and Figure 1)

Birth Weight (kg)	Survivors		Non Survivors		Total	
	Frequency	%	Frequency	%	Frequency	%
< 1	2	2.9	4	14.0	6	**6.3**
1 - 1.5	11	16.4	8	28.0	19	**20**
1.5 – 2.5	41	61.2	11	39.0	52	54.7
A)1.5 - 2.0	20	29	7	25.0	27	**28**
B)2.0 - 2.5	21	31	4	21.0	25	**26.7**
2.5 +	13	19.4	5	17.0	20	**26.7**
Total	67	100.0	28	100.0	95	**100.0**
Mean ± SD	2.6 ± 0.2		1.8 ± 0.6		**2.4 ± 0.8**	
Significance	**"t" = 3.367, df = 93 P = 0.010.**				**Range = 0.88-3.20 kg**	

Table 2:Comparison of Birth Weight between the Survivors and Non Survivors

2.6 ± 0.2 kg was the mean birth weight in the group of survivors and 1.8 ± 0.6 kg was the mean birth weight in the group of non-survivors. The difference in birth weight among non survivors and survivor babies was found to be significant statistically. (Table 2 and Figure 2)

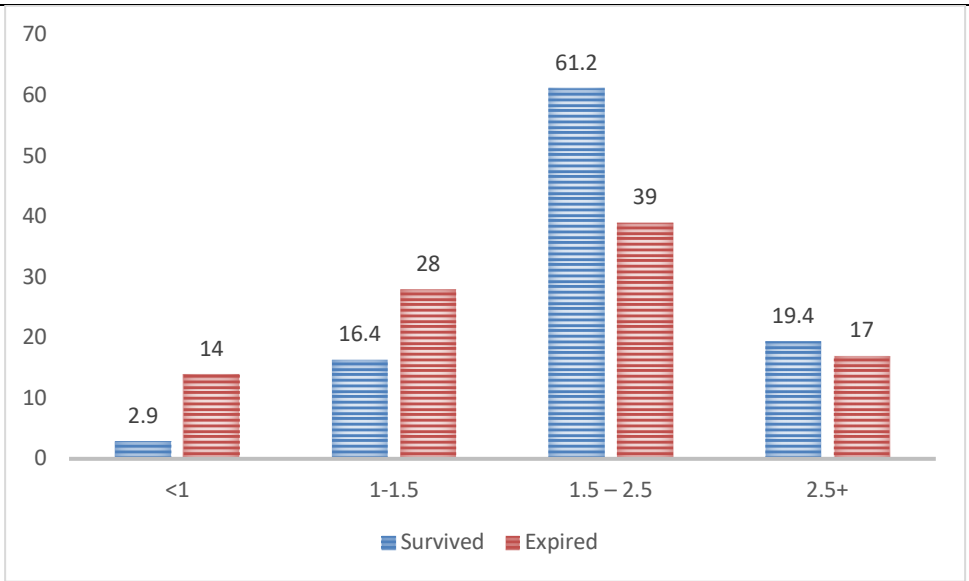

Figure 2:Comparison of Birth Weight among the Survivors and Non Survivors

Gestational Age (Weeks)	Survivors		Non Survivors		Total	
	n	%	n	%	N	%
≤ 28	4	6.0	8	28.6	12	12.6
29 - 32	13	19.4	14	50.0	17	17.9
33 - 36	32	47.8	2	7.1	34	35.8
≥ 37	18	26.9	4	14.3	32	33.7
Total	67	100.0	28	100.0	95	100.0
Mean ± SD	36.7 ± 2.9		33.8 ± 3.2		33.6 ± 2.8	
Significance	"t"=4.319, df = 93,P = 0.010				Range = 24 - 41	

Table 3:Comparison of Gestational Age among the Survivors and Non Survivors

Among survivors, 26.9 % and 47.8 % were less than or equal to 37 weeks and 33 - 36 weeks respectively, and only 6.0 % were ≤ 28 weeks. On the other hand, among the non survivors, 50.0 % were ≥ 29 to 32 weeks and 28.6 % were ≤ 28weeks respectively. The mean of gestational age of survivors group was 36.7 ± 2.9 weeks and 33.8 ± 3.2 weeks was the mean

gestational age of the non survivors' group. The difference in the mean gestational age between the two groups was found to be significant (P < 0.05). (Table 3 and Figure 3)

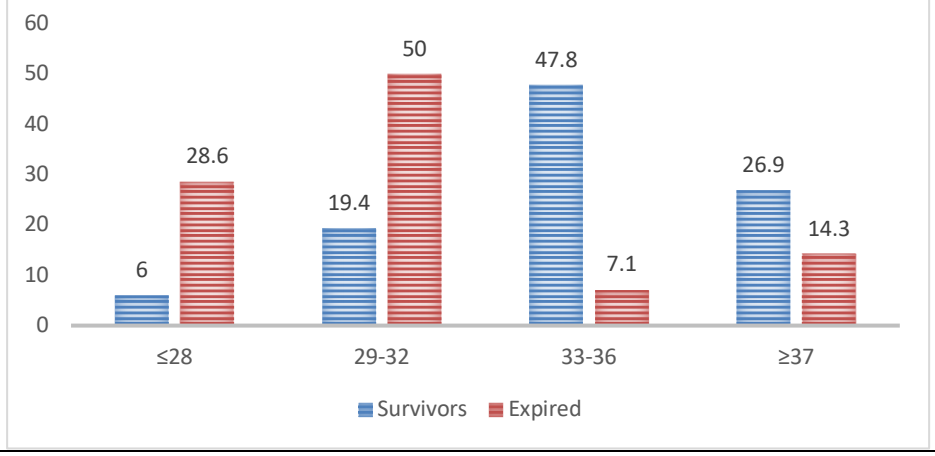

Figure 3:Comparison of Gestational Age between the Survivors and Non Survivors

Mode of Birth	Survivors		Non Survivors		Total		Results
	No	%	No	%	No	%	
LSCS	33	49.3	9	32.1	42	44.2	χ^2=2.344
NVD	34	50.7	19	67.9	53	55.8	df = 1
Total	67	100.0	28	100.0	95	67	P = 0.174

Table 4:Comparison of Mode of Birth between Survivors and Non Survivors Group

The comparison with respect to mode of birth between the two groups is depicted in Table 4 and Figure 4. Majority of the babies were born through vaginal delivery (NVD) in our study and the babies delivered by LSCS had a better survival rate. But with respect to their mode of birth (P > 0.05) , the association was not statistically significant. (Table 4 and Figure 4)

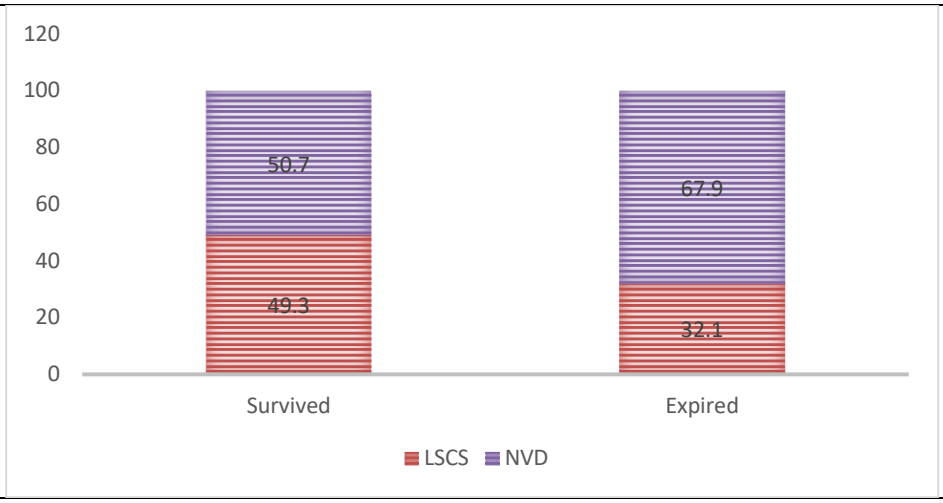

Figure 4:Comparison of Mode of Birth between the Survivors and Non Survivors Group

APGAR Score	Survivors		Non Survivors		Difference b/w Means	"t"	df	Sig
	Mean	SD	Mean	SD				
1st Min	3.1	1.1	2.7	1.3	0.5	1.530	93	P = 0.129
5th Min	6.3	1.9	5.7	2.0	0.6	1.381	93	P = 0.170

Table 5:Comparison APGAR Score between the Survivors and Non Survivors

The APGAR scores at 1st min and 5th min were compared between the two groups. The mean APGAR scores at 1st and 5th minutes of survivors group were 3.1 ± 1.1 and 6.3 ± 1.9. The non survivors groups mean APGAR (Appearance, Pulse, Grimace, Activity and Respiration) scores at 1st and 5th minute was 2.7 ± 1.3 and 5.7 ± 2.0. It was statistically insignificant($P > 0.05$). (Table 5 and Figure 5)

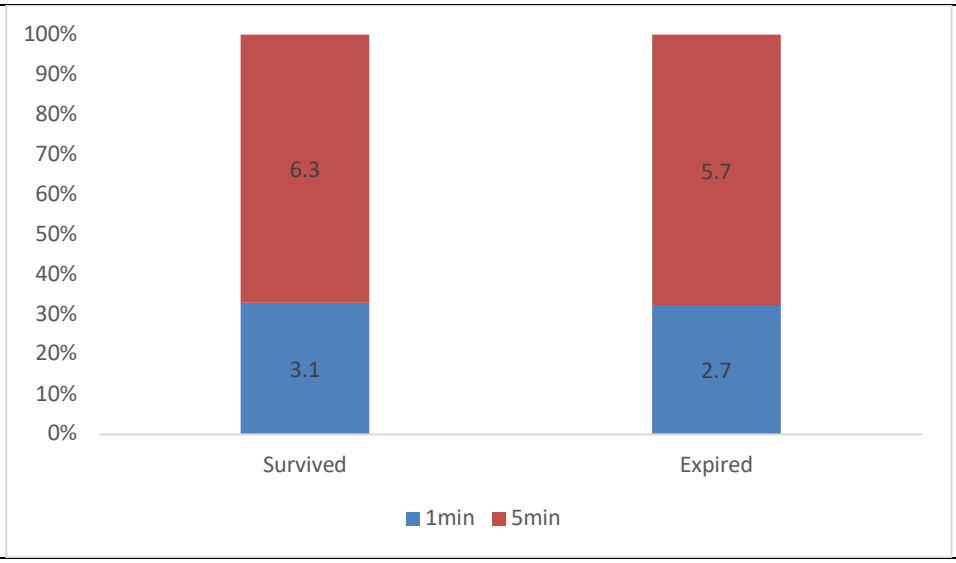

Figure 5: Comparison APGAR Score between the Survivors and Non Survivors

APGAR	Category	Survivors		Non Survivors		Total		Results
		No	%	No	%	No	%	
1Min	0-3	2	3.0	20	71.4	22	23.1	$\chi^2 = 51.9$ df = 1 P = 0.001
	4-7	65	97.0	8	28.6	73	76.9	
5 Mins	0-3	1	1.5	19	67.8	20	21.0	$\chi^2 = 56.8$ df = 1 P = 0.001
	4-7	24	35.8	8	28.6	32	33.7	
	8-10	42	62.7	1	3.6	43	45.3	

