NeoBlaze

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National Neonatology Forum

Gujarat State Chapter

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Greetings for Editors desk

Dr Ravi Parikh

I am proud and thankful to Gujarat NNF to release second edition of Neoblaze. It was privilege to get on board the authors who are expert in their field and have done excellent job in writing this article. They have given very good messages for each topic. I am thankful to whole Gujarat NNF team with special reference to Dr Prashant Karia and Dr Dipen Patel for their constant efforts and Neoblaze team to always be ready for the instructions and changes.

This edition brings diverse topics so that you can enjoy reading and learn from each. Neonatal echocardiography is now one of the mainstay tool as a POCUS in NICU. The details of indications and right way to perform focused echocardiography with help of pediatric cardiologist in structural lesions has been highlighted to understand and keep in practice so it is safe as a neonatologist to use this modality. Hypoxic ischemic encephalopathy and therapeutic hypothermia topic has very important messages highlighted. Another important topic of lactation has been written in very easy and spectacular way with understanding of the physiology of lactation. Meconium aspiration management has been highlighted in detail with all management stepwise till nitric oxide in very understandable way. I would especially mention the topic of neonatal seizure which has been written with extreme focus to neonatal population. I urge everybody to go ahead and read all the important articles and its message. I am very much sure that you will enjoy and learn from each articles.

With Warm regards,

Dr Ravi Parikh

Consultant Neonatologist

Setu Newborn Care Centre

Ahmedabad.

President's Message

Dr Dipen Patel

Dear Friends,

नमस्ते !

It is indeed a pleasure to release the 2nd Issue of NeoBlaze for the year 2023, the official quarterly E-Bulletin of NNF, Gujarat State Chapter.

I am extremely grateful to the Editor of NeoBlaze Dr. Ravi Parikh; Associate Editor Dr Prashant Kariya; Members Dr. Ronak Patel, Dr. Manan Parikh, Dr. Reshma Pujara, Dr. Jatin Unadkat, all the office bearers and the Executive Board Members for this milestone. This issue is exceptionally relevant as it incorporates recent upgrades with points of interest for the clinical conditions encountered in day to day practice. I thank all the Authors for profoundly enlightening articles.

I thank all the NNF members of Gujarat for involving themselves in the upliftment of care of newborns through actively taking part in various academic activities organized in the second quarter.

I thank Dr. Shilpi Shah, Shriji Healthcare and Century Surgical Instruments for providing generous funding for NeoBlaze.

We seek your continuous support and blessings in future issues of NeoBlaze.

धन्यवाद।

Prof. (Dr.) Dipen V. Patel

President,

NNF Gujarat State Chapter

Hon. Secretory's Message

Dr Prashant Kariya

Dear Esteemed Members and Supporters of NNF Gujarat

I hope this message finds you all in good health and high spirits. It is with immense gratitude and enthusiasm that I write to you today, as we compile another edition of our bulletin. This bulletin serves as a testament to the collective efforts and unwavering commitment of our dedicated members and contributors.

First and foremost, I would like to extend my heartfelt thanks to all the contributors who have generously shared their knowledge, insights, and experiences. Your valuable contributions have enriched our bulletin and continue to inspire our community.

I would also like to express my profound appreciation to our Executive Board members for their active involvement and tireless dedication to our cause. Your leadership and guidance are the driving forces behind our success, and I am grateful for the privilege of working alongside such a dedicated team.

I am excited to extend an invitation to all of you for the upcoming state-level conference, Gujneocon 2023 at Anand. This event promises a unique blend of academic excellence and camaraderie. It will be an opportunity for us to come together, share our knowledge, and forge lasting connections within our community.

In our relentless pursuit of improving neonatal care, NNF Gujarat has made significant strides. During Breastfeeding Week in August, we launched awareness campaigns that have played a pivotal role in reducing neonatal mortality rates. Your unwavering support and participation have made these initiatives possible, and we are determined to continue this essential work.

I am delighted to announce the launch of the E-module for Kangaroo Mother Care (KMC). This milestone reflects our commitment to advancing neonatal care through innovation and education. It is a testament to our collective dedication to improving the lives of newborns and their families.

In closing, I leave you with a quote that embodies our mission: "**Alone, we can do so little; together, we can do so much." - Helen Keller**. Let us continue to work together, strive for excellence, and make a meaningful impact on neonatal care in Gujarat.

With warm regards,

Dr. Prashant Kariya

Honorary Secretary, NNF Gujarat

National Neonatology Forum - Gujarat State Chapter

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Clinical indications of focussed neonatal echocardiography

Dr Ravi Parikh
Consultant Neonatologist
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Introduction

Echocardiography in the neonatal intensive care unit (NICU) has been a regular modality since past few years[1]. Previously Paediatric cardiologists used to diagnose or monitor congenital heart disease (CHD) and screen forpatent ductus arteriosus (PDA).More recently, neonatologists have become interested in theechocardiographic assessment of hemodynamic instability in infants. The terms functional echocardiography and point-of-care echocardiography have been introduced to describe the use of echocardiography as an adjunct in the clinical assessment of the hemodynamic status in neonates. In addition, newborns in the NICU are unique in that they are in the process of transition from fetal to postnatal circulation.

Clinical approaches to circulatory management in the neonatal unit vary significantly. The North American ELGAN study has demonstrated that the rate of vasopressor use in infants of less than 28 weeks gestation varies between 6% and 64% in different centres[7], and that this range is not due to differences in illness severity between populations. Similar variability is seen in rates of intervention to close a patent ductus arteriosus[8].

Improving circulatory management is therefore a research priority in preterm infants[9]. However, understanding of the pathophysiology and optimal treatment of circulatory failure is hampered by the limited tools available to monitor circulatory function[10]. In particular, monitoring of circulatory status in the neonatal unit still relies heavily on arterial blood pressure. However, systemic arterial blood pressure is the product of systemic blood flow and systemic vascular resistance, and cannot itself distinguish between the two. While clinicians presumably feel that monitoring systemic blood pressure is a screening tool for low systemic perfusion, in fact blood pressure is at best weakly predictive of volume of blood flow[11], and some studies have suggested no[12] or even an inverse relation[13] between BP and flow in newborn preterm infants. Other clinical assessments, such as capillary refill time, volume of urine output, etc also have limited value in indicating circulatory health[11].

A number of research tools, including near infra-red spectroscopy have produced important advances in the understanding of circulatory physiology, but echocardiography certainly has the clearest role in the

assessment of circulatory status at the cot-side. Echocardiography is cheap, safe, non-invasive and provides instant results with minimal or no post-processing of images.

Thus focussed neonatal echocardiography includes structural echocardiography to rule out congenital heart disease and functional echocardiography for circulatory management. Neonatologist doing echocardiography needs to understand the difference of indication as the role and responsibility differs when doing each examination.

Clinical applications of the focused neonatal echocardiography

The applications have been listed as functional and structural applications for better understanding.

