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Lalana Newborn Resuscitation

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Abstract

Background: Birth asphyxia is a leading cause of perinatal and neonatal morbidity and mortality with intrapartum factors rather than antepartum or post-partum having a major impact, hence effective resuscitation of newborns takes on priority in saving lives. Newly born resuscitation is unique in that displacement of fetal fluid filled lung requires continuous positive pressure ventilation by sustained nasal oxygen inflation as opposed to intermittent positive pressure ventilation to initiate breathing facilitating vital cardio-vascular changes from fetal to adult life.

Aim: Quick and safe resuscitation of hypoxic/asphyxiated newborns transiting from fetal fluid filled lungs to well aerated neonatal lungs with onset of rhythmic respiration triggering large reduction in Pulmonary Vascular Resistance (PVR), facilitating a series of cardiovascular changes essential for survival after birth.

Method: The study comprised of 1,383 consecutive singleton live births during 14-month period from 1st April 2016 to 31st May 2017, wherein 60% (n=830/1383) deliveries were attended, exclusion criteria 12 twin pregnancies and 10 stillbirths. Resuscitation of hypoxic/asphyxiated newborns involves one to three steps.

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STEP I: Assessment of score zero to +5 by pulse oximetry based on peripheral oxygen saturation.

STEP II: Classification as “Normal” and hypoxic/asphyxiated newborns Graded I-V based on SpO₂, pattern of respiration, rate/min and heart rate (bpm).

STEP III: Lalana Newborn Resuscitation (LNR) Protocol I and II by sustained nasal oxygen inflation, proven both scientifically and physiologically to initiate rhythmic respiration.

Results: Incidence of birth asphyxia was 21.4%, all 178 hypoxic/asphyxiated newborns Graded I-V within 20-60 seconds of birth, were successfully resuscitated by sustained nasal inflatory, oxygen flow at rate of 4-15 Litres/minute directed to baby's nostrils through a wide bore tube for up to 1 to 3 minutes, initiating rhythmic breathing, respiratory rate 30-60/min, heart rate 120-160 beats per minute (bpm) and, SpO₂>96% monitored continuously by Pulse Oximeter.

Conclusion: ‘Lalana Newborn Resuscitation’ (LNR) proved effective in all 178 asphyxiated newborns, Grade I-V by continuous positive pressure ventilation with sustained nasal oxygen inflation at flow rates of 4-15 L/min, commenced rhythmic breathing within 1-3 minutes of birth, successfully resuscitated with respiratory rate 30-60/min, heart rate 120-160 bpm and Zero pulse oximetry score, SpO₂ >96%.

Keywords

Lalana Newborn Resuscitation (LNR); Continuous Positive Pressure Ventilation (CPPV); Sustained Nasal Oxygen Inflation; Pulse Oximetry Score; SpO₂; Grade I to V

Aims for Effective Resuscitation of Hypoxic/Asphyxiated Newborns

- Effective resuscitation of asphyxiated newborns takes on priority in newborns in saving lives as well as without any residual neurological or any other adverse sequelae so children should be normal.
- Unique newborn resuscitation is in transition of fetal fluid filled lungs to well aerated neonatal lungs by safe and quick resuscitation by sustained inflatory nasal oxygen flow proven both scientifically and physiologically to generate hydrostatic pressure gradient between airways and lung tissue to overcome the high resistance of moving fetal lung liquid through the airways and across the alveolar wall into the interstitial tissue to the lymphatic and thence to circulation.

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- Effective resuscitation requires continuous distending pressure that results in uniform recruitment of alveoli with functional residual capacity, preventing alveolar collapse, atelectasis and V/Q mismatch, to achieve optimal gas exchange.
- Prevention of hypoxemia and hypercapnia that result in rise of arterial carbon dioxide causing reduced blood flow to the brain with ischemia resulting in altered mental status and ill effects.
- Stabilizing newborns at birth with low fraction of inspired oxygen (FiO₂) is difficult, as hypoxia is a potent inhibitor of spontaneous respiration, thus higher FiO₂ mitigates hypoxia-induced inhibition of breathing, stimulating the central respiratory center in initiating rhythmic respiration.
- Oxygenation mitigates hypoxia-induced inhibition of breathing and stimulates the central respiratory center to initiate rhythmic respiration, reducing Pulmonary Vascular Resistance (PVR) causing reflex physiological mechanism promoting vital cardiovascular changes adapting to extra-uterine life.
- The primary measure of adequate initial ventilation is the prompt improvement in heart rate is a primary measure of adequate initial ventilation, based on the concept that a low heart rate indicates vagal-induced bradycardia in response to perinatal asphyxia. Pulse oximetry plethysmograph pulsatile waveform indicates cardiac function and pumping of oxygenated blood throughout the body.
- Monitoring of peripheral tissue oxygenation (SpO₂) with Pulse oximeter allows for real time assessment of newborns and classification as 'Normal' or hypoxic/ asphyxia newborns Grade I-V to determine oxygen flow rate within 20-60 seconds. Oxygen discontinued at 96% SpO₂.
- Prevention of lung injury by avoiding potentially harmful Intermittent Positive Pressure Ventilation (IPPV) considered both physiologically and scientifically weak in transition of fetal fluid filled lungs to well aerated neonatal lungs, as entire tidal volume will only enter previously aerated regions due to much lower airway resistance causing overexpansion with intermittent collapse, surrounding atelectasis and V/Q mismatch with right to left shunting, Persistent Pulmonary Hypertension (PPH) thereby perpetuating hypoxia with poor outcome in neonates.

Introduction

Newborn deaths comprise 47% of all under five years deaths, globally declined by half from 5 million in 1990 to 2.4 million in 2019 with about one-third dying on the day of birth and close to three-quarter dying within the first week of life [1-3]. Global incidence of birth asphyxia of 28 per 1000 live births accounts for around 29% ranging from 20-40% neonatal

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deaths with three-fold higher risk of asphyxiated infants dying in the neonatal period compared to non-asphyxiated infants; however the major impact during perinatal period on larger and more mature babies, as 66.1% of term babies suffer HIE, 58.6% being of normal birth weight who have good chances of survival and would otherwise have been healthy [4-11].

Thus neonatal health in the first 28 days of life is the most vulnerable period in the life of a child has now taken on importance with highest neonatal deaths 62% being in Africa and South Asia when compared to 54% under five deaths in developed European and Northern American countries among Sustainable Development Goal (SDG) regions, in fact 98% of all reported perinatal deaths occur in Low and Middle Income Countries (LMICs) [11,12]. Worse still for every newborn baby that dies mainly by birth asphyxia at least another twenty newborn suffer birth injuries etc. Also misclassification of live born, apneic, cyanotic neonate with pulse who die due to non or inadequate resuscitation are labelled as stillbirth is actually a viable newborn as unskilled birth attendants are unable to distinguish between the two, has significant implications on national health policies and global strategies for reducing perinatal mortality, as even 1 in 100 stillbirths if effectively resuscitated will result in more than 30,000 lives that could potentially be saved each year [13,14].

Apnea is defined as cessation of breathing for greater than 20 seconds. Longer hypoxic episode resulting in delayed onset of respiration indicate recent hypoxic cerebral injury and as such defines birth asphyxia or failure to initiate and sustain breathing at birth or within 90 seconds will require immediate and effective resuscitation in the delivery room to reverse the effects of asphyxia and avoid death [15,16]. Birth asphyxia is a potent inhibitor of spontaneous respiration requires immediate and proper resuscitation by sustained positive ventilation with oxygenation at birth to reverse effects of hypoxia and decrease central apnoea to initiate rhythmic breathing [16-21].

Unique resuscitation in the newly born is the transition from fetal lungs containing about 30 ml/kg body weight of fluid consisting of low protein content 25 mg/dl, that differs from both ultra-filtrate of plasma and amniotic fluid [22] to well aerated lungs, requires newborns to make high forceful inspiratory efforts up to 60 cm H₂O at birth usually with the first cry, to overcome the resistance of inspiration of air into the liquid filled lungs [23], stretching the alveolar epithelial pore radius of 0.5 mm impermeable to solute [24] to 3.5 mm in radius [25] that allows flow of fetal alveolar liquid down a protein osmotic pressure gradient into the interstitial tissue and thence absorbed via lymphatic's into the circulation. Subsequently the pores in the alveolar epithelium contract back towards their fetal size [26]. In fact meconium staining of amniotic fluid often results from normal labor contractions with hypoxia or even infection, inhibits fetal lung fluid reabsorption at birth, disturbing the ability of the lungs in vital transition to extra-uterine life [27,28].

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Thus rapid and complex physiological changes take place at birth and the recognition that application of fast pressure to the airways especially in asphyxiated newborns, helps overcome the high resistance of moving liquid through the airways and across the distal airway wall where it is absorbed into the lung interstitial and then into lymphatic, results from generation of hydrostatic pressure gradients between airways and lung tissue [23,26] is based on the scientific principle that a soft steady jet of oxygen to baby's nares, in the form of Positive Pressure Ventilation (PPV) in both breathing and non-breathing neonates is a type of respiratory support that not only allows for recruitment of lung alveoli, but also helps to prevent the collapse of alveoli for effective ventilation and oxygen therapy is the only specific treatment to prevent or mitigate effects of hypoxia, that is key in decreasing central apnea to stimulate respiratory centre promoting a regular pattern of breathing. Thus successful resuscitation requires quick reversal of hypoxia by sustained oxygenation [19-21].

Stabilizing asphyxiated newborns with low fraction of inspired oxygen (FiO_2) is difficult since lack of oxygen cause anaerobic glycolysis with the development of metabolic acidosis due to accumulation of lactic acid. The combination of hypoxia and acidosis impairs cardiac function and increases pulmonary vascular resistance with right to left shunting [16,21]. Thus a higher FiO_2 reduces the risk of hypoxia-induced inhibition of breathing leading to a more stable breathing [20]. As any delay in initiation of breathing can result in devastating consequences in the newborn causing damage to heart, brain and other organs sometimes even death [29].

Neonatal Resuscitation Program (NRP) introduced world-wide by the American Academy of Paediatrics and American Heart Association lacks scientific clarity regarding the physiology of transition fluid filled fetal lungs to well aerated neonatal lungs is the major contributing factor [30-32]. The attempt to inflate the lungs through the application of short intermittent bursts of air/oxygen, with Intermittent Positive Pressure Ventilation (IPPV) is considered scientifically weak and potentially harmful with regard to shifting of fetal lung fluid, since entire tidal volume will only enter previously aerated regions due to the much lower airway resistance causing overexpansion with intermittent collapse of alveoli with uneven alveolar ventilation during each single breath of oxygen, predisposes to lung injury and permits lung fluid re-entering the airways with little further lung aeration, resulting in ventilation perfusion mismatch, shunting of deoxygenated blood through un-expanded lung with right to left cardiac shunt through foramen ovale and ductus arteriosus, thus perpetuating hypoxia with resultant Persistent Pulmonary Hypertension (PPH), bradycardia with reduced cardiac output and poor peripheral tissue oxygenation [29-33].

In contrast sustained oxygen inflation at 5 cm H_2O pressure up to maximum 20 cm H_2O pressure with flow rates of 8-25 litres/min that extends over 1 to 3 minutes allows for uniform alveolar recruitment, achieving better post manoeuvre lung mechanics consistent with the

concept that lung aeration is a function of applying an elevated pressure over an extended period resulting in significant improvement of oxygen saturation [19-21]. However despite significant ($p<0.01$) decline in neonatal asphyxial deaths with NRP [30, 34,35], the degree of morbidity remains high in 25% up to 40% of survivors affecting quality of life who may suffer from permanent neurological deficits such as cerebral palsy, mental retardation, seizures, blindness or severe hearing impairment, to mild effects of autism, cognitive impairment, inability to develop fine motor skills, memory and mood disturbances depending on the extent of insult showing no decline in incidence of meconium aspiration syndrome or seizures due to HIE with no overall decrease in neonatal mortality [29,36-42], however trend in western developed countries with low perinatal mortality rates report low rates of asphyxia in about 2/1000 births with resultant mortality rate around 10% in NICU with 15% cerebral palsy rate among survivors [37,43]. As mild perinatal hypoxia occurs more frequently than severe events, there is substantial long-term effect on the population with a larger proportion of adults at increased risk of low IQ score < 80 and poor scholastic performance [38,39].

Intrapartum-related complication is one of the most common causes of neonatal death; all foetuses do experience some degree of hypoxia during labor contractions due to placental insufficiency [44]. Thus intrapartum events have more impact than antepartum factors more so in most developing Asian countries due to higher incidence of serious complications in labour with reduced availability of skilled care during delivery wherein majority of world population reside. Thus preventive strategies should be aimed at intrapartum period rather than antepartum or post-partum period in achieving substantial reduction of early neonatal deaths which will also impact reducing intrapartum stillbirths. Perinatal asphyxia is estimated to be the fifth largest causes of under-five child mortality after pneumonia, diarrhoea, neonatal infections and complications of preterm births, is in reality much higher as non-breathing viable newborns, termed as stillbirth are left without resuscitative efforts at birth in fact are actually early neonatal deaths [1,2,13,14].

Thus in spite of concerted global and national efforts to improve child mortality in the post neonatal phase with key child health interventions such as oral rehydration therapy, care seeking for acute respiratory infections and improved immunization rates has however resulted in neonatal mortality gradually increasing as a percentage of total under-five child mortality with less attention being given to determinants of perinatal and neonatal mortality despite new found focus on neonatal health with the annual rate of reduction in Neonatal Mortality Rate (NMR) and Early Neonatal Mortality Rate (ENMR) still lagging behind Infant Mortality Rate (IMR) and Under Five Mortality Rate (U5MR) with resultant slow decline in perinatal mortality rates [45-47].

Many parts of low income Asian and African countries with limited resource, still lack skilled birth attendants and well outfitted resuscitation teams as even essential resuscitation

equipment such as bulb syringes, bag and mask devices etc. may be substandard or unavailable emphasizing that ethnic Asian babies deserve better. Though problems in perinatal and neonatal phases have been reported in India, little progress has been made towards implementing large-scale effective solutions and interventions, as even essential newborn care and their implementation still has not resulted in a rapid reduction in perinatal and neonatal mortality rates [30,31,45-47]. Effective resuscitation is the need of the hour that will prove to be the single most significant strategy in reducing perinatal and neonatal mortality and morbidity as well as under-five years mortality rate, presently further compounded by the negative impact of COVID-19 pandemic with hundreds of thousands more fatalities expected due to lack of medical facilities more so in Asian countries [48].

Sustained nasal oxygen inflation rather than IPPV by bag and mask or endotracheal intubation is the basis for effective resuscitation resulting in quick initiation of rhythmic respiration, saving lives, improving outcome with decrease of adverse life-long ill sequelae [21]. Around 200 million children throughout the world majority in Asian countries do not accomplished their age appropriate development due to birth asphyxia [37]. Thus the neonatal period or the first 28 days is the most vulnerable period in the life of a child especially in the first one week, more so within the first 24 hours, having the highest risk of mortality per day than any other period during childhood.

Problem Definition

1. Unique resuscitation in newly borns requires continuous distending pressure to remove fetal lung fluid for generation of hydrostatic pressure gradient between airways and lung tissue to overcome the high resistance of moving fetal lung liquid through the airways and across the alveolar wall into the interstitial tissue for well aerated neonatal lungs.
2. Sustained oxygenation counteracts hypoxia and stimulates the central respiratory center to initiate respiration facilitating smooth physiological cardiovascular transition from fetal to neonatal circulation.
3. Enable real time continuous assessment of the newborn status by SpO₂ and heart rate by Pulse oximetry.
4. Avoidance of harmful Intermittent Positive Pressure Ventilation (IPPV) that predisposes to lung injury and inadequate ventilation considered scientifically and physiologically weak in newly born resuscitation that is best suited by anesthetists for cardiorespiratory arrest in infants, children and adults with previously well aerated lungs.

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Materials and Methods

The study comprised 1,383 consecutive singleton live births during 14-month period from 1st April 2016 to 31st May 2017 at Shifa Hospital, a multispecialty centre in the metropolitan city of Bangalore. I attended 830 (60%) deliveries including vaginal deliveries both vertex and breech presentation, instrumental-vacuum and low/outlet forceps deliveries as well as surgical Lower Segment Caesarean Sections (LSCS) both Elective and Emergency surgery. Sources of data were Labor room records, neonatal charts and NICU register. Data was entered into EPI data version 3.1 and then exported to SPSS Version 21 for analysis and statistical significance, the threshold of significance was set at 0.05.

A sample pilot study was carried out on 30 newborns with the new innovative resuscitation technique using sustained nasal oxygen inflation at flow rates 2-15 Litres/minute (L/min) proved eminently successful with uneventful observation in NICU, who were then shifted to mother's side for initiation of early breast feeding. A study was then undertaken on 830 newborns whose delivery I attended during the 14 month study period.

The status of all newborns are preferably assessed within 20-60 seconds after birth i.e. following complete expulsion of newborn, with immediate clamping and cutting of umbilical cord in hypoxic newborns or may be delayed 1-3 minutes in spontaneously breathing newborns. The Pulse Oximeter is placed across the foot of newborn to monitor peripheral tissue oxygen saturation (SpO₂), heart rate, plethysmograph waveform noting adequate cardiac output, the pattern of breathing observed or respiratory rate derived from photoplethysmogram (PPG).

Lalana Newborn Resuscitation (LNR) proves ideal and consists of three steps:-

STEP I: Pulse oximetry score zero to +5 based on peripheral oxygen saturation (SpO₂).

STEP II Step 2: Classification as "Normal", healthy newborns with spontaneous onset of rhythmic respiration while hypoxic/ asphyxiated newborns Graded I-V based on SpO₂, pattern of breathing and heart rate.

STEP III : includes Protocols I and II, application of continuous positive pressure ventilation by sustained nasal oxygen inflation at flow rates 2-15 L/min determined by Pulse oximetry score, SpO₂, pattern of breathing and heart rate for upto 1-3 minutes or till onset of rhythmic respiration, SpO₂ >96% and heart rate >120 bpm.

Step-1

Newborn Pulse Oximetry Score

Pulse oximeter automatically provides an estimate of newborn health status within seconds. Zero score Pulse Oximetry score SpO₂ 96%-100% indicates 'Normal' healthy newborns, +1 Pulse Oximetry score, SpO₂ 94%-95%, mild birth asphyxia, +2 Pulse Oximetry score, SpO₂ 92%-93%, moderate birth asphyxia, +3 Pulse Oximetry score, SpO₂ 90%-91%, severe birth asphyxia, +4 Pulse Oximetry score, SpO₂ 89%-50%, Secondary apnea with absent breathing and +5 Pulse Oximetry score Terminal apnea with absent breathing or 'flat baby'. Zero to +5 Pulse Oximetry score based on SpO₂, for all newborns is seen in Table 1.

Score	SpO ₂ Pulse Oximeter Reading	Newborn Status
Zero	96%-100%	Normal / Healthy
1	94%-95%	Mild Asphyxia
2	92%-93%	Moderate Asphyxia
3	90%-91%	Severe Asphyxia
4	89%-50%	Secondary Apnea
5	<50%	Terminal Apnea

Table 1: Newborn pulse oximetry scoring for SpO₂.

Step-II

Classification of Newborns as Normal, Healthy or Hypoxic/Asphyxia Grade I to V

The status of all newborns assessed within 20-60 seconds of birth classified as Normal, healthy newborns or hypoxic/asphyxiated newborns further graded into I-V based on Pulse Oximetry Score, SpO₂, breathing pattern and heart rate. Normal, healthy newborns, have Zero Pulse oximetry Score SpO₂ 96%-100%, with spontaneous onset of rhythmic breathing, respiratory rate 30-60/min and heart rate 120-160 bpm.

Mild birth asphyxia, Grade I newborns +1 Pulse oximetry Score, SpO₂ 94% -95%, with regular/irregular breathing pattern, respiratory rate $\pm$ 20-30/ min and heart rate 100-119 bpm. Moderate birth asphyxia, Grade II newborns +2 Pulse oximetry Score, SpO₂ 92-93%, with irregular breathing, respiratory rate $\pm$ 15-20/min and heart rate 100-80 bpm. Severe birth asphyxia, Grade III newborns +3 Pulse oximetry Score, SpO₂ 90-91%, irregular or gasping

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breathing, respiratory rate 10-15/min and heart rate 80-60 bpm. Secondary apnea Grade IV newborns +4 Pulse oximetry Score, SpO₂ 89%-50%, absent respiration and heart rate 60-35 bpm, while Terminal apnea Grade V newborns +5 Pulse oximetry Score, SpO₂ <50%, absent respiration and heart rate < 35 bpm is also referred to as “flat baby”.

However grading of hypoxic newborns into Grade I-V by criteria of +1 to +5 pulse oximetry score, SpO₂ <96%, respiratory rate <30/ min and heart rate <120 bpm, may vary as real time assessment of newborn's SpO₂ is constantly changing due to continuous monitoring. The classification of newborns at birth based on Pulse oximetry score, SpO₂, respiratory rate and heart rate is seen in Table 2.

Normal Healthy Newborn	Grade I Mild Asphyxia	Grade II Moderate Asphyxia	Grade III Severe Asphyxia	Grade IV Secondary Apnea	Grade V Terminal Apnea
Zero Pulse oximetry Score - SpO ₂ 96% - 100% Spontaneous onset respiration, rate 30- 60/min. Normal Heart Rate 120-160 bpm.	+1 Pulse oximetry Score - SpO ₂ 94% - 95% Regular/ irregular Respiration, rate 20-30/min Heart Rate 100- 119 bpm.	+2 Pulse oximetry Score - SpO ₂ 92-93% Regular/ irregular Respiration, rate 15-20/min Heart Rate 100 - 80 bpm.	+3 Pulse oximetry Score - SpO ₂ 90-91% Irregular /gaspings Respiration, rate 15-20/min Heart Rate 80- 60 bpm.	+4 Pulse oximetry Score - SpO ₂ 89%- 50% Absent respiration Heart Rate 60-40 bpm.	+5 Pulse oximetry Score - SpO ₂ <50% Absent respiration 'Flat baby' Heart Rate <40 bpm.

Table 2: Classification with grading of newborns within 20-60 seconds of birth

Step-III

Effective aeration of the newly born's lung is a function of applying an elevated pressure over sustained periods of 1-3 minutes that results in significant improvement of lung mechanics in efficiently overcoming the high resistance of moving liquid from lungs to interstitial with uniform lung aeration and prompt increase in heart rate monitored continuously by pulse oximetry with circulation of oxygenated blood throughout the body.

Newborns timed at birth at complete expulsion of fetus, the umbilical cord ligated and cut immediately in hypoxic/asphyxiated newborns, which may be delayed upto 1-3 minutes in “Normal” newborns with spontaneous onset of respiration. The pulse oximeter is placed on the baby's foot to monitor superficial oxygen saturation (SpO₂), heart rate and the

plethysmograph tracing records how well heart is pumping oxygenated blood throughout the body indicated by pulsatile changes. All newborns are classified as 'Normal' or hypoxic who are further Graded I to V, based on pulse oximeter score +1 to +5 respectively, SpO₂, pattern of respiration and heart rate within 20-60 seconds of birth.

