DEDER GENERAL HOSPITAL

PERINATAL ASPHYXIA (PNA)

MANAGEMENT PROTOCOL

PREPARED BY: HSQU

July 2016E.C

Deder, Eastern Ethiopia

PROTOCOL APPROVAL SHEET

TIT

Title: Perinatal Asphyxia (PNA) Management Protocol 2017E.C

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Draft Date: July 15, 2016E.C **Approved Date: July 20, 2016E.C**

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SECTION 1: INTRODUCTION & BACKGROUND

1.1 Background

Perinatal asphyxia (PNA) is one of the leading causes of preventable neonatal morbidity and mortality globally and in Ethiopia. It refers to a condition where a newborn fails to initiate or sustain spontaneous breathing at birth, leading to a combination of hypoxemia, hypercapnia, and metabolic acidosis. This insult can cause multi-organ dysfunction, with the brain being most vulnerable to injury (hypoxic-ischemic encephalopathy – HIE).

Globally, about **2.4 million neonatal deaths** occur annually, and **23–25% are due to intrapartum-related events** (birth asphyxia). In Ethiopia, PNA remains one of the "big three" causes of neonatal death along with sepsis and prematurity.

1.2 Relevance to Ethiopia

- **National Neonatal Mortality Rate (NMR):** ~33 per 1,000 live births.
- PNA contributes to **over 30% of in-hospital neonatal deaths** in many Ethiopian facilities.
- Long-term consequences include cerebral palsy, epilepsy, hearing and vision impairment, and developmental delay.

1.3 Impact

- **Immediate:** Increased risk of death in the first week of life.
- **Short-term:** Prolonged NICU stay, need for intensive monitoring, higher healthcare costs.
- **Long-term:** Neurodevelopmental disability, increased caregiver burden, loss of economic productivity.

1.4 Purpose of This Protocol

This protocol provides a **standardized, evidence-based guide** for the prevention, early identification, and management of perinatal asphyxia at Deder General Hospital. It is intended for **midwives, NICU nurses, general practitioners, IESO, Gyn/OBS Specialist, and pediatricians** working in the delivery room and NICU.

SECTION 2 – DEFINITIONS & CLASSIFICATIONS

2.1 Key Definitions

We will use both **WHO** and **FMOH Ethiopia** definitions to maintain alignment with international and national guidelines.

2.1.1 Perinatal Asphyxia

- **WHO Definition:**

  "Failure to initiate and sustain breathing at birth, leading to impaired gas exchange resulting in hypoxemia, hypercapnia, and metabolic acidosis."

- **FMOH Ethiopia Definition (NICU Participants Manual, 2024):**

  "A clinical condition in which a newborn fails to establish spontaneous respiration after birth, accompanied by metabolic acidosis (umbilical arterial pH < 7.0) and often followed by signs of hypoxic ischemic encephalopathy."

Key points:

- Diagnosis is clinical and supported by laboratory findings.
- Not all low Apgar scores mean asphyxia — they must be interpreted with context.

2.1.2 Hypoxic-Ischemic Encephalopathy (HIE)

A syndrome of disturbed neurological function in the earliest days of life in an infant born at ≥35 weeks gestation, manifested by:

- Altered level of consciousness.
- Abnormal muscle tone and reflexes.
- Possible seizures.
- Difficulty initiating and maintaining respiration.

Cause: Result of acute or subacute brain injury due to intrapartum or peripartum hypoxia-ischemia.

2.1.3 Birth Asphyxia vs. Perinatal Asphyxia

- **Birth Asphyxia:** Refers specifically to oxygen deprivation occurring **at the time of delivery**.
- **Perinatal Asphyxia:** Broader term — includes events **before, during, and immediately after birth** (perinatal period = 28 weeks gestation to 7 days postnatal).

2.2 Diagnostic Criteria

Diagnosis of perinatal asphyxia is made when **at least three of the following** are present:

1. **Apgar score** ≤ 5 at 5 minutes.
2. Umbilical artery **pH < 7.0** or base deficit ≥ 12 mmol/L.
3. Neurologic manifestations consistent with HIE (seizures, coma, hypotonia).
4. Evidence of multi-organ dysfunction (e.g., renal, cardiac, hepatic).