Table 6: APGAR Score Category between the Survivors and Non Survivors

There was a significant association between the APGAR score at 1 min and 5 mins between survivors and non survivors group. (Table 6 and Figure 6)

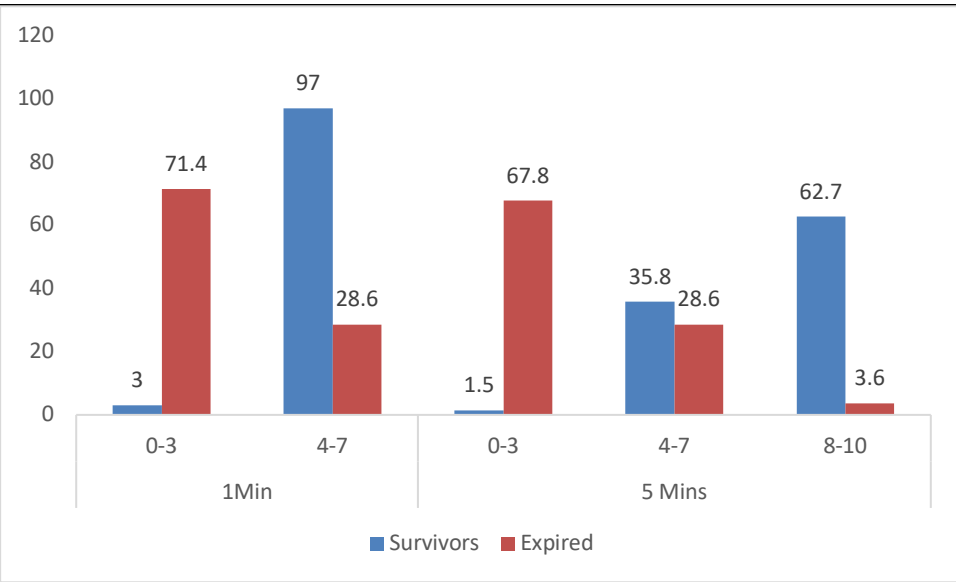

Figure 6: APGAR Score Category between the Survivors and Non Survivors

Inborn/ Outborn	Survivors		Non Survivors		Total		Results
	No	%	No	%	No	%	
Inborn	47	70.1	14	50.0	61	64.2	$\chi^2 = 3.489$
Outborn	20	29.9	14	50.0	34	35.8	df = 1
Total	67	100.0	28	100.0	95	67	P = 0.068

Table 7:Comparison of Inborn and Outborn between the Survivors and Non Survivors

Table 7 and Figure 7 shows the comparison of outcome between inborn and out-born categories. It was statistically insignificant (P > 0.05).

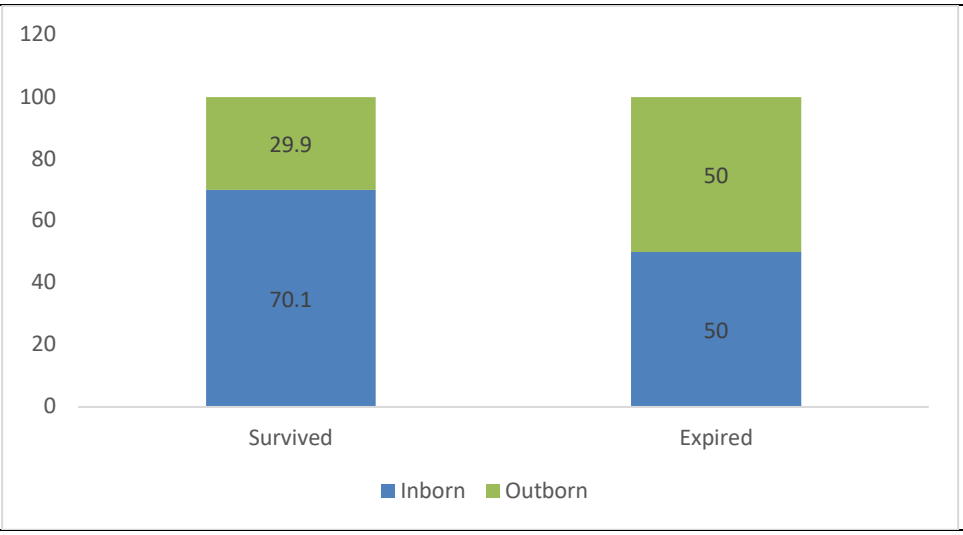

Figure 7:Comparison of Inborn and Outborn between the Survivors and Non Survivors

Indication For Ventilation	Survivors		Non Survivors		Total		Results
	No	%	No	%	No	%	
Congenital pneumonia	2	3.0	0	0.0	2	2.1	
MAS	11	16.4	4	14.3	15	15.8	$\chi^2 = 3.410$
Aspiration	3	4.5	1	3.6	4	4.2	P = 0.213
HMD	32	47.8	19	67.9	51	53.7	
Birth asphyxia	19	28.4	4	14.3	23	24.2	
Total	67	100.0	28	100.0	95	100.0	

Table 8: Comparison of the Indications between the Survivors and Non Survivors

Hyaline membrane disease (HMD) (47.8 %) was the most common indication, followed by intubation due to birth asphyxia (28.4 %) and in meconium aspiration syndrome

(MAS) (16.4 %) survivors group. Although, indication of ventilation did not affect survival statistically. (Table 8 and Figure 8)

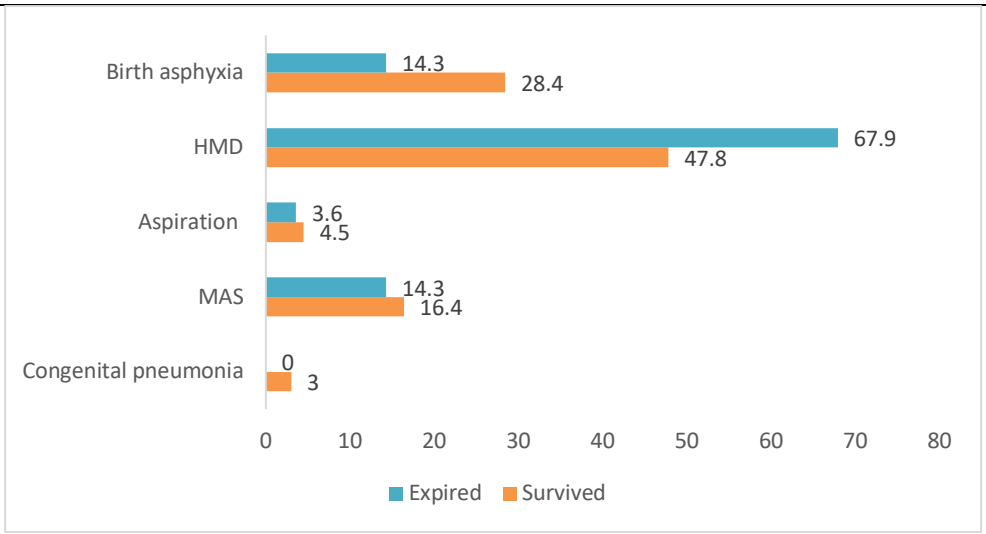

Figure 8: Comparison of the Indications between the Survivors and Non Survivors

Day of Ventilation	Survivors		Non Survivors		Total		Results
	No	%	No	%	No	%	
1st	29	43.3	15	53.6	44	46.3	$\chi^2 = 14.012$ df = 4 P = 0.001
2nd	27	40.3	8	28.6	35	36.8	
3rd	8	11.9	2	7.1	10	10.5	
4th	3	4.5	1	3.6	4	4.2	
5th	0	0.0	2	7.1	2	2.1	
Total	67	100.0	28	100.0	95	100.0	

Table 9:Comparison Day of Ventilation between the Survivors and Non Survivors

The difference among the two groups, outcome based on the days of ventilation was statistically significant ($P < 0.05$) especially the difference between 1st and 2nd day of ventilation was statistically significant.

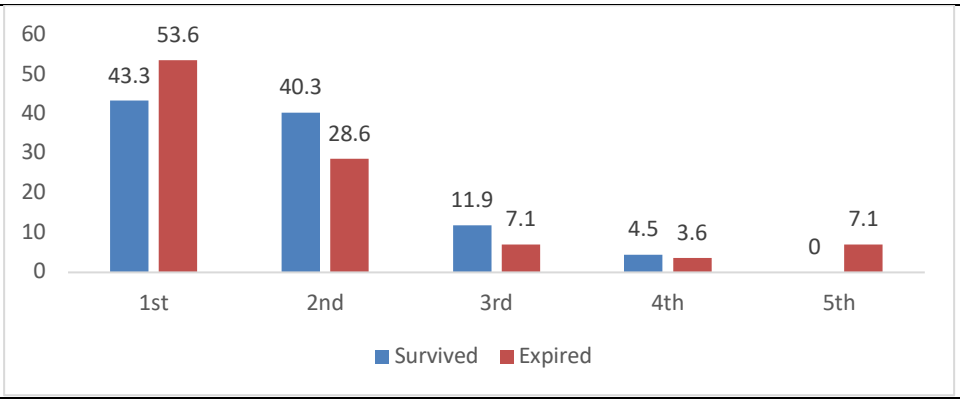

Figure 9:Comparison Duration of Ventilation between the Survivors and Non Survivors

Duration of Ventilation	Survivors		Non Survivors		Difference b/w Means	"t"	df	P
	Mean	SD	Mean	SD				
Hours	83.04	20.2	101.76	19.6	18.7	1.532	93	0.129

Table 10: Duration of Ventilation between the Survivors and Non Survivors

Comparison of the duration of ventilation between the two groups was done. 83.04 ± 20.2 days was the mean duration of survivors group. 101.76 ± 19.6 days was the mean duration of non-survivors group. This was statistically insignificant (P > 0.05). (Table 10 Figure 10)