Majority are 'Normal' Healthy newborns, active, pink in color and vigorous cry initiate spontaneous rhythmic respiration within 20-60 seconds of birth, Zero Pulse oximetry Score, SpO₂ >96% respiratory rate 30-60/min and heart rate of 120-160 bpm. Routine newborn care based on regional NICU protocol, baby wiped dry under radiant warmer to prevent hypothermia, nasal and oral suction maintaining asepsis, stomach wash after LSCS, Vitamin K 0.5-1 mg IM. Baby wrapped in warm clothing and cap, transferred to mother's side and breast feeding initiated within one hour of normal delivery and four hour after LSCS.

Lalana Newborn Resuscitation (LNR) for hypoxic newborns are further graded into Grade I - V based on +1 to +5 Pulse oximetry score, peripheral oxygen saturation (SpO₂) <95%, pattern of breathing and heart rate. Mild birth asphyxia, Grade I newborns, +1 Pulse oximetry Score, SpO₂ 94%-95% with regular/irregular respiratory rate $\pm$ 20-30/ min and heart rate 100-119 bpm, require resuscitation by sustained nasal oxygen inflation at flow rate 2-4 L/min (FiO₂ 28% to 36%), upto 60 seconds till rhythmic pattern of respiration, oxygen is discontinued with normal pulse oximeter reading SpO₂ >96%. If however SpO₂ decreases to <94% oxygen flow rate is increased to 5-8 L/min or more (FiO₂ up to 50%), for upto 1-3 minutes till Zero Pulse oximetry Score, SpO₂ >96%, and heart rate >120 bpm.

Moderate birth asphyxia, Grade II newborns have +2 Pulse oximetry Score, SpO₂ 92%-93%, regular/ Irregular respiration, rate $\pm$ 15-20/min and heart rate 100 - 80 bpm, resuscitated by sustained nasal oxygen inflation at flow rate of 5-8 L/min, (FiO₂ 40% to 52%) for upto 60-90 seconds till onset of rhythmic respiration, oxygen is discontinued with normal pulse oximeter reading SpO₂ >96% % and heart rate >120 bpm.

Severe birth asphyxia Grade III, +3 Pulse oximetry Score, SpO₂ 90%-91%, irregular/gasping respiration, rate $\pm$ 10-15/min and heart rate 80-60 bpm, resuscitation by sustained nasal oxygen inflation at flow rate of 10-12 L/min, (FiO₂ 56% to 64%) applied for 90-120 seconds till onset of rhythmic respiration, oxygen is then discontinued with normal pulse oximeter reading SpO₂ >96 and heart rate >120 bpm.

Secondary apnea Grade IV, +4 Pulse oximetry Score, SpO₂ 89%-50%, absent respiration and heart rate 60-40 bpm, ventilation by sustained nasal oxygen inflation at flow rate of 12-14 L/min, (FiO₂ 64% to 76%) up to 120-180 seconds till onset of rhythmic respiration, rate 30-60/min and heart rate >120 bpm, oxygen is discontinued with normal pulse oximeter reading SpO₂ >96%. However if heart rate is <45 bpm, endotracheal intubation undertaken with continuous distending pressure ventilation by sustained oxygen at flow rate of 15 L/min

(FiO₂ 100%) for 120-240 seconds or more till onset of breathing and heart rate increases to > 100bpm extubate and continue with nasal oxygen inflation flow rate of 10-12 L/min (FiO₂ 60-68%).

Grade IV newborns are usually subjected to severe perinatal asphyxia and are at greater risk of intrauterine (stillbirth) or early neonatal death often delivered by judicious quick obstetric intervention by emergency LSCS, may be effectively resuscitated by sustained nasal oxygen with tactile stimuli, back rubs, nasal and oral suctioning till onset of rhythmic respiration. Oxygen discontinued at Zero pulse oximetry score, rhythmic pattern of respiration, rate 30-60/min SpO₂ >96% and heart rate >120 bpm.

Terminal apnea, Grade V newborns +5 Pulse oximetry score, SpO₂ <50%, with absent respiration and heart rate <40 bpm 'flat baby' may respond to continuous distending pressure ventilation by sustained nasal oxygen inflation up to 15 L/min (FiO₂ 64% to 76%) for 120-240 seconds or more till onset of rhythmic respiration or if no recording on pulse oximeter of SpO₂ or heart rate, immediately intubate with endotracheal tube and resuscitate by positive airway distending pressure with oxygen flow at rate of 15 L/min upto 25 L/min (FiO₂ 100%), if no breathing or heart beat within 60 seconds give cardiac compression about 120/min with medication of epinephrine, volume expander 5% dextrose saline at 10 ml/kg and Carbicarb 2.5 meq/kg, a mixture of Na₂CO₃/NaHCO₃ which does not generate CO₂ slow infusion over one hour. If heart rate increases > 100 bpm with onset of breathing, shift to sustained nasal oxygen inflation up to 12 L/min (FiO₂ 64%) for 60-120 seconds and oxygen discontinued with rhythmic breathing, rate 30-60/min, normal SpO₂ >96% and heart rate >120 bpm.

However, Grade V newborns are at high risk of intrauterine death (fresh stillbirth) or if born with signs of life who are often un-responsive to resuscitative intervention and dying shortly after birth termed early neonatal deaths, are usually delivered by emergency LSCS, which impacts the more mature, larger newborns with birthweight around 3500-4000 g, at 39 weeks or later, most of whom on survival may be associated with high incidence of HIE and serious neurological sequelae with lifelong disability, cerebral palsy etc.

Hence it is recommend that resuscitative efforts may be aborted if neonate unresponsive after three to five upto ten minutes of resuscitation with sustained nasal oxygen inflation at 15 L/min (FiO₂ 76%) or through endotracheal tube FiO₂ (100%).

Preterm <32 weeks and <1250 g with incidence of 1% among 830 deliveries attended comprised a total of nine preterms, VLBW five had gestation of 28-32 weeks and four being less than 28 weeks gestation. Three preterm weighed <1000 g and remaining six between 1000-1250 g. Five delivered normally and three by emergency LSCS and one preterm 32 weeks gestation, weighing 1180 g born by assisted breech delivery had placenta previa.

Of the two preterm neonatal deaths, one preterm <28 weeks gestation weighing 740 g died on 2nd post-natal day due to extreme immaturity and RDS. Another preterm <28 weeks weighing 960 g had obstetric complications of MSAF and PROM, died on 3rd post-natal day of RDS and sepsis, the remaining seven preterm VLBWs survived. However one preterm 28 weeks gestation and 1010 g birthweight had polyhydramnios with duodenal atresia was surgically corrected at a referral hospital.

LNR Protocol II for resuscitation of preterm newborns, gestational age <32 weeks and birthweight <1250 g by CPPV by sustained nasal oxygen inflation at flow rates of 2 L/min up to 12 L/min (FiO₂ 28-60%), through wide bore oxygen tube or nasal prongs till heart rate improves and regular respiration established following which bubble CPAP with blender and FiO₂<30% through nasal prongs started, to maintain oxygen saturation around 96%, if less than 28 weeks gestation the SpO₂ maintained between 95-96%, the preterm is then transferred to NICU for observation and further management/treatment of complications.

Preterm with secondary or terminal apnea requires endotracheal intubation with sustained oxygen flow at rate of 12 L/min (FiO₂ 100%), administered for 2-3 minutes or more till rhythmic breathing established and heart rate >120 bpm followed by CPAP (FiO₂<30%) through nasal prongs and baby shifted to NICU for observation and management/treatment of any complications. Extremely preterms are more prone to birth asphyxia as opposed to term babies and extremely immature preterms are at greater risk of cerebral palsy, delay in development, hearing and sight problems, hence it is recommend that resuscitative efforts may be aborted in terminal apnea. Delivery room use of sustained nasal oxygen inflation during resuscitation triggers physiological transition of fetal fluid filled lungs to well ventilated neonatal lungs initiating rhythmic breathing.

In spontaneously breathing preterm <32 weeks and <1250 g birth weight, Continuous Positive Airway Pressure (CPAP) with blender and FiO₂<30%,with oxygen bubbling through 5 cm water in a bottle producing small airway pressure oscillations which on reaching the newborn's lung results in improved gas exchange and lung function. Preterm <32 weeks and VLBW <1250 g require CPAP (FiO₂<30%) to maintain oxygen saturation around 95-96%, extremely preterm <28 weeks gestation, SpO₂ maintained between 94-96%. The present study all 178 hypoxic newborns Grade I-V were effectively resuscitated by Lalana Newborn Resuscitation (LNR) outlined in Protocol I and Protocol II resulted in the wellbeing of neonates with normal pulse rate, respiratory rate and oxygen saturation during observation in NICU, blood glucose, CBC, serum electrolytes checked as well as blood gas study for acid base imbalance with temperature control to prevent hypothermia and minimize oxygen consumption and shifted to mother's side for initiating early breast feeding with uneventful observation for four hours in NICU. However two extremely preterm <28 weeks gestation expired on second and third day of life weighing 740 g and 960g respectively. One preterm

with 28 weeks gestation, birthweight 1010 g was surgically treated for congenital duodenal atresia.

Lalana Newborn Resuscitation (LNR) provides for quick and safe resuscitation by application of continuous positive airway pressure by sustained nasal oxygen inflation monitored by Pulse oximetry SpO_2 in determining oxygen flow rates of 2 L/min up to 15 L/min (FiO_2 28%-76%), blown to the baby's nostrils through wide bore oxygen tube for up to 60-180 seconds to initiate rhythmic respiration, $SpO_2 > 96\%$ and heart rate 120-160 bpm. Higher FiO_2 stabilizes hypoxic newborns at birth, reducing risk of hypoxia-induced inhibition of breathing and leading to a more stable breathing pattern with better aeration of the lung and increased lung volume and pulmonary compliance with Functional Residual Capacity (FRC), facilitating reflex cardiovascular changes compatible with adult life.

The primary measure of adequate initial ventilation is the prompt improvement of heart rate and adequate cardiac output noted on plethysmograph wave form on pulse oximeter with circulation of oxygenated blood throughout the body resulting in aerobic cellular metabolism and removal of lactic acid, mitigating hypoxia with minimal ill effects, decreasing morbidity and mortality associated with hypoxic ischemic tissue (brain, heart, gut and kidney) injury with untoward long-term sequel. Thus sustained oxygen nasal inflation positively impacts postnatal adaptation of newborns so children should be normal.

Lalana Newborn Resuscitation - Methodology

Protocol I

Lalana Newborn Resuscitation (LNR) Protocol I, provides quick and safe resuscitation by sustained nasal oxygen inflation oxygen flow rates 2 L/min up to 15 L/min (FiO_2 28% to 76%) determined by Pulse oximetry score (L/min-Litres per minute, HR / beats per minute-Heart Rate/bpm, ET- Endotracheal Tube) (Table 3).

Classification	Protocol I
'Normal' Healthy Newborn	Zero Pulse oximetry Score - SpO_2 96% - 100%, spontaneous onset of rhythmic breathing, rate 30-60/min and $HR \pm 120-160$ bpm. Routine newborn care maintaining asepsis and thermo-control. Shift newborn to mother's side and initiate early nutrition, by breast-feeding within an hour in normal delivery and four hours after LSCS.

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<p>Grade I Mild Asphyxia</p>	<p>+1 Pulse oximetry Score - SpO₂ >96%, regular/ irregular respiration, $\pm$ rate 20-30/min and HR $\pm$100-119 bpm. Sustained nasal oxygen at flow rates 2-4 L/min (FiO₂ 28% to 36%), directed towards the nostrils through the wide bore tube for up to 60 seconds. Newborn wiped dry under radiant warmer with tactile stimuli, nasal and oral suction.</p> <p>Discontinue oxygen with Zero Pulse oximetry Score SpO₂> 96%, with rhythmic breathing pattern, rate 30-60/min and HR 120-160 bpm.</p> <p>Shift newborn to NICU for observation for 4 hours and then to mother's side and institute breast-feeding.</p>
<p>Grade II Moderate Asphyxia</p>	<p>+2 Pulse oximetry Score - SpO₂ 92-93%, irregular/ respiration $\pm$ rate 15-20/min and HR $\pm$100-80 bpm. Sustained nasal oxygen flow rate 5-8 L/min (FiO₂ 40% to 52%), directed towards the nostrils through the wide bore tube for up to 60-90 seconds. Neonate wiped dry under radiant warmer with tactile stimuli, nasal and oral suction.</p> <p>Discontinue oxygen with Zero Pulse oximetry Score SpO₂> 96 with rhythmic breathing pattern, respiratory rate 30-60/min and HR >120 bpm.</p> <p>Shift to NICU for observation for 24 hours or management and treatment of complications if any.</p>
<p>Grade III Severe Asphyxia</p>	<p>+3 Pulse oximetry Score - SpO₂ 90-91%, gasping respiration, $\pm$ rate 10-15/min and HR $\pm$ 80-60 bpm. Sustained nasal oxygen flow rate 8-12 L/min (FiO₂ 52% to 64%) directed towards the nostrils through the wide bore tube for up to 90-120 seconds or more. Neonate wiped dry under radiant warmer with tactile stimuli, back rubs, nasal and oral suction.</p> <p>Discontinue oxygen with Zero Pulse oximetry Score SpO₂>96% with rhythmic breathing pattern, respiratory rate 30-60/min and HR >120 bpm.</p> <p>Shift newborn to NICU for observation, management and treatment of complications if any.</p>

<p>Grade IV Secondary Apnea</p>	<p>+4 Pulse oximetry Score - SpO₂ 89%-50%, absent respiration and HR $\pm$60-40 bpm. Sustained nasal oxygen flow rate 12-15 L/min (FiO₂ 64% to 76%), directed towards the nostrils through the wide bore tube for up to 120-180 seconds or more. Neonate wiped dry under radiant warmer with tactile stimuli, back rubs, nasal and oral suction.</p> <p>Discontinue oxygen with Zero Pulse oximetry Score, SpO₂>96%, with rhythmic breathing pattern, respiratory rate 30-60/min and HR >120 bpm.</p> <p>Shift newborn to NICU for observation, management and treatment of complications if any.</p>
<p>Grade V Terminal Apnea</p>	<p>+5 Pulse oximetry Score - SpO₂ <50%, absent respiration and HR <40 bpm, 'Flat baby' or no recording on pulse oximeter of SpO₂ and heart rate and occasional heart beats on auscultation, immediately intubate with endotracheal tube and ventilate with continuous distending airway pressure by oxygen flow at rate of 15 L/min (FiO₂ 100%) for 120-240 seconds or more, if no onset of breathing within 60 seconds start cardiac compression around 120/min and medications of epinephrine or volume expanders, 5% dextrose saline at 10 ml/kg also combats hypoglycemia and Carbicarb 2.5 meq/kg (a mixture of Na₂CO₃/NaHCO₃) but without the generation of CO₂ slow infusion over one hour to combat metabolic lactic acidosis secondary to hypoxemia and cardio pulmonary disturbances or Sodium bicarbonate if newborn is breathing diluted at 8 ml eq/kg.</p> <p>Onset of rhythmic respiration and heart rate increases to >100 bpm, remove ET tube and continue with sustained nasal oxygen inflation at flow rate 12-15 L/min (FiO₂ 64% to 76%) directed towards the nostrils through the wide bore tube till onset of rhythmic breathing under radiant warmer with tactile stimuli, back rubs, nasal and oral suction.</p> <p>Discontinue oxygen with Zero Pulse oximetry Score, SpO₂>96%, rhythmic breathing pattern, respiratory rate 30-60/min and HR >120 bpm.</p> <p>Shift newborn to NICU for observation, management and treatments of any complications.</p> <p style="text-align: center;">OR</p> <p>Abort resuscitation after 5-10 minutes if unresponsive either fresh stillbirth or early neonatal death.</p>

Table 3: Classification of newborns within 20-60 seconds of birth

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Protocol II

Lalana Newborn Resuscitation (LNR) - Protocol II - Preterm newborns with gestational age <32 weeks and birthweight <1250 g followed by bubble CPAP with blender and $\text{FiO}_2 < 30\%$ through nasal prongs to maintain SpO_2 around 95-96%, (Litres per minute - L/min, Heart Rate / beats per minute - HR /bpm, Endotracheal Tube - ET) (Table 4).

Classification	Protocol II
Normal Healthy Preterm	<p>Zero Pulse oximetry Score - $\text{SpO}_2 > 96\%$ with onset of with rhythmic breathing pattern, respiratory rate 30-60/min, HR 120-160 bpm. Routine newborn care given.</p> <p>Start Bubble CPAP at 5 cm H_2O, $\text{FiO}_2 < 30\%$ with blender through nasal prongs or Oxygen Hood with oxygen flow rate to maintain SpO_2 at 95-96%. Shift to NICU for observation, management and treatment of complications if any.</p>
Grade I Mild Asphyxia	<p>+1 Pulse oximetry Score - SpO_2 94% - 95%, regular /irregular breathing, respiratory rate $\pm$ 20-30/min, HR $\pm$ 100-120 bpm. Sustained nasal oxygen at flow rate at 2-4 L/min (FiO_2 28% to 36%), directed towards the nostrils through the wide bore tube for up to 60 seconds, gently wipe dry with tactile stimuli, nasal and oral suction under radiant warmer till $\text{SpO}_2 \sim 96\%$, with rhythmic breathing pattern, respiratory rate 30-60/min and Heart Rate 120-160 bpm.</p> <p>Discontinue oxygen flow when $\text{SpO}_2 \sim 96\%$, with rhythmic breathing pattern, respiratory rate 30-60/min and Heart Rate > 120 bpm.</p> <p>Start Bubble CPAP at 5 cm H_2O, $\text{FiO}_2 < 30\%$ with blender through nasal prongs or Oxygen Hood with oxygen flow rate to maintain SpO_2 between 95-96%. Shift to NICU for observation, management and treatment of complications if any.</p>

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Grade II Moderate Asphyxia	<p>+2 Pulse oximetry Score - SpO₂ 92-93%, regular/ irregular breathing, respiratory rate $\pm$ 15-20/min and HR $\pm$ 100-80 bpm, Sustained nasal oxygen flow rate 4-6 L/min (FiO₂ 36% to 44%), directed towards the nostrils through the wide bore tube for up to 60-90 seconds, gently wipe dry with tactile stimuli, nasal and oral suction under radiant warmer. Discontinue oxygen flow when SpO₂ ~96%, with rhythmic breathing pattern, respiratory rate 30-60/min and Heart Rate >120 bpm.</p> <p>Start Bubble CPAP at 5 cm H₂O, FiO₂<30% with blender through nasal prongs or Oxygen Hood with oxygen flow rate to maintain SpO₂ between 95-96%. Shift to NICU for observation, management and treatment of complications if any.</p>
Grade III Severe Asphyxia	<p>+3 Pulse oximetry Score - SpO₂ 90-91%, irregular/gasping breathing, respiratory rate $\pm$ 10-15/min and Heart Rate $\pm$ 80 - 60 bpm. Sustained nasal oxygen flow rate 6 -8 L/min (FiO₂ 44% to 52%) directed towards the nostrils through the wide bore tube for up to 90-120 seconds, gently wipe dry with tactile stimuli, nasal and oral suction under radiant warmer.</p> <p>Discontinue oxygen flow when SpO₂ ~96%, with rhythmic breathing pattern, respiratory rate 30-60/min and Heart Rate >120 bpm.</p> <p>Start Bubble CPAP at 5 cm H₂O, FiO₂<30% with blender through nasal prongs or Oxygen Hood with oxygen flow rate to maintain SpO₂ 95-96%. Shift to NICU for observation, management and treatment of complications if any.</p>
Grade IV Secondary Apnea	<p>+4 Pulse oximetry Score - SpO₂ 89%-50%, absent respiration and HR 60-35 bpm. Sustained nasal oxygen flow rate of 8 - 10 L/min (FiO₂ 52% to 60%), directed towards the nostrils through the wide bore tube for up to 120-180 seconds or more, gently wipe dry with tactile stimuli, back rub, nasal and oral suction under radiant warmer.</p> <p>Discontinue oxygen flow when Pulse oximetry SpO₂ ~ 96%, with rhythmic breathing pattern, respiratory rate 30-60/min and Heart Rate >120 bpm.</p> <p>Start Bubble CPAP at 5 cm H₂O, FiO₂<30% with blender through nasal prongs or Oxygen Hood with oxygen flow rate to maintain SpO₂ between 95-96%.</p> <p>Shift to NICU for observation, management and treatment of complications if any.</p>

Grade V Terminal Apnea	<p>+5 Pulse oximetry Score - SpO₂ <50%, absent respiration and HR <35 bpm 'Flat baby' or no reading on pulse oximeter of SpO₂ and heart rate, with occasional heart beats on auscultation, immediately intubate with endotracheal tube and ventilate with continuous distending airway pressure by oxygen flow at rate of 12-15 L/min (FiO₂ 100%) if no breathing attempts by 60 seconds start cardiac compression around 120/min and medications of epinephrine or volume expanders, 5% dextrose saline at 10 ml/kg and start Carbicarb 2.5 meq/kg, a mixture of Na₂CO₃/NaHCO₃ safer with no generation of CO₂ slow infusion over one hour that does not affect cerebral blood flow.</p> <p>If onset of rhythmic respiration and heart rate increases to >100 bpm, remove ET tube and continue with sustained nasal oxygen inflation at flow rates of 4-6 L/min (FiO₂ 36% -44%), directed towards the nostrils through the wide bore with tactile stimuli, back rubs, nasal and oral gently wipe dry with tactile stimuli, back rub, nasal and oral suction under radiant warmer.</p> <p>Discontinue oxygen flow when SpO₂ ~ 96%, rhythmic breathing pattern, respiratory rate 30-60/min and HR>120 bpm.</p> <p>Start Bubble CPAP at 5 cm H₂O, FiO₂<30% through nasal prongs or Oxygen Hood with oxygen flow rate to maintain SpO₂ between 95-96%. Shift to NICU for observation and management / treatment of complications.</p> <p style="text-align: center;">OR</p> <p>Resuscitative efforts may be aborted after 5-10 minutes in the event the preterm is unresponsive due high incidence of neurological, deficits cerebral palsy etc. or other organ deficits and untoward ill life-long sequelae.</p>
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Table 4: Classification of preterm <32 weeks and <1250 g within 20-60 seconds of birth.

Results

The incidence of birth asphyxia was 21.4% among 1,383 consecutive singleton live births, during 14-month period from 1st April 2016 to 31st May 2017, I attended 60% (n=830/1383) deliveries, including vaginal deliveries both vertex and breech presentation, instrumental-vacuum and low/outlet forceps deliveries and surgical Lower Segment Caesarean Sections (LSCS) both emergency and elective.

