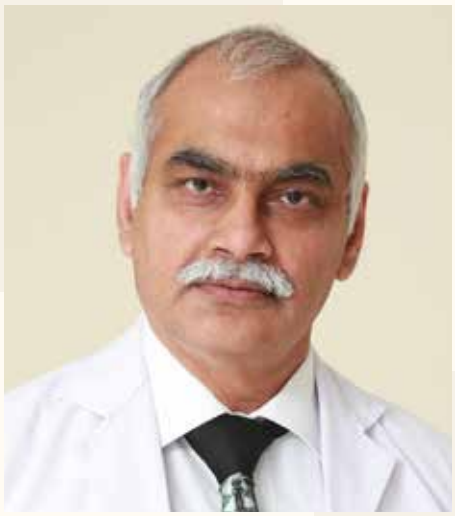
NICU TIMES

KONDAPUR

VOLUME-6

KIMS CUDDLES™
MOTHER & CHILD CENTRE

Foreword



Dear Doctors,

Kudos to KIMS Cuddles on its sixth issue of NICU TIMES. It is the result of marvelous effort and abiding passion of the team. The issue features some of the challenging and rare cases handled exceptionally.

I am glad that KIMS Cuddles has blossmed into a highly charged and dedicated team for whom Quality has become a way of life. It has carved a special niche for professional excellence and personalised care.

I compliment Dr. Aparna C. and the entire team for their untiring efforts. I wish them all success in their journey ahead.

I am sure you will find the issue informative and inspiring.

Warm regards,

Dr. B. Bhaskar Rao
Chairman & Managing Director
KIMS Group of Hospitals

Editor's Note

Dear Esteemed Readers,

It brings us immense joy to introduce the **sixth edition** of our publication, the "**NICU Times**," crafted by the dedicated team at KIMS Cuddles, Kondapur.

In this addition, we are glad to present a collection of challenging cases as case reports. We have attempted to describe our experience in managing neonates who were admitted to the Neonatal Intensive Care Unit, KIMS Cuddles, Kondapur with cyanotic critical congenital heart disease, persistent pulmonary hypertension, perinatal asphyxia or extreme prematurity. The issue also briefly captures the rationale and clinical algorithms for managing these conditions using newer treatment modalities such as inhaled nitric oxide.

We extend heartfelt gratitude to the experts from various allied departments like cardiology, cardiothoracic surgery, pediatric surgery, dermatology, and pediatric hemato-oncology. Additionally, we acknowledge the invaluable support from our branding team, instrumental in bringing this issue to fruition.

As pediatricians and neonatologists, we shoulder the significant responsibility of steering our nation towards enhanced neonatal survival rates and holistic well-being to achieve the ambitious goal of a single-digit Neonatal Mortality Rate (NMR) by 2030. At KIMS Cuddles, Kondapur we are unwaveringly committed to this objective. We firmly believe that sharing knowledge and collective learning will propel us closer to achieving this pivotal milestone.

Dr Aparna C
Clinical Director Neonatology
& Senior Consultant
Neonatology and Pediatrics

Dr. Aravinda Lochani T
Consultant Neonatologist
& Pediatrician

Dr. P. Goutami Reddy
Consultant Neonatologist &
Pediatrician

OUR EXPERIENCE IN THE MANAGEMENT OF LATE PRETERM AND TERM NEONATES WITH BIRTH ASPHYXIA AND ROLE OF THERAPEUTIC HYPOTHERMIA

Introduction:

The World Health Organization describes birth asphyxia as failure to initiate and sustain breathing at birth. Birth asphyxia can result in profound systemic and neurologic sequelae due decreased blood flow and oxygen to newborn baby during the peripartum period. If the decreased oxygen flow is severe enough, the tissues and vital organs (muscle, liver, heart, and ultimately the brain) will develop an oxygen debt. Anaerobic glycolysis and lactic acidosis will result. Neonatal hypoxic-ischemic encephalopathy refers specifically to the neurologic sequelae of birth asphyxia (Fig. 1). 1,2

Asphyxia accounts for 23% of neonatal deaths globally, and 8% of all deaths in children under five years of age3 .Survivors of birth asphyxia are at risk for neurodevelopmental sequelae including motor and cognitive disabilities4-8. The severity of perinatal asphyxia can be graded by Sarnat and Sarnat

staging system in which Stage-1 has excellent prognosis while stage-3 has high mortality and poor neurological outcome. Outcome in neonates of stage 2 can be variable and may range from mild, moderate, or severe developmental delay or even deaths in few cases.

Therapeutic Hypothermia is currently only proven neuroprotective therapy for HIE.

Research shows that moderate therapeutic hypothermia within 6 hours of asphyxia improves survival without cerebral palsy or other disability by about 40% and reduces death or neurological disability by nearly 30%.

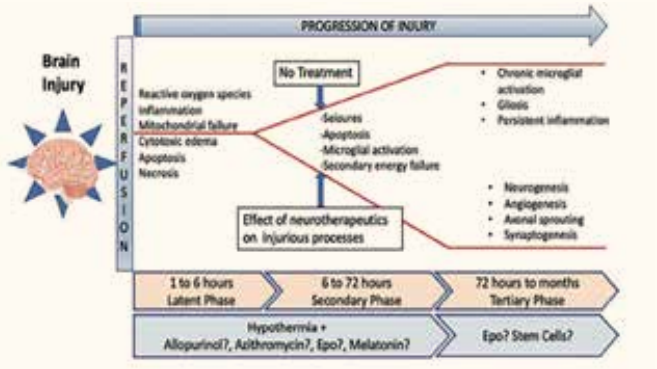


Figure-1 Pathogenesis of asphyxia related encephalopathy

Our Experience With Neonatal Asphyxia

We managed total of 61 neonates with birth asphyxia over period of 12 months from April 2022 to March 2023. Newborns beyond 35 weeks' gestational age and birth weight > 1800 grams with birth asphyxia defined as requirement of positive pressure ventilation (PPV) beyond 1 minute of life admitted to NICU (neonatal intensive care unit) of KIMS Cuddles Kondapur from April 2022 up-to March 2023, were eligible for the current study. Neonates with major congenital malformations including central nervous system (CNS) malformations, birth weight < 1,800 gm, severe hyperbilirubinemia bordering on threshold requiring exchange transfusion, pyogenic meningitis and refusal of parental consent were excluded from the study.

Demographic data, such as maternal age, mode of delivery, APGAR score at 1, 5 and 10 minutes, mode of delivery, details of resuscitation, duration of PPV, birth weight, gestational age, gender, need for mechanical ventilation, presence of seizures, stage of HIE, mortality, duration of hospital stay, other morbidities during NICU stay such as shock, MODS, requirement of inotropes and other relevant data was collected from enrolled neonates. All the neonates were treated as per uniform protocol.

Selection criteria for therapeutic hypothermia:

1. Gestation ≥ 35 weeks, birth weight ≥ 1800 grams
2. Postnatal age at presentation ≤ 6 hours since birth
3. Evidence of birth asphyxia: Any one of the following
   - I. Apgar score at 5 minutes ≤ 5
   - II. Need for IPPV till 5 minutes after birth
   - III. Cord arterial blood or blood obtained within one hour of birth pH < 7.0
   - IV. Cord arterial blood or blood obtained within one hour of birth base deficit ≥ 16.0
4. Staging of Encephalopathy: Any one of the following
   - I. Clinical seizures
   - II. Altered state of consciousness (Lethargy, stupor or coma) AND any of the following
     - a. Hypotonia
     - b. Abnormal reflexes including oculi motor or pupillary abnormalities

The World Health Organization describes birth asphyxia as failure to initiate and sustain breathing at birth. Birth asphyxia can result in profound systemic and neurologic sequelae due decreased blood flow and oxygen to newborn baby during the peripartum period. If the decreased oxygen flow is severe enough, the tissues and vital organs (muscle, liver, heart, and ultimately the brain) will develop an oxygen debt. Anaerobic glycolysis and lactic acidosis will result. Neonatal hypoxic-ischemic encephalopathy refers specifically to the neurologic sequelae of birth asphyxia (Fig. 1). 1, 2.