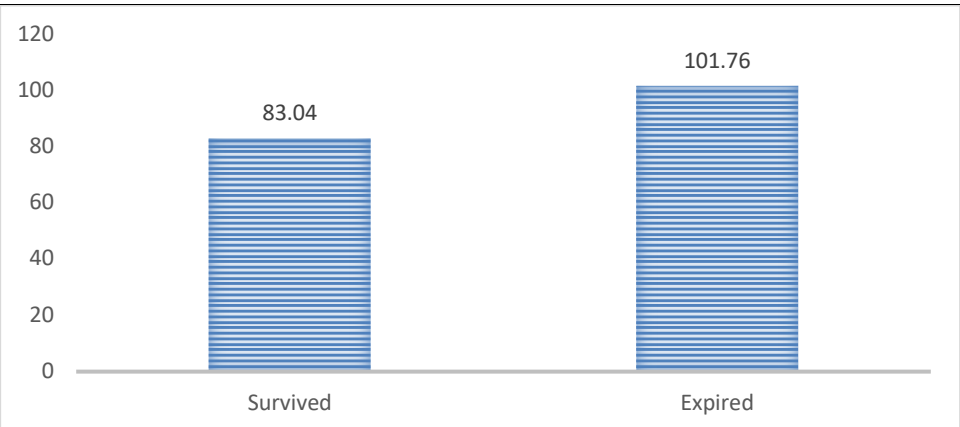

Figure-10:Duration of Ventilation between the Survivors and Non Survivors

Duration of Ventilation (in Hours)	Survivors		Non Survivors		Total		Results
	No	%	No	%	No	%	
< 24	19	28.4	6	14.3	25	24.2	$\chi^2 = 12.116$
25 - 48	11	16.4	7	17.9	18	16.8	
49 - 72	11	16.4	5	17.9	16	16.8	df = 4
73 - 96	14	20.9	3	10.7	17	17.9	P = 0.001
97 - 120	6	9.0	0	3.6	6	7.4	

➢	6	9.0	10	35.7	16	16.8
Total	67	100.0	28	100.0	95	100.0

Table 11:Duration of Ventilation among Survivors and Non Survivors

The survival rate was more if the duration of ventilation is < 24 hours followed by 73 - 96 hours from the duration of ventilation and survival rate data.

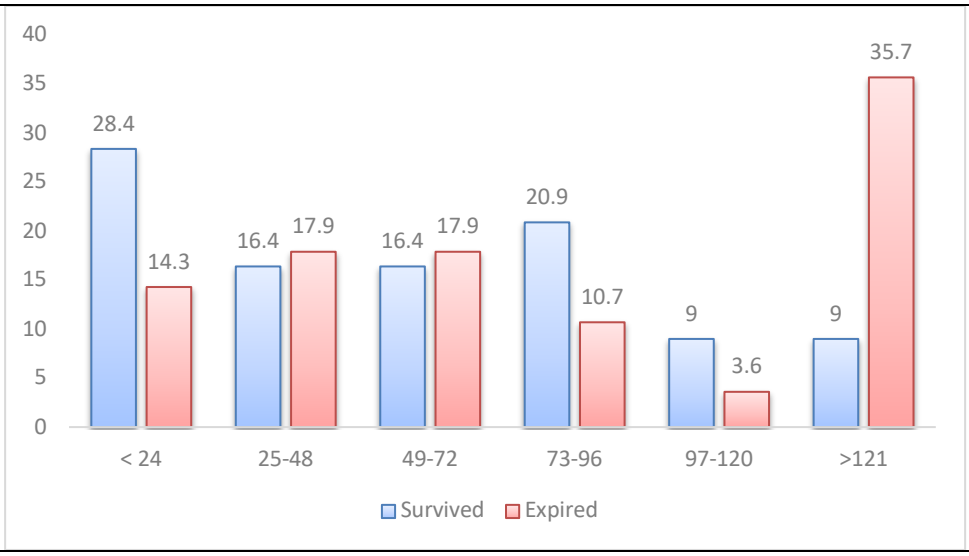

Figure 11:Duration of Ventilation among Survivors and Non Survivors

Air Leak	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Yes	2	2.5	3	10.1	5	5.4	$\chi^2 = 0.281$
No	65	97.5	25	89.9	90	94.6	df = 1
Total	67	100.0	28	100.0	95	100.0	P = 0.978

Table 12: Incidence of pneumothorax between the Two Groups – Survivors and Non Survivors

The incidence of pneumothorax in both the groups is shown in Table 12 and Figure 11. There was no statistically significant difference in the incidences of pneumothorax (P > 0.05).

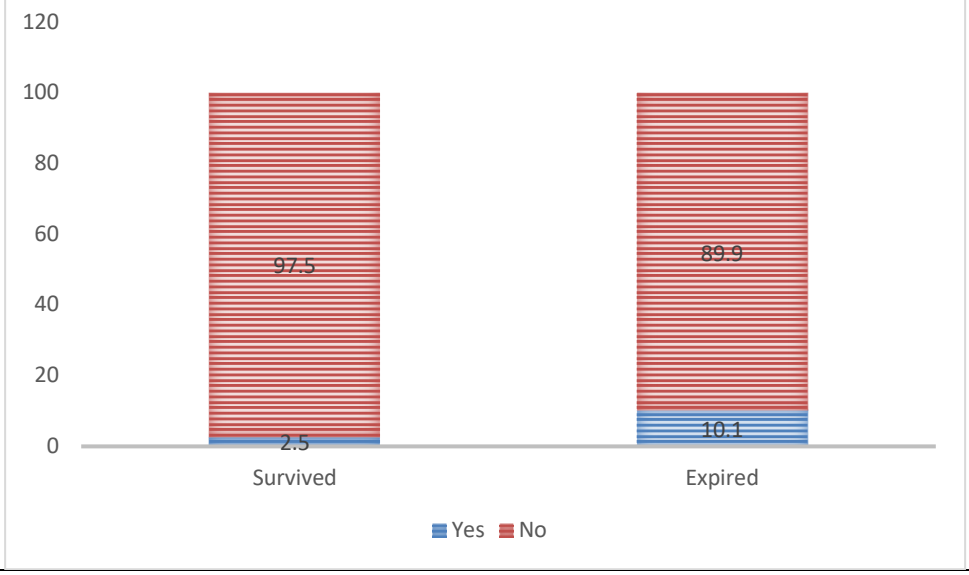

Figure 12: Incidence of Air Leak between the Two Groups, Survivors and Non Survivors

Septicaemia	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Yes	12	18.0	7	25.0	19	20.0	χ^2=0.620 P=0.430
No	55	82.0	21	75.0	76	80.0	
Total	67	100.0	28	100.0	95	100.0	

Table 13: Incidence of Septicaemia between the Survivors and Non Survivors

Table-13 and Figure-13 show the incidence of septicaemia in the two groups. There was no statistically significant difference in the incidence of septicaemia between the two groups (P > 0.05).

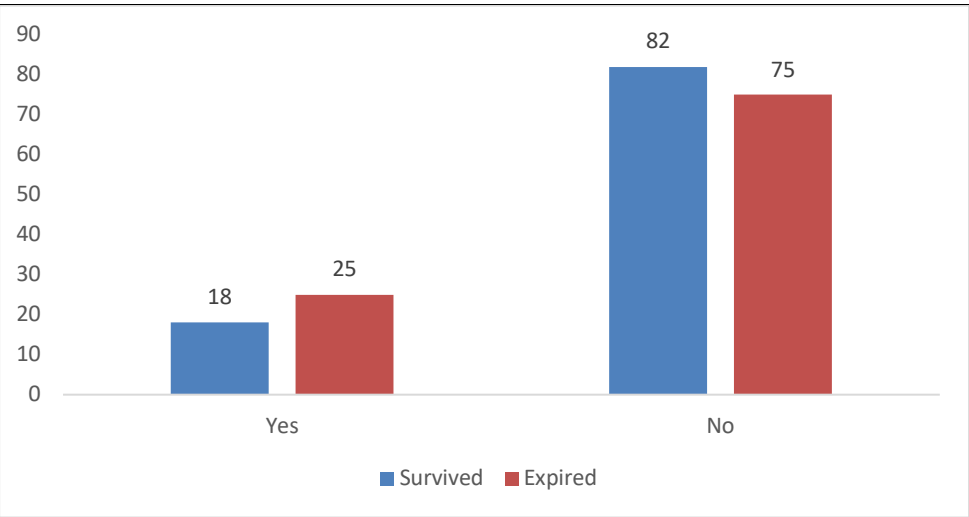

Figure 13: Incidence of Septicaemia between the Two Groups

Shock	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Yes	1	1.5	6	21.4	7	7.3	$\chi^2 = 8.763$
No	66	98.5	22	78.6	88	92.7	df = 1
Total	67	100.0	28	100.0	95	100.0	P = 0.003

Table 14: Incidence of Shock between the Two Groups

The incidence of shock in the two groups is shown in the (Table 14 and Figure 14). We observed lesser incidence of shock in survival groups (1.5 %) compared to non survivors group (21.4 %).

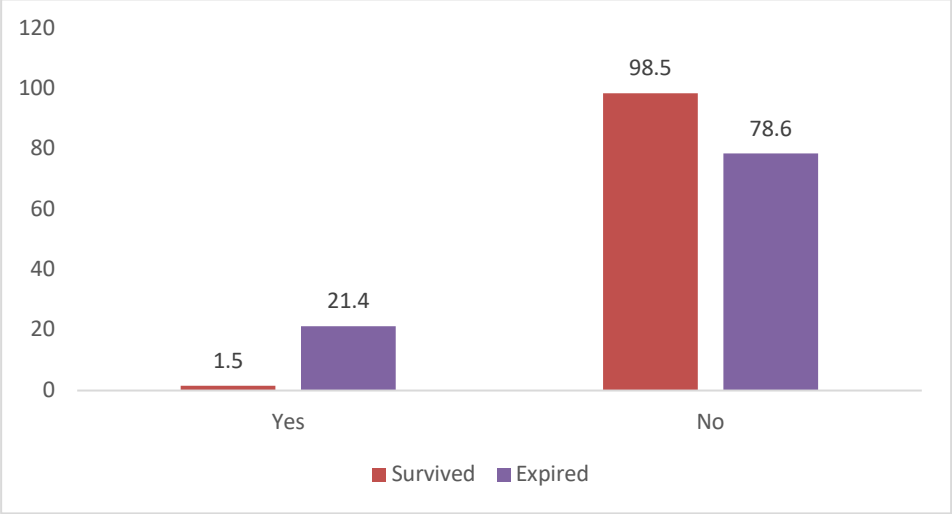

Figure 14:Incidence of Shock between the Survivors and Non Survivors

Pulmonary Haemorrhage	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Yes	1	1.5	6	21.4	7	7.3	$\chi^2 = 8.763$
No	66	98.5	22	78.6	88	92.7	df = 1
Total	67	100.0	28	100.0	95	100.0	P = 0.003

Table 15:Incidence of Pulmonary Haemorrhage between the Two Groups, Survivors and Non Survivors

The incidence of pulmonary haemorrhage in the two groups was shown in the Table 15 and Figure 5. The incidence of pulmonary haemorrhage is significantly higher in non survivors. (P > 0.05).