Clinical applications of functional echocardiography

1. Newborn baby with hypotension
2. Preterm baby with sepsis and other reason of circulatory failure
3. Premature babies with Patent Ductus Arteriosus (PDA)
4. Baby with poor oxygenation (PPHN) when CHD is ruled out.
5. The very preterm baby during the transitional period.
6. Premature babies with chronic lung disease

Clinical applications of structural echocardiography

1. Blue baby (cyanotic baby)
2. Baby with heart murmur
3. Preterm baby with unexplained collapse having central access with TPN
4. Antenatal diagnosed structural cardiac problem – To confirm or get additional diagnosis
5. Babies with chromosomal abnormalities – high chance of congenital heart disease.
6. Baby born of mother with IDDM (Insulin dependent diabetes mellitus)

These indications for echocardiography will ultimately need Paediatric Cardiologist to be involved and will not be discussed here.

Role of functional echocardiography in the Infant with hypotension

It is a common assumption in neonatology that normal blood pressure equates to normal SBF, and improving blood pressure means that blood flow must also have improved. However, in very preterm infants during the first postnataldays, mean blood pressure does not correlate well with the simultaneously measured left ventricular output. Consequently, the ability to repeatedly assess SBF at the bedside is of considerable importance. Hypotension in the neonate can be due to several underlying scenarios with variable hemodynamic physiology.Very preterm infants may initially have low SBF and/or a large PDA. Term infants may have poor myocardial function as the result of asphyxia, pathological vasodilatation in septic

shock or asphyxia, or, less frequently, hypovolemia with cardiac under filling caused by fluid or blood loss. Each of these situations potentially results in low blood pressure. However, the appropriate management and logical choice of therapy in each varies.

Echocardiography can be used to differentiate between these situations, combining measurement of cardiac output, assessment of cardiac filling, and myocardial function and even exclusion of life-threatening pathology, such as a pericardial effusion, tamponade from an extravasation of a central line or from other causes.

Role of functional echocardiography in assessment and monitoring of the Patent Ductus Arteriosus (PDA) in the preterm Infant

In some preterm infants, the ductus arteriosus effectively constricts within a few hours of birth, although others have a persisting large ductus arteriosus with no evidence of constriction. Many of the strategies for treating patent ductus arteriosus (PDA) are based on the incorrect assumption that early ductal shunting is of limited hemodynamic significance. The dominant direction of ductal shunting in the early postnatal period is left to right, and, in those ducts that fail to constrict, large volumes of blood move from the systemic to pulmonary circulation. The early left-to-right shunting results in consequences such as reduced systemic blood flow and blood pressure, increased ventilatory requirements, and pulmonary hemorrhagic edema. These hemodynamic effects may paradoxically be more important in the early hours after birth rather than later in the clinical course. These findings lend indirect support to the emerging suggestions regarding early/prophylactic therapy of the PDA and subsequent tolerance of the PDA in older infants who do not have cardiac failure. Functional echocardiography to assess early ductal constriction in infants during circulatory transition allows prediction of the likely closure of the ductus arteriosus and potential targeting of early treatment of the ductus arteriosus rather than nonspecific prophylaxis. Functional echocardiography provides the clinician with further information to aid decision-making beyond just the presence or absence of the PDA. Longitudinal assessment of changes with time allows a judgment regarding the likely closure of the ductus arteriosus. In addition, measurement of hemodynamic effects such as the direction of diastolic flow in the descending aorta and an estimate of the pulmonary blood flow give an indication of the impact of the ductus arteriosus on the cardiovascular status.

Role of functional echocardiography in assessment and response to treatment of an infant with high oxygen requirements

Babies with suspected persistent pulmonary hypertension present the neonatal intensivist with a range of respiratory and hemodynamic therapeutic challenges. As with the group of infants with hypotension, functional echocardiography reveals a broad range of

pathology underlying the clinical presentation. These babies are often assumed to have pulmonary hypertension with right-to-left ductal shunting. In fact, such assumptions are often erroneous and can lead to inappropriate management.

The ductus in such babies usually constricts early in the course and may close after the first 24hours.

In addition, there is a complex interplay between pulmonary and systemic blood flow, with both often measuring low. Because congenital heart disease can present in this way, these infants must have this possibility excluded early on in their course by a pediatric cardiologist. Another important aspect of the use of functional echocardiography in these infants is monitoring changes over time and responses to treatment such as vasopressor-inotropes, inotropes, and vasodilators. Variability in response to treatments such as inhaled nitric oxide probably relates to this hemodynamic spectrum. This group of infants also underscores the importance of having functional echocardiography readily available in the NICU.

The Very Preterm Infant during the Transitional period

The first 24 postnatal hours after birth of the very preterm infant is a period of unique circulatory vulnerability. A significant number of babies develop not only hypotension but also low systemic blood flow (SBF)[17] During this period, low SBF is often not recognized by measurement of blood pressure and has been associated with a range of adverse out comes, both short and long term[18-19]. The usual measurements of cardiac output used in older children and adults, such as the left ventricular output, are affected by the transitional circulation, so other measures of SBF such as measurement of superior vena cava flow have been developed. Low SBF also relates to larger ductal shunts, so assessment of the early constriction of the ductus arteriosus is important inearly echocardiographic assessments. After the transitional first 24 hours, where low SBF predominates, hypotensivebabies usually have normal or high SBF, indicating low peripheralvascular resistance that is probably due to abnormalregulation of vascular tone. This change from low to higher SBF has been implicated in the development of peri Intraventricular hemorrhage in the very preterm neonate.

Summary

Focused neonatal echocardiography is definitely very useful non invasive tool for additional clinical information especially in sick babies. Its usefulness and limitation as a neonatologist should be fully understood in day to day clinical practice. For functional echocardiography to fulfill its clinical potential, it needs to be available at any time in the NICU. Neonatologists should be ableto develop echocardiographic skills in close collaboration

with their cardiologist colleagues. It is skill which should be a part of the curriculum for a neonatal trainee.

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Hypoxic Ischemic Encephalopathy and its management in term newborn babies

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Introduction

Perinatal asphyxia (PA) is a major cause of neonatal and under 5 years mortality, especially in developing countries like India. Perinatal asphyxia is among the 3 most common causes of neonatal deaths. As per the NNPD (National Neonatal Perinatal Database), the incidence of PA was 8.4 % of all live births and it was responsible for 28.8% of all neonatal deaths. Hypoxic Ischemic Encephalopathy (HIE) manifestations were seen in approximately 1.4% of live births.HIE refers to CNS dysfunction associated with PA. HIE is of prime concern as it is associated with high risk of mortality and those who survive have a high risk of serious long-term neurodevelopmental sequelae.