Asphyxia accounts for 23% of neonatal deaths globally, and 8% of all deaths in children under five years of age3 .Survivors of birth asphyxia are at risk for neurodevelopmental sequelae including motor and cognitive disabilities4-8. The severity of perinatal asphyxia can be graded by Sarnat and Sarnat.

staging system in which Stage-1 has excellent prognosis while stage-3 has high mortality and poor neurological outcome. Outcome in neonates of stage 2 can be variable and may range from mild, moderate, or severe developmental delay or even deaths in few cases.

Therapeutic Hypothermia is currently only proven neuroprotective therapy for HIE.

Research shows that moderate therapeutic hypothermia within 6 hours of asphyxia improves survival without cerebral palsy or other disability by about 40% and reduces

Figure-2 Therapeutic Hypothermia device

Therapeutic hypothermia was administered using servo controlled cooling device (Brammi, Phoenix medicals Ltd, Fig.2) using a core temperature target of 33.5oC (+/- 0.5.

In our study, Newborn babies with stage 2 HIE or greater with cord blood gas showing pH < 7.0 and base excess > -12 were initiated on therapeutic hypothermia using a standard servo controlled whole body cooling device for 72 hours followed by gradual rewarming.

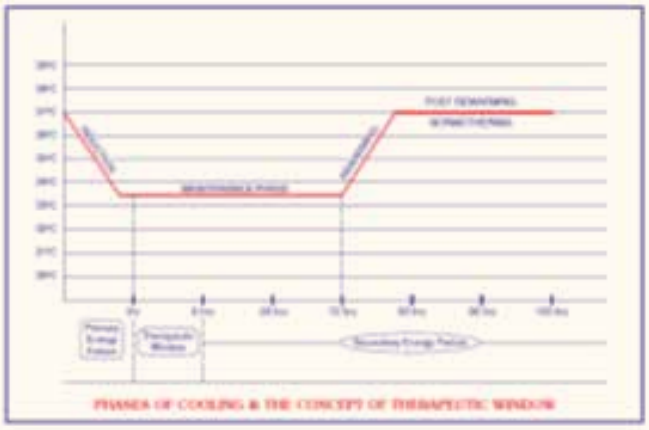


Figure-3 Phases of cooling

Results

Of the total 61 neonates who had birth asphyxia during the study period, 37 (60.6%) neonates developed Hypoxic ischemic encephalopathy (HIE). Baseline characteristics of the study group is described in Table 1. The mean gestational age and birth weight of the study population was 38.6 (3.4) weeks and 2894 (914) grams respectively. Ratio of male and female neonates was almost equal. 7 out of 61 (11.4%) were small for gestational age.

Morbidities during NICU stay

The morbidity profile of study subjects has been described in Table 2. Common morbidities in study group were respiratory distress in 50.8%, acute kidney injury in 18%, shock in 31%, and multi-organ dysfunction in 21%.

Neurological outcomes during NICU stay

Of total 61 newborns with birth asphyxia, 44 babies developed no neurological sequale. Majority of babies without HIE and grade I HIE could be discharged by 48 hours. Most (33/34; 97%) neonates with no or mild HIE had normal neurological examination at discharge.

Primary outcome

Of total 61 newborns with birth asphyxia, 44 babies had normal neurological examination at discharge.

Upto 97% newborns with no HIE or HIE 1 had normal findings on neurological assessment when assessed at discharge/ on day 7 whichever was later. Babies with grade I HIE and no HIE had good neurological outcome.

Of the total 61 cases, 17 babies with HIE stage 2 and HIE stage 3 were given therapeutic hypothermia. 9 /17 babies had good neurological outcome.

On univariate analysis, APGAR score ≤ 5 at 5 minute (odds ratio 8.182, 95% CI: 1.866-35.872), acute kidney injury (odds ratio 38.494, 95% CI: 6.807-217.673), presence of

multi organ dysfunction (MODS) (odds ratio 103.2, 95%CI: 10.983-969.740) therapeutic hypothermia (odds ratio 3.457, 95% CI: 1.040 – 11.494) were associated with death and/or abnormal neurological examination at discharge ($p < 0.05$).

Conclusion

Majority of the babies with no or mild HIE had normal neurological examination at discharge.

Therapeutic hypothermia is beneficial in neonates with severe stages of HIE. As per current guidelines it is mandatory to offer therapeutic hypothermia in all term neonates with evolving moderate to severe HIE.

Table 1: Baseline characteristics of neonates with birth asphyxia (n=61)

	Variable	
1	Birth weight, grams*	2894 (914)
2	Gestational age, weeks*	38.6 (3.4)
3	Gender	
	Male	33 (54.1%)
	Female	28 (45.9%)
4	Weight category	
	SGA (Small for gestational age)	7 (11.4%)
	AGA (Appropriate for gestational age)	54 (88.5%)
5	Duration of PPV (positive pressure ventilation)	
	60 seconds	2 (3.2%)
	60-90 seconds	35 (57.3%)
	90-120 seconds	17 (27.8%)
	>120 seconds	7 (11.4%)
6	APGAR < 5	
	At 1 minute	52 (85.2%)
	At 5 minute	20 (32.7%)
	At 10 minute	3 (4.9%)
7	Cord pH*	7.03 (0.18)

Table 2: Morbidities among the included neonates

	Morbidities	Percentage
1	Respiratory Distress	50.80%
2	Acute Kidney injury	18%
3	Shock	31%
4	HIE	60.60%
5	Sepsis	37.70%
6	DIC	22.90%
7	Seizures	42.60%
8	Hypoglycemia	9.80%
9	Hypocalcemia	1.60%

Table 3: Neurological morbidities in the study population

HIE staging	No HIE (n=24)	HIE 1 (n=10)	HIE 2 (n=15)	HIE 3 (n=12)
APGAR				
<5 at 5min	24-Jan	10-Apr	15-Aug	12-Jul
<5 at 10min	0/24	0/10	0/15	12-Mar
Duration of PPV				
30 seconds	0/24	10-Jan	15-Jan	0/12
30-60 seconds	17/24	10-Feb	15-Oct	12-Mar
60-120 seconds	24-Jun	10-Apr	15-Mar	12-Apr
>120 seconds	24-Jan	10-Mar	15-Jan	12-May
Mortality	0	0/10	15-Jan	12-Apr
Abnormal neurological examination at discharge	0	10-Jan	15-Mar	12-Sep

Table 4: Risk factors for mortality and/or abnormal neurological nexamination at discharge (n=61)

	Survived (n=43)	Death/ abnormal neurological examination (n=18)	P-value	Odds ratio	95% CI
Male Gender	23	10	0.56	1.304	0.420-4.048
Weight					
SGA	5	2	0.973	1.923	0.207-17.850
Apgar at 5min < 5	11	9	**0.0009**	**0.122**	0.028-0.536
Apgar at 10 min < 5	0	3	**0.9524**	**999.9**	**0.001-999.99**
Therapeutic hypothermia	9	8	**0.04**	**3.457**	**1.040-11.494**
MODS	1	12	**0.001**	**103.2**	**10.983-969.740**
AKI	2	11	**0.001**	**38.49**	**6.807-217.673**

MODS - Multi organ dysfunction, AKI - acute kidney injury

Discussion:

Unique challenges faced with therapeutic hypothermia in low middle income nations (LMICs).