All newborns were assessed and classified within 20-60 seconds of birth as normal, healthy neonates or hypoxic/asphyxia Grade I-V who were all, successfully resuscitated utilizing continuous positive pressure ventilation by sustained nasal oxygen inflation at varying flow

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rates of 2-15 L/min for upto 1-3 minutes or more based on pulse oximetry readings. Among 830 deliveries attended, a majority 78.5% (n=652/830) were 'Normal', healthy newborns with zero Pulse oximetry score, SpO₂ 96%-100%, heart rate 120-160 bpm with spontaneous onset of rhythmic respiratory rate of 30-60/min. There was a high incidence 21.4% (n=178/830) of birth asphyxia. The distribution of 830 deliveries attended revealed 78.5% Normal, healthy newborns and 21.4% hypoxic/asphyxiated newborns Grade I to V is seen in Table 5.

Distribution	Live Births	Deliveries Attended	Normal Healthy Newborns	Birth asphyxia Grade I to V
Number	1,383	830/1383	652/830	178/830
Percentage	100%	60.0%	78.5%	21.4%

Table 5: Distribution of live births

Majority 78.5% (n=653/830) of births attended, were normal, healthy newborns who established spontaneous rhythmic respiration at birth, while 15.1% (n=126/830) Grade I suffered from mild asphyxia, and 3.7% (n=31/830) Grade II had moderate asphyxia, only 2.1% (n=17/830) Grade III newborns had severe asphyxia while 0.4% (n=3/830) Grade IV had secondary apnea. Just 0.1% (n=1/830) Grade V newborn had terminal apnea. Sex distribution revealed five females to four males. The distribution of 830 births attended according to Grade I-V is seen in Table 6.

Distribution	Normal Healthy Newborns	Grade I Mild Asphyxia	Grade II Moderate Asphyxia	Grade III Severe Asphyxia	Grade IV Secondary Apnea	Grade V Terminal Apnea	Total Birth Asphyxia
Number	652/830	126/830	31/830	17/830	3/830	1/830	830
Percentage	78.5%	15.1%	3.7%	2.1%	0.4%	0.1%	100%

Table 6: Distribution among 830 deliveries attended as normal and grade I to V.

Distribution of 21.4% (n=178/830) hypoxic/asphyxiated newborns among births attended, revealed a majority 70.8% (n=126/178) Grade I newborns had mild birth asphyxia, +1 Pulse oximetry score, SpO₂ 94%-95%, while 17% (n=31/178) Grade II newborns with moderate asphyxia, +2 Pulse oximetry score, SpO₂ 92%-93% and only 9.5% (n=17/178) Grade III newborns had severe asphyxia, +3 Pulse oximetry score, SpO₂ 90-91% while 1.7% (n=3/178).

Grade IV newborns suffered from secondary apnea, +4 Pulse oximetry score, SpO₂ 89%-50%, with absent respiration and Heart Rate <50 bpm while 0.5% (n=1/178) with Grade V had terminal apnea, +5 Pulse oximetry Score, SpO₂ <50%, with absent respiration and Heart Rate <30 bpm or 'Flat baby'. Distribution of 178 newborns with birth hypoxia/asphyxia, Grade I to V is seen in Table 7.

Distribution	Grade I Mild Asphyxia	Grade II Moderate Asphyxia	Grade III Severe Asphyxia	Grade IV Secondary Apnea	Grade V Terminal Apnea	Total Birth Asphyxia
Number	126	31	17	3	1	178
Percentage	70.8%	17.4%	9.5%	1.7%	0.5%	100%

Table 7: Distribution of newborns with birth asphyxia Grade I to V.

In the present study all 178 hypoxic newborns Grade I-V were effectively resuscitated with onset of rhythmic breathing, by Lalana Newborn Resuscitation (LNR) outlined in Protocol I and Protocol II based on birthweight and gestation with wellbeing of the newborns during observation in NICU for four hours to monitor pulse, respiratory rate and oxygen saturation on pulse oximeter, temperature maintained to prevent hypothermia and minimize oxygen consumption, blood glucose checked as well as blood gas study for acid base balance etc newborn then shifted to mother's side and breast feeding instituted with uneventful early neonatal period.

Discussion

Children face the greatest risk of death in their first 28 days with emphasis of neonatal health receiving increased recognition in contributing towards the high proportion of global child survival even though the global number of newborn deaths declined from 5 million in 1990 to 2.04 million in 2019 [1,2], Early Neonatal Mortality Rate (ENMR), has not shown the same reduction as late neonatal mortality since 50% of all early neonatal deaths are due to birth asphyxia with 30.9% sepsis and 18.7%, prematurity [11,12], four to nine million babies experience birth asphyxia per year and only 1 to 2 million successfully resuscitated with an estimated 2.6 upto 3 million third trimester stillbirths [49,50].

Thus intra-partum hypoxia not only accounts for 46% of all maternal deaths as well as 40% of neonatal deaths within the first 24 hours but also comprises 1.02 million stillbirths who may be inadequately resuscitated and misclassified as stillbirths more so in low and middle income countries [13,14,45]. Birth asphyxia accounts for 97.2% of early neonatal deaths, of

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which 70% occur within 24 hours, compared to less than 50% sepsis deaths in first week with 30% in second week, while three fourths of deaths caused by malformations occur in first week, half within 24 hours [11,12]. Thus 50% of neonatal deaths are due to birth asphyxia followed by sepsis 30.9% and prematurity 18.7%, in spite of improved perinatal services like antenatal care for mothers and world-wide introduction of NRP that was supposed to have reduced early neonatal mortality with high incidence of disability in survivors [11,30,49, 50].

However western developed countries have almost eliminated birth asphyxia with low rate of 2/1000 births [37,43] though over 40% survivors following NRP suffer from considerable impairments such as blindness, deafness, autism, seizures and cognitive impairments with inability to develop fine motor skills, memory and mood disturbances etc [36-42]. Thus the degree of morbidity remains high affecting quality of life in survivors and the rate of Hypoxic Ischemic Encephalopathy (HIE) has remained the same over previous decades [41].

Thus the short term goal is to reduce neonatal mortality rate to 5/1000 live births thereby reducing overall 3-8 million annual deaths by 3 million requires renewed efforts and effective strategic plans since decrease in number of stillbirths lowered more slowly than maternal mortality and under five years mortality with target plan to reach 12 or less stillbirths per 1000 births in every country by 2030 [11, 12]. Thus effective strategic plans aimed at reviving asphyxial births will not only reduce early neonatal mortality but also decrease intrapartum stillbirths as approximately 60 million annual births occur outside of health facilities attended by unskilled birth attendants, thus providing effective neonatal resuscitation for 90% of deliveries currently taking place in health facilities would save more than 93,000 newborn lives each year [50-52].

Thus birth asphyxia constitutes one of the leading causes of preventable perinatal and neonatal morbidity and mortality predominantly in India and many other Low and Middle Income Asian countries. Developed countries report about 1% neonatal mortality with vast technological advances in antenatal and neonatal critical care and low Perinatal Mortality Rate (PMR) of 6.1/1,000 births with incidence of birth asphyxia less than 0.1% over the past decades, however stillbirth rates have remained unchanged constituting 70% of perinatal deaths with 30% early neonatal deaths. The corrected PMR 4.1/1,000 births excluding congenital anomalies, an unavoidable proportion of perinatal mortality, constituting the leading cause in 34% [43,53]. In UK intrauterine growth retardation (IUGR) is the single largest contributor to perinatal mortality in non-anomalous fetuses with an eight-fold increased risk of stillbirth 19.8 versus 2.4/1,000 births with over 50% of deaths being SGA having birth weight below 10th customized centile of whom only 30% were suspected antenatal [43]. In a study from Vellore, South India, birth asphyxia 24.1% was the leading cause of early neonatal deaths, followed by respiratory distress syndrome 20% and lethal congenital malformations in 18.2% ranked third [7].

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The current world population of 7.6 billion is expected to reach 8.6 billion in 2030 [54]. India has around 25 million births each year that comprises nearly one-fifth of world's annual childbirths comprise 47% of 8.2 million under five years deaths with death of one of these babies every minute, three fourths in the first week, 50% being in first 24 hours, while 13.5% occur in the 2nd week and 13.5% in last two weeks [11,47,55,56], indicating that effective strategies aimed at reduction of asphyxial early neonatal deaths including intrapartum stillbirths will substantially reduce perinatal and neonatal mortality to effectively further reduce under-five child mortality rate. The most common cause of neonatal death is intrapartum-related complications in most developing countries due to higher incidence of serious complications in labour and reduced availability of skilled care during delivery.

Birth asphyxia is due to deficient oxygenation that results in respiratory failure with persistence of fetal circulation and pulmonary arterioles remain constricted with right to left shunting through foramen ovale and ductus arteriosus with only about 10% of cardiac output perfuse the lungs [16,1729, 44] that further perpetuates hypoxia requires effective resuscitative intervention to establish rhythmic respiration by sustained nasal inflatory oxygen with flow rate of 4-15 litres/min or 5 cm H₂O pressure up to maximum 20 cm H₂O pressure extending over 1 to 3 minutes with the goal to open collapsed alveoli to achieve better post manoeuvre lung mechanics to keep lungs open and improve oxygenation that is proven both scientifically and physiologically resulting in uniform alveolar recruitment [33,57] causing exponential increase in alveolar surface area and alveolar pressure above atmospheric pressure measured as Functional Residual Capacity (FRC) that enables the generation of intrinsic hydrostatic pressure gradient between airways and lung tissue to overcome the high resistance of moving liquid through the airways and across the alveolar wall keeping air sacs to remain open, achieving optimal gas exchange, thereby triggering large reduction in Pulmonary Vascular Resistance (PVR), vasodilation of pulmonary arteriole, left to right shunt causing reflex physiological mechanism converting fetal circulation to adult type [17,18,21,57].

Dramatic transition takes place at birth, as organ of gas exchange switch from placenta in the fetus to the lungs in the newborn occur at onset of rhythmic breathing, often within 10 seconds of birth or even after 60-90 seconds. Normally at birth newborns make forceful inspiratory efforts up to 60 cm H₂O in order to overcome the resistance to the inspiration of air into the fetal liquid filled lung [23,24]. The inspiratory efforts not only sucks air into the alveoli but also stretch the alveolar epithelium allowing passage of fetal alveolar liquid down a protein osmotic pressure gradient into the interstitial tissue into lymphatic's and into the circulation [26]. Once fetal lung fluid has been displaced from the alveoli, lungs can be inflated at lower 1-15 cm H₂O [58,59].

This increase in airway and alveolar pressure facilitating clearance of alveolar fluid, even as baby starts breathing efforts helps improve oxygenation in hypoxemic respiratory failure. Hypoxemia with hypercapnia or rise in arterial carbon dioxide results in reduced blood flow to the brain with ischemia causes altered mental status [15-17,60]. Lalana Newborn Resuscitation (LNR), is the most safest physiological resuscitative method proven by studies showing that a high pressure of 25-30 cm H₂O is required for the first inflation in fluid filled lungs for about five seconds so whole lung becomes inflated displacing foetal lung fluid from alveoli, thereafter lungs can be inflated at a lower 10-15 cm H₂O [21,58,59].

The incidence of Birth asphyxia in present study was 21.4%, effective newborn resuscitation by Lalana Newborn Resuscitation (LNR) was successful in all 178 asphyxiated newborns. Oxygen therapy is the only specific treatment to prevent or mitigate the effects of hypoxia resulting in rapid reduction for the need of high FiO₂ [21]. Continuous monitoring by Pulse Oximeter gives accurate insights to peripheral oxygenation (SpO₂), heart rate (bpm) in matter of seconds, empowers one to respond quickly and confidently to abnormal SpO₂ reading to determine supplemental oxygen, SpO₂ 96% indicate successful resuscitation, oxygen discontinued [61-63].

Randomized controlled trials have demonstrated the benefit of sustained inflation decreasing the need for intubation and mechanical ventilation [20] also animal studies scientifically establishes that longer sustained inflation is beneficial in establishing residual capacity during transition from fluid filled to air filled lungs after birth [64] by generating a hydrostatic pressure gradient between airways and lung tissue to overcome the high resistance of moving liquid through the airways, across the alveolar wall into the interstitial tissue.

When oxygen/air enters the lungs the normal fetal partial pressure of oxygen (P_AO₂) levels of 25 mmHg rises sharply to above 60 mmHg, resulting in dilation of pulmonary vasculature at birth [65] facilitated by development of surface tension forces in the alveoli that exert radial traction on blood vessels [66] with increase in blood flow through the lungs and pressure in left atrium rises, along with cessation of umbilical circulation, the right atrial pressure falls slightly resulting in closure of foramen ovale, the increased pulmonary arteriolar oxygenation (P_AO₂) constricts ductus arteriosus once breathing has started, the entire cardiac output must flow through the lungs allowing for full oxygenation of blood [67] that initiates a series of cardiovascular reflex physiological mechanism changes that are essential for survival after birth converting fetal circulation to adult type [17, 18].

Thus sustained nasal inflation with oxygen at flow rates of 2-15 Litres/min (FiO₂ 21% to 76%), is key to effective resuscitation in quick reversal of hypoxia with onset of rhythmic respiration with prompt increase in heart rate as being a sign of adequate lung aeration based on the concept that a low heart rate indicates vagal-induced bradycardia in response to perinatal asphyxia [68] facilitates smooth physiological cardiovascular changes transiting

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from fetal to neonatal life monitored continuously by Pulse oximetry SpO_2 achieve aim of effective resuscitation with reduction in perinatal and neonatal mortality and morbidity in reducing adverse long-term hypoxic neurodevelopmental ill sequelae, so children should be normal.

In contrast non-physiological resuscitation by NRP by bag and mask or invasive endotracheal intubation with short intermittent inflation lacks scientific clarity regarding transition of fluid filled fetal lungs to well aerated neonatal lungs, as short intermittent bursts of air/oxygen does not generate adequate intrapulmonary pressure but also proves potentially harmful as entire tidal volume will only enter previously aerated regions due to the much lower airway resistance predisposes to lung injury with overexpansion and intermittent collapse of alveoli causing ventilation perfusion (V/Q) mismatch, persistent pulmonary hypertension and bradycardia that further perpetuates hypoxia with delay in onset of breathing [30-32]. Thus continuous distending airway pressure by sustained oxygen inflation is the key to mitigating hypoxia-induced inhibition of breathing [19-21].

High FiO_2 results in exponential increase of surface area measured as Functional Residual Capacity (FRC) with positive end-expiratory pressure or alveolar pressure above atmospheric pressure helps keeps air sacs open, improving ventilation triggering large reduction in Pulmonary Vascular Resistance (PVR) with vasodilation of pulmonary arteriole causing increased blood flow through the lung and circulation of oxygenated throughout the body [21,57,58]. Thus continuous distending airway pressure by sustained oxygen inflation is the key to mitigating hypoxia-induced inhibition of breathing at flow rates of 5-15 Litres/min (FiO_2 up to 76%) results in quick reversal of hypoxia by stimulating the respiratory centre with rhythmic respiration and prompt increase of heart rate being a sign of adequate lung aeration, SpO_2 monitored by Pulse oximetry, achieves the aim of effective resuscitation [68,69], thus saving lives thereby reducing perinatal and neonatal mortality with adverse long-term hypoxic neurodevelopmental sequelae, so that children should be normal.

Lalana Newborn Resuscitation (LNR) includes three steps:-

Step I: Newborns at birth timed after complete expulsion and pulse oximeter placed on the baby's foot monitors superficial oxygen saturation (SpO_2), heart rate and the plethysmograph tracing records how well heart is pumping oxygenated blood throughout the body indicated by pulsatile changes. Immediate clamping and cutting of umbilical cord is recommended in newborns with birth asphyxia, however umbilical cord ligation may be delayed up to 60-90 seconds in healthy newborns with rhythmic respiration [71].

Delayed clamping of cord however predisposes to hypoxia that acts as a major stimulus to breathing including chilling of the skin at birth and stimulation of receptors near larynx when airways are cleared of liquid and increased sensitivity of carotid chemoreceptors to hypoxia

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have shown to augment breathing [72-74] despite which these newborns require active resuscitation for healthy outcome as fetal asphyxia first evokes gasping but prolonged asphyxia depresses central nervous system including respiratory centre, such that newborns do not respond to normal stimuli augmenting onset of breathing [60].

Step II: Classification of newborns based on pulse oximetry score of SpO₂ 'Normal' healthy newborns have Zero Pulse oximetry score, SpO₂ 96%-100% with spontaneous onset of rhythmic respiration, rate 30-60/min and heart rate of 120-160 bpm. Hypoxic/asphyxiated newborns are further graded into Grade I to V based on Pulse oximetry score +1 to +5 with SpO₂ <96%, either presence or absence of breathing $\pm$ respiratory rate <30/min and heart rate <120 bpm, with poor peripheral perfusion. However grading of hypoxic newborns into Grade I-V by criteria of +1 to +5 pulse oximetry score to determine oxygen flow rate may vary as real time assessment of newborn's SpO₂ is constantly changing due to continuous monitoring, oxygen and oxygen discontinued at SpO₂ 96%.

Step III: Lalana Newborn Resuscitation Protocol I and II, by continuous positive pressure ventilation by sustained nasal oxygen inflation, flow rates determined by Pulse oximetry score, facilitate onset of rhythmic breathing with effective resuscitation maintaining thermo-control and asepsis [70].

Among 830 deliveries attended, majority 78.5% (n=653/830) were normal, healthy newborns who established spontaneous rhythmic respiration, rate 30-60/min at birth with Zero Pulse oximetry score, SpO₂ >96%-100%, heart rate of 120-160 beats per minute (bpm). Only 15.2% (n=126/830) newborns with mild birth asphyxia Grade I, +1 Pulse oximetry Score, SpO₂ 94% -95%, regular/irregular respiration, rate $\pm$ 20-30/ min and heart rate $\pm$ 100-119 bpm. While 3.7% (n=31/830) comprised Grade II moderate birth asphyxia +2 Pulse oximetry Score, SpO₂ 92-93% with regular/ irregular breathing, respiratory rate 15-20/min, $\pm$ heart rate 100-80 bpm and 2.1% (n=17/830) Grade III severe birth asphyxia, +3 Pulse oximetry Score SpO₂ 90-91% with irregular or gasping respiration, rate 10-15/min, heart rate 80-60 bpm and newborns 0.4% (n=3/830) Grade IV secondary apnea, +4 Pulse oximetry score, SpO₂ 89-50%, heart rate < 60 bpm with absent respiratory effort.

Just 0.1% (n=1/830) constituted Grade V terminal apnea +5 Pulse oximetry score, SpO₂<50%, absent breathing and very low heart rate < 30 bpm. Grade V newborns with terminal apnea are often subjected to severe perinatal asphyxia especially after prolonged labour due to failed trial due to undetected cephalo-pelvic disproportion are associated with high risk of intrauterine death termed as fresh stillbirth if born without signs of life or early neonatal death with signs of life and failed resuscitation including endotracheal intubation upto 5-10 minutes with CPPV and 100% oxygen flow at 15 L/min (FiO₂ 100%) are usually delivered due to quick obstetric intervention by emergency LSCS, however resuscitative efforts may be aborted after 5-10 minutes in view of the high incidence of HIE with lifelong

untoward neurological sequelae [16]. However the more severe the perinatal asphyxia the longer it will take longer for the newborn to breathe more so with Neonatal Resuscitation Program (NRP) that requires a team of usually four or more skilled birth attendants for cardiopulmonary resuscitation by administering IPPV with bag and mask or endotracheal intubation and if heart rate remains below 60 beats per minute, external cardiac massage using two fingers to depress the lower sternum at approximately 120 times a minute while continuing with respiratory assistance in a ratio of 1:5. Medications such as Epinephrine or volume expanders are recommended at dose of 10ml/kg of saline may be repeated to increase cardiac output [30].

Basic principles include minimal handling of the infant, monitoring for vital signs including blood gases, temperature maintenance with correction of metabolic abnormalities. Strict calculated fluid requirement necessary according to body weight and age in days after birth, usually 5% dextrose only 50 to 70% of the calculated volume is given which varies from 50 to 100 ml/kg body weight as these infants often may have hypoxic renal failure myocarditis and gut injury, so fluid administration should be carefully monitored anticonvulsants and early initiation of physiotherapy may be instituted. Feeding is preferably delayed in severe cases till 48 to 72 hours.

A severely asphyxiated and acidotic infant usually requires a slow (1 ml/min) administration of sodium bicarbonate (3 to 4 mEq/kg) through the umbilical vein of the umbilical cord with prompt correction of hypoglycaemia and hypocalcaemia is needed. If the baby is hypoglycemic 10% glucose infusion should be given. Hypocalcaemia infant should receive 2 ml/kg body weight of 10% calcium gluconate diluted with equal volume of 10% dextrose as IV bolus. Maintenance calcium at the rate 200 mg/kg/day should be added to the IV infusion.

The clinical signs of asphyxia or hypoxic ischemic encephalopathy vary with a lethargic, hypotonic infant to flaccidity and coma. In severe asphyxia hypoxic ischemic encephalopathy, there may be convulsions with onset at 6 to 24 hours of age. Respiratory abnormalities include apnea and cyanosis, temperature instability, blood pressure instability-circulatory insufficiency also may present at birth as a result of intracranial or other internal haemorrhage. The early administration of phenobarbitone helps in controlling the seizures and also reduces cerebral edema. A loading dose of 10-20 mg/kg body weight as a bolus IV or IM followed by 3-5 mg/kg divided into two doses given orally/ IV/IM or Dilantin in the same dosage. Dexamethasone in severe cases is given at 2 mg/kg in two divided doses IV or IM. However the use of cortisone has been disputed. The infant may require oxygen, antibiotics, total parenteral nutrition and mechanical ventilation if indicated [30].

Resuscitatory efforts aborted after 10-20 minutes, justified due to association of high mortality or morbidity in survivors with severe neuro-developmental disability [30], hence a coordinated approach by obstetrician, neonatal team and parents is important. Resuscitation

may also be withheld in extremely immature newborns with gestational age <23 weeks and birth weight below 400 g or in newborns with lethal congenital malformations, not compatible with life, for example anencephaly etc. However despite Neonatal Resuscitative Program (NRP) resulting in significant worldwide reduction in asphyxial neonatal deaths [34-35] nearly half of survivors suffered from permanent neurological deficits depending on the extent of insult, varying from mild ill effects to severe hypoxic ischemic encephalopathy or multi-organ complications and death within the first few days [36-42].

Since mild perinatal hypoxia occurs more frequently than severe events, it is associated with substantial long-term effect on the population, who are at increased risk of low intelligence or Intelligent Quotient (IQ) scores <80 affects a large proportion of adults with poor scholastic performance [38,39] and other deficits include impaired cognition, mild autism, lack of development of fine motor skills, memory and mood disturbances etc. with over 200 million children not attaining age appropriate development [35] while severe perinatal asphyxia in survivors may result in hypoxic ischemic encephalopathy which over past few decades has remained the same [41].