Definition of Perinatal Asphyxia

WHO

Failure to initiate and sustain breathing

NNPD Network

Moderate PA : Slow/gasping breathing or an Apgar score of 4-6 at 1 minutes of age

Severe PA : No breathing or an Apgar score of 0-3 minutes at 1 minutes of age

American Academy of Pediatrics (AAP)

Presence of following criteria:

- Profound metabolic or mixed acidemia (pH < 7.0) in umbilical cord blood
- Persistence of low Apgar scores less than 3 for more than 5 minutes
- Signs of neonatal neurologic dysfunction (e.g., seizures, encephalopathy, tone abnormalities)
- Evidence of multiple organ involvement (such as that of kidney, lungs, liver, heart and intestine)

Table: Modified Sarnat & Sarnat Staging

Severity	Stage 1	Stage 2	Stage 3
Level of Consciousness	Hyperalert	Lethargic	Stupor/coma
Activity	Normal	Decreased	Absent
Neuromuscular control ● Muscle tone ● Posture ● Tendon reflexes	 ● Normal ● Mild distal flexion ● Overactive	 ● Mild hypotonia ● Strong distal flexion ● Overactive	 ● Flaccid ● Intermittent decerebration ● Decreased or absent
Neonatal reflexes ● Suck ● Moro ● Tonic neck	 ● Weak ● Strong ● Slight	 ● Weak/absent ● Weak, incomplete ● Strong	 ● Absent ● Absent ● Weak
Autonomic nervous system ● Pupils ● Heart rate ● Respiratory rate	 ● Dilated pupils ● Tachycardia ● Regular	 ● Constricted pupils ● Bradycardia ● Periodic breathing	 ● Variable, unequal ● Variable ● Apnea
Seizure	None	Common, focal or multifocal	Uncommon

Evolution of HIE changes

There is a gradual evolution of HIE over time from the start of insult to hours and days later. The initial hypoxic ischemic insult results in infarction of the brain tissue i.e. primary energy failure. Secondary injury is subsequently mediated by reperfusion and free radicals in the area surrounding the ischemic area called as penumbra. Penumbra area undergoes apoptosis even after the hypoxic insult is over. Hence, we get a window between these two phases (< 6 hours) to institute specific therapeutic intervention.

Management

Management of PA is mainly supportive and involves optimizing oxygenation, ventilation, perfusion, metabolic milieu and control of seizures.

Delivery room management

- Resuscitation should be started as per the latest NRP guidelines
- Resuscitation should be initiated with 21 % oxygen for term and near-term neonates
- For preterm neonates resuscitation should be started with 21 to 30 % oxygen
- Try to obtain cord blood ABG. (pH <7.0 and base deficit of ≥ 16 mmol/L is associated with short & long term adverse outcomes

NICU admission

- Apgar score of <3 at 1 minute
- Prolonged (> 60 seconds) of bag and mask ventilation
- Requirement of chest compression

Care in NICU

1. **Maintain normal temperature**
   - Maintain normal temperature by placing the baby in radiant warmer (except for babies requiring therapeutic hypothermia)
   - Avoid hyperthermia
2. **Maintain normal oxygenation & ventilation**
   - Maintain saturation between 90-95%; avoid hypo or hyperoxia
   - Assisted ventilation is required in neonates with apnea, inadequate spontaneous respiration, hypoxia or hypercarbia
3. **Maintain normal tissue perfusion**
   - Ensure normal CRT, BP and urine output; ensure absence of tachycardia and metabolic acidosis
   - Start intravenous fluid in neonates with Apgar < 4 at 1 min or < 7 at 5 minutes. Avoid routine restriction of IV fluids
   - Do ECHO for assessing cardiac dysfunction
   - Start inotropes - Dobutamine or Adrenaline
4. **Maintain normal metabolic milieu and hematocrit**
   - Maintain blood glucose levels between 75-100 mg/dL
   - Maintain hematocrit between 45-55 %
   - Maintain pH > 7.30
   - Check ionic Calcium and give IV calcium if levels are low

5. **Treat seizures**

- Phenobarbitone is the first line drug and if seizures persist then treat as the neonatal seizure protocol

6. **Nutrition**

- Start oral feeding as soon the baby is hemodynamically stable and off vasopressor support.

Special investigation

Electroencephalography (EEG) / aEEG

Not routinely indicated in all babies, but helps in diagnosis and management of seizures and prognosticating long term outcome. aEEG is a simple, reliable, non-invasive technique which can be applied in NICU for monitoring EEG continuously.

Cranial Ultrasound

Cranial USG is not good for detecting changes of HIE. Resistive Index (RI) measurement can be done and if it is ≤ 0.55 within first 24 hours; it indicates poor prognosis.

Magnetic Resonance Imaging (MRI)

MRI is the best imaging modality for determining prognosis in term neonates with HIE. Diffusion weighted MRI can detect abnormalities within 24-48 hours after birth, whereas conventional MRI can detect abnormalities in first 3-4 days.

Specific Management – Therapeutic hypothermia (TH)

Initiation of therapeutic hypothermia (33-34°C) within first 6 hours has been shown to reduce mortality and neuro-morbidity by 18 months of age. It has to be continued for 72 hours and then there should be gradual rewarming over next 12 hours. Cochrane review has shown that TH used in term neonates with moderate to severe HIE, reduces the combined outcome of mortality or major neurodevelopmental disability by 24% at 18 months of age. TH has been shown to be protective at critical cellular and vascular sites of cerebral injury. TH reduces the secondary injury in penumbra area.

Follow up

All neonates with moderate to severe PA, especially those with stage II and III HIE should be followed up. They should have complete neuro developmental assessment and early intervention therapy. Among survivors with severe HIE, there can be sequelae like mental retardation, epilepsy and cerebral palsy.

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- All Type of Disposable Ventilator circuit
- All Type of Reusable Ventilator circuit
- Bubble CPAP Circuit
- All Type of Sensor of Monitor
- All Type Battery
- Disposable Spo2 Sensor
- Ambu Bags
- Weights Scales
- Oxygen Hood
- Laryngoscope
- Nebulizer Machine















Latching and Breastfeeding
Do we know the basics?

Dr Prachee Shah
Pediatrician and Lactation Consultant.
Founder:NEEV Breastfeeding clinic

Common scenario:

27 year Primi mother comes at 1 month postpartum with complains of not having enough milk and baby not latching directly. I truly wished that same old story does not repeat. But history revealed the same events [failure to latch at birth or earlier, use of shield, use of top milk]. What could be done to avoid this?? Let's begin from beginning.

Stages of Lactogenesis:

Stage	Duration	Hormones	Mechanism
Mammogenesis	Woman's birth to her pregnancy	Estrogen, progesterone	Proliferation Of duct and glandular system
Lactogenesis I	16 weeks of pregnancy to day 2-3 postpartum	Human placental Lactogen, Prolactin, Estrogen, Progersterone	Differentiation of alveolar cells into secretory cells. Colostrum production starts.
Lactogenesis II	Day 3-8 postpartum	Progesterone ↓ Prolactin increased ↑	Prolactin- ↑alpha lactalbumin-↑ lactose-water drawn- copious milk production
Lactogenesis III/ galactopoiesis	Day 9postpartum to beginning of involution	Autocrinemode. work on demand and supply mechanism.	Feedback inhibitor of lactation {FIL} collects in full breast and slows rate of milk production

In nutshell, breast starts making milk from 2ndtrimester. First 3 days after birth small quantity of colostrum is formed and between 3-5 day postpartum milk production increases and 9th day onwards milk production works on demand –supply principle and frequency of breast emptying.

Ideal time to start latching and breastfeeding.

Immediately after birth, babies are in quiet alert stage for 2 hours and then sleep for next 4 hours.If babies are placed on mother's abdomen or chest immediately after birth,they go through 9 instinctive stages and self latch on mother's breast.Thisself latch will be deep and willcause imprinting of mother's nipple in baby's mouth.