The benefits of therapeutic hypothermia evidenced in studies in high income nations may not be extrapolated to low and middles income countries for a number of reasons. Firstly, a large number of deliveries occur outside the hospital settings or in settings with sub optimal intrapartum fetal surveillance and may be associated with chronic maternal and fetal under-nutrition. The exact duration of hypoxia may not be determinable in these circumstances. The newborn may be well into the phase of secondary energy phase of neuronal death making cooling ineffective.

Secondly, the high incidence of maternal infections like human immunodeficiency virus (HIV) and puerperal sepsis complicate the picture. The effect of lowering of core body temperature on impaired neutrophil function may well explain the association of cooling with an increased mortality in LMICs. However the reported incidence of blood culture positivity from studies conducted in India was low, which was similar in number in TOBY trial and ICE trial. TH trials did not show any increased risk of infection, but this could have been masked by use of intravenous antibiotics.

Third, studies from developed nations involve sick neonates with multi-organ dysfunction, who may not survive in some units in the developing world apparently reducing the efficacy of cooling. On the other hand, even neonates with mild encephalopathy may be at risk of developing sequelae. This difference in case mix may pose challenges in understanding the implication of TH as a neuro protective strategy.

The lack of dedicated neonatal transport systems, poor quality of neonatal transport and high incidence of accidental uncontrolled hypothermia in developing nations complicates the picture.

Finally, the use of therapeutic hypothermia must be accompanied by standard intensive care management for thrombocytopenia, complications like persistent pulmonary hypertension which may accompany meconium aspiration syndrome, sedation, ventilatory management etc. Limitations in delivering quality intensive care may lead to more harm than benefit with the use of therapeutic hypothermia. Optimal cardiovascular support and neurological monitoring is an important part of TH. In places with lack of basic neonatal facilities, lack of trained medical personnel, lack of sedation leads to deleterious consequences.

Evidence Of Benefit of Therapeutic Hypothermia in LMICs.

The implications of therapeutic hypothermia in low middle income countries (LMIC) in various studies have shown contrasting results. Three RCT conducted from LMIC's (India & China) reported improved neurodevelopmental outcomes beyond 12 months of age as well as in school age children and all concluded with beneficial effect of TH. In contrast, systematic reviews of 7 trials from LMIC's using low cost cooling techniques did not show a significant reduction in mortality. Furthermore there was insufficient long term follow up to allow assessment of whether hypothermia had improved neurological outcomes. Population characteristics with higher incidence of fetal growth restriction and different comorbidities like sepsis and meconium aspiration, facility characteristics and limited staffing with limited training and unavailability of expensive controlled cooling equipment have been suggested as reasons for why TH can still not be recommended as standard of care for moderate to severe HIE in low resource settings. HELIX trial is one of the largest trials conducted in India which showed the feasibility and effectiveness of a low cost servo controlled cooling device which showed significant population differences in infants with encephalopathy in LMIC's. There were significant number of babies with meconium aspiration and the mean birth weight was less compared to babies in high income countries. There was also higher incidence of gastric bleeding in infants with neonatal encephalopathy who received cooling. MR biomarkers used for babies with neonatal encephalopathy who received cooling therapy showed high prevalence of white matter injury which is contrast to findings seen with high income countries where deep grey matter injuries were more common.

References:

1. Sugiura-Ogasawara M, Ebara T, Yamada Y, Shoji N, Matsuki T, Kano H, Kurihara T, Omori T, Tomizawa M, Miyata M, Kamijima M, Saitoh S., Japan Environment, Children's Study (JECS) Group. Adverse pregnancy and perinatal outcome in patients with recurrent pregnancy loss: Multiple imputation analyses with propensity score adjustment applied to a large-scale birth cohort of the Japan Environment and Children's Study. Am J ReprodImmunol. 2019 Jan;81(1):e13072. [PMC free article] [PubMed].
2. Hakobyan M, Dijkman KP, Laroche S, Naulaers G, Rijken M, Steiner K, van Straaten HLM, Swarte RMC, Ter Horst HJ, Zecic A, Zonnenberg IA, Groenendaal F. Outcome of Infants with Therapeutic Hypothermia after Perinatal Asphyxia and Early-Onset Sepsis. Neonatology. 2019;115(2):127-133.
3. Simon NP. Long-term neurodevelopmental outcome of asphyxiated newborns. Resuscitation Fetus Newborn. 1999;26:767-78.
4. Robertson CMT, Finer NN. Term infants with hypoxic-ischemic encephalopathy: outcome at 3.5 years. Dev Med Child Neurol. 1985;27:473-84.
5. Robertson CMT, Finer NN, Grace MGA. School performance of survivors of neonatal encephalopathy associated with birth asphyxia at term. J Pediatr. 1989;114:753-60.
6. Cioni G, Prechtl HFR, Ferrari F, Paolicelli PB, Einspieler C, Roversi MF. Which better predicts later outcome in full term infants: quality of general movements or neurological examination? Early Hum Dev. 1997;50:71-85.
7. Brookhouser PE. Sensorineural hearing loss in children. PediatrClin North Am. 1996;43(6):1195- 216.

8. Ellis M, Manandhar N, Shrestha PS, Shrestha L, Manadhar DS, Costello AM. Outcome at 1 year of neonatal encephalopathy in Kathmandu, Nepal. Dev Med Child Neurol. 1999;41:689-95.
9. Thornberg E, Thiringer K, Milson I. Birth asphyxia:incidence, clinical course and outcome in aswedishpopulation. ActaPediatr. 1995;84(8):1927-32.
10. Monebenimp F, Tietche F, Eteki N. Asphyxieneonatale au centre hospitalieret. universitaire de yaounde, cameroun. Clinics Mother Child Health.2005;2(2):335-8.
11. Airede AI. Birth asphyxia and hypoxic ischemicencephalopathy(HIE)incidence and severityAnnTrop Pediatr. 1991;11(4):331-5.
12. Chandra S, Ramji S, Thirupuram S. Perinatalasphyxia: multivariate analysis of risk factors in hospital births. Indian Pediatrics. 1997;34(3):206- 12.
13. Gonzales DJ, Moya M. Perinatal difference in asphyxia full term newnorns: an epidemiological study. Rev Neurol. 1996;24:812-9.
14. Glass HC, Glidden D, Jeremy RJ, Barkovich AJ, Ferriero DM, Miller SP. Clinical neonatal seizures are independently associated with outcome in infants at risk for hypoxic-ischemic brain injury. J Pediatrics. 2009;155(3):318-23.

Dr. Sneha
Neonatal Fellow

Dr. Aparna C
Clinical Director Neonatology & Senior Consultant Neonatology and Pediatrics

Dr. Aravinda Lochani T
Consultant Neonatologist & Pediatrician

Dr. P. Goutami Reddy
Consultant Neonatologist & Pediatrician

SHORT TERM OUTCOMES OF EXTREMELY PRETERM NEONATES (23^{+0} TO 27^{+6} WEEKS) IN OUR TERTIARY NICU

Introduction:

Extremely preterm (EP) neonates with gestational age (GA) <28 weeks are at high risk of morbidity and mortality. Prematurity is associated with increased risk of respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), periventricular leukomalacia (PVL), infections, necrotizing enterocolitis (NEC), retinopathy of prematurity (ROP) and so forth. The majority of them require advanced life support during the initial few days.