In contrast resuscitation by LNR that may be administered by a single birth attendant is unparalleled, due to the impact of oxygenation with quick onset of regular breathing and reversal of hypoxic pulmonary vasoconstriction converting to left to right shunting and neonatal life. Air has 21% oxygen (FiO_2 0.21) which mixes with oxygen increasing concentration of oxygen in air by approximately 4% per litre flow. Peak inspiratory flow required is 20-30 L/min at the first breath, oxygen at flow rate of 10 L/min, mixes with 20 L/min of air (FiO_2 0.60) on inspiration, hence pure 100% oxygen is not inhaled [57, 58]. In contrast NRP with use of tight fitting face mask or endotracheal intubation increases FiO_2 up to 100% [30].

The oxygen saturation of newborn by NRP seems unacceptable with long delay in hypoxic new-borns achieving SpO_2 >90%, with 40-45% at 1 minutes, SpO_2 65-75% at 2 minutes, SpO_2 70-75% at 3 min, SpO_2 75-85%, at 4 minutes, SpO_2 80-85% at 5 minutes and SpO_2 85-95% 10 minutes [30-33]. In contrast LNR results SpO_2 >96% within 1-3 minute, even nasal inflation oxygen at flow rate of around 2-4 L/min creates continuous distending pressure throughout respiratory cycle provides intrinsic Positive End Expiratory Pressure (PEEP) that helps keep alveoli open to achieve optimal gas exchange [57] facilitating quick physiological transition from fetal to neonatal life soon after birth [17,18], continuously monitored by pulse oximetry requiring minimal training of unskilled birth attendants that will prove to be beneficial in many Asian countries, contrasts to highly skilled trained birth attendants a major prerequisite for NRP.

In fact NRP recognizes several significant gaps in knowledge related to neonatal resuscitation as current recommendation are based on weak evidence lacking well designed large well

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controlled trials (RCTs) [30-32], in contrast large controlled study of LNR versus NRP may be undertaken in the delivery room. Positive outcome in LNR is the quick reversal of hypoxia by supplementary oxygenation that is key to successful resuscitation in newborns with perinatal asphyxia.

Lack of oxygen causes anaerobic glycolysis and metabolic acidosis that result in primary energy failure or deprivation of high energy phosphate, causing cellular damage and multi-organ failure with renal failure, hypoxic myocarditis and neurological damages etc [16, 29,75-77]. Also severe hypoxia causes interference with the production of clotting factors from the liver and may initiate DIC also a strong association between hypoxia and intraventricular hemorrhage as cause of death in preterm as increase in intravascular pressure ruptures vulnerable vessels in the germinal matrix [78]. Hypoxia is also a major factor in necrotizing enterocolitis with interference of blood to the gut [79].

The longer the hypoxemia, the more severe the sequelae, thus combating with oxygen supplementation in LNR results in prompt increase of heart rate with circulation of oxygenated blood throughout the body improving cellular function, converting anaerobic to aerobic metabolism utilizing glucose with generation of 38 mols of adenosine triphosphate (ATP) instead of 2 mols of ATP and removal of lactic acid from tissues [75,80]. Thus Lalana Neonatal Resuscitation (LNR) with oxygenation is a safe resuscitative method physiologically and scientifically effective by sustained nasal oxygen inflation with higher fraction of inspired oxygen (FiO_2), quickly reverses hypoxial injury, initiating rhythmic respiration with circulation of oxygenated blood throughout the body, reducing adverse hypoxic neurological or organ deficits serves to achieve the aim of effective resuscitation such that children should be normal [70].

Thus continuous positive pressure ventilation with sustained nasal oxygen inflation facilitates lung fluid reabsorption at birth, enabling smooth transition from fetal to extra uterine life. Hypoxia is a potent inhibitor of spontaneous respiration and lack of oxygen causes anaerobic glycolysis with metabolic acidosis due to accumulation of lactic acid and acidosis which impairs cardiac function and increases pulmonary vascular resistance. The low pH worsens pulmonary vasoconstriction with right to left shunting through foramen ovale and ductus arteriosus and venous blood bypasses lungs to enter aorta with serious consequences causing life threatening hypoxial injury in newborns [75,76,81].

Oxygen therapy in hypoxic newborns is the only specific treatment to prevent or mitigate the effects of hypoxia in decreasing central apnoea and promoting regular breathing pattern that is the key to successful resuscitation [19-21,58]. Oxygen flow rate varying from 4 to 15 L/min (FiO_2 36% to 75%) determined according to Grade I-V of hypoxic newborns creates distending pressure to achieve optimal gas exchange stimulating central respiratory centre to initiate rhythmic respiration, counteracting hypoxemia and hypercapnia that is associated

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with rise in arterial carbon dioxide causing reduced blood flow to the brain and ischemia with altered mental status [60,76]. Some newborns that remain short of breath have transient tachypnoea (wet lung) for a few hours even occasionally one to two days later after birth due to delayed removal of fetal lung fluid [82].

IPPV with bag and mask provides insufficient transpulmonary pressure for clearance of lung fluid [30,32] that remains in the alveolar walls and adequate oxygenation is required for reversal of hypoxia being the only specific treatment. In LNR 100% oxygen (FiO_2 1.0) is blown through the oxygen tube to baby's nostril however the first breath requires a peak inspiratory flow of around 20-30 L/min or 8 L/kg/min, eg. a baby weighing 3500 g, requires approximately 28 L/min, to meet the first inspiratory flow. Oxygen at flow rate of 10 L/min, therefore another 18 L/min of air (FiO_2 0.21 or 21%) is sucked in from surrounding atmosphere, therefore $(10 \times 100) + (18 \times 21) = 1378$, 1378 divided by 28 = FiO_2 0.49 or 49%. Therefore the air/oxygen mixture FiO_2 0.49 or 50% is inhaled, obviating both hyperoxia and mitigating hypoxia [83].

In contrast NRP with tight fitting mask or endotracheal intubation and supplemental oxygenation increases FiO_2 , to 1.0 or 100% oxygen that predisposes to hyperoxia with its deleterious effect on slowing cerebral blood flow in both term and preterm infants, since even brief periods of supplemental, uncontrolled exposure of 100% oxygen result in generation of oxygen free radicals that play a role in reperfusion injury, contributing to eye (ROP) with blindness, lung injury and altered mental status in preterm [60,76,77,84-88].

Also intermittent inflation with bag and face mask does not generate adequate intrapulmonary pressure to displace alveolar fluid with un-even alveolar ventilation during a single breath in addition gastric distension occurs precludes satisfactory oxygenation in fetal fluid filled lungs despite administration of oxygen [32] resulting in decreased pulmonary perfusion, V/Q mismatch further perpetuating hypoxia, hypercarbia and acidosis since pulmonary arterioles remain constricted with right to left shunt through foramen ovale and ductus arteriosus that interferes with the transition of fetal to neonatal cardiopulmonary circulation [57,81].

Importantly newborns remain extremely vulnerable to reopening of fetal right to left shunts for several days to even weeks after birth due to pulmonary vasoconstriction and if PO_2 of lung tissue falls, further compounded by low pH, worsens pulmonary vasoconstriction has serious consequences with bypass of venous blood into the aorta probably being the single most life threatening result of hypoxia in the neonatal period as anatomical closure usually takes place by about two weeks of age [15-17,57,81].

Fetal lungs secrete surfactant by the 7th month of gestation while Type II pneumocytes in neonatal lungs, secrete a thin lining of alveolar fluid that combines with surfactant to form an aqueous protein containing hypophase with overlying phospholipid film composed mainly of

dipalmitoyl phosphatidylcholine to create a moist surface conducive to gas exchange by lowering surface tension, open alveoli and prevent atelectasis, gases first dissolve in the alveolar lining fluid and then diffuse across type I, extremely thin squamous alveolar cells and pulmonary arteriolar capillary membrane to combine with haemoglobin [57].

Oxygenation is the process of taking oxygen from inspired air that diffuses passively from the alveoli to pulmonary capillaries where it binds to haemoglobin forming oxyhaemoglobin and a small amount dissolves in plasma. In the newborn gas exchange occurs in the lungs by two mechanisms for oxygen delivery to the body, 98.5% oxygen is bound to haemoglobin, which is assessed by Pulse oximetry SpO_2 , being almost similar to SpO_2 measured by Arterial Blood Gas (ABG) analysis, while some oxygen is dissolved in the plasma, accounts for only 1.5% transport to tissues [89].

Each haemoglobin molecule carries four molecules of oxygen bound to the iron of the heme prosthetic group. There are about 270 to 300 million haemoglobin molecules present in one-third of erythrocyte cytoplasm which are relatively short lived about 100 to 220 days. As the first oxygen molecule binds to haemoglobin tetramer, it induces a change in shape of haemoglobin that increases its ability to bind to three other molecules of oxygen, reflecting cooperative interaction between haemoglobin and oxygen molecules, thus each haemoglobin tetramer binds to four molecules of oxygen and a gram of haemoglobin can combine with 1.34 ml of oxygen. Hence blood with normal haemoglobin concentration of 15 g/dl, 100 ml carries approximately 20 ml of oxygen in addition a small quantity of oxygen is dissolved in blood. If haemoglobin tetramer binds to only three molecules of oxygen instead of four, it leads to hypoxia and deoxyhaemoglobin. However if the partial pressure of oxygen in the alveoli is high, then four molecules of oxygen binds haemoglobin binds to form oxyhaemoglobin [89].

However a lack of oxygen in the blood means that body tissues will not be oxygenated adequately causing damage to the organs is an indication of serious pulmonary issues. While oxygen delivery is the rate of oxygen transported from the lungs to the peripheral tissue and oxygen consumption is the rate at which oxygen is removed from the blood for use by the tissues. Oxygenated blood sustains aerobic cellular metabolism throughout the body, wherein oxygen is used to convert glucose to adenosine triphosphate (ATP). Insufficient oxygenation is termed hypoxemia, causes low partial oxygen tension that refers to abnormally low oxygen content in tissue or organ with residual neurological and organ deficits.

Arterial blood flows from the heart to parts of the body laden with oxygen where it diffuses to the surrounding tissues with low partial pressure of oxygen and oxyhaemoglobin releases oxygen to cells to form deoxyhaemoglobin. Diffusion of oxygen is related to partial pressure of oxygen (P_{AO_2}) from the alveoli into the pulmonary capillaries (P_aO_2), depends on the Alveolar-arterial (A-a) gradient, normal range of difference between P_{AO_2} - P_aO_2 being 5-10

mmHg, but collapsed, fluid filled or unventilated alveoli with VQ mismatch shunt, reflects rising A-a gradient that impairs oxygen diffusion across the alveolar-pulmonary arteriolar capillary membrane into the blood stream [89].

The normal partial pressure of oxygen in the alveoli (P_{AO_2}) is FiO_2 0.21 or 21% at atmospheric pressure of 760 mmHg, breathing in room air at sea level is around 80 to 100 mmHg, therefore about 90% of oxygen in healthy lungs makes it to the blood. However when P_{AO_2} is >90%, the increase in P_{AO_2} has relatively little impact on oxygen saturation by haemoglobin, as there can be no further increase in saturation however high the P_{AO_2} rises with supplemental oxygen. If however the alveolar partial pressure (P_{AO_2}) falls to 60 mmHg, less oxygen binds to haemoglobin with rapid fall of oxygen in red blood cells. Hence alveolar partial pressure of oxygen at 100 mmHg is much better than the alveolar partial pressure of oxygen of 80 mmHg even though oxygen saturation of haemoglobin in blood will not change very much despite increments in oxygen supplement [89].

The relationship between the partial pressure of oxygen and oxygen saturation is shown by the oxygen disassociation curve. The sigmoid shape of the disassociation curve reflects the cooperative interaction between haemoglobin and oxygen molecules which is initially steep and then flattens out, being a graphical representation of haemoglobin affinity to oxygen or percentage saturation of oxyhemoglobin at various alveolar partial pressures (P_{AO_2}) of oxygen. The X-axis of the oxygen disassociation curve represent dissolved oxygen in linear relationship to its partial pressure result in a straight line on the horizontal axis and the proportion of haemoglobin in its saturated (oxygen-laden) forms the vertical Y-axis against the prevailing oxygen tension.

High alveolar partial pressure (P_{AO_2}) at 100 mmHg drives oxygen on to the haemoglobin until 95-100% saturated. Haemoglobin (Hb) releases oxygen as the blood passes through the tissues with lower partial pressure of oxygen and mixed venous blood (PvO_2) returning from the tissues is much lower, PvO_2 at 40 mmHg compared to arterial blood. The most important aspect of the oxygen disassociation curve is that if Pulse oximeter reading falls below 90%, the partial pressure of oxygen in the blood (P_aO_2 or SaO_2) drops very rapidly and oxygen delivery to tissues is reduced that may lead to cardiac arrest, requiring quick resuscitative intervention [89].

Pulse Oximeter provides a rapid tool in assessing adequate peripheral oxygenation or percentage of hemoglobin that is saturated with oxygen. In addition the plethysmograph indicates cardiac function by pulsatile changes as prompt increase in heart rate indicates adequate lung aeration since vagal-induced bradycardia is response to perinatal asphyxia. Continuous Pulse Oximetry therefore empowers one to respond quickly and confidently to abnormal readings to determine supplemental nasal oxygen flow if SpO_2 falls <96% [61-63, 89]. Various factors affect haemoglobin's affinity for oxygen, while right shift decrease

oxygen affinity influenced by factors of lower pH, higher temperature, PCO_2 and high concentration of 2,3 diphosphoglycerate (2,3 DPG) produced from phosphoglyceraldehyde in response to hypoxia in red blood cells is an intermediate metabolite in the glycolytic pathway that binds to the beta chains of deoxyhaemoglobin and rearranges it into the T state thereby decreasing affinity for oxygen, hence more oxygen is released into tissue, indicating that haemoglobin allows more oxygen to be available to tissues but it is also more difficult for oxygen to bind with haemoglobin in lungs. Fetal haemoglobin is the main oxygen transport protein in the human fetus during the last seven months of development and persists in newborn until six months of age to later form adult haemoglobin which has two alpha and two beta subunits.

Fetal haemoglobin is composed of two alpha and two gamma subunits which shifts oxygen disassociation curve to the left compared to that of adult haemoglobin, resulting in greater affinity for oxygen allowing the fetus to extract oxygen from maternal circulation, however oxygen disassociation curve in relation to partial pressure of oxygen is lower than normal adult haemoglobin based on lower P_{50} value or 6-8 mmHg (Torr) difference due to decrease affinity of 2,3 Diphosphoglycerate in that oxygen released from red blood cells requires a lower P_{aO_2} with left shift compared to adult Hb [89,90].

Also hypoxia with anaerobic metabolism produces lactic acid causing metabolic acidosis that decreases pH and shifts the curve to the right, referred as Bohr effect, as the higher hydrogen ion concentration causes an alteration in amino acid residues that stabilises deoxyhaemoglobin in a T (taut or tense) state that has lower affinity for oxygen, thus further promotes hypoxia. While left shift indicates increase affinity of haemoglobin for oxygen binding at any given P_{aO_2} with increase oxygen transport to tissues, as blood passing through the lungs gives CO_2 and H^+ ions in the form of carbonic acid that increases oxygen binding to haemoglobin. Tissues have low oxygen concentration and oxyhemoglobin releases oxygen to form deoxyhaemoglobin, thus diffusion of oxygen from haemoglobin to tissue cells is enhanced by this process and corresponds to the steep portion of the 'S' shaped curve [89,90].

Oxygenation in LNR is from high pressure sources such as cylinder or piped wall supply that first passes through a pressure regulator to a lower pressure which then flows through the flow meter, controlled by a valve for litre flow per minute. Sustained positive pressure is maintained according to the flow rate of oxygen dialled on flow meter usually between 1-15 Litres per minute (L/min) while FiO_2 is defined as the percentage concentration of oxygen inhaled or fraction of inspired oxygen.

Air contains 21% oxygen equivalent to FiO_2 0.21 and flow rate of 1 L/min gives an oxygen increment of approximately 4% or FiO_2 0.04 for each increased in litre/min flow i.e., when 1 L/min oxygen flow is mixed with air gives FiO_2 24%. Thus flow rate of 6 L/min gives

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oxygen concentration FiO_2 0.45 or 45% volumetric fraction of oxygen that mixes with air during inhalation [83].

Therefore even though oxygen with FiO_2 1.0 or 100% through flow meter which is connected to either medical wall supply or oxygen cylinder is bubbled through a bottle containing 5 cm water for humidification and through the wide bore oxygen tube, mixes with room air on inspiration resulting in lower FiO_2 of 0.24 up to 0.72 i.e. 24% to 72% oxygen but never 100% oxygen inspired by the newborn. In addition the wide bore oxygen tube proves advantageous in allowing for quick adjustment in varying oxygen flow rates between 2 L/min up to 15 L/min without the requirement for changing low to high flow oxygen delivery devices [70].

Low flow oxygen delivery devices allows oxygenation of $\text{FiO}_2 < 35\%$, while moderate oxygen flow delivery devices allows FiO_2 35% -60% and high oxygen flow delivery devices allows $\text{FiO}_2 > 60$. Low flow oxygen delivery devices includes paediatric nasal cannula consisting of a thin tube with two small nozzles that inserts into the nostrils and allows oxygen at flow rate of 2-4 L/min with approximate FiO_2 0.28-0.36. Simple face mask allows oxygen flow of 5-6 L/min with FiO_2 0.32-0.36. Higher oxygen flow rate require humidification with minimum 5 L/min to flush carbon dioxide (CO_2) from mask in breathing patients and to protect mucosa of nostrils from drying. Partial rebreather allows oxygen flow of 6-8 L/min sufficient to keep reservoir bag from deflating during inspiration, does not have a one way valve [59,83].

High oxygen flow device, the Venturi mask has a one way valve over port that limit entrainment of room air with humidified oxygen flow rate of 6 L/min upto 15 L/min and approximate FiO_2 0.44 to 0.78. The non-breather mask is high flow device with one-way valves to exit exhaled air and draw oxygen from attached reservoir bag with oxygen flow rates of 10 L/min to 15 L/min, delivers approximate FiO_2 0.70 up to 1.0, if mask is properly fitted to 100% oxygen as with endotracheal intubation. An aerosol generating device will deliver FiO_2 anywhere from 0.21 to 1.0, depending on the set up usually at 10 L/min and desired FiO_2 is selected by adjusting an entrainment collar located on the top of the aerosol container with humidity device connected to the flow meter through wide bore tubing that connects to patient's mask [59,83].

While Continuous Positive Airway Pressure (CPAP) is a non-invasive nasal type of respiratory support in spontaneously breathing preterm newborns, CPAP with blender to maintain $< 30\%$ FiO_2 in delivery room is recommended for early rescue of preterm babies < 32 weeks and < 1250 g for respiratory acidosis, respiratory distress syndrome, recurrent apnoea, atelectasis etc [87-90]. CPAP is constant air pressure directed through nasal prongs or oxygen tube directed to baby's nostrils, helps to open air sacs in the lungs and stay open prevents apnea and at higher oxygen flow rate helps build up intrinsic Positive End-Expiratory Pressure (PEEP) in lungs (alveolar pressure) above atmospheric pressure which is caused by

incomplete expiration, such that they do not need mechanical ventilation. Bubble CPAP with blender allows for increase in FiO_2 with increasing oxygen saturation in newborn but not in oxygen flow rate, preterm <32 weeks have periodic breathing and may have short attacks of apnea due to immaturity and inadequate control of breathing provides respiratory support in preterm at birth with CPAP and oxygen bubbled through 5 cm water in a bottle with FiO_2 <0.3 or <30% produces small airway pressure oscillations on reaching the neonate's lung results in improved gas exchange and lung function is considered as an excellent alternative to routine intubation and mechanical ventilation in management of respiratory distress syndrome [87-90].

Prolonged apnea results in hypoxia, so instead of breathing air by CPAP, increasing P_{aO_2} by 23%-25% or 50 mmHg raises P_{AO_2} to about 70-80 mmHg that improves gas exchange and lung function to maintain oxygen saturation between 90%-96% . Delivery room use of CPAP for early rescue routinely used for preterm babies with spontaneous breathing avoids routine intubation in management of respiratory distress syndrome, as well as decreases surfactant need by 50% and provides post extubation respiratory support effectively reducing broncho-pulmonary dysplasia [89,90]. However in low income Asian countries with limited resources, oxygen hood may also be used instead of CPAP machine, the oxygen flow rate adjusted to maintain preterm SpO_2 around 95% managed with parenteral fluids and treatment of any complications. A BMJ study found nasal CPAP, a non-invasive technique more effective than intubation in reducing BPD [91].

CPAP titrated pressure is prescribed pressure of steady oxygen flow rate between 4-8 L/min ensures bubbling through 5 cm H_2O to deliver constant air pressure into the baby's nose through nasal prongs, enabling air sacs to stay open by increasing Functional Residual Capacity (FRC) and maintaining lung volume during expiration, prevents atelectasis by building up Positive End Expiratory Pressure (PEEP) or alveolar pressure above atmospheric pressure that exists till the end of expiration preventing alveolar collapse and apnea, making it easier for the baby to breathe independently [57]. Also any decrease in lung volume (FRC) decreases surface area for gas exchange with intrapulmonary shunting or V/Q mismatch perpetuating hypoxia.

While extrinsic PEEP is applied by ventilator, intrinsic PEEP is facilitated by sustained nasal oxygen flow with higher FiO_2 as well as by incomplete expiration. Pressure support PEEP or pressure applied during inspiration causes progressive air trapping (hyperinflation). This accumulation of air increases alveolar pressure at the end of expiration. Alveolar gas equation used to calculate alveolar pressure with any given FiO_2 is $\text{P}_{\text{AO}_2} = \text{FiO}_2 (\text{P}_{\text{BAR}} - \text{P}_{\text{H}_2\text{O}}) - \text{P}_{\text{ACO}_2}/\text{RQ}$. If $\text{P}_{\text{H}_2\text{O}}$ at $37^\circ\text{C} = 47$ mmHg, P_{BAR} or barometric pressure at sea level varies from 745 to 765 mmHg or 747 mmHg, P_{ACO_2} = arterial P_{aCO_2} = 40 mmHg and $\text{RQ} = 1$, then $\text{P}_{\text{AO}_2} = \text{P}_{\text{AO}_2} (700) - \text{P}_{\text{aCO}_2}$. Infant breathing 30% O_2 , ($\text{FiO}_2 = 0.3$) has arterial CO_2 of 40

mmHg then Alveolar oxygen tension: $P_{AO_2} = 0.3(700) - 40 \text{ mmHg} = 170 \text{ mmHg}$ [57,69,83]. Pre-oxygenation is dangerous and an ineffective practice [92]. Effective oxygen therapy is delivering the lowest FiO_2 to achieve normal oxygen saturation and heart rate, based on Pulse oximetry SpO_2 thus preventing hypoxia and hyperoxia, LNR with sustained positive pressure ventilation establishes an ideal approach in resuscitating hypoxic/asphyxiated newborns, transiting from fluid filled fetal lungs to uniformly well aerated neonatal lungs by avoiding inappropriate high supplemental oxygen therapy determined by Pulse oximetry SpO_2 , oxygen flow discontinued at zero score $SpO_2 > 96\%$ [70].