2.3 Classification Systems

2.3.1 Apgar Score

Parameter	0 Points	1 Point	2 Points
Appearance	Blue/pale	Pink body, blue limbs	Completely pink
Pulse	Absent	< 100 bpm	≥ 100 bpm
Grimace	No response	Grimace	Cough, sneeze, cry
Activity	Limp	Some flexion	Active motion
Respiration	Absent	Weak, irregular	Strong cry

Interpretation:

- **7–10:** Normal.
- **4–6:** Moderate depression.
- **0–3:** Severe depression.

Note: Apgar score alone does not diagnose asphyxia but helps assess the newborn's initial status.

2.3.2 Sarnat and Sarnat Staging of HIE

Stage	Consciousness	Neuromuscular Control	Pupils	Seizures	Duration
Stage I	Hyperalert, irritable	Normal or hypertonia	Mydriasis	None	< 24 hrs
Stage II	Lethargic, obtunded	Hypotonia, weak suck	Miosis	Common	Days–weeks
Stage III	Coma	Flaccid	Variable	Frequent	Hours–days

SECTION 3 – EPIDEMIOLOGY & RISK FACTORS

3.1 Epidemiology

Global:

- Perinatal asphyxia accounts for **~23–25%** of the world's neonatal deaths (WHO).
- Estimated **~900,000 deaths annually** due to intrapartum-related events.
- Incidence of HIE in developed countries: **1–3 per 1,000 live births**.
- Incidence of HIE in low- and middle-income countries: **10–26 per 1,000 live births**.

Ethiopia

- **Neonatal Mortality Rate (NMR):** 33 per 1,000 live births (EDHS 2019).
- PNA is among the top **three causes of neonatal death** alongside sepsis and prematurity.
- Hospital-based studies report PNA prevalence ranging from **15% to 30%** among NICU admissions.
- Higher incidence observed in rural areas with limited skilled birth attendance.

3.2 Public Health Significance

- **Mortality:** High case fatality rate if not promptly managed.
- **Morbidity:** Significant proportion of survivors develop neurodevelopmental disabilities.
- **Economic impact:** Long-term disability increases family and health system costs.
- **Preventability:** Majority of cases can be prevented through improved **intrapartum care** and **timely neonatal resuscitation**.

3.3 Risk Factors

Perinatal asphyxia is often the result of **reduced placental or pulmonary gas exchange** before, during, or after delivery. Risk factors can be grouped into **maternal**, **fetal**, and **intrapartum** categories.

*Table 1:**Maternal Risk Factors***

Risk Factor	Mechanism / Explanation
Pregnancy-induced hypertension	Reduced placental perfusion → chronic fetal hypoxia
Antepartum hemorrhage (APH)	Acute maternal blood loss → compromised fetal oxygenation
Severe anemia	Reduced oxygen-carrying capacity
Prolonged rupture of membranes with infection	Maternal fever, chorioamnionitis → fetal hypoxia
Malnutrition	Fetal growth restriction, reduced reserves
Advanced maternal age (>35 years)	Associated with obstetric complications
Eclampsia	Seizures → interrupted placental blood flow

*Table 2: **Fetal Risk Factors***

Risk Factor	Mechanism / Explanation
Intrauterine growth restriction (IUGR)	Reduced placental nutrient and oxygen transfer
Post-term pregnancy (>42 weeks)	Placental insufficiency
Multiple gestation	Increased risk of cord accidents, preterm birth
Fetal anemia (e.g., Rh disease)	Low oxygen delivery capacity
Congenital anomalies (cardiac, CNS)	Impaired adaptation to extra-uterine life

*Table 3:**Intrapartum Risk Factors***

Risk Factor	Mechanism / Explanation
Prolonged or obstructed labor	Fetal exhaustion, hypoxia
Malpresentation (breech, face)	Cord compression, difficult delivery
Umbilical cord prolapse	Acute interruption of fetal blood supply
Shoulder dystocia	Delayed delivery of fetal head
Meconium-stained amniotic fluid	Risk of aspiration, hypoxia
Instrumental delivery	Risk of trauma and delayed initiation of breathing
Uterine rupture	Acute fetal distress, severe hypoxia

*Table 4: **Postnatal Risk Factors***

Risk Factor	Mechanism / Explanation
Prematurity (<37 weeks)	Immature lungs and poor adaptation
Severe respiratory distress	Hypoxia secondary to lung disease
Sepsis	Poor perfusion, metabolic acidosis
Persistent pulmonary hypertension of the newborn (PPHN)	Failure of normal circulatory transition

3.4 Key Messages for Clinical Teams

- **Antenatal care** is critical to identify and manage maternal risk factors.
- **Continuous fetal monitoring** during labor reduces intrapartum hypoxic events.
- **Timely and effective neonatal resuscitation** is the single most important intervention to prevent deaths.