(1) The WHO explains preterm birth as any birth that occurs before 37 full weeks of pregnancy. Based on gestational age, preterm birth can be further classified as follows: extremely preterm (under 28 weeks), very preterm (between 28 weeks to 31 weeks), and moderately preterm (between 32 to 33 weeks) and late preterm (34 week to 36 weeks). Until 1970, the long-term survival rates were often under 10%. Additionally, around a quarter of extreme preterm neonates born in the 1990s had a significant impairment during the preschool age, such as cerebral palsy, blindness, deafness, or cerebral palsy. (2)

Due to significant advancements in perinatal care, such as antenatal steroids, surfactant replacement therapy, non- invasive breathing support, aggressive parenteral and enteral nutrition therapy, caffeine therapy and infection prevention, the outcomes of extreme preterm infants have significantly improved in recent decades. Preterm delivery complications are the leading direct cause of newborn fatalities, accounting for 2.6 million annual deaths worldwide. They are also the most prevalent cause of death for children under the age of five. (3)

Our experience at KIMS Cuddles NICU, Kondapur:

In the period of 2 years from June 2021 to May 2023 we managed 55 neonates born between 23+0/7 to 27+6/7 weeks of gestational age, out of which 41 were inborn and 14 were out born. Nearly 40% (23) neonates born via Normal vaginal delivery (NVD) and 32 via cesarean section.

	Number of neonates (n=41)
Inborn	41
Outborn	14
NVD	23
Cesarean section	32

There were 6 neonates born between 23+1/7 to 24+0/7 weeks of gestation age, 43 between 24+1/7 to 26+0/7 weeks of gestational age and 6 between 26+1/7 to 27+6/7 weeks of gestational age.

Gestational age	Number of neonates
$23^{+1/7}$ to $24^{+0/7}$	6
$24^{+1/7}$ to $26^{+0/7}$	43
$26^{+1/7}$ to $26^{+6/7}$	6

Complete course of antenatal steroids was covered in 40 neonates and magnesium sulfate was given in 27 neonates.

	Received	Not Received
Antenatal steroids (complete course)	40	15
MgSO4	27	28

The most common complication seen in extreme preterm neonates was respiratory distress syndrome. While 9 neonates required HFOV mode of ventilation, 32 neonates with conventional mechanical ventilation for more than 24 hours and 14 neonates with non-invasive mode of ventilation like NIPPV and Bubble CPAP. All neonates received early surfactant therapy. Reduction of respiratory distress syndrome, length of respiratory support can be reduced by giving antenatal steroids and early surfactant therapy.

Mode of ventilation	Number of neonates
HFOV	9
Conventional Ventilation	32
Non-Invasive Ventilation	14

After respiratory distress, sepsis was the most common complication involving 14 neonates. NEC was seen in 9 neonates. Only 3 neonates developed ROP requiring intervention like anti VEGF/ Laser. According to few studies done showed sepsis 15.7% in neonate, NCE in 10.1%, ROP in 45.1%.(4,5,6) In extreme preterm sepsis is seen frequently as they are prone to catch infections due low immunity, extended hospital stays, invasive procedures. To bring down sepsis rate in our unit we are implementing staff education, bundled approach and training in infection control.

In our unit ROP is seen significantly low when compared with other studies. It has been achieved with judicious use of oxygen and using oxygen blenders for same.

Complications	Number of neonates
Sepsis	14
NEC	9
ROP	3

Intraventricular hemorrhage was seen in 17 neonates out of which 8 had gargle I GMH, 6 had grade II, 1 had grade III and 2 had grade IV intraventricular hemorrhage.

Grade of Intraventricular Hemorrhage	Number of neonates
Grade I	8
Grade II	6
Grade III	1
Grade IV	2

Average duration of stay in the unit is 7 weeks. Mortality among this group is 32.7% which is comparable with other Indian studies.

Figure-1 Preparation of TPN

Figure-2 Delayed cord clamping

Figure-3 Human milk banking

Conclusion

For extreme preterm neonates, survival is progressively getting better. Care for extreme preterm neonates and conversations with families may be made easier with an understanding of short- and long-term outcomes. In the end, lowering preterm delivery is required to significantly lower the mortality and morbidity burden for extreme preterm neonates. RDS, NEC, and sepsis were all rather low. The rate of mortality was consistent with averages across various centers in our country. To increase the quality of a premature neonate's result, we still plan to implement adequate prenatal and postnatal care.

References

1. Patel RM, Kandefer S, Walsh MC, et al. Causes and timing of death in extremely premature infants from 2000 through 2011. N Engl J Med. 2015; 372(4):331–340.
2. Horbar JD, Carpenter JH, Badger GJ, et al. Mortality and neonatal morbidity among infants 501 to 1500 grams from 2000 to 2009. Pediatrics. 2012; 129(6):1019–1026.
3. Ancel PY, Gofnet F, Group E-W, et al. Survival and morbidity of preterm children born at 22 through 34 weeks' gestation in France in 2011: results of the EPIPAGE-2 cohort study. JAMA Pediatr. 2015; 169(3):230–238.
4. Bahadue FL, Soll R. Early versus delayed selective surfactant treatment for neonatal respiratory distress syndrome. Cochrane Database Syst Rev. 2012; 11: CD001456
5. Stevens TP, Finer NN, Carlo WA, et al. Respiratory outcomes of the surfactant positive pressure and oximetry randomized trial (SUPPORT). J Pediatr. 2014; 165(2):240–249. e244.
6. Wyllie J, Perlman JM, Kattwinkel J, et al. Part 7: Neonatal resuscitation: 2015

   International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation. 2015; 95:e169–201.

Dr. Bhargav R
Neonatal Fellow

Dr. Revanth Reddy
Neonatal Fellow

Dr. Aparna C
Clinical Director Neonatology & Senior Consultant Neonatology and Pediatrics

Dr. Aravinda Lochani T
Consultant Neonatologist & Pediatrician

Dr. P. Goutami Reddy
Consultant Neonatologist & Pediatrician

OUR EXPERTISE IN SURGICAL INTERVENTION FOR NEONATES WITH CRITICAL CONGENITAL HEART DISEASES AND POST OPERATIVE MANAGEMENT

Over a 12 month period at KIMS CUDDLES , we cared for a total of 21 Neonates diagnosed with Critical congenital heart diseases (CCHDs) .These infants had an average gestational age of 37.8 weeks with standard deviation of 1.8 weeks and an average birth weight of 2625 grams with a standard deviation of 521 grams . Among these infants, 14 were male, and 7 were female. Out of 21 neonates with CCHDs, surgical procedures were performed on all of them, and 19 were subsequently discharged to their homes

Introduction:

Congenital heart diseases (CHDs) are the most common birth defects which are responsible for 33% of all congenital birth defect. the incidence of CHDs is 8-12/1000 live birth globally.Critical congenital heart diseases (CCHDs) are 20-25 % of CHDs which requires early surgical intervention1.

CCHD was defined as any infant with a ductal dependent lesion that required intervention or resulted in death before 3 months of age 2.

CCHD was further categorized into four groups: (1) duct-dependent pulmonary circulation for pulmonary atresia or critical pulmonary stenosis (PS);

(2) duct-dependent systemic circulation for severe left ventricular outflow tract (LVOT) obstruction (aortic stenosis, coarctation of the aorta, interrupted aortic arch); (3) parallel circulation for d-transposition of the great arteries (D-TGA); and (4) critical but non-duct-dependent lesion, such as total anomalous pulmonary venous drainage, truncus arteriosus, or complex lesion associated with total anomalous pulmonary venous drainage.2,3

Significant mortality and morbidity are seen in late diagnosis. There are several factors that could potentially contribute to a delay in diagnosis. These factors include the absence of a murmur, mild hypoxemia, and early discharge from the hospital after birth4.

The purpose of screening for CHD, particularly critical congenital heart disease (CCHD), is to prevent late diagnoses, as the timing of diagnosis can greatly impact the outcome. Failure to diagnose these conditions early on after birth may result in acute cardiovascular collapse and death. If the cardiovascular system cannot be observed by echocardiography, then treatment with continuous prostaglandin-E1(PGE1) infusion should be started in any newborn whose condition deteriorates in the first few days of life5.

Indications, timing and methods for Critical congenital heart diseases mentioned in table 1, 2, 3, 4.