The dangers of Neonatal Resuscitation Program (NRP) [30,32] in that tight fitting face mask or endotracheal intubation predisposes to hyperoxia with FiO_2 upto 0.8-1.0 has deleterious effect of slowing cerebral blood in both term and preterm infants and reperfusion injury with generation of oxygen free radicals may cause high regional cerebral oxygen saturation that predisposes to periventricular haemorrhage and increased incidence of necrotizing enterocolitis [75-79,93]. However randomized trials (SUPPORT) and benefits of oxygen saturation targeting (BBOT), the best oxygen profiles to reduce ROP while optimizing the health of preterm and their development remain unknown [94-96]. Neurodevelopmental impairment may be observed among extremely premature infants at 18-22 months of age [93]. However SpO_2 85-89% in extremely preterm was associated with increased mortality at the time of discharge compared to higher 91-95% (19.9% vs 16.2%; $p=0.045$) [97].

Initiation with room air remains controversial as current Neonatal Resuscitation Program (NRP) guidelines suggest using air or blended oxygen to titrate oxygen to meet preductal saturation SpO_2 at 85-94%, but there are no studies to justify any particular starting oxygen concentration [98]. In LNR maximum FiO_2 of upto 68% is administered through sustained nasal inflation and at no time is 100% oxygen administered except with endotracheal intubation with Grade V Terminal apnea with poor circulation resulting in low arterial oxygen saturation. These asphyxiated babies are subject to high mortality rates though to date no randomized trials [98] of strategies to achieve the NRP-recommended interquartile range of preductal saturations have been conducted in preterm neonates, perhaps maintaining SpO_2 around 95-96% would be ideal in preventing both hypoxia and hyperoxia.

Pulse oximetry SpO_2 readings follow the Gaussian curve of normal distribution and equipment bias, within ± 1 Standard Deviation (SD) of true arterial oxygen saturation 68% of the time, then SpO_2 90% is 1% on equipment bias then true arterial saturation will be 89%, 90%, or 91%. If monitor has 3% bias then SpO_2 90% will be between 87% and 93% for 68% of the time and as such, a difference of 1-2% may be inconsequential. The alarm limits should preferably be set at 1% or 2% above or below the chosen target range, high alarm should be set at 95% and lower limits should be set at $\geq 85\%$ when breathing supplemental oxygen with $FiO_2 > 0.21$ with careful attention on averaging and sensitivity of monitors.

Since there is no definite conclusive evidence for ideal oxygen saturation in extremely premature newborns, it is perhaps best to avoid both hypoxia as well as hyperoxia by maintaining oxygen saturation of SpO₂ around 95% to 96% [99,100, 101].

The assessment of peripheral oxygen saturation by Pulse Oximeter was developed by Takuo Aoyagi in 1935; specifically monitors' peripheral arterial blood is a small non-invasive portable device, of particular value in neonatal units to prevent hypoxia and hyperoxia [61]. It is also known as the fifth vital sign, the first four being temperature, pulse, respiratory rate and blood pressure (TPR and +BP). The Pulse oximeter probe consists of two parts, Light Emitting Diodes (LEDs) and light detector called photo-detector which notes how much light at each red and infrared wave length has been absorbed to determine the ratio of the two wave lengths.

Light is emitted from light source passes through the body part to a photo detector that measures amount of light absorbed according to Beer's law that states that the amount of light absorbed is proportional to the concentration of light absorbing substance and Lambert's law, which states that the amount of light absorbed is proportional to the length of the path light has to travel in the absorbing substance. Red light is absorbed by deoxyhemoglobin and infrared light by oxyhemoglobin. The ratio of the absorbed red light and infrared light differs and the microprocessor calculates a value for the oxygen saturation in the pulsing arterial blood excluding venous blood, skin, bone, muscle fat etc. providing percentage of haemoglobin saturated with oxygen within seconds, being almost similar to arterial oxygen saturation (SaO₂) measured by Arterial Blood Gases (ABG) analysis [61-63].

Pulse oximeter also provides reading of heart rate, the increase in heart rate indicating adequate lung aeration and circulation of oxygenated blood. Also pulsatile change in absorbance due to pressure changes in arteriolar blood volume in the skin represented in graphical form is called plethysmography trace. The variation of pulsatile changes in transmission of light signal received by the sensor indicates cardiac function and how well the heart is pumping oxygenated blood through the body [61, 102].

The perfusion index quantifies the amplitude of the peripheral plethsmograph waveform and helps to predict early adverse respiratory outcome in neonates, also any irregularity of cardiac rhythm improves detection of critical congenital heart disease in newborns, is implemented as screening test for critical congenital heart diseases [102-104]. Pulse oximetry photoplethysmogram (PPG) is measurement of respiratory rate by application of digital band filters to allow the removal of cardiac component on the PPG waveform. PPG signal should have two distinct peaks, one low frequency corresponding to respiratory rate and another higher frequency corresponding to heart rate. A recent method titled 'ARspec' (Acute Regressive specral median) yielded most reliable respiratory rate estimation [105].

Apgar's simple clinical practical score at 1, 5 and 10 minutes of birth, 0-3 severe asphyxia, 4-7 as moderate asphyxia and 8-10 in normal healthy newborns in terms of cardio-respiratory status, reflex irritability, muscle tone and colour of newborn, is now rendered redundant and obsolete as it is subjective and poorly reproducible and as such the need for active resuscitation being a specific sign of delayed onset of respiration could therefore indicate recent cerebral injury especially hypoxic-ischemic [106-108]. Also umbilical blood gases for metabolic acidosis is difficult, as pH below 7.18 and a base excess more negative than -8 despite being indications of oxygen deprivation in newborn is a poor predictor of significant perinatal brain injury [109]. While Pulse oximetry automatically provides condition or status of newborns within seconds, significantly detecting more cases of hypoxia requiring immediate resuscitative measures [63]. Pulse oximeter also guides oxygen flow rate based on grading I-V among hypoxic newly borns [70].

The definition for birth asphyxia due lack of oxygen during antepartum, more so intrapartum period or just before birth as mild, moderate, severe birth asphyxia, secondary and terminal apnea based on Grade I-V classification respectively within 20-60 seconds of birth monitored by pulse oximetry scoring of SpO₂. Pulse oximetry scoring of SpO₂<96% is an accurate indicator of hypoxia in newborns as delayed onset of respiration indicate recent cerebral injury especially hypoxic-ischemic. Resuscitation best achieved by sustained positive airway pressure with nasal inflatory oxygen at flow rates 2-15 L/min for 1-3 minutes result in uniform lung aeration with increased pulmonary compliance, regular breathing and prompt increase in heart rate and cardiac output with circulation of oxygenated blood throughout the body [70].

The practice of resuscitation IPPV with bag and mask or endotracheal intubation was undertaken at Christian Medical College and Hospital, (CMCH), Vellore, South India, the then, premier institution in South East Asia, decades earlier to my post graduate residency in early eighties at the neonatal unit, that was later implemented world-wide after introduction of 'Neonatal Resuscitation Program' (NRP) published in 1988 [30]. I immediately resuscitated severely asphyxiated newborns by endotracheal intubation with intermittent positive pressure ventilation and adjunct therapy of epinephrine as well as sodium bicarbonate administered in ventilated breathing newborns for severe perinatal asphyxia with Apgar score of 0-3 at 1 min to 5-10 min. However if newborn is properly oxygenated, metabolic acidosis due to anaerobic glycolysis will rapidly resolve, precluding use of hypertonic bicarbonate solution that may overload circulation causing increase risk of cerebral haemorrhage [110]. Though Carbicarb (equimolar mixture of sodium bicarbonate and sodium carbonate) may also be used but has no added advantage [111].

It wasn't until 2016 that I discovered the application of continuous positive pressure ventilation by sustained nasal oxygen inflation at flow at rates of 8-15 litres/min resulted in

almost immediate revival of the central respiratory centre, initiating rhythmic respiration resulting in minimal ill or none adverse hypoxial injury in newborns, who on observation for four hours in Neonatal Intensive Care Unit (NICU) fared well, were then shifted to mothers side, breast feeding initiated and discharged 3-5 days later. I have since used LNR with sustained nasal oxygen inflation successfully in all asphyxiated newborns till date, without the resort to IPPV by bag and mask ventilation or endotracheal intubation and good neonatal outcome.

I earlier published a study from CMCH, Vellore that reported high Perinatal Mortality Rate (PMR) of 40.7/1000 among 21,585 consecutive total live-births during 1979-1983, being one-half contemporary national PMR of around 80/1000 total births, CMCH is a tertiary referral centre for high-risk cases. The stillbirth rate (SBR) 23.6, featured higher, one and a half times more than Early Neonatal Mortality Rate (ENMR) of 17.5 per 1000 live births [112]. There were 509 stillbirths accounting for 58% of 878 perinatal deaths with 369 Early Neonatal Deaths (ENDs), indicating that most asphyxiated babies died before birth as fresh stillbirths and nearly half 48% (n=119/369) within two hours of life as early neonatal death suffering severe perinatal asphyxia who would have been fresh term stillbirth were it not for obstetric intervention often with emergency LSCS with then skilled resuscitation by IPPV using bag and mask or endotracheal intubation etc. could therefore constitute a total 72% stillbirths of perinatal deaths. The adage “masterful inactivity and watchful expectancy” proves detrimental in ethnic Asian-Indian population with high perinatal mortality rates, hence active management of labour with quick obstetric intervention is important to rescue endangered fetuses, which has however resulted in a sharp increase of LSCS from 14% in the eighties to 42% in twenties, as fetal distress was the commonest indication for emergency LSCS indicating undetected CPD in cases of prolonged labor [113,114].

Among 369 early neonatal deaths within the first seven days of life, birth asphyxia comprised 31.4% (116/369) ENDs being the leading cause in 1979-1983 at CMCH Vellore. Majority 41.7% (154/369) were severely asphyxiated with Apgar scores 0-3 at 1 min of birth, only one-third 32.8% were lethally malformed constituting an absolutely unavoidable cause of perinatal deaths, ten other newborns died later of intracranial haemorrhage, nine had hyaline membrane disease, five fulminant sepsis, two massive meconium aspiration syndrome and one second birth, infant had hydrops fetalis [7].

First day deaths constituted two-thirds 67.2% (n=248/369) of ENDs, main causes being severe birth asphyxia 30% (n=74/248) followed by Lethal Congenital Malformation (LCM) 24% (n=59/248) and Respiratory Distress Syndrome (RDS) 21% (n=52/248) while intracranial haemorrhage (ICH) 7.6% (n=19/248) and neonatal infections 3.2% (n=8/248) ranked fourth and fifth with remaining ‘miscellaneous’ 14.5% (n=36/248) including extreme prematurity, pulmonary haemorrhage, liquor aspiration and Rh iso immunization, constituted

first day deaths [8]. Distribution of first day deaths according to time in hours revealed nearly half 48% (n=119/248) took place within two hours of birth, 29% (n=72/248) between 2-12 hours with 23% (n=57/248) in the next 12-24 hours [8]. Other studies have noted that more than half, 57% of neonatal mortality occurred within 24 hours [115]. Seven out of every eight stillbirths is due to severe birth asphyxia with stillbirth rate figuring one and a half times that of early neonatal mortality rate, reveals the magnitude of asphyxiated stillbirths [5,8,112], emphasizing that birth asphyxia continues to be a major cause of preventable proportion perinatal and neonatal deaths and that the neonatal period, more so the first one week, especially within the first 24 hours of life being critical for survival in the life of a child with prevailing high mortality rates predominately in developing Asian countries. I also observed during my neonatal residency at CMCH, Vellore that more babies born by normal vaginal delivery were hypoxic when compared to those delivered by LSCS, though those delivered by outlet forceps delivery, cutting short the second stage of labor had least perinatal mortality rate [113].

However during 1982 increased sensitization of obstetrician to fetal hypoxial injury by avoiding prolonged labor with active management of labor and judicious obstetric intervention, reduced the incidence of birth asphyxia which ranked fourth with lethal congenital malformations, an absolutely unavoidable proportion of perinatal mortality ranking as the first cause, followed by respiratory distress syndrome and intracranial haemorrhage as second and third cause of early neonatal deaths. Another observation noted during 1982 was an increase in outlet forceps deliveries indicating judicious obstetric intervention, associated with least PMRs [113].

The low mean birth weight in ethnic Asian-Indian population among 4426 live births in 1983 cohort was 2881 g, compared to 2873 g three decades later among 2750 singleton live births in 2015-'17 cohort being almost similar. The decrease of -8 g is attributed to a shift in demography with small family norm and over 50% being young primigravida mothers having lower birth weight babies [114,116]. The distribution of birth weight between Asian and Caucasian newborns is remarkable, Caucasian newborns have higher mean birth weight of 3470 g reported in a British study, most 82.7% weighed above 3000 g with only 17.3% weighing below 3000 gm [117] that contrasted to low Asian mean birth weight of 2874 g with around one-fourth 27.7 % weighing more than 3000 g and a high 72.3 % weighing 3000 g and below. In fact almost two thirds 65.6% of Asian Indian births weighed between 2000-3000 g, while almost similar 68% Caucasian births weighed higher 3000-4000 g, indicates why ethnic Asian population have to set standards and perinatal definitions of their own for international comparison [114,117,118].

Norway reported highest mean birth weight of 3575 g being statistically significant (p=0.001) when compared to other Asian countries such as D.R. Congo, Egypt and Thailand with 400 g

less birth weight median, while Argentina, Brazil and France had birth weight less than 200 g and Denmark, Germany with mean birth weights approximately 100 g less. WHO has also observed that differences in birth weight when adjusted to gestational age at birth between other countries is highly significant for all percentiles at birth, $p=0.0018$ at 5th percentile to $p<0.001$ for 10th, 25th, 50th, 75th, 90th and 95th percentiles [119], reveals the wide variation in human fetal growth across ethnic Asian and Caucasian population, indicating that Asian and Caucasian perinatal definitions are mandated, taking into consideration the wide variation in mean birth weight gestation at birth and intrauterine growth pattern with new perinatal guidelines based on ethnicity that will result in improved perinatal and neonatal outcome as ethnic Asian population presently comprises majority four-fifths of world's nearly 8 billion population [120, 121].

The Asian-Indian peak births 32.6 % in 1983 cohort occurred at 39 weeks gestation with mean gestation of 38.86 weeks $SD \pm 1.29$ weeks while peak births 27.4% among 2015-'17 cohort took place at 38 weeks with mean gestation of 38.2 $SD \pm 2$ weeks, contrasted with Caucasian births which peaked 31% at 41 weeks gestation, mean gestation of 41.03 weeks $S.D. \pm 1.32$ weeks, with a highly statistically significant difference of 3-4 weeks ($p=0.001$), while Norway reported mean gestation of 40 weeks 3 days [114,116,119]. The shortened gestation of 38 weeks at peak births among Asian-Indian babies results in smaller babies with lower average birth weight 2881 g when compared to Caucasian newborns who with a longer gestation upto 42-44 weeks continue to gain weight by deposition of subcutaneous fat have birth weight around 3500 g [118,117,122].

The early peak births at 38 weeks gestation in 2015-17 study compared to 39 weeks in 1983 study is due to judicious quick surgical obstetric intervention in rescuing jeopardized fetuses that resulted in increased incidence of LSCS deliveries 41.6 %, when compared to low 14.8% during the 1979-1983 [1183,1140. Not only shortened gestation but also decreased intrauterine fetal growth potential contributes to the low mean birth weight in ethnic Asians as intrauterine growth curves for Asian- South Indian newborns, constructed for 1983 cohort when compared three decades later to 2015-'17 cohort revealed almost similar low growth potential for 10th, 25th, 50th, 75th, and 90th percentile curves till 32 weeks gestation, thereafter the 2015-'17 study gained 100-300 g weight from 32 to 37 weeks, following which there was catch-up growth by the 1983 cohort at 40 weeks with 200-300 g growth spurt 40-42 weeks gestation and thereafter 500 g in 2015-'17 study demonstrating an inherent genetic predisposition at play rather than environmental factors despite vast increase of socioeconomic reforms and technological advances in the country [116].

The comparison of national and international intrauterine growth chart revealed that the 10th percentile in the 2015-'17 South Indian cohort and the All India National Neonatal Perinatal Data base (NNPD) study with Lubchenco's were closely related, thereafter the Lubencho

curve diverged from 32-33 weeks gestation with rapid weight gain of around 500 g at 37 weeks, then decreased by 200- 300 g weight gain at 40 weeks gestation. However the 50th percentile curve in 2015-'17 study and 50th percentile NNPD study revealed low growth potential corresponded to the 10th percentile International WHO curve [116,123,124].

However though the 90th percentile growth curve in 2016-'17 Indian study was similar only till 32 weeks gestation to 90th percentile World Health Organization (WHO) curve, it thereafter declined to below the 50th percentile WHO curve at 40 weeks in contrast the 90th percentile WHO curve continued to gain more than 1000 g at 40 weeks gestation [116,119]. Also the 90th percentile all India NNPD had the least growth potential corresponding to 50th percentile WHO curve up till 37 weeks gestation and thereafter fell by over 250 g less but had a catch-up growth to the 90th percentile South Indian 2015-'17 curve at 40 weeks gestation [119,123].

Comparison of the 10th percentile intrauterine growth curve reported in UK study corresponded to the 50th percentile of 2015-'17 South Indian curve till 36 weeks, which thereafter flattened by almost 800 g to meet the 10th percentile of 2015-'17 South Indian curve at 41-42 weeks gestation. However the 50th percentile South Indian curve was lower by around 200 g compared to 50th percentile UK curve at all gestation, though the 90th percentile South Indian curve corresponded to 90th percentile UK curve till 38 weeks, the UK curve thereafter increased by almost 500 g greater weight gain by 41-42 weeks gestation [116,125]. The 50th percentile US curve though similar to the 50th percentile South Indian curve till 30 weeks, thereafter diverged with rapid intrauterine fetal growth up to 34 weeks corresponding to 90th percentile Indian growth curve and gaining more than 1000 g at 40 weeks gestation [116,126].

Birth weight for gestational age is a commonly assessed perinatal outcome parameter and Small for Gestational Age (SGA) defined as weighing less than 10th percentile of birth weight for that gestation is also an indicator for Intrauterine Fetal Growth Restriction (IUGR), its importance is due to high associated perinatal and infant morbidity and mortality as well as future adult chronic non-communicable diseases such as cardiovascular disease, stroke, type II diabetes, obesity, other endocrine and metabolic disorders prominently linked to Small for Gestational Age (SGA) [127, 128].

Thus invalidating international reference WHO percentile intrauterine growth curves which differs from the Indian intrauterine growth curves such that the 10th percentile is almost 800 g below WHO 10th percentile curve, while 50th percentile South Indian curve corresponds to 10th percentile WHO curve, that would mistakenly identify a large proportion of Appropriate for Gestational Age (AGA) Indian babies as Small for Gestational Age (SGA) who have differing morbidity and mortality, emphasizing further why ethnic Asians require specific intrauterine growth charts to avoid inaccurate labelling of SGA newborns and their

management, however these Asian small for dates after birth feed avidly and gain weight [116,119]. Also pooling of data as in the international WHO intrauterine growth curves do not represent variations among populations in a single intrauterine growth chart but only partially reflect the individual population included [119], hence ideally two separate ethnic Asian and Caucasian intrauterine growth charts are recommended [116,125,126].

Therefore despite the vast technological and economic revolution that has influenced all sections of society in India including improved obstetric care, ethnic Asian fetuses will continue to have low intrauterine growth velocity with low average birth weight as a result of asymmetrical intrauterine growth retardation as well as shortened gestation when compared to Caucasian counterparts is primarily due to inherent genetic predisposition [116,122,124]. Hence ethnic Asian specific intrauterine growth curves is mandated for accurate identification of small for dates for institution of early management of treatment of complication for SGA as well as LGA newborns [116,129].

I have also earlier reported incidence 21.2% of birth asphyxia, comprising 583 cases among 2750 singleton live births who required resuscitation at birth to establish rhythmic respiration, majority were born at 39 weeks gestation by emergency LSCS, OR 4.91, [CI 95%] 3.94-6.10 times compared to normal delivery being highly statistically significant $P=0.0001$. In contrast elective LSCS deliveries was associated with low risk 9.1% of birth asphyxia with OR 1.67 [CI 95%] 0.84-1.63, not statistically insignificant $P=0.358$. Though vacuum extraction comprised 11% of births, it was associated with a significantly higher risk of birth asphyxia, OR 8 [CI 95%] 5.58- 11.69, being highly statistically significant $P=0.0001$ [9].

Also Meconium Staining of Amniotic Fluid (MSAF) was associated with 84% asphyxiated newborns, OR 8.42 [CI95%] 5.1-14 being 30 times higher compared to newborns with clear liquor ($P=0.0001$) [129]. One-fifth 21% newborns with MSAF develop MAS, one-third requiring intubation and mechanical ventilation. MSAF is usually present in 8-20% of all deliveries increasing to 23-52% by 42-44 weeks of gestation but rarely found in amniotic fluid before 34 weeks gestation. MSAF with increasing gestation beyond 39 weeks, predisposes to maternal complications such as meconium laden amniotic fluid embolism, intra-partum chorio-aminioitis, puerperal endometritis, wound infection etc. with increasing morbidity and mortality in both the newborns and their mothers. Peak delivery 38% of newborns with thick MSAF, 35% MAS, and 32% newborns with thin MSAF occurred at 39 weeks delivered mainly by emergency LSCS [130-134].

WHO has stated that LSCS performed when necessary can effectively reduce maternal and neonatal mortality, the ideal rate of LSCS being 10% for a given population with a rise towards is 10-15%. However if LSCS rates go above 10% there is no evidence to indicate that mortality rates will improve. Though emergency LSCS is a boon for mothers and babies, there is no similar evidence for elective LSCS, which in fact could become life threatening.

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However the benefits of elective LSCS in high risk cases is associated with improved neonatal and maternal outcome and other benefits such as decreased perineal pain and urinary incontinence at three months [135].

In India, the National Family Health Surveys (NFHS) 4 and 5 in 2015 and 2019 respectively reported an overall incidence of 17.5% LSCS, being higher than WHO recommended limits with a high 60.7% in Telangana followed by 42.2% in Andhra Pradesh, 41.7% in Jammu and Kashmir, 39.5% in Goa and 37.6% in Ladakh [136,137]. I reported LSCS of 42% in Bangalore in 2015-'17 with up to 80% in some centres [114,138]. In fact Turkey has the highest caesarean section rate of 54.9% followed by Korea 45%, Poland 38.9%, US 32%, UK 28.5%, Canada 27.7% [139].