SECTION 4 – PATHOPHYSIOLOGY & ORGAN MANIFESTATIONS

4.1 Overview

Perinatal asphyxia occurs when **oxygen delivery to the fetus or newborn is reduced or interrupted**, leading to **hypoxemia**, **hypercapnia**, and **metabolic acidosis**. This combination causes **cellular energy failure** and a cascade of injury affecting multiple organs.

4.2 Pathophysiology Cascade of Injury

Step 1 – Hypoxic Event

- Reduced oxygen supply due to impaired placental gas exchange (antenatal/intrapartum) or ineffective lung function (postnatal).
- Causes: placental insufficiency, cord accidents, delayed resuscitation.

Step 2 – Hypoxemia & Hypercapnia

- Low PaO_2 and high $PaCO_2$ due to poor gas exchange.
- Leads to respiratory and metabolic acidosis.

Step 3 – Initial Compensation

- Redistribution of blood flow ("brain-sparing effect"):
  Blood shunted to vital organs — brain, heart, adrenals — at the expense of kidneys, liver, skin, GI tract.

Step 4 – Decompensation

- Prolonged hypoxia depletes glycogen and ATP stores.
- Ion pump failure → cellular swelling, calcium influx → cell death.
- **Step 5 – Reperfusion Injury**

- When oxygen is restored, free radicals and inflammatory mediators cause secondary injury.
- Especially damaging in the brain → **Hypoxic-Ischemic Encephalopathy (HIE)**.