1. Four different variants in TOF have been described with differences in anatomy, clinical presentation and surgical management; (a) Classical TOF, (b) TOF with pulmonary atresia (c)TOF with aortopulmonary collaterals, (d) TOF with absent pulmonary valve. 6, 7.

Table - 1

	TOF/ TOF like(VSD-PS) physiology	***Single ventricle with PS***	***Ebstein anomaly of tricuspid valve***
Indications	☑ Cyanosis (Saturation <80%), ☑ Recurrent cyanotic spells *Contraindication* Hypertensive MAPCAS, ventricular dysfunction	☑ Cyanosis (Saturation <80%), ☑ Recurrent cyanotic spells *Contraindications* Severe ventricle dysfunction or mitral regurgitation	Desaturation – SpO_2 - <90%, Cardiomegaly (CTR >0.65), RV dysfunction on ECHO
Timing and methods	*<4 months with severe cyanosis/ Recurrent cyanotic spells* –Palliative modified Blalock – Taussig shunt Or RVOT/PDA stenting *4–6 months with cyanosis:* TOF repair surgery (VSD closure + RVOT resection +/– transannular patch) *Pink TOF:* 6–12 months of age (institutional policy)	*Stage 1:* Neonatal period – PDA stenting/Modified BT shunt (if neonatal severe hypoxia) *Stage 2:* At 6–12 months – bidirectional Glenn shunt *Stage 3:* At 4–6 Years – Fontan completion	*Surgical repair:* Tricuspid valve repair – 'cone repair' at 1–3 years of age *Severe forms:* Needs individualized care (single ventricle protocol)

2. There are two major subsets for TGA
   Simple TGA: In this subset, TGA is not associated with VSD or outflow tract obstruction
   Complex TGA: In this subset, TGA is associated with large VSD/PDA and/or outflow tract obstruction.
3. TA-Congenital cyanotic CHD in which, a single great vessel arises from heart as a common arterial trunk giving origin to aorta and pulmonary artery.
4. TAPVC-Pulmonary veins drains anomalously into right heart via different routes. There are 4 variants depending on site of drainage namely supra-cardiac, cardiac, Infra-diaphragmatic or mixed TAPVC If there is obstruction in pulmonary venous drainage, its can present immediately at birth. In symptomatic neonates, it may mimic like persistent pulmonary hypertension and HMD (reticular pattern on chest X-ray). 6, 7.
5. Coarctation of aorta (CoA) is a congenital defect in which narrowing of Aorta is present.
6. Aortic stenosis: AS can be valvular (80–85%), subvalvular (15%) or supravalvular (least common).
7. Pulmonary stenosis: It can be valvular (80–90%), supravalvular or subvalvular. 8, 9, 10.

Table - 2

	TGA	***Truncus arteriosus***	***TAPVC***
Indications	All patients with TGA except in those with irreversible pulmonary vascular disease presenting at later age	All patients except in irreversible pulmonary vascular disease presenting at later age	All patients diagnosed with TAPVC except in those with irreversible pulmonary vascular disease presenting at later age
Contra-indication	Irreversible PVD	Irreversible PVD	Irreversible PVD—very unlikely
Ideal age	*Simple TGA* ☑ *Asymptomatic:* Within 2–4 weeks of life ☑ *Symptomatic:* As early as possible *TGA with large VSD* At 6–8 weeks (more risk of early PVD) *TGA with CoA-* - As early as possible *Those presenting late after ideal age* Elective closure, if operable	*Uncontrolled heart failure* - As early as possible *Controlled heart failure* At 3–6 weeks (more risk of early PVD) *TA with CoA/IAA-* - As early as possible *Those presenting late after ideal age* Elective repair, if operable clinically or on cardiac catheterization	*Obstructed TAPVC* - Emergency surgery *Unobstructed TAPVC:* Electively as early as possible when diagnosed—might present late even after 1 year of age *Those presenting late after ideal age* Elective repair, if operable clinically or on cardiac catheterization
Methods	☑ *Arterial switch operation (ASO):* If LV not regressed ☑ *Two stage arterial switch operation:* Borderline LV regression presenting between 2 and 4 months of age ☑ *Atrial switch operation(Senning/ Mustard operation):* Those presenting late with regressed LV	☑ *Truncus repair – VSD closure + RV to PA conduit +/– truncal valve repair* ☑ Need for future conduit revision *surgeries to be discussed*	*Surgical TAPVC repair*

Table - 3

	CoA	AS	PS
Indications, ideal age and methods	*Indications:* CoA gradient ≥20 mm Hg (peak) with diastolic tailing LV dysfunction LVH Upper limb HTN >50% narrowing relative to aortic diameter at diaphragm *Ideal age:* ☑ LV dysfunction, CHF, severe upper limb HTN: immediate ☑ Normal LV function, no CHF, mild upper limb HTN: 3–6 months ☑ Normal LV function, no CHF/HTN: 1–2 years *Methods:* *Surgical:* Neonatal presentation, infants with native CoA *Balloon angioplasty:* Critically ill neonates, infants with recoarctation, children <25 kg with native CoA/ recoarctation *Catheter based stenting:* Children >25 kg with native CoA/recoarctation	*Valvular AS* *Immediate surgery or balloon dilation:* Neonates with critical AS, LV dysfunction *Elective balloon dilation:* ☑ Peak gradient >64 mm Hg, mean gradient >40 mm Hg, ☑ Symptomatic AS or ST changes in ECG even with lower gradients *Subvalvular AS* *Surgery:* ☑ Peak gradient ≥50 mm Hg ☑ If peak gradient is less than <50 mm Hg, if associated AR of more than mild severity, LV dysfunction or plan to involve in competitive sports *Supravalvular AS* *Surgery:* ☑ Peak gradient ≥64 mm Hg/ mean gradient ≥50 mm Hg, ☑ Lower gradients but symptomatic , LV dysfunction, severe LVH, evidence of MI	*Valvular PS* *Immediate surgery or balloon dilation:* Neonates with critical PS, RV dysfunction *Elective balloon dilation:* Peak gradient >64 mm Hg, mild RV hypoplasia causing hypoxia, dysplastic pulmonary valve *Subvalvular and supravalvular* PS *Surgery:* All indications same as mentioned above for valvular PS *Peripheral PS* Focal branch and/or peripheral PA stenosis with >50% diameter narrowing, RV systolic pressure >50 mm Hg (or >50% of systemic pressure), difference in perfusion of both lungs of >20% (on lung perfusion scan), symptomatic child *Balloon dilatation ± stenting* – treatment of choice *Surgery* – when not amenable to balloon dilatation

Echocardiography diagnosed CCHDs were included, all patients were reviewed by a paediatric cardiologist, Neonates with syndromic features were not included, CHDs were not falling into criteria of CCHDs were excluded.

Table - 4

	ASD	VSD	PDA
Indications and ideal age	L to R shunt with RV volume overload *Asymptomatic:* 2–4 years *Symptomatic in infancy:* early closure *Those presenting late after ideal age:* Elective closure, if operable	*Small VSD:* Only if associated complications like endocarditis or cusp prolapse with AR, RVOT obstruction *Moderate VSD:* Asymptomatic (2–5 yrs); symptomatic and controlled with medications (1–2 yrs) *Large VSD:* Poor growth/ uncontrolled CHF (as early as possible); controlled CHF (6 months) Any VSD with cusp prolapse and AR–immediate surgery	*Small PDA:* 12–18 months *Moderate PDA:* No CHF (6 months – 1 year); CHF (early, by 3 months) *Large PDA:* Early by 3 months
Contra-indication	Irreversible PVD (pulmonary vascular disease)	Irreversible PVD	Irreversible PVD, Silent PDA (PDA with no murmur)
Methods	*Device closure:* For OS-ASD with good rims *Surgical repair:* All other types of ASD, OS-ASD with deficient rim <5 mm	*Device closure:* Mid and anterior muscular VSDs, postoperative residual VSD, PM-VSD at least 4 mm away from aortic valve *Contraindicated* in associated AR, conduction abnormalities *Surgery:* Patch closure (standard method); *Staged PA banding* –only in multiple VSDs, inaccessible VSDs and children with contraindications for CPB	*Device closure:* Preferred in child with weight >4 kg to 6 kg (institutional policy) *Surgical ligation:* Child with weight < 4–6 kg, PDA with unusual shape, progressively enlarging or symptomatic ductal aneurysm, endarteritis

Case of neonates with CCHDs were collected and demographic, clinical presentation, antenatal history related factors and post-natal resuscitation details what factors might influence one's chance of survival were collected.