Instinctive stages	Time after birth
Birth cry	
Relaxation	
Awakening slowly opens eyes,moves head and shoulder	3 min
Activity. stronger movements and sucking and rooting motions.	8min
Crawling. approach breast and nipple	35 min
Resting. several times during first hour	
Familiarization. becomes acquainted with mother by licking the nipple and touching and massaging breast. Droolsand places tongue on floor of mouth	45 min later and last for 20 min.
Sucking. opens mouth wide,lips latch on areola and takes the areola in mouth and suckle.	Within an hour
Resting after self attachment and sucking fallsasleep	2 hours after birth.

Advantages of early skin to skin and latching for mother:

- Reduces birthing stress.
- Stabilizes heart rate and blood pressure.
- Releases oxytocin from posterior pituitary
- Oxytocin also called love hormone or cuddle hormone, helps in bonding and molding maternal behaviour.
- Oxytocin -----> Uterine contraction-expels placenta-reduces PPH.
- Release prolactin from anterior pituitary. More sucking – more prolactin- more milk
- Prolactin is also mothering hormone which stimulates a feeling of yearning for baby and also calms the mother.
- Baby sucking in initial days causes expression of prolactin receptors in breast tissue.

More sucking -----> more receptors-more milk.

- Reduce incidences of engorgement.
- Increased maternal confidence with interaction with baby.

Advantages for baby:

- Temperature regulation
- Reduce birthing stress-stabilize heart rate, respiratory rate and improved oxygen saturation
- Cry less and improved sleep and wake cycle Reduce incidence of hypoglycaemia
- Reduce stress-reduce cortisol-better glucose metabolism.
- Less weight loss/faster weight gain.

- Babies naïve gut colonise with healthy bacteria from mother's skin.
- Enhance brain development.
- Skin to skin contact and latching-stimulation of all 5 senses in baby-increase synaptic connections-increase IQ
- Receives colostrum.

  Colostrum is thick, gellike yellow colour milk secreted for first 5 days after birth.It is rich in protein, Vitamin A, Vitamin E, sodium, zinc, chloride and potassium, Ig s espsIg A, white cells espPMN, epidermal growth factors, cytokine, TNF, Interleukins, lymphocytes, macrophages, lactoferrin, lysozyme.

Benefits of colostrum:

Growth factors in colostrum help to seal gaps in intestinal cells.

Colostum coats the gut and prevents adhesion of pathogen to intestine.

Swallowing of colostrum -----> peristalsis-passage of meconium -----> reduce enterohepatic circulation of bilirubin-decrease incidence of jaundice

Feeding requirement and maternal milk production:

Days	Milk requirement /day	Stomach capacity /feeding	Average milk produced /day
1	30 ml	5-7ml	37ml
3		22-27ml	
5			500ml
7	300-450ml		
10		60-80ml	
1 month	750-1050ml	60-120ml	800ml

Signs of adequate latch:

- Wide open mouth[angle between upperjaw and lowerjaw>120]
- Everted lips
- No hollowing of cheeks
- Asymmetric latch[more areola visible from upper than lower]
- Audible swallows
- Painless for mother.

As per study from Ghana,***16%***of neonatal deaths can be prevented if babies were breastfed from day 1 and ***22%*** if babies were breastfed within an hour after birth.

Key messages:

- Early initiation of breastfeeding within 2 hours has positive impact on duration of exclusive breastfeeding.
- Frequent removal of breastmilk by baby or pump in initial days helps to increasebreastmilk production.
- Uninterrupted skin to skin contact between mother and baby till 1st feeding.
- Avoid forcing the baby to latch as it can disturb normal rooting reflex and position of tongue.
- Avoid suctioning in normal neonate as it can cause nasal edema, stuffiness and even oral defensiveness.
- Avoid use of shields, pacifiers as it may affect imprinting to the mother's nipple..

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MECONIUM ASPIRATION SYNDROME- AN UPDATED OVERVIEW

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Introduction:

Meconium aspiration syndrome (MAS) is a clinical condition characterized by respiratory distress / failure in neonates born through meconium-stained liquor. Coordination of care between the obstetric and neonatal team is important to reduce the incidence of MAS and to identify and provide urgent therapy in those who develop MAS to reduce morbidity and mortality.

This article offers an updated overview of the etiopathogenesis, diagnosis and management of infants with meconium aspiration syndrome.

Reported risk factors for MSAF and MAS include:

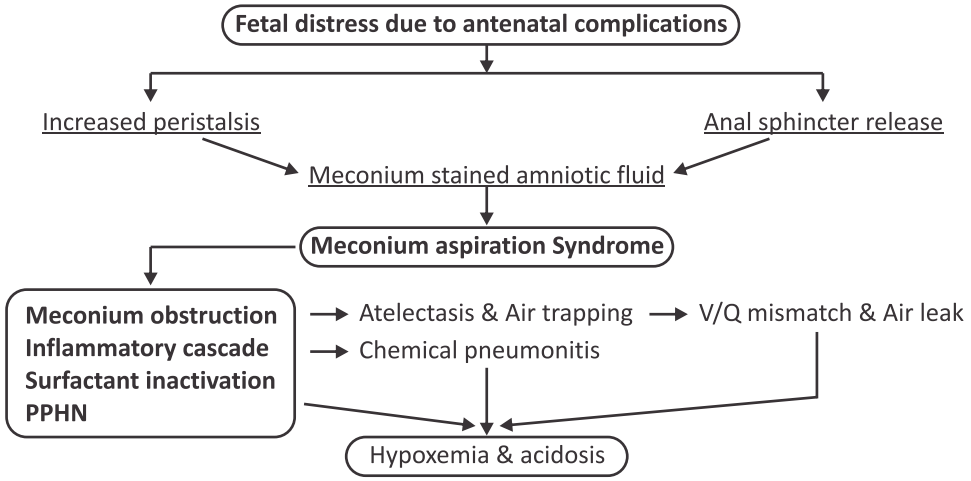
- Postterm infants (GA >41 weeks) especially those who have intrauterine growth restriction
- Vaginal breech delivery
- Caesarean delivery
- Nonreassuring fetal heart rate based on fetal heart monitoring
- Black and South Asian ethnicity
- Maternal fever and intraamniotic inflammation and infection

How to prevent?

Because of the potential for poor outcome, Intrapartum care to reduce the incidence or prevention of MAS includes:

- **Intrapartum fetal heart (FHR) monitoring** – Continuous or periodic FHR monitoring of higher risk for intrapartum fetal hypoxemia
- **Prevention of postterm (>41 weeks gestation) delivery** –Induction of labor can be offered to women at 39 weeks gestation .
- **Management of postterm delivery** –>41 weeks of gestation, induction of labor reduces the risk of MAS compared with expectant management.

What is etiopathogenesis of MAS?



How to diagnose MAS?

Maternal history: post-term pregnancy, perinatal distress, the presence of MSAF

Clinical features: respiratory distress andhyperexpansion of the chest

Chest X-ray:pulmonary hyperinflation with cottony and patchy infiltrates alternating with areas of hypertransparency

A lung ultrasound: B-pattern interstitial coalescent or sparse consolidations, dynamic sings in lung ultrasound

An echo-Doppler: to investigate the presence of PPHN in MAS patients. Hemodynamic features of PPHN: (i) a decreased pulmonary flow with a ventilation-perfusion mismatch; (ii) systo-diastolic right ventricular (RV) dysfunction; (iii) RV dilatation with a D-shape; (iv) systo-diastolic LV dysfunction; (v) right-to-left shunting

Oxygenation index (OI):

Respiratory severity may be assessed by using (OI):

OI = (mean airway pressure × FiO_2 × 100)/preductal PaO_2

The severity range	OI
Mild	<15
Moderate	15 to 25
Severe	25 to 40
Very Severe	>40

An OI over 40 for more than 4 h is included among the indications for initiating extracorporeal membrane oxygenation.