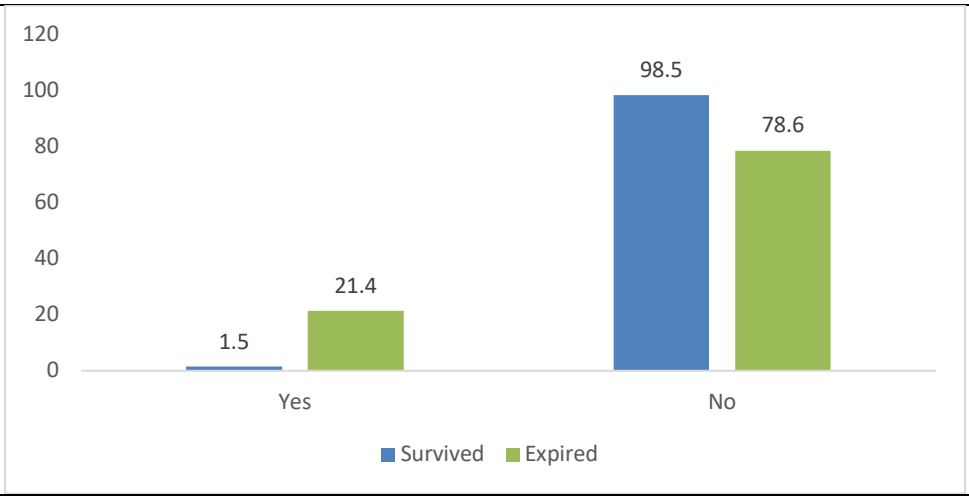

Figure 15:Incidence of Pulmonary Haemorrhage between the Two Groups, Survivors and Non Survivors

DIC	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Yes	5	7.8	9	32.1	14	14.7	$\chi^2 = 19.36$
No	62	98.5	19	67.9	81	85	df = 1
Total	67	100.0	28	100.0	95	100.0	P = 0.001

Table 16:Incidence of DIC between the Survivors and Non Survivors

The incidence of disseminated intravascular coagulation (DIC) in the two groups is shown in the Table 16 and Figure 16. The incidence of DIC was higher in non survivors group compared to non survivors group (P > 0.05).

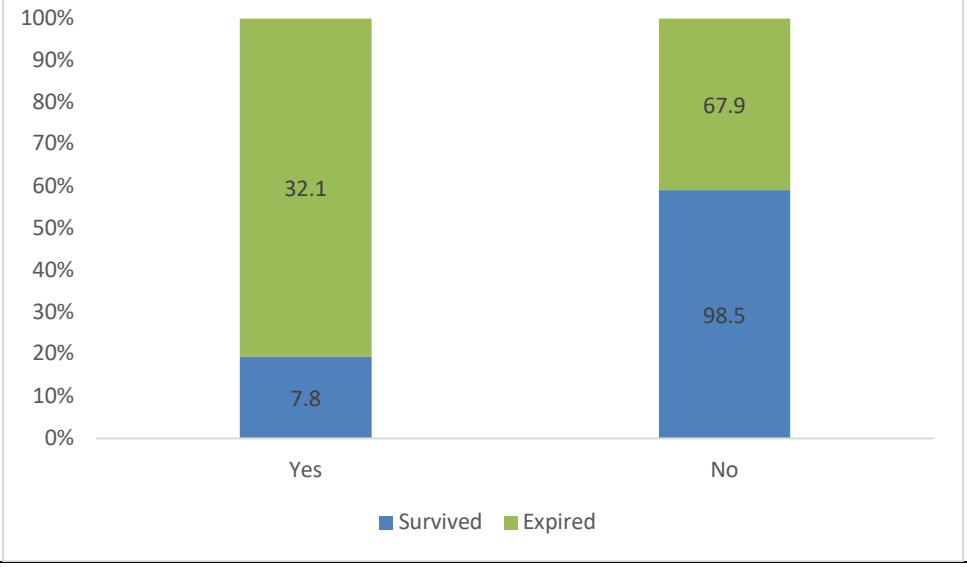

Figure 16: Incidence of DIC between Survivors and Non Survivors

Downe's Score	Survivors		Non Survivors		Difference b/w Means	"t"	df	P
	Mean	SD	Mean	SD				
Downe's Score	5.04	1.48	4.96	1.76	0.075	0.199	93	0.843

Table 17: Comparison of Downe's Score between Survivors and Non Survivors

Table 17 and Figure 17 show Downes score between both groups. This was statistically insignificant.

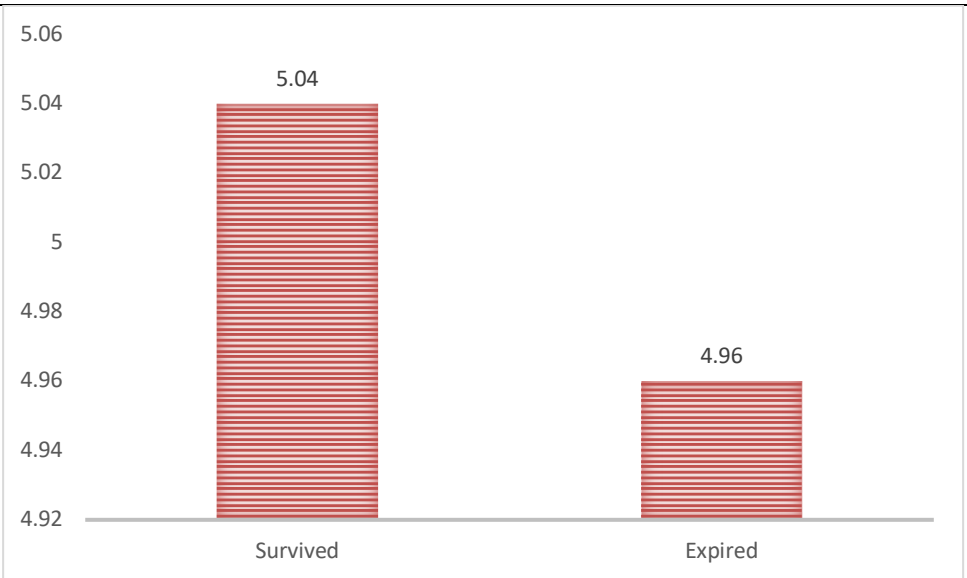

Figure 17: Comparison of Downe's Score between Survivors and Non Survivors

IVH	Survivors		Non Survivors		Total		Results
	n	%	n	%	N	%	
Yes	2	2.9	2	7.1	4	4.2	$\chi^2 = 4.26$
No	65	97.1	26	92.9	91	95.8	df = 1
Total	67	100.0	28	100.0	95	100.0	P = 0.03

Table 18: Incidence of IVH between Survivors and Non Survivors

The incidence of intraventricular haemorrhage (IVH) was 2.9 % among survivors and 7.1 % among non survivors. The IVH in no survivors was found to be statistically significant. (Table 18 and Figure 18)

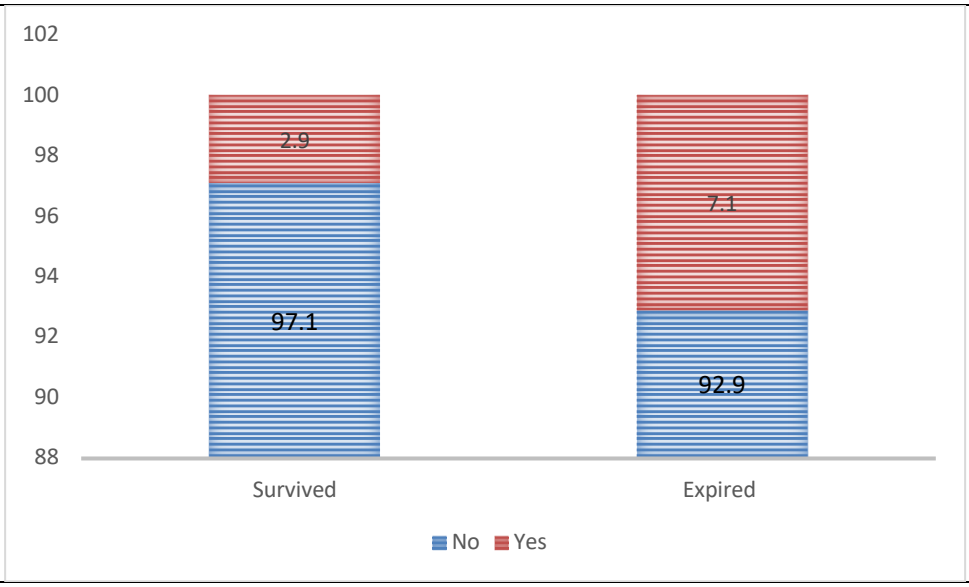

Figure 18: Incidence of IVH between Survivors and Non Survivors

Table 19: Summary of the Risk Factors Associated with Outcome among Neonates on Ventilator

Table 19: Risk Factors Affecting Ventilator Outcome

Mortality Predictors		Survivors (%)	Non Survivors (%)	P Value
Gender	Male	43.3	60.7	0.120
	Female	56.7	39.3	
Gestational Age	< 28weeks	6	28.6	0.010
	29 - 32	19.4	14.3	
	33 - 36	47.8	7.1	
	> 37 weeks	26.9	50	

Birth Weight	**> 2.5 kg**	**22**	**17**	**0.010**
	1.5 - 2.5 kg	**61**	**39**	
	< 1.5 kg	**16.9**	**28**	
	< 1 Kg	**2.9**	**14**	
Mode of Delivery	**LSCS**	**49.3**	**32.1**	**0.174**
	Vaginal delivery	**50.7**	**53**	
Place of Delivery	**Out-born**	**29.9**	**50**	**0.060**
	Inborn	**70.1**	**50**	
APGAR-Mean	**1st min**	**3.1**	**2.7**	**0.129**
	5th min	**6.3**	**5.7**	**0.170**
Maternal Risk Factors	**Hypertension**	**32.6**	**43.1**	**0.370**
	Diabetes mellitus	**12.9**	**10.0**	
	PROM	**17.0**	**12.0**	
	No risk factor	**37.5**	**34.9**	
Indication for Mechanical Ventilation	**HMD**	**44**	**53.1**	**0.213**
	MAS	**12**	**14**	
	Birth asphyxia	**28**	**14**	
	Congenital pneumonia	**3**	**0**	
Disease Pattern and Associated Complications	**Persistent-pulmonary -artery hypertension**	**6.2**	**22**	**0.002**
	Disseminated-intravascular coagulopathy	**7.8**	**32.1**	**0.001**
	Shock	**8.9**	**28.5**	**0.032**
	Sepsis	**18**	**25**	**0.430**
	Air leak	**4.5**	**7.1**	**0.978**
	Pulmonary haemorrhage	**1.5**	**21.4**	**0.003**
	IVH	**2.9**	**7.1**	**0.043**

DISCUSSION

"The use of mechanical ventilation is an integral part of any nursery catering to sick neonates in both developed and developing countries. However, even today, mechanically ventilated neonates have high fatality reported all over the world,[76-78] the fatality being higher in the tertiary referral neonatal units receiving most cases as out born neonates."[79] In order to determine the determinants of death in ventilated newborns, we conducted a prospective observation research using this scenario. The study was carried out at the RL Jalappa Hospital and Research Center's Pediatric Department in Tamaka, Karnataka. Ninety-five neonates under mechanical ventilator support admitted to the Department of Paediatrics admitted to RLJH were included in the study. Along with comparison with other studies the results of the present study are discussed below.