Thus it may be observed that caesarean section rates are highest in Asian countries as compared to western countries due to the increased vulnerability of ethnic Asian foetuses to asphyxial birth injury often with prolonged labor due to undetected CPD during vaginal delivery as compared to Caucasian foetuses who have remarkably low perinatal mortality rates, having almost eliminated birth asphyxia with low incidence of around 1% [43, 53,114,136].

Planned elective LSCS or active management labor and cutting short second stage of labor with outlet or low perineal forceps delivery in high risk cases at 38 weeks gestation will obviate complications of later delivery by 39 weeks and beyond by decreased resort to emergency surgical intervention will not only envision reduction in incidence of emergency LSCS but also improvement in outcome of both mother and baby with significant resultant fall in perinatal, neonatal and maternal mortality and morbidity [9,113,114].

Intrapartum events with placental insufficiency during labour contractions results in maximum decrease in fetal oxygen saturation especially during the latter part of the second stage of labour as fetal blood supply is diminished by uterine contractions or terminated by cord compression, resulting in asphyxia [44]. In the fetus deoxygenated blood with low oxygen saturation of 25-40% passes through umbilical arteries to the placenta which is the organ of gas exchange returns to fetus through umbilical vein with high oxygen saturation of 80-90% is first delivered to brain and myocardium as circulation is 'shunt dependent', also fetal haemoglobin (HbF) helps to maintain oxygen delivery due to shift in left of oxygen disassociation curve which after birth proves disadvantageous with impairment of oxygen extraction at tissue level [81,89].

During labor the mean fetal SpO₂ decrease to around 45-50% and during last one hour of delivery if SpO₂ falls below <30% correlates highly with fetal acidosis in cases with non-reassuring fetal heart rate [44,]. However after birth, organ of gas exchange shifts to lungs with 8-10 fold increase in pulmonary blood flow with fall in pulmonary vascular resistance as

better oxygenation of neonatal blood increases pulmonary compliance by reversing pulmonary vasoconstriction caused by hypoxia [16,17,57]. Therefore a low SpO₂ below 30%-50% may be expected if the asphyxiated newborn fails to establish breathing after birth mandating immediate resuscitation with oxygen to quickly reverse hypoxial injury and its ill sequelae or death. Thus all foetuses do experience some degree of hypoxia during labor contractions, and its intrapartum events that has more impact than antepartum factors as various studies show placental insufficiency during labor contractions, causing maximum decrease in fetal oxygen saturation recorded 92 seconds after peak contraction, taking approximately 1 minute and 30 seconds (P=0.036) to recover [44,141,142].

Hence intra-placental pressure exceeds maternal perfusion pressure thereby decreasing oxygen supply to the fetus, recorded by scalp electrode, intrauterine pressure catheter and fetal pulse oximeter, sensor placed in front of ear [143]. Placental blood flow may be completely arrested if the intra-placental pressure >30 mmHg exceeding perfusion pressure of maternal blood, also inadequate relaxation or uterine tetany resulting from administration of oxytocin does not permit placental filling. Angiographic studies reveal that uterine contraction blocks circulation through intervillous spaces with transient fetal hypoxemia monitored by intravascular oxygen electrode [145].

Placental insufficiency will decrease oxygen supply to the fetus during contractions and intravascular oxygen electrode for continuous fetal arterial PO₂ reveals transient fetal hypoxemia following uterine contractions [143]. Fetal infrared spectroscopy also demonstrated a fall in cerebral oxygenated hemoglobin after a contraction with decrease in oxygen transfer to the fetus resulting in lower oxygen levels and pH, noted more so at the end of labor rather than at the beginning, compounded by maternal pain, breath holding and maternal metabolic acidosis further reducing oxygen delivery to the fetus [44,144]. Thus uterine contractions affect uteroplacental blood flow with increase in intravillous pressure causing fall in placental perfusion resulting in decreased return of oxygenated blood to the fetus followed by transient increase in fetal oxygen saturation as the intraplacental pressure exceeds the perfusion pressure of maternal blood, thereby placental blood flow is greatly reduced and may be completely arrested if intravillous pressure is above 30 mmHg [44,141,142].

Scalp blood analysis may show acidosis with a pH of less than 7.20, comprising both of respiratory and metabolic components [146]. Fetal heart rate monitoring reveals variable late (type II dips) deceleration pattern without any variability in response to fetal movements or uterine contractions [147,148]. Consistent slowing or late deceleration of the fetal heart rate following termination of each uterine contraction is indicative of uteroplacental insufficiency also considered as positive oxytocin challenge test requires supplemental oxygen to mother before delivery [145,147]. If fetus is at high risk for asphyxia due to inadequate supply of

oxygen from the placenta detected during labor presenting with fetal distress, then emergency delivery may be attempted preferably by caesarean section or alternatively by outlet forceps delivery if head is in perineum.

Fetal anoxia few minutes to few days before delivery may be indicated by sudden increase in fetal activity followed by diminished activity or at delivery include abnormal heart rate and metabolic acidosis, with gasping respiration or absent breathing at birth with bradycardia, pale color, poor muscle tone with weak reflexes or altered sensorium requiring effective resuscitation to establish rhythmic respiration. Thus bradycardia with weak, irregular beats is indicative of fetal anoxia, usually observed a few minutes to a few days before delivery associated with sudden increase in fetal activity followed by diminished activity [16,17].

The longer the hypoxic episode the more severe the sequelae with secondary and terminal apnea require immediate effective resuscitation to avoid death by sustained nasal inflatory oxygen administration at 16 to 20 cm of H₂O for 1 to 2 seconds or flow rates of 12-15 L/min for 1-3 minutes [19-21,70]. In the initial stage of primary apnea, the fetus gasps in utero, for a short period, heart rate and blood pressure may remain constant or become slightly elevated, following which, after an interval of a few minutes the fetus commences a second period of gasping and enters terminal stage or secondary apnea wherein the heart rate and blood pressure fall quickly and satisfactory oxygenation of peripheral tissues is not possible due to decrease pulmonary perfusion furthering hypoxia with hypercarbia and acidosis that interferes in transition of fetal to neonatal cardiopulmonary circulation since pulmonary arterioles remain constricted, perpetuating right to left shunt through ductus arteriosus. Apnea is defined as cessation of breathing greater than 20 seconds and most asphyxiated infants born with primary apnea will commence spontaneous respiration if given air or supplemental oxygen to breathe, while secondary or terminal apnea require immediate resuscitative measures best achieved by LNR to establish rhythmic respiration [15,16].

Birth asphyxia initiates diving reflex; causing shunting of blood to the brain; heart and adrenals with reduce flow or hypo perfusion in organ system of lungs, gut, liver, kidney, spleen and skin. The redistribution of fetal blood in mild hypoxia with increase in heart rate and blood pressure maintains blood flow to brain, heart and adrenals with hypo perfusion of lungs, gut, liver, kidney, spleen and skin. However, satisfactory oxygenation of peripheral tissues is not possible due to decrease pulmonary perfusion furthering hypoxia with hypercarbia and acidosis, interferes in transition of fetal to neonatal cardiopulmonary circulation since pulmonary arterioles remain constricted, perpetuating right to left shunt through ductus arteriosus [16-18].

In mild hypoxia there is increase in blood pressure and heart rate to maintain cerebral perfusion - the brain sparing effect [16-18, 89]. In the initial stage of primary apnea, the fetus gasps in utero, for a short period, heart rate and blood pressure may remain constant or

become slightly elevated, following which, after an interval of a few minutes the fetus commences a second period of gasping and enters terminal stage or secondary apnea wherein the heart rate and blood pressure fall quickly and if not immediately resuscitated, will die at birth [16]. Apnea is defined as cessation of breathing greater than 20 seconds and most asphyxiated infants born with primary apnea will commence spontaneous respiration if given air/oxygen to breathe, while secondary or terminal apnea requires immediate resuscitative measures best achieved by LNR to establish rhythmic respiration [15,16].

Also, hypoxemia and acidosis cause ATP pump to fail, depleting energy reserves, among all sources of energy glucose is the sole source of energy capable of sustaining metabolism in the brain under conditions of total cerebral ischemia, because of its capacity for consumption via anaerobic glycolysis with the production of lactic acid and ATP. However, during anaerobic conditions, one molecule of glucose yields only 2 mols of ATP as opposed to 38 molecules of ATP during aerobic metabolism [75,80]. ATP depletion causes primary cellular failure and ultimately secondary energy failure due to mitochondrial dysfunction causing apoptotic cell death [17, 2, 4,10]

Production of lactic acid due to the anaerobic metabolism remains in the tissue because of poor perfusion. The concomitant acidosis leads to decreased heart rate and cardiac output with decreasing blood pressure leading to cell damage and functional abnormality - such as renal failure in the kidneys, necrotizing enterocolitis in the gut associated with high mortality rate is due to repeated or persistent redistribution of blood flow causing ischemia of the intestines in cases of severe or prolonged hypoxia with birth asphyxia or intrauterine growth restriction compounded by immature auto regulation of intestinal blood flow and enteral feeding with resulting increased metabolism predisposes to necrotizing enterocolitis more so in gut of preterm with decreased immunological defences and bacterial overgrowth [79], hypoxic myocarditis due to cardiac ischemia with metabolic acidosis results severe cardiac malfunction in impaired function and decreased contractility and cardiac arrest due to decreased ATP generation with mitochondrial hypoxic induced injury [60,68,75], Impaired myocardial function results in reduced cerebral blood flow.

Hypoxia and glycolysis during perinatal hypoxia-ischemia causing anaerobic glycolysis inhibits transport of glucose across the blood brain barrier into the brain being the sole metabolite utilized by brain and adequate ATP production relies primarily on aerobic glycolysis with cerebral dysfunction [29,149]. Perinatal asphyxia causes severe neurological deficits approximately 45% newborns die [150] and 25% survivors have permanent neurological deficits [151,152] such as cerebral palsy, developmental delays, visual and hearing problems [77], learning deficits or disabilities, and school readiness [38,39]. Decreased pulmonary perfusion, abnormalities of gas exchange and persistent pulmonary hypertension in the lungs further perpetuates hypoxia [16,57,60,].

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Asphyxia has been demonstrated while attempting to reproduce events of human asphyxia in the monkey; causes two patterns of brain damage in animals, firstly acute total asphyxia produce neuronal necrosis of the brain stem nuclei and secondly partial prolonged asphyxia results in necrosis of cerebral hemisphere [153]. However sequence of events in pathogenesis of intrauterine asphyxia in a full term neonate causes redistribution of organ blood flow, result in oxygen debt to brain cells, impaired auto regulation of cerebral blood flow, intracellular swelling, leading to focal ischemia, generalized brain swelling, increased intracranial pressure, causing cerebral necrosis and atrophic cortical sclerosis, significant intrapartum asphyxia usually results in the birth of an infant with depressed cardiac respiratory function [154].

Types of asphyxia brain damage include haemorrhagic lesions in germinal matrix, choroid plexus, cerebellum and pial matter, destructive lesions of white matter with leukomalacia of periventricular, subcortical and telencephalon areas as well as grey matter areas such as pontosubiculum, cerebral cortex, basal ganglia, thalamus and brain stem [149], during reperfusion injury with decrease in cerebral perfusion, deprivation of cerebral oxygen and glucose supply leads to ATP depletion and loss of $\text{Na}^+/\text{K}^+/\text{-ATPase}$ activity [29].

Autopsy study in preterms with repeated prolonged apnea more than 20 seconds and cyanosis revealed diffuse neuronal loss in cerebral cortex, leukomalacia in periventricular watershed zones while full terms with hypoxic episodes between 2 to 52 weeks of age noted subcortical leukomalacia related to border zones with tenuous arterial blood supply from anterior, middle and posterior cerebral arteries and in preterm as auto regulation of smooth muscle tone in vessel is not present is mainly dependant on systemic blood pressure hence these areas are extremely susceptible to hypoxic injury [155]. The mechanism of intraventricular haemorrhage is probably that an asphyxial episode causes vascular constriction followed by vascular dilation with increased intravascular pressure which ruptures vulnerable vessels in the germinal matrix especially in the periventricular area in the immature-brain [156].

Various other criteria recommended by AAP and ACOG for severe birth asphyxia include: (I) Profound metabolic or mixed acidemia, with an umbilical artery pH <7.00 , (II) Apgar score of 0-3 beyond 5 minutes, (III) Neurological involvement such as convulsions, unconsciousness and hypotonia, (IV) Multi organ system dysfunction involving various systems such as CVS, GI, kidneys, lungs etc [157]. While Sarnat and Sarnat classifies Hypoxic Ischemic Encephalopathy (HIE) as mild, moderate or severe into stages I, II or III based on level of consciousness, neuromuscular control, tendon and complex reflexes, gastrointestinal motility, presence or absence of myoclonus, electrography findings and autonomic functions, however these parameters have no predictive value for long-term neurologic injury after mild to moderate asphyxia is no longer validated as accurate assessment of birth asphyxia can be monitored by pulse oximeter [158].

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Nearly 20-40 percent of perinatal deaths are attributed to birth asphyxia especially in Asian countries [4,5]. Strictly speaking stillbirths are fetal deaths nevertheless even live born neonate who is apneic and cyanotic with pulse is set aside after birth and left to initiate respiration, or are inadequately resuscitated and die are classified as stillbirths as unskilled birth attendants may not be able to distinguish between the two conditions with inaccuracy of recording these fatalities with high stillbirth rates. Hence misclassification of stillbirths has significant implications of national health policies and global strategies for reducing perinatal mortality since effective resuscitation of viable newborn, would lower stillbirths rate, in addition a large number of stillbirths are unregistered [13,14].

The disadvantages of effective neonatal resuscitation by Neonatal Resuscitation Program (NRP) includes the requirement of highly trained skilled birth attendants and well outfitted resuscitation teams and as such is not universally applicable in many parts of low income Asian countries with limited resource settings and unskilled birth attendants who lack essential resuscitation equipment and in addition bulb syringes, bag and mask devices may be substandard [30-32].

Lalana Newborn Resuscitation is a new novel non-invasive approach in management of all cases of birth asphyxia requires minimal infrastructure for oxygen supply from either piped oxygen of cylinder with flow meter and wide bore oxygen tube requires minimal training of even unskilled birth attendants. The increase in airway and alveolar pressure by sustained nasal oxygen inflation even as baby starts breathing efforts helps improve oxygenation in hypoxemic respiratory failure [70].

Thus perinatal asphyxia results in lack of oxygen in newborns with hypoxemia with hypercapnia that causes rise in arterial carbon dioxide reduces blood flow to the brain causing ischemia and altered mental status [60]. LNR will prove to be of vital importance by improving individual outcome as quick oxygenation of tissues by initiating regular breathing pattern reverses hypoxial insult, not only reviving viable apneic newborns who otherwise would be termed as stillbirths but also saving millions of lives by reducing asphyxial neonatal deaths with minimal or none or minimal residual sequelae in survivors, so children should be normal [70].

India with a population of over 1.3 billion has 124, 419, 96 thousand births each year [55,56], approximately 10% of newborns require assistance to breathe with incidence of birth asphyxia ranging around 20-30 percent [8,159] with 6.61 lakh newborn dying in the neonatal period of whom 5.1 lakhs die within the first week of life with Early Neonatal Mortality Rate (ENMR) 20 per 1000 live births and Neonatal Mortality Rate (NMR) 26 per 1000 live births is concerning, however in India NMR is not uniform across the country with Kerala and Tamil nadu with low NMRs of below 20/1000, Odisha, Madhya Pradesh and Uttar Pradesh have high NMRs of more than 35/1000, though Haryana and Gujarat have similar or higher

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per capita GDP than Tamil Nadu but almost double NMR. In fact four states, Uttar Pradesh, Madhya Pradesh, Bihar and Rajasthan alone contribute to 55% of total neonatal deaths in India and up to 15% of global neonatal deaths occur every year [160].

Nigeria ranked second with 270, 000 newborn deaths being almost one half neonatal deaths reported in India, next Pakistan 248, Ethiopia 99, Democratic Republic of the Congo 97, China 64, Indonesia 60, Bangladesh 56, Afghanistan 43 and United Republic of Tanzania 43 per 1000 live births [160-162], indicating that strategies aimed at reduction of early neonatal deaths will substantially reduce under-five child mortality rate, perinatal mortality rate being a sensitive indicator for monitoring health care status. Lesotho (South Africa) now leads with highest neonatal mortality rate of 42.8 deaths per 1000 live births during 2019 [163]. Iceland has the lowest rate of 0.7 deaths per 1000 live births and ranked as No. 1 [165].

Thus birth asphyxia is the leading preventable cause of neonatal deaths compounded by the high stillbirth rate, those who would have had a good chance of healthy life on survival with effective resuscitation, however essential newborn care recommended by WHO reveals inadequacies in mother and child with only around four antenatal visits and skilled birth attendants is about 50% in 68 count down countries and high neonatal mortality comprising 52% of under five-year mortality [165].

Effective newly born resuscitation by LNR in many low to middle income Asian countries with limited resources would require minimal set up with availability of humidified oxygen regulated with flow meter and pulse oximeter to monitor peripheral tissue oxygenation (SpO_2), in fact portable oxygen cylinders even allows for effective domiciliary resuscitation, equipping even unskilled birth attendant, who may easily be taught detection of hypoxia/asphyxia of newborn based on pulse oximetry SpO_2 and the recording of heart beats per minute obviating the need for a stethoscope.

Simple classification of newborns should be done within 20-60 seconds of birth as 'Normal' or hypoxic newborns further graded into Grade I-V to determine sustained nasal oxygen flow rate varying from 2-15 L/min, while drying the baby, suctioning nose and mouth to clear the airway passage aided by tactile stimulation in initiating rhythmic respiration and normal heart rate 120-160 bpm, Oxygen flow to be discontinued with Pulse oximetry, zero score SpO_2 96%, body temperature control maintained with asepsis precautions and initiate early breast feeding practices [70]. In the present study LNR proved eminently effective in all 178 asphyxiated newborns resuscitated among 830 deliveries attended, three of whom had secondary apnea Grade IV and one newborn with terminal apnea Grade V initiated rhythmic respiration, smoothly transiting from fetal fluid filled lungs to well aerated neonatal lungs with vital cardiovascular transition to neonatal life.

Advantages of Lalana Newborn Resuscitation (LNR) with Continuous Positive Pressure Ventilation (CPPV) by sustained nasal oxygen inflation at flow rates varying from 2-15 litres per minute assessed by SpO₂ Pulse oximetry score

- 1). Resuscitation of the newly born is unique as the presence of fetal lung fluid prevents exchange of gases.
- 2). Sustained nasal oxygen inflatory flow provides continuous distending pressure that generates hydrostatic pressure to effectively overcome the high resistance of moving fetal lung fluid through the airways and across the alveolar wall into the interstitial tissue as well as opposes elevated interstitial pressure during expiration, preventing lung fluid from re-entering the airways promoting enhanced reabsorption of lung fluid.
- 3). Lalana Newborn Resuscitation (LNR) is safe and quick resuscitation of newborn by non-invasive technique based on CPPV by sustained nasal oxygen inflation meets the aim of effective resuscitation preventing neonatal death or adverse long term neuro-developmental sequelae in survivors, ensuring that children should be normal.
- 4). Continuous positive pressure ventilation results in uniform lung aeration, improved oxygenation with increased pulmonary compliance that increases Functional Residual Capacity (FRC), prevents atelectasis and maintains lung volume.
- 5). Stabilizing newborns with low fraction of inspired oxygen at birth is difficult since hypoxia is a potent inhibitor of spontaneous breathing; therefore increase in oxygen flow rate with higher FiO₂ determined by Pulse oximetry reading of SpO₂, help to overcome hypoxial insult with quick onset of respiration.
- 6). Oxygen is the only treatment for hypoxia that facilitates aerobic metabolic glycolysis and mitigates vagal-induced bradycardia resulting from perinatal asphyxia that perpetuates hypoxemia with hypercapnia or rise in arterial carbon dioxide, reducing blood flow to the brain with ischemia causing altered mental status.
- 7). The prompt increase in heart rate with improved cardiac output indicate adequate lung aeration and function resulting in left to right shunting triggering reflex physiological mechanism that converts fetal circulation to adult type.
- 8). Continuous Pulse oximetry monitoring gives real time assessment of newborns in maintaining SpO₂ at 96%-98%, mitigates hypoxial injury and cell damage causing multi-organ failure, neurological deficits or death.
- 9). LNR prevents both deleterious effects of hypoxia and detrimental hyperoxia especially in preterms with sustained nasal oxygen inflatory flow rate determined by Pulse oximeter SpO₂ and discontinued at SpO₂ 96%.
- 10). Lalana Newborn Resuscitation Protocol require minimal infrastructure for supply of humidified oxygen with flow meter as well as minimal training of even unskilled birth attendants, proves advantageous more so in low to middle income Asian countries

with limited resources wherein majority of the world's population reside accounting for 98% of global perinatal mortality rates.

Disadvantages of Neonatal Resuscitation Program (NRP) with Intermittent Positive Pressure Ventilation, using bag and simple mask or invasive endotracheal intubation

- 1). Short Intermittent Positive Pressure Ventilation (IPPV) is potentially harmful, causing un-even alveolar ventilation with increased susceptibility to lung injury as the entire tidal volume will only enter previously aerated regions which has important implications because during subsequent inflation air will first rapidly flow into and expand previously aerated lung regions due to much lower airway resistance.
- 2). IPPV is proven both scientifically and physiologically weak in effectively clearing lung fluid due to impaired generation of hydrostatic pressure while also permitting fluid to re-enter the airways with rising A-a gradient, poor oxygenation and circulation of deoxygenated blood to peripheral tissues.
- 3). Hypoxia is perpetuated due to increased pulmonary compliance with intermittent alveolar collapse during IPPV causing V/Q mismatch and right to left shunting of deoxygenated blood through unexpanded lung.
- 4). Peripheral oxygen saturation stated by NRP protocol by IPPV seems unacceptable advocating SpO₂ 40-45% at 1 min, SpO₂ 65-75%, at 2 min, SpO₂ 70-75%, at 3 min, SpO₂ 75-85%, at 4 min, SpO₂ 80-85% at 5 min, SpO₂ 85-95% at 10 min with long delay for hypoxic newborns to achieve SpO₂ 96%. Longer hypoxic episode result in more severe sequelae often leaving of survivors with permanent lifelong neurological deficits or multi-organ complications or death within the first few days.
- 5). Prolonged hypoxia predisposes to anaerobic metabolism and acidosis, impairs cardiac function resulting in bradycardia and poor peripheral circulation hindering smooth transition of fetal to neonatal circulation.
- 6). IPPV with bag and face mask or mouth-to mouth breathing is not effective as adequate trans-pulmonary pressure for adequate displacement of lung fluid is difficult to achieve as also because gastric distension occurs.
- 7). Also simple tight fitting of face mask or endotracheal intubation with FiO₂, up to 1.0 or 100% predisposes to hyperoxia with generation of oxygen free radicals, which have a deleterious role causing reperfusion injury contributing to eye (ROP), neurological and lung injury in preterm.
- 8). NRP requires the presence of a team of highly trained skilled birth attendants for bag and mask or invasive endotracheal intubation for Intermittent Positive Pressure

Ventilation, as more severely asphyxiated newborns require cardiopulmonary resuscitation or medication such as epinephrine or saline volume expanders etc.