How to treat and what to treat?

Maintenance of adequate oxygenation and Ventilation

Once the diagnosis of MAS has been established, target preductal SaO_2 95 to 98 percent (arterial partial pressure of oxygen [PO_2] between 55 and 90 mmHg) that provides adequate tissue oxygenation and avoids lung injury. Hypoxemia should be avoided because it contributes to pulmonary vasoconstriction and possibly PPHN. Pulse oximetry and arterial blood gases are used to monitor oxygenation.

Respiratory support ranges from oxygen therapy (for the mildest forms) to non-invasive ventilation (for the moderate forms) up to mechanical ventilation (for the most severe cases)

About 40% of infants with MAS require mechanical ventilation.The ventilatory management of these newborns is complex due to alternation of atelectaticarea which are difficult to recruit and hyperinflated area which are at risk of air leak.Because of the different time constant of each terminal respiratory unit following meconium aspiration, High frequency oscillatory ventilation (HFOV) is indicated over conventional ventilation to reduce the barotrauma, guarantee a more homogeneous recruitment and prevent the risk of air leaks in severe MAS. HFOV uses low pressure and high frequency to recruit the collapsed alveoli, and delivers a more homogenous pulmonary ventilation and gas exchange. Clinical trials have shown that HFOV reduced the need for extracorporeal membrane oxygenation (ECMO) treatment

and decreased air-leak in infants with PPHN.

Inhaled Nitric Oxide (iNO)

MAS patients with persistent pulmonary hypertension should receive iNO therapy while ensuring adequate sedation and maintaining sub-systemic pulmonary pressures. iNO therapy reduces need for ECMO and mortality in full-term or near-term infants with respiratory failure and persistent pulmonary hypertension and its effect is enhanced when using HFOV as a ventilatory strategy . In a MAS patient with an oxygenation index reaching 15–25, iNO therapy should be started at an initial dose of 20 ppm after optimizing lung recruitment and hemodynamic support. The aim is to achieve an improvement in PaO_2 by at least 20 mmHg; after that, slow weaning can be started (decrements by 5 ppm every 4 h until 5 ppm, then decrements by 1 ppm). Of note, doses > 20 ppm do not seem beneficial and iNO therapy should be interrupted to prevent side effects in infants failing to respond.

Maintenance of adequate systemic and pulmonary blood pressure (BP) and perfusion

MAS patients with hypotension or a reduced left cardiac output should receive inotropic therapy.To assess the status of the hemodynamic conditions and to choose the appropriate inotropic agent 2D Echocardiography is compulsory.

Vasopressors like Dopamine / Norepinephrine / Vasopressin can be used in hypotension due to low Left Ventricular Output with normal contractility.. In this situation hypotension mostly occurs due to peripheral vasodilatation. Inotropic agents having a pulmonary vasodilator effect (e.g. **norepinephrine, dobutamine**) should be used if LV preload is low with RV/LV systolic dysfunction..

If systemic blood pressure is stable with cardiac dysfunction**, milrinone** should be used; milrinone is a powerful vasodilator of the pulmonary circulation that also has a positive lusitropic and inotropic action while it also causes systemic vasodilation and reduces mean arterial pressure.

Sildenafil is phoshphodiesterase 5 inhibitor, increases cGMP concentration, and may result in pulmonary vasodilatation or enhance the activity of iNO.

Extracorporeal Membrane Oxygenation (ECMO)

MAS patients failing conventional therapy (such as HFOV and iNO) require ECMO. Current indications for ECMO are (i) inadequate tissue oxygen delivery despite maximal therapy (rising lactate, worsening metabolic acidosis, the sign of end organ dysfunction); (ii) severe hypoxic respiratory failure with acute decompensation (PaO_2 < 40 mmHg); (iii) an oxygenation index with sustained elevation and no improvement; iv) severe pulmonary hypertension with evidence of RV/LV dysfunction.

Surfactant

Surfactant may also be helpful in infants with radiographic evidence of surfactant dysfunction but further studies are warranted.

Steroids

Although corticosteroid therapy has been proposed to reduce the severity of MAS, there

is **no** evidence of its effectiveness in infants with MAS

Other general approach to care includes

- Correction of any metabolic abnormality including hypoglycemia and acidosis, which increase oxygen consumption
- Empirical or prophylactic antibiotic therapy
- Thermoneutral environment
- Minimal handling of the infant to avoid agitation, which exacerbates persistent pulmonary hypertension of the newborn (PPHN), if present.

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Neonatal seizures

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Introduction

Epileptic seizures are common in neonates, but can be difficult to diagnose if not associated with any clinical features. Neonates can also have other movements which may be mistaken for seizures. Accurately recognising epileptic from non-epileptic movements is paramount to clinical management. Most neonatal seizures occur due to a recognisable event like hypoxic ischaemic encephalopathy (HIE), haemorrhage, hypoglycaemia, meningoencephalitis, stroke, electrolyte imbalance, cerebral dysgenesis etc. These are called acute symptomatic seizures. However, rarely neonates can also experience recurrent unprovoked seizures and can have an epilepsy syndrome for which the list of different causes varies from metabolic to genetic.

Description of movements that are likely to be epileptic seizures

1. Myoclonic seizures – brief, shock-like jerks, repetitive and non-rhythmical. Generalised repetitive myoclonic jerks are likely to be epileptic. Myoclonic jerks of the diaphragm can lead to hiccups, and the suspicion of non-ketotichyperglycinemia.
2. Clonicseizures – rhythmical jerking involving hips, shoulders, elbow, wrist, ankle or even face are frequently epileptic. Rhythmical tongue thrusting or eye deviation also can be associated.
3. Tonic seizures – stiffening of one-sided limbs associated with eye and face deviation to one side is usually focal tonic seizure. In contrast, bilateral symmetrical tonic posturing of hands and legs can be associated with facial flushing and/or cyanosis.
4. Tonic (epileptic) spasms - are subtly longer than myoclonic seizures and cause sudden contraction of the axial and proximal muscle leading to flexion of neck, trunk, shoulders and hips mainly.
5. Other abnormal movements – apnoea, pedalling or cycling, swimming, boxing, thrashing, lip-smacking, tongue thrusting, startling can all be epileptic seizures if they occur repetitively. Need to be careful that these are not over interpreted as epileptic seizures.

Description of movements that are unlikely to be epileptic

1. Myoclonus – benign neonatal sleep myoclonus, often resolves by 3 months age.
2. Tremors and jitteriness – can be present in healthy neonates, hypoglycaemia and hypocalcaemia need to be excluded.
3. Clonus – can occur with some stiffening of limbs, usually stops when the position of the joint where it is occurring is changed.
4. Dystonia or tonic stiffening – usually feature

of hypoxic ischaemic brain injury, meningitis or neurometabolic disorders.