Neonatal morbidity and mortality rates are the reflection of a country's socioeconomic appearance. Besides the performance a good health of a care service can also be expressed by these indications.[80] In low-income countries (LICs), newborns on mechanical ventilation have a survival rate of 25% to 64%. [81] In the present study, mortality among ventilated neonates was 28 %. Whereas Thakkar et al. found the overall mortality was 52.1 % which is higher compared to the present study.[82] Similar mortality of 54% was recorded in a 2009 Surat study. [83] Additionally, Sultana et al. have observed overall survival rate of ventilated neonates as 35.8 %[84] which was strongly supported by some previous studies where the reported the overall survival of neonates as 33.3 % and 35.48 %.[85] Like other previous studies, the outcome of the neonate required mechanical ventilation was better with increasing birth weight and gestational age.[86] In the present study, among the study neonates, 48.4 % were male baby and 51.6 % were female baby. The sex distribution revealed that female babies had better survival rate (56.7 %) in comparison to male babies (43.3 %). This is consistent with the Sultana et al.

study, and a recent study that found that male babies had higher survival rates also supported this tendency. [87]

The mean birth weight of the survivors was 2.6±0.2 kg, whereas the mean birth weight of the non-survivors was 1.8±0.6 kg. Babies born to survivors and non-survivors had significantly different birth weights, according to statistics. In comparison to non-survivors, the average birth weight was substantially higher in the former group. Similar to Sultana et alstudy, .'s the enrolled neonates' mean birth weight was 1.85 51 g, and the mean birth weight of survivors was considerably greater than that of non-survivors (2.1±5.5 g vs. 1.7±4.2 g, p value 0.004). At least two earlier investigations, which are equivalent to ours, showed that the mean gestational age and mean birth weight of survivors were significantly higher than those of non-survivors. [88] In Yadav et al. "study, the most ventilated babies were of 1000–1499 g (18 %) while the mean birth weight in survived neonates was 2173 ± 665 g and expired neonates was 1611 ± 792 g."[89] Higher survival was reported by Trivedi et al.,[90] Hossain et al., and Duttet al.[91] in babies weighing > 2000 g: 56.7 %, 52.1 %, and 51 %, respectively, as in our study, it was 52.6 %.

Only 6.0% of survivors were younger than 28 weeks, whereas 26.9% and 47.8% were older than 37 weeks and 33 to 36 weeks, respectively. On the other hand, 50.0% of non-survivors were between 29 and 32 weeks old and 28.6% were under 28 weeks. The difference in mean gestational age between the survivors group (36.7±2.9 33.8±3.2 weeks) was statistically significant (P 0.05). In Takkar et al. "study, 17 % neonates were of less than 32 weeks of gestation, 31 % were between 32 to 36 weeks."[92] A similar cohort was reported in other Indian studies with 16 to 17 % for < 32 weeks gestation, 30 % to 40 % for 32 to 37 weeks respectively. 34.6 ± 3.38 weeks was the mean gestational age in Yadav et al. study of survived neonates and it was 33.4±4.5 weeks in expired neonates. Survival rates are directly proportional to the gestational age and are being highlighted as a statistically significant factor. The same

was observed in the study by Nayana et al.; The mean gestational age of the newborns who lived was 34.5 ± 3.5 weeks while it was 33.1±4.2 weeks in Hossain et al. study having a better survival than of 36.4 ± 2.7 weeks.

Babies born by normal vaginal delivery (NVD) were the majority in our study, and those babies delivered by lower segment caesarean section (LSCS) had a better survival rate. In Sultana et al. study, the mode of delivery revealed caesarean section (71.7 %) was mostly chosen approach which was subsequently followed by NVD (28.3 %). The increased rate of babies born by caesarean section in Sultana et al. This was because many high risk pregnancies were referred to this hospital

The mean APGAR scores at 1st and 5th minutes of survivors group were 3.1 ± 1.1 and 6.3 ± 1.9. The non survivors groups mean APGAR (Appearance, Pulse, Grimace, Activity and Respiration) scores at 1st and 5thminutes was 2.7 ± 1.3 and 5.7±2.0. There was a significant association of a 5 min APGAR score < 7 with mortality in the study of Arafa and Alshehri.[94] A similar finding was seen, in our series the motility was high with APGAR score < 7 at 1st min as compared to APGAR score of > 7 at 5 mins.

There was no statistically significant relationship between the two groups in respect of whether they are inborn or outborn (P > 0.05). Nearly 64.2 % babies were inborn when compared to out born 35.8 % and outcome was much better in intramural babies compared to extramural babies (70.1 % vs 20 %). These statistics were also observed in some other Indian studies.[95] These findings remind the importance of regionalization of newborn care to improve the overall outcome by the implementation of early intervention for high risk and sick inborn babies.

In our study, the most common underlying cause that required ventilation was hyaline membrane disease (HMD) (47.8 %), followed by birth asphyxia (28.4 %) and in meconium aspiration syndrome (MAS) (16.4 %) survivors group. “Similar results were seen in the studies

conducted in Nepal (indications - birth asphyxia, sepsis and MAS), in Karnataka (indications- birth asphyxia, RDS, sepsis),[96] RDS, MAS and apnoea were common indications for mechanical ventilation."[7] "The most common indication of ventilation in Yadav et al. study was HMD (38 %), but the survival rate of same was quite low (31.6 %). Dutt et al. found it as the third-most common (18.98 %) while Mathur et al.[4] as fourth-most common cause (8.4 %) of ventilation. Although, they reported a better survival rate in HMD of 53 % and 52 % respectively. Malhotra et al. showed MAS had the best outcome, 100 % and 63 %, respectively,[97] while Singh et al. had the poorest outcome where all babies in their series who were ventilated for MAS, expired." The least survival of 10 % (P = 0.03) was seen in severely asphyxiated babies (APGAR 0–3 at 5 min) similar to Trivedi et al., (20 %) whereas in Dutt et al., (40 %) and Nayana et al., (100 %), there was a higher survival rate.

The difference in the day of ventilation between the two groups was statistically significant (P < 0.05) especially the difference between 1st and 2nd day of ventilation was statistically significant. 83.04 ± 20.2 days was the mean duration of survival group and 101.76 ± 19.6 days was the mean duration of the non-survivors group. In the duration of ventilation between < 24 hours followed by 73 to 96 hours, the survival rate was more. Yadav et al. "study observed that majority of the cases - 52 (72.22 %) required ventilation for 4 - 7 days and 3 cases (4.16 %) required it for prolonged duration (> 10 days). Sheikh et al. also observed the duration of ventilation as 2 - 7 days in his study."[98]

In our study, most common mechanical ventilator modes used were PSIMV, volume-controlled ventilation and HFOV. Among those, 90 % of the neonates were put on PSIMV , only in 10 % of the neonates HFOV and volume controlled ventilation was used depending on the condition of the neonate , hence effect of mode of ventilation on the mortality could not be studied.

There was no statistically significant (P > 0.05) difference in the incidences of air leak between the two groups. Babies with shock, pulmonary haemorrhage and pneumothorax and IVH had 100 % mortality in Yadav et al study. In western literature and in West Indies, pulmonary air leak was the common complication.

Preterm babies need mechanical ventilation more than term neonates as they suffer from RDS, apnoea, sepsis, pneumonia, pulmonary haemorrhage etc. In our study, the incidence of septicaemia between the two groups was not statistically significant (P > 0.05). In Sultana et al. study sepsis was the commonest disease (in 35.8 % cases) among newborn requiring mechanical ventilation during hospital stay. Next to sepsis, respiratory distress syndrome 11 (20.8 %), congenital pneumonia 10 (18.9 %) and perinatal asphyxia in 8 (15.1 %) cases were listed as predominant disease pattern. Other conditions were meconium aspiration syndrome, meningitis and transient tachypnea of the newborn TTN in 2 (3.8 %), 2 (3.8 %) and 1 (1.9 %) cases. Likewise, the most common disease pattern in mechanically ventilated neonates was sepsis in 19 patients (37.2 %) followed by respiratory distress syndrome 9 (17.6 %), meconium aspiration syndrome 5 (9.8 %), birth asphyxia 6 (11.7 %) and congenital pneumonia in 2 according to a previous Nepalese study.[99] Similar report was observed in a previous Indian study.[100] However, perinatal asphyxia was also shown as the commonest disease pattern requiring mechanical ventilation in previous two studies.[101] In Sultana et al. study, complications of the ventilated babies were hospital acquired sepsis septicaemia (67.9 %) followed by tube block (52.8 %), ventilator associated pneumonia (26.4 %), ROP (13.2 %), BPD (11.3 %) and pneumothorax (5.7 %). Hospital acquired sepsis was the commonest complication in Sultana et al. study, as reported in other study. Due to frequent intervention like blood gases and prolonged duration of ventilation sepsis is a major complication in ventilated babies.

We observed lesser incidence of shock in survival groups (1.5 %) compared to non survival group (21.4 %). Sultana et al. revealed that shock was the commonest co-morbidity (64.2 %) which if higher compared to our study. There are other studies where the incidence of co-morbidity like shock was higher (84 %) reported in an Indian study. Shock and DIC were found significantly high in non survivors when compared with survived newborn. Besides, a significantly higher incidence of DIC was also reported by another study which is comparable to our study.

Complications like pulmonary haemorrhage and DIC has significant p value and caused adverse outcome. A similar mortality pattern was reported in a study conducted in Puducherry in relation to complications. The highest mortality was 100 % in Pnuemothorax and 94 % in pulmonary haemorrhage followed by 83.4 % in DIC, 65.6 % in pneumonia / sepsis and 43 % in shock according to them. Complications found by Thakkar were statistically significant such as circulatory disturbances, pneumothorax and pulmonary haemorrhage.

In our study, Downe's score between both groups was statistically insignificant. The most common condition of initiating mechanical ventilation in the Sultana et al. study found Downe score > 6 in 11 (20.8 %). The incidence of intraventricular hemorrhage (IVH) was 2.9 % among survivors and 7.1 % among non survivors was found to be statistically significant in our study. Several studies have investigated to find out the predictors or factors of mortality among ventilated newborn and variation exist regarding findings of the studies.