Figure 1: Pathophysiology Cascade Diagram

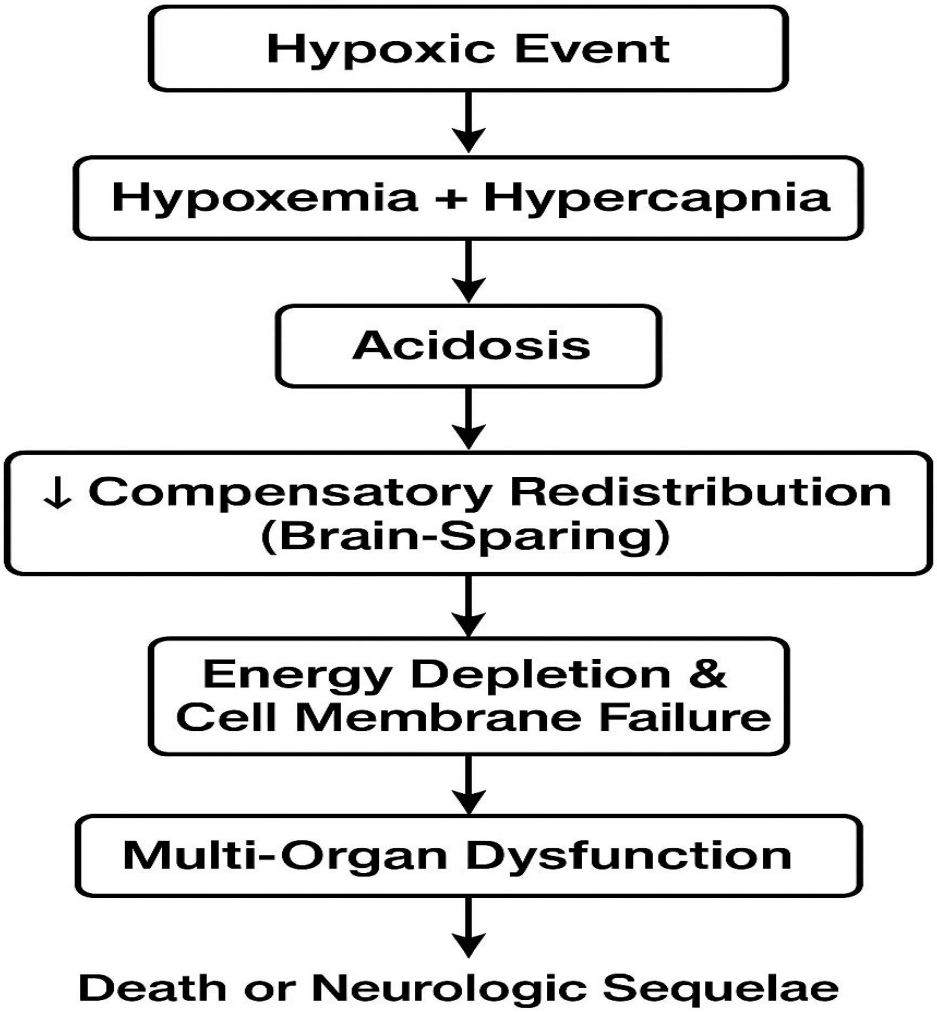


Table 5: Clinical spectrums of HIE includes mild, moderate or severe according to Sarnat stage of HIE

SIGNS	STAGE 1	STAGE 2	STAGE 3
Level of consciousness	Hyper alert	Lethargic	Stupors, coma
Muscle tone	Normal	Hypotonic	Flaccid
Posture	Normal	Flexion	Decerebrate
Tendon reflexes/clonus	Hyperactive	Hyperactive	Absent
Myoclonus	Present	Present	Absent
Moro reflex	Strong	Weak	Absent
Pupils	Mydriasis	Miosis	Unequal, poor ligh reflex
Seizures	None	Common	Decerebration
Electroencephalographic findings	Normal	Low voltage changing to seizure activity	Burst suppression to isoelectric
	<24 hr if		
Outcome	Good	Variable	Death, severe

4.3 Organ Manifestations of PNA

Hypoxia during the perinatal period can cause **multi-system injury**.

4.3.1 Brain (Central Nervous System)

- **Mild:** Irritability, hyperalertness, hypertonia (Stage I HIE).
- **Moderate:** Lethargy, hypotonia, weak suck, seizures (Stage II HIE).
- **Severe:** Coma, flaccidity, absent reflexes, recurrent seizures (Stage III HIE).
- Long-term: cerebral palsy, developmental delay, epilepsy.

4.3.2 Heart (Cardiovascular)

- Myocardial ischemia → poor contractility.
- Hypotension, arrhythmias.
- Risk of congestive heart failure due to pump dysfunction.

4.3.3 Lungs (Respiratory)

- Primary apnea, secondary apnea.

- Pulmonary hypertension.
- Risk of meconium aspiration syndrome.
- Respiratory distress requiring ventilation.

4.3.4 Kidneys (Renal)

- Acute kidney injury due to reduced perfusion.
- Oliguria/anuria.
- Electrolyte imbalances (hyperkalemia, hyponatremia).

4.3.5 Liver (Hepatic)

- Elevated liver enzymes.
- Coagulopathy (due to reduced synthesis of clotting factors).

4.3.6 Gastrointestinal

- Risk of necrotizing enterocolitis due to poor gut perfusion.

4.3.7 Hematologic

- Disseminated intravascular coagulation (DIC).
- Thrombocytopenia.

4.3.8 Metabolic

- Hypoglycemia from depleted glycogen stores.
- Metabolic acidosis from anaerobic metabolism.

4.4 Key Clinical Insight

- Organ dysfunction may **not be apparent immediately** after birth — serial monitoring over the first 72 hours is essential.
- The **brain and kidneys** are the most sensitive to hypoxic injury; renal output is a good early marker of systemic hypoxia.

SECTION 5 – CLINICAL FEATURES & DIAGNOSIS

5.1 Clinical Presentation

5.1.1 In the Delivery Room

- Failure to breathe or cry within the first minute.
- Poor muscle tone (flaccidity or hypotonia).
- Cyanosis or pallor.
- Bradycardia (HR < 100 bpm).
- Weak or absent reflexes (e.g., weak Moro, absent suck).