5. Excessive startling with tonic stiffening – Hyperekplexia is rare and caused by mutation in the glycine receptors. Neonates can present with apnoea due to stiffening of respiratory muscles. Excessive startle to the glabellar tap test with failure to habituate clinches the clinical diagnosis. Clonazepam is the treatment of choice.

Table 1: Classification of epileptic seizure types adapted from ILAE (International League Against Epilepsy), Mirzahi and Volpe——1

ILAE	Mirzahi	Volpe
• Clonic – focal, multifocal, bilateral • Tonic – focal, bilateral asymmetric, bilateral symmetric • Myoclonic – focal, multifocal, bilateral asymmetric, bilateral symmetric • Epileptic spasms – unilateral, bilateral asymmetric, bilateral symmetric • Automatisms	• Focal clonic – uniclonic, multifocal clonic • Focal tonic – asymmetrical, truncal, eye deviation • Apnoea • Myoclonic – focal, generalised • Spasms • Electrographic	• Clonic – focal, multifocal • Tonic – focal, generalised • Subtle • Myoclonic – focal, multifocal, generalised • Electrographic

Aetiology

Most neonatal seizures are acute symptomatic seizures, which occur during a systemic or brain insult including metabolic derangements. HIE is the commonest cause of acute symptomatic seizures, followed by hypoglycemia commonly occurring between day 3 and 5 of life. Neonatal sepsis and meningitis is again a common cause which needs to be promptly recognised and managed. Neonatal viral encephalitis, de novo (enterovirus) or secondary to maternal Dengue or Chikungunya, is also important to recognise. A thorough history, examination and initial screen for causes for symptomatic seizures is important. The aetiology by predominant time of onset is presented in Table 2. If the initial investigation does not reveal a clear cause, then further investigations are necessary for optimal clinical management. A list of initial investigations which can be useful in determining the cause of neonatal seizures is presented in Table 3.

History

- Family history : consanguinity, benign neonatal familial seizures
- Pregnancy : infections, rhythmic kicking or hiccups suggest in-utero seizures
- Peripartum events : peripartum fever, difficult labour, meconium-stained liquor, Apgar scores, resuscitation details
- Seizure : time of onset, description, videos

Examination

- Dysmorphism, neurocutaneous markers, rashes
- Head size
- Metabolic clues: organomegaly, cataract, acidotic breathing, hiccups, odour, jaundice
- Neurological examination: wakefulness

state, tone, movements of limbs, symmetry of face, rooting, sucking, Moro's reflex

- Holding the jerking limb to differentiate epileptic from non-epileptic movements
- Waking the baby to see for disappearance of sleep myoclonus

Table 2: Aetiology by day of onset of seizures——1

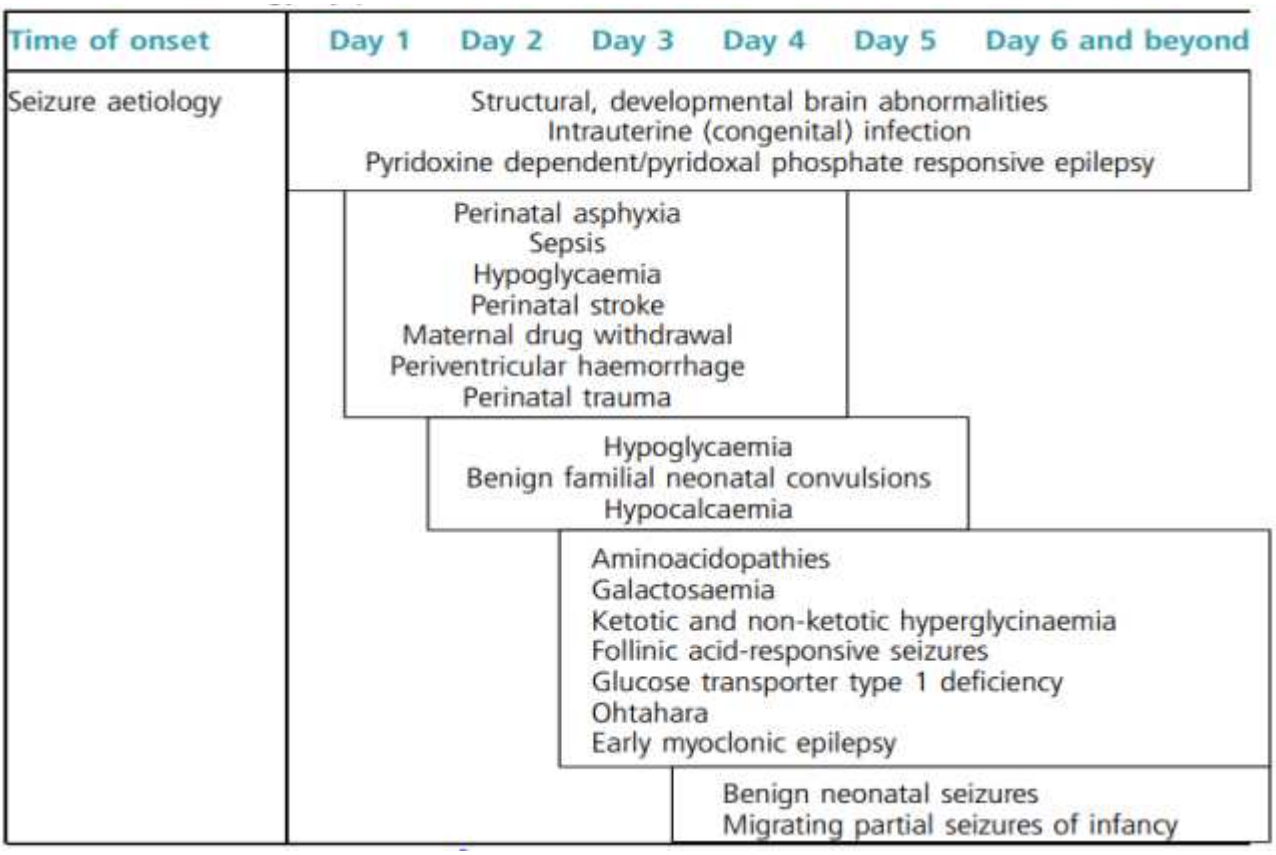

Table 3: Investigations in neonatal seizures

First line	Glucose Calcium and magnesium Liver function test Renal function test Blood culture Lumbar puncture – check for glucose (glucose transporter deficiency-GLUT1), consider storing sample to check for amino acids and lactate in suspected metabolic disorder Urine culture Cranial ultrasound scan
Second line	EEG (with neonatal montage), amplitude integrated EEG in HIE MRI brain (consider MR spectroscopy in suspected metabolic disorders) Ammonia Lactate Biotinidase Plasma amino acids (TMS) Urine organic acids (GCMS)
Third line	Copper, caeruloplasmin (Menkes disease) Urine alfa amino-adipic semialdehyde (pyridoxine dependent epilepsy) Urine sulfites (molybdenum cofactor or sulfite oxidase deficiency) Acylcarnitine, Fatty acids profile (peroxisomal disorders) TORCH serology and urine for CMV PCR Whole exome sequencing

EEG in neonatal seizures

Video-EEG would be the treatment of choice in the management of neonatal seizures. A reduced neonatal montage (Figure 1) is often used in most units and can provide useful information. The use of amplitude integrated EEG is already in place with therapeutic hypothermia in the management of neonates with hypoxic ischaemic encephalopathy. However, having a dedicated person apply the EEG appropriately and the expertise required to interpret the recording accurately are limitations in the widespread use of this technique. Electro-clinical dissociation and electrographic seizures are commonly seen in neonates, and aggressive management is known to improve outcomes.