- 9). NRP protocol is best adapted for resuscitation in cardiopulmonary arrest of infants, children and adults with previously well aerated lungs.
- 10). Neonatal Resuscitation Program (NRP) has been associated with remarkable decline in asphyxial deaths worldwide, however the degree of morbidity remains high affecting quality of life in nearly half of survivors as the rate of Hypoxic Ischemic Encephalopathy (HIE) has remained the same over the past decades [31-39].

It is the lack of scientific clarity regarding the physiology in transition of foetal fluid filled lungs to well aerated neonatal lungs, that is a major contributing factor why Neonatal Resuscitation Program (NRP) guidelines are regarded as weak [32] and the recognition that application of fast pressure to the airways in asphyxiated newborns with oxygen flow rates between 2-15 L/minute that provides Continuous Distending Pressure (CDP) allowing for uniform lung inflation and wide recruitment of alveoli [19-21], overcomes the high resistance of moving liquid through the airways to distal airway wall of alveoli, where it is absorbed into the lung interstitial and thence into lymphatic due to generation of constant hydrostatic pressure gradients between airways and lung tissue [22,23] which is not possible with IPPV which allows intermittent collapse of alveoli, atelectasis with lung fluid re-entering the airways [32]. Hence NRP is the anaesthesiologist's approach, ideally suited for resuscitation in infants with previously well aerated lungs in cases with respiratory or cardiorespiratory arrest unlike in newborn [166].

The pathophysiology of oxygen deprivation is hypoxemia with hypercarbia and acidosis result in lactic acid accumulation occurring within minutes causing cell damage and necrosis followed by 'reperfusion injury' with release of oxygen free radical by lactate dehydrogenase from damaged cells and metabolic acid imbalance with malfunction of mitochondria causing secondary energy failure, leads to progressive cerebral edema, programmed apoptosis due to accumulation of excitatory amino, encephalopathy, multiorgan complications and death [68-70].

Effective resuscitation of asphyxiated newborns by LNR will result in decreased perinatal mortality and morbidity based on scientific principle of application of Continuous Positive Pressure Ventilation (CPPV) with oxygen at varying flow rates based on degree of hypoxia in newborn at birth by sustained fast pressure over periods of 2-3 minutes that helps to overcome the high resistance of inspiration of air into fluid filled lungs and in movement of lung liquid through the airways into circulation, thereby transiting to well aerated neonatal lungs, proved 100% successful, constantly monitored by Pulse Oximetry and with asepsis, thermal control and early nutrition will ensure well-being of newborns [70].

Thus despite vast advances in perinatal care, obstetric management and improved technology in fetal monitoring etc. birth asphyxia continues to be the leading cause of the preventable high prevailing perinatal and neonatal morbidity and mortality in most Asian countries despite world-wide reduction in neonatal deaths following the introduction of Neonatal Resuscitation Program [30-31] was however associated with a high 20-40% morbidity in survivors suffering from ill effects of hypoxial sequelae ranging from mild to severe permanent neurological deficits etc with incidence of HIE showing no decline over the past decades [36-42].

I have reported that the average duration of pregnancy in ethnic Asian-Indian population is 38.2 weeks [114,116] and that peak births of healthy, non-asphyxiated newborns born normally with clear liquor took place at 38 weeks, while most asphyxiated births, majority delivered by emergency LSCS occurred at 39 weeks [9]. In fact 84% of asphyxiated newborns developed complications of Meconium Staining of Amniotic Fluid (MSAF) and some Meconium Aspiration Syndrome (MAS) [129]. This has important implications since increased emergency surgical intervention at mean gestation of $39.1 \pm \text{SD } 1.2$ weeks, occurring in more mature, high birthweights newborns weighing around 3500-4000 g with fetal distress as a common indication with prolonged labor often due to undetected CPD more so in young primigravida mothers, are at higher risk of dying from perinatal asphyxia [9, 114,116]. Thus the small Asian neonate with average low birth weight around 2800 g-3000 g attributed to asymmetrical intrauterine growth retardation due to inherent genetic predisposition rather than environmental factors have low energy reserves are less well equipped to cope with any asphyxial insults during uterine contractions especially when labor is prolonged result in increased mortality and morbidity and adverse long term sequelae in survivors [114,116].

However healthy small for dates, shortly after birth, will feed avidly and gain weight indicating effective preventive strategies now takes on priority in saving the small Asian babies, mandating the implementation of new ethnic Asian specific perinatal guidelines for well-being of Asian newborns by definition of Asian Due Date (ADD) for delivery at 38^{+6} weeks gestation correlating to peak ethnic Asian births thus preventing hypoxial birth injury more so in births at 39 weeks and later including the colossal asphyxial deaths both first day early neonatal deaths as well as intrapartum stillbirths which may be preventable both by active management of labor as well as by effective resuscitation who otherwise may have healthy outcome [114,116,120].

Thus prolonging pregnancy to 39 weeks and beyond increases the risk of stillbirth, neonatal and maternal morbidity and mortality with adverse increase in neonatal risk predisposing to high risk of perinatal asphyxia and higher risk of stillbirth as well as other outcomes such as PIH that rises with each additional week of gestation [9,167]. Postdate induction is typically not recommended prior to 41st week [167-169] a dictum followed also by Asian obstetricians in management of pregnancy in ethnic Asian women, who often

experience uterine contractions by 38 weeks of gestation, labelled as ‘false labor pains’ and instead of attempting delivery with amniotomy, which is now well accepted in acceleration of labour, or use of prostaglandin E2 for cervical ripening, instead opt for prolonging pregnancy to 40 weeks or Expected Date for Delivery (EDD) with tocolytic medication to suppress uterine contractions and/or bed rest increasing risk of stillbirths, neonatal and maternal morbidity and mortality.

Also the later delivery at term gestation, questions management of pregnancy at 39-43 weeks associated with increase obstetric intervention by emergency LSCS more so in ethnic Asian population [114,138]. In fact a substantial number of newborns could have perinatal asphyxia attenuated or removed given timely obstetric intervention and as such elective section at 38 weeks more so in high risk cases would remove the risk of intrapartum asphyxia and reduction of HIE with improved perinatal outcome and lower term stillbirth rates [9,169-171]. Hence the lowest risk of perinatal deaths was noted at 38 weeks, sharply increased among primigravida women beyond 39 weeks, because of greater risk of shoulder dystocia, foetal trauma, meconium staining of liquor, neonatal encephalopathy and intrauterine demise with higher incidence of HIE [9,129,170-174].

Other clinical studies from the west report low digit figures perinatal, neonatal and maternal mortality rates having almost eliminated birth asphyxia with low 1% incidence [43,53], yet demonstrate that delivery at 38 weeks by planned or elective LSCS in high risk groups have least risk of perinatal deaths and that prolonging pregnancy to 39 weeks up to 43 weeks noted increased perinatal risk index due to obstetric events more so in primigravidas with greater risk of antepartum stillbirth [170-172].

Also non-laboring women delivered by caesarean section before 39 weeks, obviates intrapartum events, reported 83% reduction in moderate to severe encephalopathy, a leading causes of HIE including late fetal deaths [170], signifying that early planned delivery preferably at 38 weeks more so by elective section in high risk cases before occurrence of adverse intrapartum events was associated not only with reduction in birth asphyxia cases but also fresh stillbirths due to anoxia and neonatal complications such as respiratory disorders etc, being the most effective strategy than any other so far implemented, yet still others estimate the lowest cumulative risk of perinatal deaths and advocate delivery by 37 weeks gestation [171,172].

Majority of ethnic Asian women who do experience uterine contractions before 40 weeks EDD, labor should be allowed to progress even from 36 weeks of gestation onwards, though a blanket policy for induction of labour at 38 weeks would certainly be associated with an unacceptable general increase in rate of obstetric intervention [167-169], hence it is important to take into consideration the unique ethnic diversity or genetic predisposition by implementing ethnic due dates for the two main ethnic races, Asians and Caucasians. Thus implementation of ethnic Asian Due Date at 38⁺⁶ weeks gestation will result in improved perinatal outcome as opposed to a common EDD at 40⁺⁶ weeks well adapted to Caucasian

population [175] according to the existing perinatal definitions and guidelines established by World Health Organization [176] stated in standard medical text books have not taken into consideration ethnic difference that exists between the two main Asian and Caucasian races.

It is this inability to address the unique ethnic inherent genetic predisposition in Asian population, that resulted in failure of MDGs - 4 goals with the specific aim of reducing under five child mortality rates by two-thirds, despite addressing determinants of human health and welfare including poverty, hunger and disease between 1990 to 2015 that was adopted by United Nations (UN) globally, as majority under-five child mortality occur predominantly in ethnic Asian population residing mainly in low to middle income Asian countries [177-179].

Following years of research and in-depth analysis, I have outlined up-to-date ethnic Asian perinatal standards and definitions, providing appropriate guidelines for ethnic Asian Obstetricians, Paediatricians/Neonatologists, if implemented will result in improved neonatal and maternal outcome with reduction in perinatal, neonatal and maternal morbidity and mortality, consequently infant and under-five years child mortality rates in attempting to reach target set by SDG goals. As well as the extended perinatal team including radiologists, genetic/prenatal counsellors etc in cases of fetal anomalies and paediatric surgeons if surgically correctable with in utero or immediate post natal surgical intervention will benefit by the new ethnic Asian perinatal guidelines and definitions [175].

Hospital based study suggests that 25-62% of intrapartum stillbirths can be avoided by better obstetric care with more rapid response to intrapartum complications [51,52,165], however question remains whether given present common universal guidelines and definitions, can ethnic Asian intrapartum-related deaths be reduced including disability in survivors including reduction of global caesarean section rates, which is alarmingly increasing and will the present health system be able to deliver?

The answer is 'No' as so far, present strategies for maternal and child care world over has not seen further dramatic reduction in perinatal mortality rates as well as preventable birth asphyxial deaths including maternal mortality rates including reduction of emergency LSCS envisioned, unless Asian ethnic specific perinatal guideline is clinically implemented thereby reducing majority of the global neonatal, infant mortality and under five years mortality rates by a simple Asian Due Date (ADD) for delivery at 38⁺⁶ weeks eliminating a world-wide common E.D.D at 40⁺⁶ weeks gestation, presently followed as stated in standard medical text books, ideally suited for ethnic Caucasian population, thereby avoiding adverse intrapartum events 39 weeks and beyond in Asian population ensuring not only the colossal reduction in stillbirths of otherwise healthy babies but also early neonatal deaths due to perinatal asphyxia obviating the impact of shoulder dystocia, foetal trauma, neonatal encephalopathy and intrauterine demise and other complications of labour occurring more commonly at 39 weeks delivery and beyond, circumventing adverse intrapartum problems by early delivery of the small Asian newborns at 38+6 weeks gestation [175,176].

In fact 90.3% Asian-Indian women gave birth before EDD i.e. 40 completed weeks or 280 days gestation [116] compared to only 1.7 percent at 40+6 weeks EDD and beyond [122]. The Asian Due date at 38⁺⁶ weeks is ideally appropriate for ethnic Asians and may be accurately calculated if menstrual cycles are regular at 28 days interval and ovulation occurring on Day 14, estimated by Lalana's rule, Step 1 - Determine the first day of last menstrual period, Step 2 - subtract 1 week and 3 months, Step 3 - Add 1 year to arrive at 38+6 weeks or 266 days of gestation, for eg. if LMP is 15th November, 2021, then ADD is August 8th, 2022 approximately at an interval of 38⁺⁶ weeks (or 266 days) from LMP [175].

In contrast only one-third, 32% of Caucasian- British women delivered before Expected Date of Delivery (EDD) or 40+6 weeks gestation, as significantly more British women, 68 percent delivered after 40 weeks EDD during 41-44 weeks of gestation [122]. The importance of addressing inherent genetic or racial differences Asian and Caucasian population cannot be underestimated as this important criterion has not been addressed world-wide so far, in formulating ethnic Caucasian and Asian perinatal definition and guidelines that will go a long way in catering to the well-being of ethnic Asian fetuses and neonates as well as their mothers. UNICEF, WHO, World Bank and UNDESA reports that sixty million under-five years children will die between 2017 and 2030 which will be further compounded by global covid epidemic with far greater fatalities, while only 5.6 million children died in 2016 pre-covid when compared to 9.9 million in 2000. Despite global decline in maternal mortality ratio by 37 percent between 2000 and 2015 from 451,000 in 2000 to 295,000 in 2017, that's around 808 women every day, mostly from preventable causes, reduction to less than 70 per 100,000 live births global maternal mortality ratio seems untenable more so in Asian countries, while Finland, Greece, Iceland and Poland have low 3 women per 100,000 births [180-185].

Reduction of maternal mortality with 95% occurring in Asian countries at 130/100, 000 live births during 2016-2018 or 26437 maternal deaths in 2018, commonest causes being post-partum haemorrhage, sepsis, PIH and complications of delivery in 50-98%, may be envisioned by implementing Asian perinatal guidelines by due date at optimum 38⁺⁶ weeks gestation. In fact global maternal mortality rate declined by 38% from 342 deaths to 211 deaths per 100,000 live births according to UN Inter-agency estimates translates to average annual rate of reduction by only 2.9 % [181,185]. I hope that it will not be frustratingly too long before we finally accept changes in clinical practices by implementing ethnic Asian specific perinatal guidelines with the aim for the steep reduction in perinatal, neonatal and maternal mortality and morbidity becomes a reality [175].

The current world population of 7.8 billion according to recent United Nations estimates, the majority 60% of world population comprising 4.5 billion are ethnic Asians residing mainly in Asian countries [54] report high prevailing perinatal, neonatal and maternal mortality rates as well as under five years, morbidity and mortality [12,178,179]. The MDG - 4 set out by World Health Organization (WHO) significantly reduced under-five mortality

rate by 59% between 2000-2015, WHO in 2013 reported that the number of deaths of children under five years fell from 12.7 million in 1990 to 6.3 million in 2013, majority 95% of these occur in developing low to middle income Asian countries [184].

During 2018 WHO reported that over 6 million children and adolescents died globally, comprised of 5 million under five years deaths, majority being preventable in low-middle income Asian countries despite measures for intervention in the care of newborn and their mothers such as infant and young child feeding, expanded programme on immunization with newer vaccines, prevention and case management of pneumonia, diarrhea and sepsis, malaria control and prevention and care of HIV/AIDS by appropriate home care and early treatment of complications of newborn with integrated management of childhood illnesses in under five years, complimented by interventions for maternal health and nutrition, especially skilled care during pregnancy and childbirth without further significant reduction in perinatal and neonatal mortality [177].

While in 2019, 5.4 million under five child deaths due to preventable/ treatable causes which on an average means 15,000 young children, while infants comprised 1.5 million, 1-4 years accounted for 1.3 million deaths. The remaining 2.4 million deaths occurred among neonates in the first 28 days of life, highlights importance of reducing neonatal deaths that will impact under-five year mortality rates majority among ethnic Asian population. Sustainable Development Goals (SDGs) 3.2.1, known also as Global Goals stipulate reduction to 11 million under-five child deaths between 2019-2030, is an insurmountably huge achievement and is currently seems far away, despite improvement of global health as expected 4.5 million child deaths will occur by 2030 or 86 million child deaths in SDG era, with the aim to reduce neonatal mortality to at least 12 per 1000 live births, or that 97.5% of all newborns would survive, no matter where they are born decreasing child mortality to at least 2.5%, more than 100 UN Member States being signatory as part of the 70th session of UN General Assembly with renewed commitment to children rights who will grow to become future leaders and pillars of society by protecting their healthy growth and development [180-184], emphasizes being shifted to the neonatal period or first 28 days of life as being the most vulnerable time in the life of a child with high death rates.

India with growth rate of 1.11% and 1.35 billion population is poised to become the most populated country in the world by 2027 according to new UN study of global population trends [54] is just second to China with 1.41 billion and growth rate of 0.39-0.59% with a shrinking population [186]. Indian studies representative of a vast population will be justified in setting ethnic specific perinatal Asian guidelines and definitions, envisioning reduced perinatal, neonatal and maternal deaths and improved outcomes. Including reference intrauterine growth chart for accurate identification of at risk SGA and LGA neonates that require special care. In fact the most vulnerable time in child survival is the neonatal period, more so the first 24 hours of life and first week being critical in the life of individual. Thus effective strategies aimed at the perinatal period becomes crucial in improving outcome by

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addressing inherent ethnic diversity in Asian and Caucasian population is now the need of the hour.

India has 0.386 million cases of newborn asphyxia annually and around 75% are preventable, comprising 20% of deaths, prematurity 35% and sepsis 33% being the other two causes. Congenital malformation an absolutely unavoidable cause of perinatal mortality constitutes only 9% [10-12]. Thus India had 522 neonatal deaths per 1000 live births with Early Neonatal Mortality Rate (ENMR) 20 per 1000 live births and Neonatal Mortality Rate (NMR) 26 per 1000 live births. Nigeria ranks second with 270 ‰ followed by Pakistan 248 ‰, Ethiopia 99 ‰, and Democratic Republic of the Congo 97 ‰, China 64 ‰, Indonesia 60 ‰, Bangladesh 56 ‰, Afghanistan 43 ‰ and United Republic of Tanzania 43 ‰ live births. The projected NMR of 22/1000 live birth for 2020 in India has not been attained and current NMR 28/1000 and ENMR 22/1000 live births accounts for 45% of total under-five year child deaths [159-162].

The relationship between birth asphyxia and antepartum, intrapartum or fetal risk factors was assessed to determine preventive strategies, the anticipation of high risk cases and its effective management and quick judicious obstetric intervention to reducing birth asphyxia, therefore implementation of Asian Due Date for delivery at 38⁺6 weeks [122] obviating adverse intrapartum factors with effective resuscitation at birth as well as sepsis control, thermal protection and early nutrition of newborn are important criteria. Risk factors include primigravidity with OR 1.34 (95% CI 0.92- 2.04) times risk of birth asphyxia compared to second and above gravidas, though not statistically significantly, though 44.8% newborns experienced birth asphyxia. Obstetric complications of PIH OR 2.98 (95% CI 2.14- 4.16) times risk of birth asphyxia was highly statistically significant $P=0.0001$. Though gestational diabetes did have a higher risk of birth asphyxia of 1.27 (95% CI 0.86- 1.86) times, it was not statistically significantly. PROM of more than one hour prior to onset of labor OR 1.19 (95% CI 0.85-1.7) times risk of birth asphyxia, though not statistically significant $P=0.315$. Breech presentation obviously had high risk of birth asphyxia OR 8.42 (95% CI 5.1-14) times vertex presentation was highly statistically significant ($P=0.0001$), including meconium staining of amniotic fluid OR 29 (95% CI 19.78- 42.65) times higher risk of birth asphyxia, statistically significant ($P=0.0001$) [9].

Lower Segment Caesarean Section (LSCS) rate was 41.6% with two-thirds (64.7%) majority being emergency LSCS for indication of fetal distress etc. wherein 50.3% of these newborns suffered birth asphyxia when compared to one third (35%) elective or planned LSCS with associated low 9.1% incidence birth asphyxia. The high risk of birth asphyxia in emergency LSCS OR 4.91 (95% CI 3.94-6.10) being highly statistically significant ($P=0.0001$), in comparison elective sections have low risk of birth asphyxia OR 1.67 (95% CI 0.84-1.63). In contrast vacuum extraction have high risk OR 8 (95% CI 5.58-11.69) ($P=0.0001$) [9]. Neonatal factors include prematurity <37 weeks with high risk of birth asphyxia OR 1.36 (95% CI 1.03- 1.79) ($P=0.032$), compared to term gestation with peak

births 23.7% at 39 weeks contrasted with peak births 27% among controls born at 38 weeks. Low birth weight was associated with higher OR 1.5(95% CI 1.21- 1.88) times the risk of birth asphyxia, compared to normal birth weight ≥ 2500 g ($P=0.0001$), however most infants weighing above 3500 g were asphyxiated. Male babies had slightly higher OR 1.14 (95% CI 0.95- 1.37) times birth asphyxia when compared to female babies [9].

Intrapartum events during labor causes 46% of all maternal deaths and 40 percent of neonatal death with in the first 24 hours of birth eminently preventable by active management of labor by skilled birth attendants with judicious emergency obstetric intervention as lately eight out ten women deliver in health facility as compared to earlier a decade ago with six out of ten women delivering in their homes [47,51], however unless ethnicity with its impact on maternal and neonatal mortality is taken into consideration majority occurring in Asian population with new Asian perinatal definitions and effective resuscitation by LNR targeting reduction in neonatal and maternal mortality.

Thus preventive strategies should be aimed at intrapartum period rather than antepartum or post-partum, with 814,000 neonatal deaths and 1.02 million stillbirths usually related to intra-partum related causes such as intrauterine hypoxia, more so in low -and middle-income countries [47,51]. Thus active management of labor with judicious obstetric intervention in rescuing endangered fetuses, since intrapartum events have been reported to have a major impact with adverse outcome more so in ethnic Asian population, presently lies entirely in the domain of the obstetrician following western perinatal guidelines will be circumvented by the clinical implementation of Asian Due Date (ADD) for delivery at 38+6 weeks will prove to be the single most eminently suitable guideline for Asian obstetrician to deliver healthy babies and in safe guarding their mothers with improved perinatal, neonatal and maternal outcome as opposed to a common EDD at 40 weeks gestation [120,175].

I am convinced with the aftermath of the covid pandemic affecting all aspects of life, including non-availability of health care facilities with hundreds of thousands more under-five fatality [48], Sustainable Development Goals (SDGs) 3.2.1, known also as Global Goals targeting reduction of under-five child mortality faces setbacks and requires renewed determination for the stipulated reduction to 11 million under-five child deaths between 2019-2030 that is specifically aimed at reduction of neonatal mortality to at least 12 per 1000 live births and consequently under-five years child mortality to a low 25 per 1000 live births [177,179]. As the given past endeavors and strategies have met with little or no further fall, it is important that we implement new approaches and preventive strategies in management of well-being of newborns and their mothers to envision attaining stipulated goals by 2030 [181].

Thus emphasizing the impact on short term goal to reduce neonatal mortality rate to 5/1000 live births thereby reducing 3-8 million annual deaths by 3 million [12,184] may be effected without introduction of costly high technologically advancement and medicine by simple grass root level, implementation of two effective preventive strategies viz. Asian Due

Date at 38⁺⁶ weeks gestation and effective resuscitation by LNR in asphyxiated newborns saving lives and reducing adverse outcome as well as reducing intra-partum fresh stillbirths, with regionalization of health care, free antenatal care and centralized supervised deliveries by health care personnel [51,175].