Complications such as persistent pulmonary artery hypertension, disseminated intravascular coagulopathy, shock, bleeding in the lungs and intraventricular haemorrhage were determined to be important “independent predictors” of fatality in the babies on mechanical ventilation in the present study on risk factors affecting ventilator outcomes like gestation age and low birth weight. Pulmonary haemorrhage and DIC were reported as the independent risk factors for the fatality in a study conducted in Kerala. In the mechanically

ventilated neonates, VLBW, higher FiO2 at the beginning and complications such as pulmonary haemorrhage, pneumothorax and disturbances in circulation were found to be important predictors of mortality in the observation of Thakkar et al.

Risk factors mentioned above were found to be “predictors of adverse outcome” in the babies on ventilators, which will help in prognostication.

CONCLUSION

- Neonatal patients who required mechanical ventilation had a 28% survival rate.
- HMD followed by birth asphyxia and MAS were the commonest indications for mechanical ventilation in neonates in our study.
- Among the low-birth-weight neonates and gestation age with < 28 weeks the mortality was significantly high.
- Ventilator associated complications like persistent pulmonary artery hypertension, DIC, shock, pulmonary haemorrhage and IVH contributed to the adverse outcome.
- Duration of ventilation was significantly low among the survival group compared to non survival group.

LIMITATIONS

- This is a single centre study, a multi-centre study could have included wider range of population groups, would have increased the generalizability of the study and would have given more precise results.
- Adequate follow up of Patients that went DAMA and patients that were referred to higher centre was not possible

SUMMARY

In the 1960s, intermittent positive or negative pressure mechanical ventilation for critically unwell newborns was implemented. This study was conducted in a tertiary care neonatal centre to evaluate the determinants of death in ventilated neonates. The study was carried out at the RL Jalappa Hospital and Research Center's Pediatric Department in Tamaka, Karnataka. The study comprised 95 newborns who were being supported by a mechanical ventilator and were being treated at the Department of Pediatrics at RLJH.

- In this study, about 38 (56.7 %) out of the 67 survivors were females and 17 (60.7 %) out of 28 non survivors were females. The males were 48.4 % and females were 51.6 %.The outcome did not have any statistically significant association with gender ($P > 0.05$).
- The mean birth weight of the survivors was 2.6±0.2 kg, whereas the mean birth weight of the non-survivors was 1.8±0.6 kg. Babies born to survivors and non-survivors had significantly different birth weights, according to statistics.
- Among survivors, 26.9 % were greater than or equal to $\geq$ 37 weeks and 47.5% were 33 - 36 weeks, and only 6.0 % were $\leq$ 28 weeks. On the other hand, among the non survivors, 50.0 % were $\geq$ 29 to 32 weeks and 28.6 % were < 28 weeks respectively. The mean gestational age of survivors group was 36.7 ± 2.9 weeks and non survivors group mean gestational age was 33.8 ± 3.2 weeks. The difference of mean gestational ages between the two groups was statistically significant ($P < 0.05$).
- In our study, we found that the majority of babies were delivered via normal vaginal delivery (NVD), and that kids delivered via lower segment caesarean section had a higher survival rate (LSCS). Regarding their method of birth, there was no statistically significant difference between the two groups ($P > 0.05$).
- The survivors' mean APGAR scores at the first and fifth ±1.9, respectively. The average APGAR (Appearance, Pulse, Grimace, Activity, and Respiration) scores for the non-

survivor groups were 2.7±1.3 and 5.7±2.0 at the first and fifth minutes, respectively.

- Whether they are inborn or outborn, the p value was statistically insignificant (P > 0.05).
- Hyaline membrane disease (HMD) was the most frequent cause of indication (47.8%), followed by birth asphyxia (28.4%), and in survivors of meconium aspiration syndrome (MAS) (16.4%). However, statistically, ventilation indication had no impact on survival.
- The difference between the two groups' days of survivors on ventilation was statistically significant (P 0.05), particularly the difference between the first and second day of ventilation.
- The mean duration for the group of survivors was 83.04 20.2 days, while the mean duration for the group of non-survivors was 101.76 19.6 days. Day 18.7's mean difference did not reach statistical significance (P > 0.05).
- The survival rate was higher when ventilation lasted between 24 and 72 hours and 73 to 96 hours.
- Table 12 and Figure 11 illustrate the air leak incidence in the two groups. The incidences of air leak across the two groups did not differ statistically significantly (P > 0.05).
- Between the two groups, there were no statistically significant differences in the occurrences of septicaemia (P > 0.05).
- The incidences of septicaemia between the two groups was not statistically significant (P > 0.05).
- We observed lesser incidence of shock in survival groups (1.5 %) compared to non survivors group (21.4 %).
- Instances of pulmonary haemorrhage were noticeably common in the group of non-survivors. (P > 0.05).
- When compared to the non survivors group, the incidence of DIC was considerably higher in the non survivors group (P > 0.05).

- There was no statistically significant difference in Downe's score between both groups.
- Intraventricular haemorrhage (IVH) occurred 2.9% of the time in survivors and 7.1% of the time in non-survivors. It was shown that there was a statistically significant correlation between IVH and survival status.
- Complications like persistent pulmonary artery hypertension, disseminated intravascular coagulopathy, shock, pulmonary haemorrhage, and intraventricular haemorrhage were discovered to be significant independent predictors of mortality in ventilated neonates, independent of risk factors affecting ventilator outcomes like gestational age and low birth weight.

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ANNEXURES

PROFORMA

TITILE – A PROSPECTIVE OBSERVATIONAL STUDY OF PREDICTORS OF MORTALITY IN MECHANICALLY VENTILATED NEONATES AT A TERTIARY CARE CENTRE

Patient information:

UHID number:

Name of the mother:

Name of the father:

Neonatal history:

Gestational age

Sex

Birth weight

Apgar at birth

Mode of delivery

Inborn or out-born

Blood pH at birth

Initial PO2

Heart rate at birth

Respiratory rate

Peripheral pulses

Capillary refill time

Haemoglobin

WBC-

CRP-

GRBS-

Downe's Score-

Temperature-

Maternal History:

Maternal complications

Mode of delivery

Maternal drug intake

Indications of Mechanical Ventilation:

Meconium Aspiration Syndrome

Hypoxic Ischemic encephalopathy

Persistent pulmonary Hypertension

Respiratory Distress Syndrome

Ventilator associated factors

Mode of mechanical ventilation:

Ventilator parameters:

Maximum PIP (CM of H2O)–

Maximum PEEP(CM of H2O)-

Maximum Fio2-

Indication of ventilation-

Duration of ventilation-

Complications of ventilation-

Others

Use of Inotropes

Chest X-ray findings

Mode of oxygen support used prior to mechanical ventilator:

Use of sedatives-

seizures -

No of antiepileptics used :

INFORMED CONSENT FORM

I Mr./Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is **A prospective observational study of predictors of mortality in mechanically ventilated neonates at a tertiary care centre .**

I have been explained that my clinical findings, investigations, postoperative findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Relation to patient:

Date:

Place:

PATIENT INFORMATION SHEET

STUDY TITLE: A PROSPECTIVE OBSERVATIONAL STUDY OF PREDICTORS OF MORTALITY IN MECHANICALLY VENTILATED NEONATES AT A TERTIARY CARE CENTRE

STUDY SITE:

Principal investigator: Dr NikhithaVenkiteela /Dr. J Krishnappa

I Dr. Nikhitha Venkiteela , Post graduate student in Department of Paediatrics at Sri Devraj Urs Medical College, will be conducting a study titled **A PROSPECTIVE STUDY OF PREDICTORS OF MORTALITY IN MECHANICALLY VENTILATED NEONATES AT A TERTIARY CARE CENTRE ,**my dissertation under the guidance of Dr J Krishnappa , Professor of Department of Paediatrics. The participants of this study i.e. include 93 neonates who are ventilated , admitted in the neonatal intensive care unit .

You will not be paid any financial compensation for the participation of your neonate in this research project.

All the data will be kept confidential and will be used only for research purpose by this institution. You are free to provide consent for the participation of your child in this study. You can also withdraw your child from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Principal Investigator

Dr.NikhithaVenkiteela

Date-

ರೋಗಿಮಾಹಿತಿಹಾಳೆ

ಅಧ್ಯಯನಶೀರ್ಷಿಕೆ:

ಪ್ರಾಸ್ಪೆಕ್ಟಿವ್ಆಬ್ಸರ್ವೇಷನಲ್ಮ್ಯೂಚಕರಅಧ್ಯಯನವೆಂಟಿಲೇಟೆಡ್ನವಜಾತಶಿಶುಗಳಲ್ಲಿನಮರಣದಪ್ರಮಾಣಅವಲೋಕನಅಧ್ಯಯನ

ಅಧ್ಯಯನಸ್ಥಳ:

ಮುಖ್ಯಸಂಶೋಧಕಿ: ಡಾ.ನಿಖಿತಾವೆಂಕಟೇಶ್ (ಡಾ.ಜೆ.ಕೃಷ್ಣಪ್ಪ) .

ಐಡಾ.ನಿಖಿತಾವೆಂಕಟೇಶ್ಮತ್ತುಶ್ರೀದೇವರಾಜಅರಸುವೈದ್ಯಕೀಯಕಾಲೇಜಿನಮಕ್ಕಳವಿಭಾಗದಸ್ನಾತಕೋತ್ತರವಿದ್ಯಾರ್ಥಿನಿಡಾ. ನಿಖಿತಾವೆಂಕಟೇಶ್ಅವರು, ವೆಂಟಿಲೇಟೆಡ್ನ್ಯೂರೊನೇಟ್ಸ್ನಲ್ಲಿಸಂಭವಿಸುವಮರಣದಮುನ್ಸೂಚನೆಗಳಮುನ್ಸೂಚನೆಗಳಬಗ್ಗೆಅಧ್ಯಯನನಡೆಸಿ, ನನ್ನಪ್ರಬಂಧವನ್ನು'ಪಿಡಿಯೇಟ್ರಿಕ್ಸ್' ವಿಭಾಗದಪ್ರಾಧ್ಯಾಪಕಡಾ.ಜೆ.ಕೃಷ್ಣಪ್ಪಅವರಮಾರ್ಗದರ್ಶನದಲ್ಲಿಅಧ್ಯಯನನಡೆಸಿ,ಡಾ.ಬಿ.ಆರ್.ಅಂಬೇಡ್ಕರ್ಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲಿದ್ದಾರೆ. ಈಅಧ್ಯಯನದಸ್ಪರ್ಧಿಗಳು 93 ಜನರುವೆಂಟಿಲೇಟೆಡ್ - ತೀವ್ರನಿಗಾಘಟಕದಲ್ಲಿದಾಖಲಾಗಿದ್ದಾರೆ .

ಈಸಂಶೋಧನಾಯೋಜನೆಯಲ್ಲಿನಿಮ್ಮನಿಯೋನೇಟ್ಭಾಗವಹಿಸುವಿಕೆಗಾಗಿನಿಮಗೆಯಾವುದೇಆರ್ಥಿಕಪರಿಹಾರನೀಡಲಾಗುವುದಿಲ್ಲ.