Time of presentation, cyanosis at presentation, shock before surgery, inotropes usage , need for PD(Peritoneal dialysis) after surgery , Any shock post-surgery, Antibiotic duration, sepsis screen, blood culture was included was risk factors. During the study period, all neonates were treated as per the unit's antibiotic policy which clearly laid down the empirical choice, duration, dose and criteria for downgrading or upgrading antibiotics, A dedicated cardiology team conducted the surgeries.

Of the total 21 neonates were surgically operated with critical congenital heart diseases during the study period, 19(90%) neonates were dicharged home . Baseline characteristics of the study group is described in Table 5. Morbidities of study population are mentioned in table 6.

Table 5: Base line characteristics of neonates with CCHDs

Variable	
Birth weight ,grams	2500(521)*
Gestational age,weeks	2500(521)*
Male	14(66.66%)
Female	7(33.33%)
Duration of ventilation post-surgery	3(2.3)*
Prostaglandins usage before surgery	6(28%)
Cyanosis as presentation	12(57%)
Shock before surgery	16(76%)
Early presentation	19(90%)
Hospitalized days	15.7(11)

* Expressed as mean (standard deviation); Rest as numbers (proportions)

Table 6: Morbidities among included neonates

	Morbidities	
1	Culture positive sepsis	19%
2	Peritoneal dialysis	9%
3	Post surgical shock	38%
4	VAP	9%

We are delighted to announce that KIMS Cuddles has achieved remarkable success in delivering top-tier care for cardiac cases, exemplified by a series of successful procedures. Our dedicated team of medical professionals has not only demonstrated their expertise but also their commitment to improving the lives ofour patients.

Among the noteworthy accomplishments are: Total Anomalous Pulmonary Venous Connection (TAPVC): Successfully performed 7 TAPVC surgeries, ensuring proper blood flow and function within the heart. Coarctation of the Aorta (COA): Excellently managed and treated 6 cases of COA, relieving patients from this congenital heart defect.

Ventricular Septal Defect (VSD) Closure: Skillfully closed 3VSDs, promoting better cardiac function and overall well-being for our patients.

Transposition of great arteries (TGA):we succeded in positioning the arteries which lead to normal thrivingof baby.

Patent Ductus Arteriosus (PDA) Stenting: Accomplished 2 PDA stenting procedures, aiding in the correction of this condition and enhancing the quality of life for our patients.

Seymeter syndrome again a type of TAPVC we were able to bring significant changes in life of that neonate

Taussig-Bing Anomaly: Successfully tackled a complex syndrome called Taussig-Bing Anomaly, showcasing our ability to manage intricate cardiac issues.

At KIMS Cuddles, we remain steadfast in our commitment to delivering the highest level of care, utilizing cutting-edge technology, and harnessing the expertise of our healthcare professionals. Our success stories in treating cardiac cases highlight our dedication to improving the health and well-being of our patients. We look forward to continuing this journey of excellence and making a positive impact on more lives in the future

References:

1. Wren C, Reinhardt Z, Khawaja K. Twenty-year trends in diagnosis of life-threatening neonatalcardiovascular malformations. Archives of Disease in Childhood-Fetal and Neonatal Edition. 2008 Jan1;93(1):F33-5.
2. Fixler DE, Xu P, Nembhard WN, Ethen MK, Canfield MA. Age at referral and mortality from criticalcongenital heart disease. Pediatrics. 2014 Jul 1;134(1):e98-105.
3. Eckersley L, Sadler L, Parry E, Finucane K, Gentles TL. Timing of diagnosis affects mortality in critical congenital heart disease. Archives of disease in childhood. 2016 Jun 1;101(6):516-20.
4. Brown KL, Ridout DA, Hoskote A, Verhulst L, Ricci M, Bull C. Delayed diagnosis of congenital heartdisease worsens preoperative condition and outcome of surgery in neonates. Heart. 2006 Sep 1;92(9):1298-302.
5. Knowles R, Griebsch I, Dezateux C, Brown J, Bull C, Wren C. Newborn screening for congenital heart defects: a systematic review and cost-effectiveness analysis. Health technology assessment (Winchester, England). 2005 Jan 1;9(44):1-52.

6. Moss AJ. Moss and Adams' heart disease in infants, children, and adolescents: including the fetus and young adult. Lippincott Williams & Wilkins; 2008.

7. Bernier PL, Stefanescu A, Samoukovic G, Tchervenkov CI. The challenge of congenital heart disease worldwide: epidemiologic and demographic facts. InSeminars in Thoracic and Cardiovascular Surgery: Pediatric Cardiac Surgery Annual 2010 Jan 1 (Vol. 13, No. 1, pp. 26-34). WB Saunders.8. Hoffman JI. The global burden of congenital heart disease. Cardiovascular journal of Africa. 2013 May 1;24(4):141-5.9. Saxena A, Relan J, Agarwal R, Awasthy N, Azad S, Chakrabarty M, Dagar KS, Devagourou V,Dharan BS, Gupta SK, Iyer KS. Indian guidelines for indications and timing of intervention for common congenital heart diseases: Revised and updated consensus statement of the Working group on management of congenital heart diseases. Annals of pediatric cardiology. 2019 Sep;12(3):254.10. Working Group on Management of Congenital Heart Diseases in India. Consensus on timing of intervention for common congenital heart disease. Indian pediatrics. 2008 Feb;45(2):117-26.

Dr. B. Ganesh Yadav
Neonatal Fellow

Dr. Aravinda Lochani T
Consultant Neonatologist & Pediatrician

Dr. P. Goutami Reddy
Consultant Neonatologist & Pediatrician

Dr. Anil Dharmapuram
Sr. Consultant Paediatric Cardiac Surgeon

Dr. Nagarajan R
Sr. Consultant Pediatric Cardiac Anaesthesiologist

Dr. Sudeep Verma
Consultant Pediatric Cardiologist

Dr. Aparna C
Clinical Director Neonatology & Senior Consultant Neonatology and Pediatrics

OUR EXPERIENCE IN TREATING BABIES WITH PERSISTENT PULMONARY HYPERTENSION (PPHN) REQUIRING INHALED NITRIC OXIDE (INO)

PPHN is a frequent complication of respiratory disease in neonates. It complicates the course of approximately 10% of infants with respiratory failure. PPHN can lead to severe respiratory distress and hypoxemia associated with considerable mortality and morbidity. Incidence for PPHN is 1.9/1000 live births, being highest in term and late preterms. Persistent pulmonary hypertension is a cardiopulmonary disorder leading to systemic hypoxemia secondary to failure to achieve or maintain normal decrease in pulmonary vascular resistance at birth ,with resultant right-to-left shunting through persistent fetal channels such as the ductus arteriosus and foramen ovale, bypassing the lungs. Inadequate pulmonary blood flow leads to refractory hypoxemia, respiratory distress, and finally acidosis.

PPHN is associated with variety of lung diseases like meconium aspiration syndrome, perinatal asphyxia, congenital diaphragmatic hernia (CDH), respiratory distress syndrome, usually accountable for its severe outcomes. PPHN can also occur in preterm infants if complicated with rupture of membranes and pulmonary hypolpasia. Newborns with PPHN are also at high risk of severe asphyxia and its complications, including neurological injury, multiorgan dysfunction, and death.