WHO is now calling for new sustainable development goals to continue to reduce child mortality rate to a low 2.5% in all countries by 2030, Goal 3.2 even as UNICEF works to end preventable new-born and maternal deaths. Current trends indicate that accelerated progress is needed to reach target, as 80% of under five years deaths globally occur in Asian countries with 45% of under-five child deaths being neonatal deaths, compounded by colossal stillbirth loss including maternal deaths that occur each year, 95% occurring in Asian countries [172].

Birth asphyxia continues to be the leading cause of preventable perinatal deaths and identification of various risk factors assumes importance as nearly half 44.3% of asphyxiated babies were born to primigravida mothers, in contrast to most 31% controls born to second gravida mothers, other factors include abnormal presentation, meconium staining of liquor, oligohydramnios, cord abnormalities and length of gestation with about half 48.85% of live singleton births occurring 39 weeks and beyond contributing to the high 21.2% incidence of birth asphyxia with peak 23.7% asphyxiated births at 39 weeks, in contrast peak 27% controls births took place earlier at 38 weeks. Mode of delivery in over half 50.3% asphyxiated infants was emergency LSCS, in contrast elective or planned LSCS had low 9% incidence birth asphyxia [9]. Preventive strategy by detection and effective management of high risk cases and the delivery by 38⁺⁶ weeks gestation, preferably by elective LSCS obviating intrapartum events would not only reduce birth asphyxia with significant reduction in early neonatal deaths including fresh term stillbirths.

Thus two important new criteria aimed at improving the outcome of newborns and their mothers with focus on intrapartum period, thereby reducing perinatal, neonatal and maternal morbidity and mortality by obviating several adverse factors at birth from 39 weeks onwards by implementation of delivery in ethnic Asian population by 38⁺⁶ weeks' gestation with effective resuscitation by LNR of asphyxial births will result in improved perinatal and impact neonatal outcome, more so within first 24 hours, being critical in targeting global fall in neonatal, infant and under five mortality rates.

Strategies include:-

1. Asian Due Date (ADD) for delivery at 38+6 weeks (peak births occur at 38.2 weeks SD $\pm$ 2) will prove to be the single most important strategy in reducing neonatal, perinatal as well as maternal mortality and morbidity with focus on intrapartum period also stemming colossal fresh stillbirth loss

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2. 'Lalana Newborn Resuscitation' will revolutionize resuscitation of asphyxiated newborns by sustained nasal inflatory oxygen flow up to 2-15 L/min. The superficial oxygen saturation monitored continuously by Pulse oximetry to determine Grade I-V birth asphyxia with successful ventilation indicated by Zero pulse oximetry score, SpO₂ >96%, rhythmic respiration, rate 30-60/min and heart rate 120-160 bpm with asepsis and thermo-control and early institution of breastfeeding will result in well-being of newborn, thereby ensuring that children should be normal with healthy growth and development in infancy, childhood and adolescence, the right of every individual.

Summary

Rapid and complex physiological changes occur during birth in most newborns, around 5–10% in western countries and upto 20-30% in Asian countries with birth asphyxia require assistance to begin breathing in the first minutes after delivery [9,52]. The aim of resuscitation is to prevent death and adverse long-term neurodevelopmental sequelae and that adequate ventilation is the key to success if assisted ventilation is imperative thus higher FiO₂ should be delivered by positive pressure ventilation up to 20 cm H₂O that extends over 2 to 3 minutes [21] achieves better post manoeuvre lung mechanics [57] consistent with the concept that adequate lung aeration results in significant improvement in airway liquid clearance after birth due to generation of hydrostatic pressure gradients between airways and lung tissue that overcomes the high resistance of moving lung liquid through the airways and across the distal airway wall [23], where it is absorbed into the lung interstitial and then into lymphatic and circulation [24] but also positive airway pressure prevents the lung from collapsing as well as opposes the elevated interstitial pressure enhances reabsorption of lung fluid and preventing lung fluid from re-entering the airways during expiration. Lung aeration triggers very large reduction in Pulmonary Vascular Resistance (PVR) which initiates a series of cardiovascular changes that are also essential for survival after birth [19-21,52].

Very preterm neonates commonly have partial lung aeration at birth, high FiO₂ reduces the risk of a hypoxia-induced inhibition of breathing resulting in higher respiratory rates and more stable breathing pattern with better aeration of the lung being a measure of Functional Residual Capacity (FRC). In fact hypoxia after birth at five minutes is associated with bradycardia with higher mortality before hospital discharge and higher risk of intraventricular hemorrhage including laryngeal adduction due to inhibitory effect of hypoxia on breathing pattern which may persist for weeks after birth [60,187]. Hence higher FiO₂ required to avoid

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hypoxia is an essential strategy to promote spontaneous breathing at birth further complicated by presence of airway liquid that reduces surface area for gas exchange, thus higher partial oxygen pressure gradient achieves adequate oxygen exchange resulting in exponential increase in lung surface area aeration with rapid reduction in need of high FiO_2 based on pulse oximetry maintaining SpO_2 at 96% to avoid hyperoxia [187-192].

Lalana Newborn Resuscitation is scientifically and physiologically superior to Neonatal Resuscitation Program in initiating spontaneous rhythmic breathing essential for successful non-invasive respiratory support at birth by sustained nasal oxygen inflation at high FiO_2 based on pulse oximetry, reduces risk of hypoxia, a potent inhibitor of spontaneous breathing which is further compounded by the presence of lung liquid, greatly reducing surface area for gas exchange, thus necessitating use of higher partial pressure gradient FiO_2 with oxygen flow rate at 4-15 litres/min achieves adequate oxygen exchange with exponential increase in surface area and lung aeration resulting in pulmonary vascular dilatation, left to right shunting with complex cardiovascular changes transiting from fetal to neonatal circulation essential for survival. “Successful” resuscitation with peripheral oxygen saturation levels $>96\%$ by pulse oximetry and increased heart rate indicate adequate lung aeration based on the concept that vagal-induced bradycardia in response to perinatal asphyxia causes a low heart rate.

Neonatal Resuscitation Program using air/oxygen with intermittent positive pressure ventilation through face mask or endotracheal tube causes different lung regions to aerate at different rates in contrast to sustained inflation that allows more lung regions to aerate during a single inflation, has important implications for lung injury because during subsequent short inflation with iPPV, air will rapidly flow into and expand aerated lung regions first due to the much lower airway resistance with entire tidal volume will only enter aerated regions, potentially causing overexpansion and injury in those regions with little further lung aeration perpetuating hypoxia with its ill effects and long term untoward sequelae. It is this lack of scientific clarity regarding the physiology of transition is a major contributing factor that the current neonatal resuscitation guidelines is regarded as weak and/or absent [30-32].

Conclusion

Timely delivery of ethnic Asian newborns with active management of labor more so in Asian countries wherein almost all perinatal and maternal deaths occur with effective resuscitation of asphyxiated newborns, a leading preventable proportion of perinatal and neonatal mortality with focus on events occurring during intrapartum period that has a major impact on outcome of mother and child. The key to dramatic reduction in preventable proportion of perinatal

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mortality and morbidity with birth asphyxia is effective management of high-risk cases with judicious obstetric intervention and quick reversal of hypoxial insult in newborns by scientifically proven NNR resuscitation with sustained CPPV and oxygenation over 1-3 minutes that initiates rhythmic respiration. The two most important preventive strategies include clinical implementation of Asian Due Date for delivery at 38+6 weeks gestation and effective resuscitation by Lalana Newborn Resuscitation (LNR) based on scientific principle of application of Continuous Positive Pressure Ventilation (CPPV) by sustained nasal oxygen inflation with real time SpO₂ and heart rate (bpm) monitored by pulse oximetry, in grading asphyxiated newborns to determine oxygen flow rate that will save lives and improving outcome in survivors, thereby reducing perinatal, neonatal mortality and consequently under-five child mortality rate.

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ADDENDUM

PRENATAL CHART^[193]

Name:.....Date of Birth.....Age.....
 Medical Record No.....Religion.....Occupation.....
 Qualification.....

Husband's

Name.....Occupation.....Qualification.....Income.....
 Address:.....
Pin.....

Phone

(Mob.).....(Res.).....(Off.).....

Emergency

Contact:.....Phone.....
 Insurance Company:.....Policy No.....

OBSTETRIC HISTORY

Obstetric Formula G.....P.....L.....Duration of Married
 Life.....Consanguinity.....
 Conception: Planned YesNo..... Spontaneous.....Infertility Treatment.....
 Prior Contraceptive Use.....
 Full term.....Preterm.....Abortion:
 Induced/Spontaneous.....Ectopic.....Multiple.....Living.....

MENSTRUAL HISTORY

Menarche.....Previous Cycles.....Regular - Yes.....No.....
 LMP.....DCP.....Definite USG Report.....Unknown..... On OCP at
 conception.....
 EDD.....ADD.....EaDD.....CDD.....

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PAST PREGNANCIES

	Date/Month /Year	GA Weeks LMP / DCP	Type of Delivery	Spontaneous /Induced	Duration of Labor
1.					
2.					
3.					
4.					

	Anaesthesia	Sex	Birthweight	Place	Complications/Comments
1.					
2.					
3.					
4.					

MEDICAL HISTORY

Diabetes:
 Thyroid Dysfunction:
 Drug Reactions:
 H / o Blood Transfusions:
 Operations / Hospitalization (Year & Reason):

Heart Disease:
 Hypertension:
 TB / Asthma / Seasonal Allergies:
 Tabaco / Alcohol:

Any Others:

FAMILY HISTORY

INITIAL PHYSICAL EXAMINATION:

Weight:
 Height:
 BMI:

Thyroid:
 Breast:
 Heart:
 Lungs:

Others details:

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RISK FACTORS

- 1.
- 2.
- 3.

PRENATAL CARE

Serial No	Date	Gest. Age	Weight (Kgs)	Symptoms	Pallor	Odema	Blood Pressure	CVS	RS
1									
2									
3									
4									
5									
6									
7									
8									
9									
10									
11									
12									
13									
14									
15									
16									
17									
18									
19									
20									

Serial No.	Date	Fundal Height	Presentation	Singleton/ Multiple	FHR (Beats/min)	Next Appointment	Comments
1							
2							
3							
4							
5							
6							
7							
8							
9							

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10							
11							
12							
13							
14							
15							
16							
17							
18							
19							
20							

LAB TESTS

1st TRIMESTER Results

Date
 Blood Group & Rh
 Blood Group & Rh
 (Partner)
 Blood Sugar
 Hb% / PVC
 HbSaG
 HIV Counselling/
 Testing
 VDRL
 Urine Examination
 Urine Culture
 Thyroid Profile

2nd TRIMESTER Results

Date
 OGCT

 Hb% / PVC

 Hb% / PVC

 GTT
 (If screen abnormal)

3rd TRIMESTER Results

Date
 Hb% / PVC

 Hb% / PVC

 Blood Sugar

 Additional
 Tests

TT 1st dose (Tetanus Toxoid) dt..... TT 2nd dose (Tetanus Toxoid) dt.....

ULTRASOUND - EARLY PREGNANCY SCAN

Date :
 Result :

NT SCAN & 1st TRIMESTER-ANEUPLOIDY RISK ASSESSMENT

Date :
 Result:

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ANOMALY SCAN

Date :

Result :

GROWTH SCAN

Date :

Result :

OTHER INVESTIGATIONS:**PLAN OF MANAGEMENT****EDUCATION****DELIVERY PROFORMA ^[193]**

Onset of labor	Time	Date
Duration of Labor 1 st Stage	2 nd Stage 3 rd Stage	
No. of Vaginal Examinations		
Duration of rupture of membranes onset of Labor	1) Before onset of Labor	2) after
Aminotic fluid 1. Clear 2. Meconium stained	3) Foul smelling	
Quantity : Adequate/inadequate/excess		
Pregnancy Singleton/multiple	L.M.P.	Weeks Dating by USG
Presentation: Vx/Breech		
Any complications during Labour:		
Prenatal/Intranatal/Postnatal	Fetal Conditions	

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Partogram: Cervical dilatation charted against hours in labor for active management of labor and judicious obstetric intervention.

Mode of Delivery Normal Vaginal

LSCS: Elective/Emergency

Indication

Low Perineal Forceps Delivery

Vacuum Extraction

Others

MOTHER'S LAB INVESTIGATIONS:

Hb.....Blood Gr....Rh.... Coomb's test.....

Prenatal fetal assessment: Clinical

VDRL HIV Au Ag

Biochemical: Biophysical Others

Urine Microscopy /Alb, sugar

Other tests

If Induction of labor

Method	1.	Mechanical
	2.	Medical a)Local Pessary (Estrogen, prostaglandin E ₂ etc)
b) Systemic (IV Oxytocin, prostaglandin E ₂ etc)
c) Surgical (artificial rupture of membranes-time)

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BISHOP SCORE

	0	1	2	3
1. Cervical dilation	0	1-2 cm	3-4cm	5-6 cm
2. Cervical effacement	0-40%	40-50%	60-80%	80%+
3. Consistency	Firm	Medium	Soft	-
4. Station of head	-3	-2	-1	1,+1,+2,+3
5. Position of cervix	Posterior	Mid	Anterior (Sacral os)	

TOTAL SCORE = 13**RESULTS**

- > 9 score - Very favorable for induction,
- 6-9 Favorable -70-80% success
- < 6 - Unfavorable- more than 20% failure

Remarks

Management of Preterm Labor

a) Tocolysis b) Nifedipine c)Antiprostaglandins

Adjunctive therapy for preterm labor

Corticosteroids: Betamethasone 12 mg IM for 2 doses 24 hours apart administered and repeated weekly until 36 Weeks.

Maternal/Obstetric Risk Factors:

1. _____
2. _____
3. _____
4. _____
5. _____

Post Partum Period:-

Placenta removed intact Yes/No weight_____

Post partum bleeding: Normal/Excessive

Eventful_____

Uneventful_____

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Diagnosis**Management****NEWBORN PROFORMA** ^[193]**IDENTIFYING PARTICULARS**

Mother's name & Occupation _____ Age _____ Hosp. No. _____

Baby's Hosp. No. _____ Sex _____ Date of birth _____

Father's name & Occupation _____

Family History: Nuclear/Joint family

Consanguineous/non-consanguineous

Married for how many years _____ /single

Siblings No. _____ Age _____ Sex _____

ASSESSMENT OF NEWBORN AT BIRTH WITHIN 20-60 SECONDS

Time of birth _____ Date _____

A. Pulse oximetry SpO₂ _____ Time _____

B. Pattern of breathing:-

1. Spontaneous Rhythmic respiration Rate _____/min
2. Regular _____ Rate _____/min
3. Irregular _____ Rate _____/min
4. Gasping _____ Rate _____/min
5. Absent

C. Heart Rate _____ bpm

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Classification of Newborn:

1. Normal/Healthy
2. Grade I (Yes/No) II (Yes/No) III (Yes/No) IV (Yes/No) V (Yes/No)

Lalana Newborn Resuscitation (LNR) :- Yes/No if yes Duration _____minutes

Onset of respiration at _____minutes

Oxygen flow rate :- L/min Duration _____

Neonatal Resuscitation Program (NRP)

1. IPPV Bag & Mask _____ if yes onset of respiration.....minute
2. IPPV Endotracheal intubation _____ if yes onset of respiration.....minutes

ANTROPOMETRY

Birthweight _____grams Gestation _____weeks+days

Head circum _____Chest circum _____Length _____

Any congenital malformations: Yes/No

If yes specify

MATERNAL DETAILS**MOTHER'S MEDICAL/OBSTETRIC HISTORY**

Past Obstetric history: G__P__L__

Details of previous pregnancy

G1 (age, sex, dead/alive? complication)

G2 Abortions/still births/Congenital

Malformations:

No. of vaginal examinations.....

Other details: Prenatal care: Yes/No

Mother's Height..... Weight.....B.P.....Odema etc

DELIVERY NOTES

Mode of delivery: Normal/operative:

Duration of labor: 1st stage.....2stage.....

Rupture of membranes: Spontaneous/

Artificial Duration:

Liquor: clear/meconium stained/foul

smell

Quantity: adequate/inadequate/excess

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Maternal ingestion of drugs alcohol/smoking

Pregnancy Singleton/multiple

L.M.P./D.C.P/A.D.D./C.D.D./Ea.D.D.

Presentation: Vx/Breech

Any Medical/Obstetric `complications: Prenatal/Intranatal/Postnatal

Fetal Condition

Any fetal distress:

FHR monitoring

MOTHER'S LAB INVESTIGATIONS:

Hb.....Blood Gr....Rh.... Coomb's test.....

Prenatal fetal assessment: Clinical

VDRL HIV Au Ag

Biochemical: Biophysical Others

Urine Microscopy /Albumin, sugar

BABY'S LAB INVESTIGATIONS:

Hb.....Blood Gr....Rh.... CBC.....Platelet countetc

Maternal/Obstetric Risk Factors:

1. _____
2. _____
3. _____
4. _____
5. _____

Newborn Diagnosis:

Normal/ hypoxic Singleton/ Twin 1/2, live, Term/preterm, boy/girl baby born by normal vertex vaginal delivery/emergency LSCS/Elective LSCS/LP/Vacuum Extraction, breech delivery, and established spontaneous respiration at birth.

Birth asphyxia: Grade I/II/III/IV/V resuscitated by LNR Protocol/NRP Protocol and established rhythmic respiration after _____minutes

Management of complication if any:

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Final Outcome:-

1. Live birth Yes

2. Early Neonatal Death - Time of death/Age: _____hours____minutes Date:dt/m/year

NEONATAL CHART ^[193]

NAME..... Age..... Sex.....

Age at first examination of the baby..... (In hours/minute)

Pulse oximetry SpO₂_____ Time _____

Pattern of breathing:- Regular/irregular/ gasping (rate/min) /absent

Heart Rate _____bpm

Assessment of Newborn within 20-60 seconds of birth

Normal, Healthy baby:- Yes... No...

Birth Asphyxia/hypoxia:- Pulse oximetry SpO₂ Heart rate.....(bpm) Respiratory rate...../min

Classification of Newborn at Birth:

Normal/Healthy Newborn

Birth Asphyxia

Grade I (Yes/No)

Grade II (Yes/No)

Grade III (Yes/No)

Grade IV (Yes/No)

Grade V (Yes/No)

Resuscitation Yes/No

Lalana Newborn Resuscitation (LNR) ☐ Neonatal Resuscitation Program (NRP) ☐

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Lalana Newborn Resuscitation: CPPV with Oxygen flow rate ____ L/min

Duration _____minutes Onset of respiration Time.....minutes____

Neonatal Resuscitation Program _____ IPPV

1. Bag & Mask (Yes/No) if yes onset of respiration.....minutes
2. Endotracheal intubation (Yes/No) if yes onset of respiration.....minutes

Outcome: Live/NICU (diagnosis/management)

Duration of resuscitation:- _____minutes

Anthropometry:-

Birth weight..... grams

Gestational age.....weeks

Head circumference..... cms

Chest circumference.....cms

Length (crown-heel)cms

Risk score code.....

APGAR score at 1 min..... 5min.....10 min.....

NEWBORN GENERAL AND SYSTEMATIC EXAMINATION:

Eyes	Heart	Genitalia
Ears	Lungs	Anus/Rectum
Nose	Abdomen	Passed meconium/urine
Mouth	Hips	Head
Skin	Spine	Any congenital malformations If yes specify
Umbilicus	Limbs	

Resp. rate

Heart rate

B.P.

Body temp.

MATERNAL RISK FACTORS

- 1.
- 2.

FETAL RISK FACTORS

- 1.
- 2.

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NEWBORN RISK SCORE AND COLOR CODING INDEX

DIAGNOSIS

Hospital course:	Uneventful	Eventful (specify).....
Investigations:	Blood group etc.	
Treatment:		
Condition at discharge:		
Weight at discharge:		

IMMUNIZATION : BCG date given

OPV

HBV

RECOMMENDATIONS:

FOLLOW - UP:

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LALANA NEWBORN RESUSCITATION (LNR) ^[70]

PROTOCOL I

GRADE NEWBORNS I-V WITHIN 20 - 60 SECONDS OF BIRTH

SUSTAINED NASAL OXYGEN INFLATION

Grade I SpO ₂ 94% -95%	Grade II SpO ₂ 92-93%	Grade III SpO ₂ 90-91%	Grade IV SpO ₂ 89%-50%	Grade V SpO ₂ <50%
↓	↓	↓	↓	↓
O ₂ 2-4 LPM, FiO ₂ 28% to 36% for up to 60 seconds	O ₂ 5-8 LPM, FiO ₂ 40% to 52%, for 60-90 seconds.	O ₂ 8-12 LPM, FiO ₂ 52% to 64%, for 90- 120 seconds	O ₂ 12-15 LPM, FiO ₂ 64% to 76% up to 120-180 seconds	O ₂ 12-15 LPM, FiO ₂ 64% to 76% up to 180-240 seconds or more

ROUTINE NEWBORN CARE

Transfer to NICU for observation and further assessment for at least four hours. Temperature, SpO₂, Heart rate (bpm), respiratory rate, blood pressure, colour and activity are recorded and blood glucose levels, blood gas analysis checked.

IF UNEVENTFUL SHIFT TO MOTHER'S SIDE TO INITIATE BREAST FEEDING.

OR

KEEP IN NICU FOR OBSERVATION, MANAGEMENT AND TREATMENT, IF ANY
COMPLICATIONS

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LALANA NEWBORN RESUSCITATION (LNR) ^[70]

PROTOCOL II

PRETERM NEWBORNS <32 WEEKS AND <1250G

GRADE NEWBORNS I-V WITHIN 20 - 60 SECONDS OF BIRTH

SUSTAINED NASAL OXYGEN INFLATION

Grade I SpO ₂ 94% - 95%	Grade II SpO ₂ 92% - 93%	Grade III SpO ₂ 90% - 91%	Grade IV SpO ₂ 89% - 50%	Grade V SpO ₂ <50%
↓	↓	↓	↓	↓
O ₂ 2-4 LPM, FiO ₂ 28% to 36%, for 60 seconds	O ₂ 5-8 LPM, FiO ₂ 40% to 52%, for 60- 90 seconds.	O ₂ 8-12 LPM, FiO ₂ 52% to 64%, for 90-120 seconds.	O ₂ 12-15 LPM, FiO ₂ 64% to 76% up to 120- 180 seconds	O ₂ 12-15 LPM, FiO ₂ 64% to 76% up to 180- 240 seconds or more

START CPAP AT 5 cm H₂O, FiO₂<30%, THROUGH NASAL PRONGS

SHIFT TO NICU

FOR OBSERVATION OR MANAGEMENT AND TREATMENT OF ANY
COMPLICATIONS

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