ಎಲ್ಲಾದತ್ತಾಂಶಗಳನ್ನುಗೌಪ್ಯವಾಗಿಇಡಲಾಗುತ್ತದೆಮತ್ತುಈಸಂಸ್ಥೆಯಿಕೇವಲಸಂಶೋಧನಾಉದ್ದೇಶಕ್ಕಾಗಿಮಾತ್ರಬಳಸಲ್ಪಡುತ್ತದೆ. ಈಅಧ್ಯಯನದಲ್ಲಿನಿಮ್ಮಮಗುವಿನಪಾಲ್ಗೊಳ್ಳುವಿಕೆಗೆಸಮ್ಮತಿಯನ್ನುಒದಗಿಸಲುನೀವುಸ್ವತಂತ್ರರು. ಯಾವುದೇಕಾರಣಗಳನ್ನುನೀಡದೆಯಾವುದೇಸಮಯದಲ್ಲಿನೀವುನಿಮ್ಮಮಗುವನ್ನುಅಧ್ಯಯನದಿಂದಹಿಂದೆಗೆದುಕೊಳ್ಳಬಹುದು. ನೀವುಭಾಗವಹಿಸಲುನಿರಾಕರಿಸುವುದರಿಂದಈಸಂಸ್ಥೆಯಲ್ಲಿಯಾವುದೇವರ್ತಮಾನಅಥವಾಭವಿಷ್ಯದಆರೈಕೆಗೆನೀವುಪೂರ್ವಗ್ರಹಪೀಡಿತರಾಗುವುದಿಲ್ಲ.

ಪ್ರಧಾನಪರಿಶೋಧಕರಹೆಸರುಮತ್ತುಸಹಿ

ಡಾ.ನಿಖಿತಾವೆಂಕಿಟೇಲ

ದಿನಾಂಕ-

ಮಾಹಿತಿಸಮ್ಮತಿನಮೂನೆ

ನಾನುಶ್ರೀ / ಶ್ರೀ. _____ ಅನ್ನುನನ್ನಸ್ವಂತಅರ್ಥವಾಗುವಭಾಷೆಯಲ್ಲಿವಿವರಿಸಲಾಗಿದೆ, ಇದುನನ್ನನ್ನುಅಧ್ಯಯನದಲ್ಲಿಸೇರಿಸಲಾಗುವುದು,

ಇದುವಾತಾಯನನವಜಾತಶಿಶುಗಳಲ್ಲಿನಮರಣದಮುನ್ಸೂಚಕರನಿರೀಕ್ಷಿತಅವಲೋಕನಅಧ್ಯಯನವಾಗಿದೆ.

ನನ್ನಕ್ಲಿನಿಕಲ್ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯನಂತರದಸಂಶೋಧನೆಗಳನ್ನುಮೌಲ್ಯಮಾಪನಮತ್ತುಅಧ್ಯಯನದಉದ್ದೇಶಕ್ಕಾಗಿದಾಖಲಿಸಲಾಗುತ್ತದೆಎಂದುನನಗೆವಿವರಿಸಲಾಗಿದೆ.

ಈಅಧ್ಯಯನದಲ್ಲಿನನ್ನಭಾಗವಹಿಸುವಿಕೆಯುಸಂಪೂರ್ಣವಾಗಿಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆಎಂದುನನಗೆವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತುನಾನುಯಾವುದೇಸಮಯದಲ್ಲಿಅಧ್ಯಯನದಿಂದಹಿಂದೆಸರಿಯಬಹುದುಮತ್ತುಇದುನನ್ನವೈದ್ಯರೊಂದಿಗಿನನನ್ನಸಂಬಂಧಅಥವಾನನ್ನಕಾಯಿಲೆಗೆಚಿಕಿತ್ಸೆಯಮೇಲೆಪರಿಣಾಮಬೀರುವುದಿಲ್ಲ.

ನನ್ನಸ್ವಂತಅರ್ಥವಾಗುವಭಾಷೆಯಲ್ಲಿ, ಮಧ್ಯಸ್ಥಿಕೆಗಳಕಾರಣದಿಂದಾಗಿಸಂಭವನೀಯಪ್ರಯೋಜನಗಳುಮತ್ತುಪ್ರತಿಕೂಲತೆಗಳಅಗತ್ಯವಿರುವಮಧ್ಯಸ್ಥಿಕೆಗಳಬಗ್ಗೆನನಗೆವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದಸಮಯದಲ್ಲಿಕಂಡುಬರುವನನ್ನಎಲ್ಲಾವಿವರಗಳನ್ನುಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆಮತ್ತುಸಂಶೋಧನೆಗಳನ್ನುಪ್ರಕಟಿಸುವಾಗಅಥವಾಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನವಿವರಗಳನ್ನುಮರೆಮಾಚಲಾಗುತ್ತದೆಎಂದುನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿನನ್ನಬಳಿಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿಮೊಬೈಲ್ಸಂಖ್ಯೆಇದೆ.

ಈಅಧ್ಯಯನದಭಾಗದಲ್ಲಿಸೇರಿಸಲುನನ್ನಸಂಪೂರ್ಣಮನಸ್ಸಿನಲ್ಲಿನಾನುಸಂಪೂರ್ಣಒಪ್ಪಿಗೆನೀಡುತ್ತೇನೆ.

ರೋಗಿಯಸಹಿ:

ಹೆಸರು:

ರೋಗಿಗೆಸಂಬಂಧ:

ದಿನಾಂಕ:

ಸ್ಥಳ:

ಸಾಕ್ಷಿಯ ಸಹಿ:

ಹೆಸರು:

KEY TO MASTER CHART

ABBREVATIONS

S.No: Serial Number

UHID: Unique Hospital Identification Number

NVD : Normal Vaginal Delivery

LSCS: Lower Segment Cesarean Section

DIC: Disseminated Intravascular Coagulopathy

DKA: Diabetic ketoacidosis (in mother)

PROM: Prolonged rupture of membranes

HMD – Hyaline Membrane Disease

MAS- Meconium Aspiration Syndrome

HFOV – High Frequency Oscillatory Ventilation

PSIMV- Pressure controlled synchronized intermittent mandatory ventilation

VC- Volume controlled ventilation

A - S.No

B – Name of the patient

C – Gender of the patinet

D – gestational age

E – Birth weight

F – APGAR at birth

G – Mode of delivery

H – Inborn or out born

I – Blood pH at birth

J – initial pO2

K – Downes score

L – Maternal complications or drug intake

M – Indication of ventilation

N – Maximum PIP

O – Maximum PEEP

P – Maximum Fio2

Q – Duration of ventilation

R – Complications

S – Use of inotropes

T – Mode of oxygen support prior to ventilation

U – Outcome (extubated or death)

MASTERCHART

	Name	Sex	Gestational age	Birth weight	Apgar at birth	Mode of delivery	Inborn or Outborn	Blood pH at birth	Initial pO2	Downe's score	Maternal complications or drug intake	Indication for mechanical ventilation	Mode of mechanical ventilation	Maximum PIP	Maximum PEEP	Maximum Fio2	Duration of mechanical ventilation	Complications	Use of ionotropes	Mode of oxgen support prior to ventilation	Outcome
1	Baby of gayathri	female	preterm	2kg	7	LSCS	inborn	7.36	82	4	nil	HMD	PSIM	18	6	70	2	SEPSIS	no	CPAP	extubated
2	Baby of Ayesha	male	late preterm	2.4kg	6	NVD	inborn	7.3	90	2	DKA	HMD	PSIM	17	6	60	4	SEPSIS AND DIC	yes	no	DEATH
3	Baby of ayesha sultana	female	term	2.5kgs	7	LSCS	outborn	7.6	72	7	Anemia	MAS	PSIM	18	6	75	4	SEPSIS	no	Hfnc	extubated
4	Baby of jyothi	female	late preterm	1.54 kgs	7	LSCS	inborn	7.02	82	5	Eclampsia	HMD	PSIM	18	5	100	7	SEPSIS	No	Hfnc	extubated
5	Baby of swethashree	female	preterm	0.980 kgs	6	LSCS	inborn	6.99	88	4	Eclampsia	HMD	PSIM	17	6	100	7	SEPSIS	Yes	No	DEATH
6	Baby of venkatratna	female	term	2.5kgs	7	NVD	inborn	7.32	88	4	nil	MAS	PSIM	18	7	100	3	SEPSIS AND DIC	No	No	extubated
7	Baby of Tejaswini	male	very preterm	2.36kgs	7	LSCS	inborn	7.6	80	4	Rh negative	HMD	PSIM	17	6	75	2	SEPSIS AND HYPOCALCEMIA	NO	CPAP	Extubated
8	Baby of Shabana	male	pretem	2.4kgs	6	NVD	inborn	7.54	60	4	nil	HMD	PSIM	16	5	65	4	SEPSIS	No	CPAP	Extubated
9	Baby of Gangothri	male	term	2.1kgs	7	NVD	outborn	7.34	88	3	Thrombo cytopenia	birth asphyxia	PSIM	15	5	70	3	SEPSIS	no	CPAP	Extubated
10	baby of Kernitha	male	preterm	2kgs	6	LSCS	outborn	7.6	86	4	nil	HMD	PSIM	16	5	80	7	Nil	no	CPAP	Extubated
11	Baby of Sowmya twin 1	female	pretrm	1.9kgs	8	LSCS	outborn	7.34	84	6	nil	HMD	PSIM	16	5	60	8	SEPSIS AND DIC		Hfnc	Extubated
12	baby of sowmya twin 2	female	extreme preterm	0.890kgs	6	LSCS	outborn	7.3	88	5	nil	HMD	PSIM	15	6	50	3	Shock	yes	no	DEATH
13	baby of Abhilasha	male	term	2.3kgs	7	NVD	outborn	7.33	78	5	nil	MAS, Birth	PSIM	15	5	90	1	SEPSIS	yes	no	DEATH
14	baby of Harish	male	term	2.6kgs	7	NVD	outborn	7.4	79	4	nil	MAS	PSIM	15	5	70	9	SEPSIS	yes	CPAP	extubated
15	baby of isha sultana	female	term	2.5kgs	6	LSCS	outborn	7.24	89	4	nil	MAS	PSIM	16	6	80	5	nil	no	CPAP	extubated
16	baby of Dhivya	male	term	1.94kgs	7	LSCS	outborn	Not known	80	4	Hyper tension	MAS	PSIM	17	5	100	4	Pulmonary haemorrhage	Yes	no	DEATH
17	baby of Sujatha	male	extreme preterm	0.990kgs	6	LSCS	inborn	7.22	60	6	Eclampsia	HMD	HFOV	15	5	100	2	DIC SEPSIS	yes	no	DEATH
41	baby of Chandana	male	term	2.6kgs	4	NVD	inborn	7.42	78	6	nil	aspiration	VC	16	5	100	4	NIL	no	CPAP	extubated
42	baby of Vasantha	female	preterm	1.8kgs	6	LSCS	inborn	7.22	79	8	nil	Apnoea of prematurity	VC	17	5	75	2	SEPSIS AND	no	Hfnc	extubated
43	baby of Jyothi	female	term	2.82kgs	8	NVD	inborn	7.22	89	4	nil	MAS	PSIM	16	5	65	1	NIL	no	Hfnc	extubated
44	baby of Manjuka	female	pretem	2.1kgs	6	NVD	outborn	Not known	80	4	nil	HMD	PSIM	16	5.5	70	1	NIL	no	Hfnc	extubated
45	baby of Monica	female	very preterm	1.68kgs	7	LSCS	outborn	Not known	60	3	nil	HMD	VC	17		80	2	NIL	yes	no	DEATH
46	baby of Bhavani	female	preterm	2.56kgs	7	NVD	outborn	Not known	70	4	PROM	HMD	HFOV	16	5	60	8	Pneumothorax	yes	no	DEATH
47	baby of Pavithra	female	preterm	2.4kgs	6	NVD	outborn	Not known	60	6	nil	Birth asphyxia	PSIM	16	6	50	4	NIL	no	Hfnc	extubated
48	baby of Krishna veni	male	late preterm	2.52kgs	7	LSCS	outborn	Not known	80	5	nil	MAS	PSIM	16	7	90	4	NIL	no	Hfnc	extubated
49	baby of Fiza kouser	female	late preterm	2.62kgs	7	LSCS	outborn	Not known	40	5	nil	HMD	HFOV	17	6	70	7	NIL	no	Hfnc	extubated
50	baby of Samreen khanum	female	preterm	2.16kgs	7	NVD	outborn	Not known	70	4	Ards	HMD	VC	18	5	80	7	Pneumothorax	no	CPAP	extubated
51	baby of Ayana	male	term	2.9kgs	2	NVD	outborn	Not known	80	4	nil	birth asphyxia	PSIM	17	5	100	3	NIL	yes	CPAP	extubated
52	baby of Lavanya	male	very preterm	1.98kgs	6	NVD	outborn	Not known	60	4	nil	HMD	PSIM	16	5	100	2	Shock pulmonary	yes	CPAP	DEATH
53	baby of Varalakshmi	male	late preterm	2.43kgs	6	LSCS	outborn	Not known	88	5	nil	HMD	PSIM	16	5	50	5	NIL	no	CPAP	extubated
54	baby of Prathiba	male	preterm	2.42kgs	not known	LSCS	outborn	Not known	86	4	PROM	HMD	PSIM	16	6	60	6	NIL	no	CPAP	extubated
55	baby of Sirisha	male	term	2.68kgs	7	NVD	outborn	Not known	84	6	PROM	MAS	HFOV	15	5	70	7	DIC	no	no	DEATH
56	baby of Ramya	male	preterm	2.4kgs	7	NVD	outborn	Not known	88	6	CHD	HMD	PSIM	16	5	70	4	Hypo glycaemia	yes	Hfnc	extubated
57	baby of Vaseema	female	very preterm	1.6kgs	5	LSCS	outborn	Not known	78	5	nil	HMD	PSIM	15	5	30	3	NIL	yes	Hfnc	extubated
58	baby of Gangothri	female	late preterm	2.56kgs	7	NVD	outborn	Not known	79	6	nil	RDS	HFOV	15	5	80	5	NIL	no	Hfnc	extubated
59	baby of Sabira	male	preterm	2.12kgs	7	NVD	outborn	Not known	89	6	nil	HMD	VC	16	5	90	3	DIC AKi	no	no	DEATH
60	baby of Gayathri	female	preterm	2.3kgs	7	LSCS	outborn	Not known	80	6	nil	Congenital Pnemonia	PSIM	16	5	50	2	NIL	no	Hfnc	extubated
61	Baby of Kavitha	female	preterm	1.75kgs	7	LSCS	Inborn	7.45	80	6	Eclampsia	HMD	PSIM	17	6	70	2	SEPSIS AND DIC	Yes	no	DEATH
62	Baby of Pooja	male	term	3.2kgs	8	NVD	Inborn	6.57	66	7	Nil	HMD	PSIM	18	6	100	7	IEM	Yes	no	DEATH