MRI in neonatal seizures

MRI detects cases of cerebral dysgenesis like pachygyria or schizencephaly, or vascular problems like acute arterial ischaemic stroke or cerebral venous sinus thrombosis and venous infarct (hypernatremic dehydration). Aspirin is not usually given in stroke, but low molecular weight heparin can be helpful in preventing further propagation of the venous thrombosis and is well tolerated. Many metabolic disorders can have characteristic imaging features (Figure 2).

Clearly defined neonatal epilepsy syndromes—"——2

1. **Self-limiting neonatal seizures**: previously called benign neonatal seizures or fifth day fits. The seizures can be unilateral, or bilateral clonic jerking of limbs and face lasting minutes or even occurring in clusters, sometimes associated with apnoea. There is no family history and it is a diagnosis of exclusion after other causes of symptomatic seizures are excluded. They respond well to any medications like phenobarbitone or levetiracetam.

   In contrast, benign familial neonatal seizures are characterised by frequent, short, tonic seizures with apnoea and autonomic changes like cyanosis. There can be unilateral or bilateral clonic jerking as well associated with tonic seizures. There is a family history of similar seizures during the neonatal period, and the seizures respond well to phenobarbitone or phenytoin. Medications are usually continued till 1 year as some of them can continue to have seizures till 1 year of life. Genetic studies have identified few mutations which can help choice of medications. EEG is either normal, or shows some focal abnormalities.

2. **Early infantile epileptic encephalopathy (Ohtahara syndrome)**: rare condition with onset in the first week of life with usually tonic seizure or tonic (epileptic) spasms, occurring in clusters and can be symmetrical or asymmetrical, and occur during wakefulness and sleep. The EEG is typically associated with burst-suppression pattern which is present during both wakefulness and sleep (Figure 3). The aetiology can be varied with structural brain problems and genetic disorders being the commonest. Certain genetic mutations in SCN2A and SCN8A can respond to supra-therapeutic doses of phenytoin and should

always be tried in refractory neonatal seizures. Thorough work-up for metabolic disorders should also be considered. Seizures remain difficult to treat and overall neurodevelopmental prognosis is poor, but dependent on the underlying aetiology.

3. **Early myoclonic encephalopathy :** rare and another early infantile epileptic encephalopathy, however the main seizure type is myoclonic, occurring in clusters shortly after birth. The EEG again shows burst suppression, which is more prominent during sleep. Metabolic aetiology is more common in this disorder followed by genetic disorders. Non-ketotichyperglycinemia and pyridoxine dependent epilepsies should be considered early in the work-up of such neonates.
4. **Epilepsy of infancy with migrating focal seizures :** is usually seen in early infancy, but can also be seen in neonates. The seizures are usually focal clonic or tonic with autonomic features like flushing, tearing and/or cyanosis. They migrate from one region of the body to the other, with simultaneous correlation on EEG. Genetic aetiology, particularly KCNT1 related, are likely. Seizures are refractory to management. Quinidine has been tried with some success.
5. **Vitamin responsive disorders :** Investigations and a therapeutic trial should be given in any refractory neonatal seizures where a cause is not known. Pyridoxine can be given intravenously (100 mg) but usually causes apnoea. Hence, oral pyridoxine at 15mg/kg/day in two divided doses is usually effective in these disorders. In some cases, where there is no response to pyridoxine, changing to pyridoxal phosphate at 50mg/kg/day in two divided doses proves to be useful due to the possibility of pyridoxamine 5'-phosphate oxidase (PNPO) deficiency. Biotin is given as 5mg twice a day (Biotinidase deficiency), and Folinic acid is given (Folinic Acid responsive seizures) as 5mg twice a day (available as calcium leucovorin).

Treatment of neonatal seizures

No standardised guidelines exist and most protocols are individual neonatal unit based. An algorithm for management of neonatal seizureis proposed (Figure 4). Phenobarbitone remains the first line anti-seizure medication in most units. Levetiracetam has become more popular due to its favourable side effect profile, mainly with less sedation and ease of conversion to oral preparation at discharge. The loading doses of levetiracetam are usually higher in neonates with doses ranging from 30-50mg/kg as being effective. Phenytoin, although associated with risk of hypotension and extravasation related problems, is important to consider in certain epilepsy syndromes where the possibility of response is expected (channelopathy due to sodium channel mutations like SCN2A or SCN8A). Benzodiazepines are not commonly used unless there is refractory seizures and usually cause respiratory depression with the need for ventilation.

As discussed above, a trial of vitamins is

essential in refractory cases where a clear cause is unknown. Consultation and discussion with the paediatric neurologist are paramount in the epilepsy syndromes to decide further medications to be used in management. In most cases, neonates would be discharged on anti-seizure medications and then followed up by the neonatologist or neurologist to decide the duration of treatment. In most acute symptomatic seizures, medicines are continued for a period of 6-8 weeks and then discontinued. However, this is dependent on the extent of brain injury and the clinical acumen in deciding the probability of recurrence. In the severe neonatal epilepsy syndromes, treatment is likely to continue long term and needs to be evaluated on case-by-case basis.

Conclusion

Most neonatal seizures are acute symptomatic and are controlled with medications like phenobarbitone or levetiracetam. Consider channelopathy related neonatal epilepsy syndrome and the use of phenytoin, as well as the vitamin responsive disorders as these can be readily treated. Video recording, selected investigations and the use of video-EEG is helpful in the optimal management of neonatal seizures.

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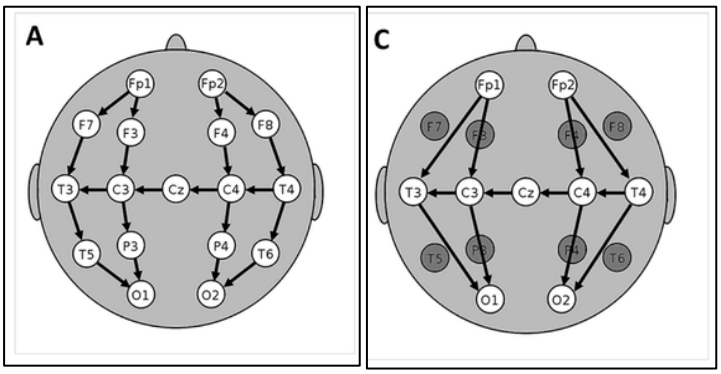


Figure 1: Full 10:20 montage in older children (A) and reduced neonatal montage in neonates till 6 weeks age (C)