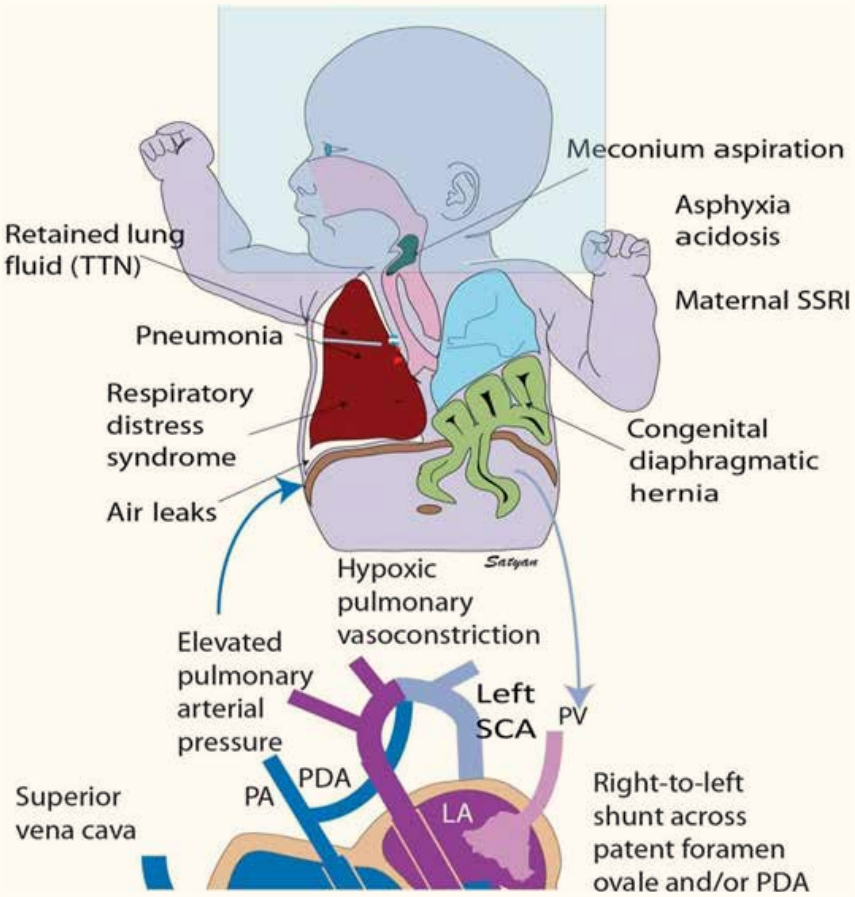


Figure-1 Etiology and pathophysiology of persistent pulmonary hypertension of the newborn (PPHN). Secondary PPHN can be due to various lung diseases, such as retained lung fluid or transient tachypnea of newborn (TTN), pneumonia, aspiration syndromes, respiratory distress syndrome (RDS), and congenital diaphragmatic hernia with lung hypoplasia

Response to inhaled NO may depend upon the aetiology of neonatal acute respiratory failure.iNO improves the oxygenation in most newborns with severe hypoxaemic respiratory failure including preterm neonates.

Inhaled Nitric Oxide (iNO) is a vascular endothelial relaxing agent.

Mechanism of action : NO is generated from the vascular endothelium of lung by the action of NO Synthase on L-Arginine which is converted to L-Citrulline. NO diffuses through vascular endothelium activates guanylate cyclase, increases cGMP, which triggers cGMP kinase and opens calcium activated potassium channels and thereby relaxes vascular smooth muscle (Fig. 2).

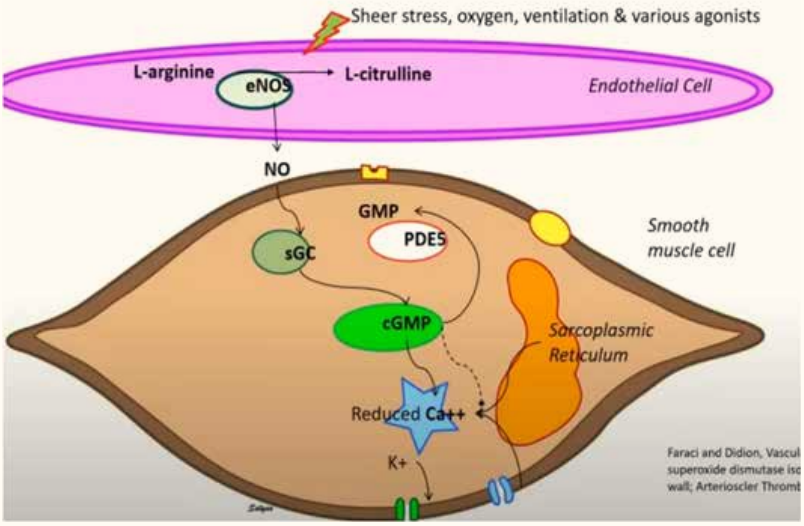


Figure-2 Action of inhaled nitric oxide

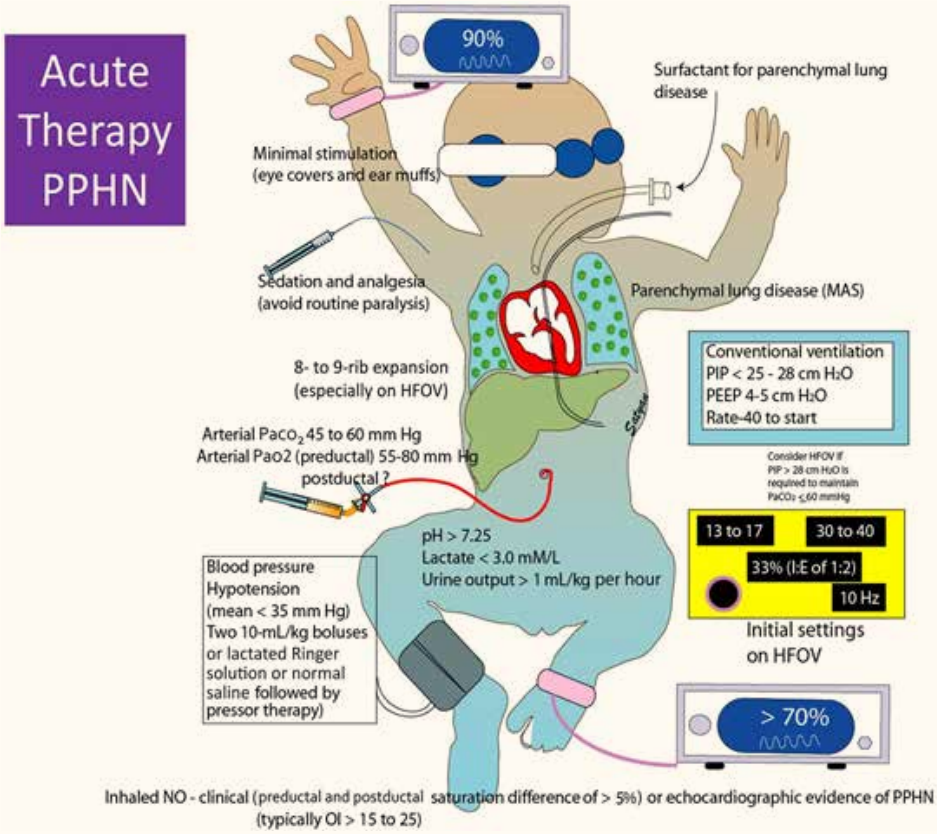


Figure-3 Summary of management of PPHN

Actions of iNO :

1. Micro- selective pulmonary vasodilation. Selectively distributed to the ventilated segments of lung and redistributes pulmonary blood flow away from non-ventilated segments and decreases pulmonary artery pressure
2. Improves ventilation perfusion (VQ) mismatch
3. Decreases right to left , extra and intra cardiac shunting and intra pulmonary shunts
4. Reduces inflammation by decreasing the cumulation of neutrophils in lung
5. Overall enhancement in oxygenation in hypoxic respiratory failure Half life : 3-5 seconds with brisk, prolonged and prominent action.