63	Baby of Vimala	female	Late preterm	1.28kgs	7	NVD	outborn	notknown	64	7	Nil	HMD	PSIM	16	6	100	1	pulmonary haemorrhage	Yes	Hfnc	DEATH
64	Baby of Kodimalar	Male	Very preterm	1.03	6	LSCS	Inborn	notknown	90	6	Hypertension	HMD	PSIM	18	6	80	1	Nil	No	Cpap	Extubated
65	Baby of Ahila	male	Term	3kgs	7	NVD	Inborn	7.54	90	7	Nil	MAS	PSIM	16	7	60	3	nil	No	Hfnc	Extubated
66	Baby of Mounika	female	Very preterm	1.85	Notknown	NVD	outborn	notknown	100	7	Nil	HMD	PSIM	17	6	100	1	pulmonary haemorrhage	No	no	DEATH
67	Baby of Lavanya	male	Term	2.88	Notknown	NVD	outborn	notknown	100	7	Nil	Birth asphyxia	PSIM	15	5	40	1	Intracranial bleed and shock	Yes	no	DEATH
68	Baby of Asma aja	Female	Term	2.2	Notknown	NVD	outborn	notknown	70	6	Nil	No	PSIM	16	5	50	2	Nil	No	no	Extubated
69	Baby of Sumana taj	female	term	3	Notknown	NVD	outborn	notknown	76	8	Nil	Aspiration	HFO	17	6	100	7	pulmonary haemorrhage and	Yes	Hfnc	DEATH
70	Baby of Nithyasree	Female	Late preterm	2.02kgs	Notknown	LSCS	outborn	notknown	60	7	Placenta previa	HMD	PSIM	16	6	80	6	nil	no	Hfnc	Extubated
71	Baby of Vaniashree	female	Late preterm	1.70kgs	Notknown	LSCS	Inborn	7.2	90	5	Nil	HMD	PSIM	16	6	70	2	Nil	No	Cpap	Extubated
72	baby of Farzana khanum	female	Late preterm	2.02	7	LSCS	inborn	7.34	80	6	Nil	HMD	PSIM	17	6	60	4	Pnuemothorax	No	Cpap	Extubated
73	baby of Manjula	female	Late preterm	2.11	7	LSCS	inborn	7.44	90	3	Nil	Birth asphyxia	PSIM	16	6	75	4	Nil	No	Hfnc	Extubated
74	baby of Roopa	female	Late preterm	2.1	6	NVD	inborn	7.3	90	7	Nil	MAS	PSIM	16	5	100	7	nil	no	Hfnc	Extubated
75	baby of Usha	female	Very preterm	1.2	7	NVD	inborn	7.22	90	7	Hypertension	HMD	PSIM	16	6	100	7	Nil	No	Cpap	Extubated
76	baby of Kavitha	male	Late preterm	2.2	6	LSCS	inborn	7.04	90	6	Nil	HMD	PSIM	14	7	100	3	Nil	No	Cpap	Extubated
77	baby of Bhavani	male	term	2.8kgs	Notknown	NVD	outborn	notknown	80	2	Nil	Birth asphyxia	PSIM	18	6	75	2	pulmonary haemorrhage	no	no	DEATH
78	baby of Kavya	male	Term	2.74kgs	Notknown	NVD	outborn	notknown	80	4	Prom	Sepsis	PSIM	15	5	65	7	Shock	Yes	no	DEATH
79	baby of Anupama	female	Late preterm	2	7	LSCS	inborn	6.97	80	5	nil	HMD	PSIM	16	5	70	4	DIC	no	no	DEATH
80	baby of Sowmya	female	Very preterm	1.5	7	NVD	inborn	7.24	60	6	Nil	HMD	PSIM	18	5	80	7	Intracranial bleed	No	Hfnc	Extubated
81	baby of Sumathi	male	Late preterm	2.2	3	LSCS	inborn	7.2	88	2	Nil	Birth asphyxia	PSIM	16	5	60	3	Nil	No	Hfnc	Extubated
82	Baby of Rose philomina	female	Late preterm	1.85	7	AVD	inborn	7.4	86	6	Nil	HMD	HFO	17	6	50	2	DIC/pulmoary haemorrhage	No	Cpap	Extubated
83	Baby of Chandana	male	Term	2.2	5	NVD	inborn	7.24	84	6	nil	Aspiration	PSIM	15	5	90	5	Shock	No	Cpap	Extubated
84	Baby of Vasantha	male	Late preterm	2	4	NVD	inborn	7.4	88	6	nil	HMD	PSIM	16	5	70	3	Nil	No	Cpap	Extubated

85	Baby of Jyothi	male	term	3.1	2	NVD	inborn	7.3	78	3	nil	Birth asphyxia	PSIM	17	6	80	2	Nil	No	no	DEATH
86	Baby of Manjuka	female	term	2.9	8	NVD	inborn	7.22	79	7	nil	Congenital pneumonia	PSIM	16	5	100	4	Nil	No	Hfnc	Extubated
87	Baby of Monica	female	Very preterm	1.5	6	NVD	inborn	7.04	89	6	Prom	Apnoea of prematurity	PSIM	16	5	100	2	nil	No	Hfnc	Extubated
88	Baby of Bhavani	female	Very preterm	1.2	7	NVD	inborn	7.1	80	7	nil	Birth asphyxia	PSIM	17	5	50	1	IVH	No	Hfnc	Extubated
89	Baby of Pavithra	female	term	3.2	7	NVD	inborn	7.22	40	6	nil	Birth asphyxia	PSIM	16	5	60	1	shock	Yes	no	DEATH
90	baby of krishna veni	male	term	2.8	6	NVD	inborn	6.97	70	7	nil	Birth asphyxia	PSIM	16	5.5	100	2	Pnuemothorax	No	no	DEATH
91	baby of fiza kouser	male	term	2.7	3	NVD	inborn	6.29	80	3	PROM	Birth asphyxia	PSIM	16	6	70	6	nil	no	Cpap	Extubated
92	baby of samreen khanum	male	Late preterm	2.3	7	AVD	inborn	7.22	60	7	Nil	Birth asphyxia	PSIM	17	5	80	4	Pnuemothorax	No	Hfnc	Extubated
93	baby of ayana	female	Very preterm	1.9	2	NVD	inborn	7.34	88	6	Prom	Apnoea of prematurity	PSIM	18	5	89	4	Nil	No	Hfnc	Extubated
94	baby of lavanya	female	Late preterm	2	7	NVD	inborn	7.2	86	6	nil	Birth asphyxia	PSIM	16	6	88	3	Nil	No	Hfnc	Extubated
95	baby of varalakshmi	female	term	2.17	2	NVD	inborn	7.22	84	2	nil	Birth asphyxia	HFO	15	6	90	3	IVH	No	no	DEATH