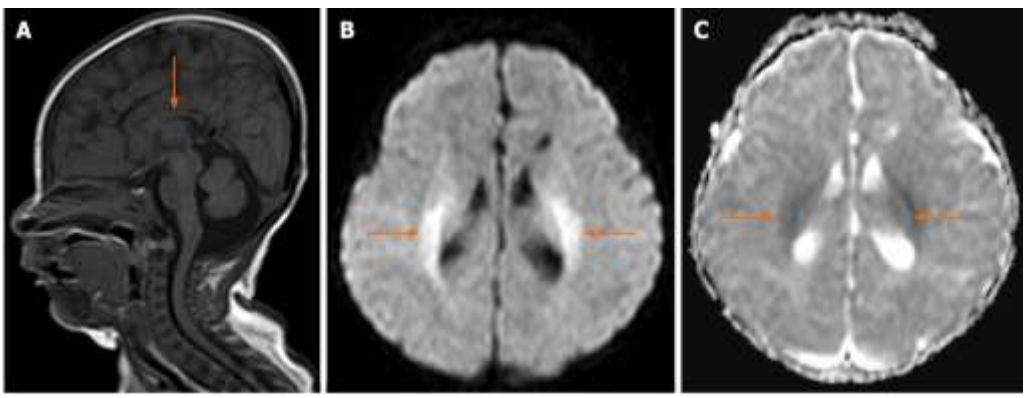


Figure 2: MRI brain in non-ketotichyperglycinemia (thin corpus callosum, bilateral symmetrical diffusion restriction in both periventricular white matter, red arrows)

Figure 3: bursts of high amplitude spike/sharp waves followed by complete suppression, burst-suppression encephalopathy in Ohtahara syndrome

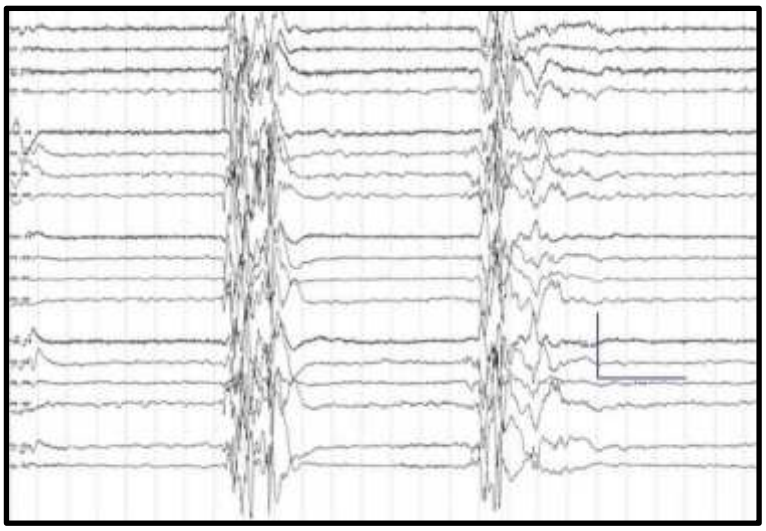
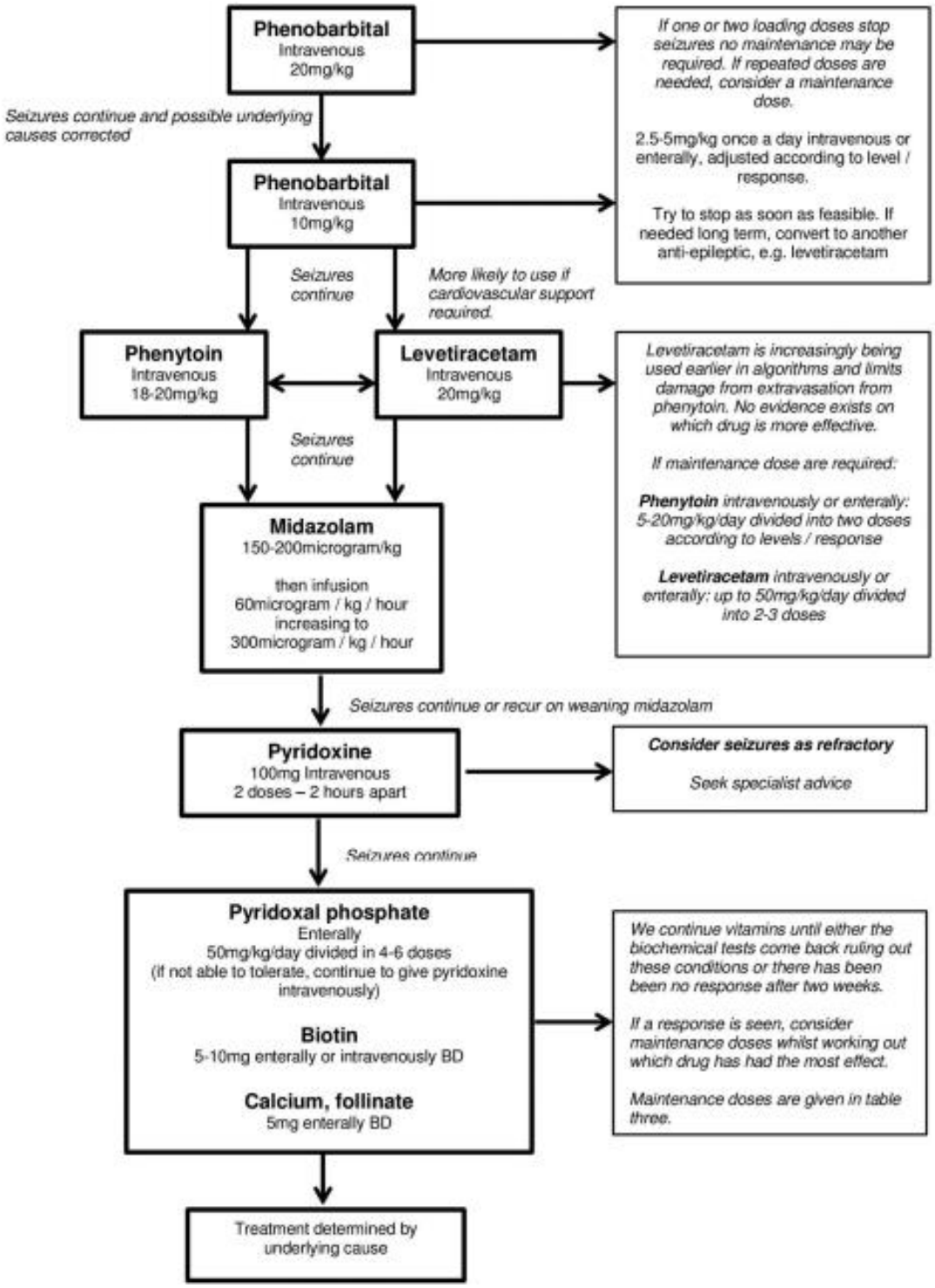


Figure 4: Algorithm for the management of neonatal seizures—"——2



Key Message:

1. Identifying epileptic from non-epileptic movements is paramount.
2. Acute symptomatic seizures are the commonest cause for neonatal seizures. (HIE, hypoglycemia, hypocalcaemia, meningitis and vascular)
3. Neonatal epilepsies are rare, but identification of the epilepsy syndrome and seizure type is key.
4. Systematic approach to investigations, focus on key metabolic and treatable factors.
5. Rational management with step wise escalation. Phenytoin a useful medication in epilepsy related to channelopathies. Therapeutic trial of vitamin essential in refractory cases.