Pre-requisite before starting iNO:

Always do echocardiography to assess for

1. Cyanotic congenital heart disease.
2. Left ventricular dysfunction
3. Pulmonary hypertension.
4. Cardiac function.

Rule of 20-20-20:

Start iNO at Oxygenation index (OI) 20 - Initial dose 20 ppm, expected rise in PaO2 / FiO2 - 20 mm Hg Initiation at OI 15-20:

1. Decreased progression to severe criteria (RR-0.66 with CI- 0.55 to 0.79; 512 infants)
2. Reduced worsening of respiratory failure
3. Reduced need for ECMO/death (RR-0.88 with CI- 0.62 to 1.27; 495 infants)

While on iNO, monitor

1. Clinical – SpO2 (pre & post ductal), FiO2
2. Labs - Blood gas- PaO2, OI, Methemoglobin
3. Echo – Right ventricular afterload, septal position, PDA, direction of shunts at foramen ovale and PDA.

Weaning:

1. Rule of 60-60-60 Wean when FiO2 is 60% PaO2 > 60 mm hg (SpO2 >90% / OI5%, high NO2 levels in blood.
2. Avoid rapid weaning
3. Reduce by 5ppm every 4 hrs
4. After reaching 5ppm, reduce by 1ppm every 4 hours
5. Stop after reaching 1 ppm. Endogenous NOS activity is usually restored in 30-60minutes after stopping iNO

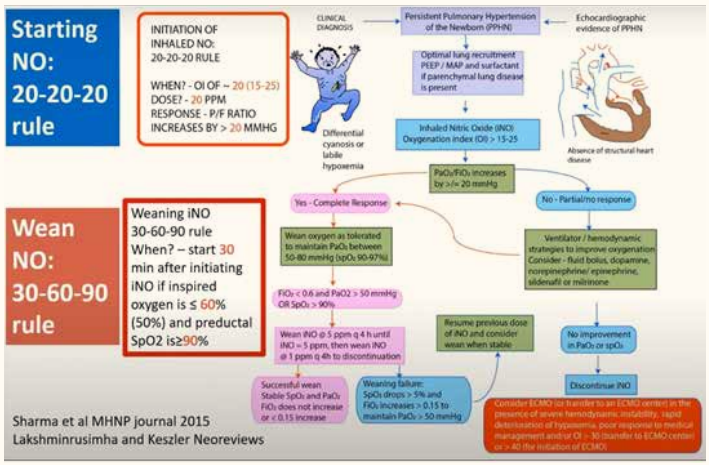


Figure-4 Protocol for titrating inhaled nitric oxide

However, response to iNO is disease-specific. iNO when combined with adequate alveolar recruitment and limited barotrauma using exogenous surfactant (Fig. 3 & 4) and HFOV may obviate the need for extracorporeal membrane oxygenation in many term infants.

Our experience with PPHN

In our hospital at KIMS (Krishna Institute of Medical sciences) CUDDLES neonatal intensive care unit, during the period of 1 year from August 2022 to July 2023, 25 neonates with PPHN were admitted requiring HFOV with inhaled nitric oxide (I-NO). All these neonates presented with hypoxemic respiratory failure and were mechanically intubated and connected to ventilator. Most of the babies were changed to HFOV i/v/o failure of conventional ventilation (MAP >10 in PT and >12 in Term). Neonates were started on I-NO if they develop postnatal OI index (>20) and were monitored strictly. The average birth weight was 2474 (+-938) grams and mean gestational age in weeks was 35.9 (4.9) weeks. Of the total study population 14 were male and 11 were female with 8 babies being SGA and 11 babies being AGA. During hospital stay multiple variables were recorded from history,workup and examination like gestational age, birth weight, gender, mode of conception, mode of delivery, abnormal antenatal and intrapartum history, blood gas for pH, initial OI, maximum OI, duration on I-NO, vasoactive inotrope score (VIS), X ray findings and ventilator settings like PIP,MAP,drugs like milrinone, sildenafil and Bosentan (Table 1). The average duration on ventilation for the babies was 6 days. Gestational age was an important risk factor for mortality. We discharged 20 babies of the study group with normal respiratory and neurological outcome at discharge.

Table 1: Base line characteristics of neonates with PPHN on iNO

Variable	
Birth weight ,grams	2474 (938)
Gestational age,weeks	35.9 (4.9)
Gender	
Male	14 (56%)
Female	11 (44%)
Weight	
SGA	8 (32%)
AGA	17 (68%)
APGAR <5	
At 1 minute	15 (60%)
PH	7.18 (0.11)

Table 2: Morbidities among included neonates

	Morbidities	
1.	Primary PPHN	20%
2.	RDS	60%
3.	MAS	0
4.	Post surgical PPHN	20%
5.	Sepsis	80%
6.	Encephalopathy	20%

Dr. N. Aruna Devi
Neonatal Fellow

Dr. Aravinda Lochani T
Consultant Neonatologist & Pediatrician

Dr. P. Goutami Reddy
Consultant Neonatologist & Pediatrician

Dr. Aparna C
Clinical Director Neonatology & Senior Consultant Neonatology and Pediatrics

SCOPE OF SERVICES - PEDIATRICS & NEONATOLOGY

Pediatrics Department Comprises:

- Outpatient Clinics
- Pediatric Emergency Area
- Children's Ward & Rooms
- Neonatal Intensive Care Unit (NICU)
- Pediatric Intensive Care Unit (PICU)
- OT Complex

Outpatient Clinics:

- Well Baby Clinics
- Antenatal Counselling
- Growth Monitoring & Immunization
- General Pediatric Clinics

Specialty Clinics:

- Pediatric Surgery & Urology
- Pediatric Neurology & Neurosurgery
- Pediatric Hemato-oncology & BMT
- Pediatric Pulmonology
- Pediatric Cardiology & Cardiac Surgery
- Pediatric Endocrinology
- Pediatric Nephrology
- Pediatric Gastroenterology & Hepatology
- Pediatric ENT
- Pediatric Ophthalmology
- Pediatric Dentistry
- Pediatric Nutrition
- Orthopaedic Services
- Plastic Surgery
- Developmental Pediatrics
- Pediatric Psychiatry

Other Services:

- MRI Scan
- CT Scan
- Blood Bank
- Human Milk Bank
- 24/7 Transport Services Available

KIMS CUDDLES™
MOTHER & CHILD CENTRE

Our Network Hospitals:

KIMS Cuddles, Kondapur, Hyderabad
Address: KIMS Hospitals Premises, Near RTA Office, Kondapur, Hyderabad.

KIMS Cuddles (KIMS Hospitals Campus), Gachibowli
Address: Dargah Road, Raidurg, Gachibowli, Hyderabad.

KIMS Cuddles (KIMS Hospitals Campus), Secunderabad
Address: Minister Road, Secunderabad, Telangana.

KIMS Cuddles (KIMS-ICON Hospital Campus), Visakhapatnam
Address: Sheela Nagar, BHPV Post, Visakhapatnam, Andhra Pradesh.

KIMS Cuddles (KIMS-KINGSWAY Hospitals Campus), Nagpur
Address: 44, Parwana Bhawan, Kingsway, Nagpur, Maharashtra.

For consultation & admission, contact: 040 - 4243 4243
24/7 Helpline no.: 91006 55997
hello@kimscuddles.com | www.kimscuddles.com