5.1.2 In the NICU (Post-Resuscitation)

- **Neurologic signs:** Altered level of consciousness (irritability, lethargy, coma), abnormal tone, seizures.
- **Respiratory signs:** Apnea, grunting, nasal flaring, need for supplemental oxygen.
- **Cardiovascular signs:** Hypotension, poor perfusion (capillary refill > 3 sec), arrhythmias.
- **Renal signs:** Oliguria (< 1 mL/kg/hr) or anuria.
- **Metabolic signs:** Hypoglycemia, metabolic acidosis.

5.2 Early Warning Signs Table

For rapid bedside screening in delivery room and NICU

Domain	Early Signs	Late Signs
Breathing	Irregular respiration, grunting	Apnea, gasping
Color	Mild cyanosis (peripheral)	Central cyanosis, pallor
Tone	Mild hypotonia	Severe flaccidity
Reflexes	Weak Moro/suck	Absent primitive reflexes
Heart Rate	80–100 bpm	< 80 bpm
Neurology	Irritability, jitteriness	Seizures, coma

5.3 Diagnosis

Diagnosis is **clinical**, supported by laboratory and imaging findings.

5.3.1 Diagnostic Criteria

Presence of **≥ 3** of the following:

1. Apgar score ≤ 5 at 5 minutes.
2. Need for resuscitation > 1 minute after birth.
3. Umbilical artery pH < 7.0 or base deficit ≥ 12 mmol/L.
4. Signs of neurologic dysfunction (HIE).
5. Evidence of multi-organ failure.

5.3.2 Laboratory Support

- **Blood gas analysis:** Metabolic acidosis.
- **Serum glucose:** Hypoglycemia common in PNA.
- **Renal function tests:** BUN/Creatinine for AKI.
- **Liver function tests:** Elevated transaminases.
- **Coagulation profile:** Detect DIC.
- **Electrolytes:** Identify hypo/hyperkalemia, hyponatremia.

5.3.3 Imaging

- **Cranial ultrasound:** Useful for ruling out intraventricular hemorrhage.
- **MRI (if available):** Gold standard for detecting HIE brain injury.
- **Chest X-ray:** If respiratory distress or MAS suspected.

SECTION 6 – IMMEDIATE MANAGEMENT IN THE DELIVERY ROOM

6.1 Principles

- **Time is brain:** The first **60 seconds after birth ("Golden Minute")** are critical.
- **Most PNA-related deaths are preventable** if appropriate resuscitation is initiated immediately.
- Management must be **team-based**, with a clearly assigned leader and roles before delivery.

6.2 Pre-Delivery Preparation Checklist

(To be completed before every high-risk delivery)

Category	Items
Warmth	Pre-warmed radiant warmer, warm towels, hat
Airway	Suction device (bulb or mechanical), oxygen supply, bag-mask device (term & preterm sizes)
Breathing	Self-inflating bag, flow-inflating bag, T-piece resuscitator
Circulation	Stethoscope, pulse oximeter, neonatal ECG (if available)
Drugs/Fluids	Adrenaline (1:10,000), normal saline, IV access kits
Miscellaneous	Clock/timer, clean scissors/blade for cord, gloves

6.3 Delivery Room Resuscitation Algorithm

Step 1 – Rapid Assessment (Immediately after birth)

Ask:

1. Is the baby term?
2. Is the baby breathing or crying?
3. Is there good muscle tone?

- **If YES to all:** Routine care (dry, keep warm, skin-to-skin, early breastfeeding).
- **If NO to any:** Proceed to initial steps.

Step 2 – Initial Steps (within 30 seconds)

- Place under radiant warmer.
- Position head in "sniffing" position.
- Clear airway if secretions are present and obstructing breathing.
- Dry the baby and remove wet towels.
- Stimulate (rub back, flick soles).

Step 3 – Evaluate Breathing & Heart Rate

- If **apneic/gasping** OR **HR < 100 bpm** → Start Positive Pressure Ventilation (PPV).
- Use room air (21% O_2) for term infants; 21–30% O_2 for preterm infants.
- Use correct mask size and ensure good seal.

Step 4 – Reassess after 30 seconds of PPV

- **HR ≥ 100 bpm & breathing:** Stop PPV, monitor closely.
- **HR < 100 bpm:** Continue PPV and check technique.
- **HR < 60 bpm:** Start chest compressions (3:1 ratio with PPV).

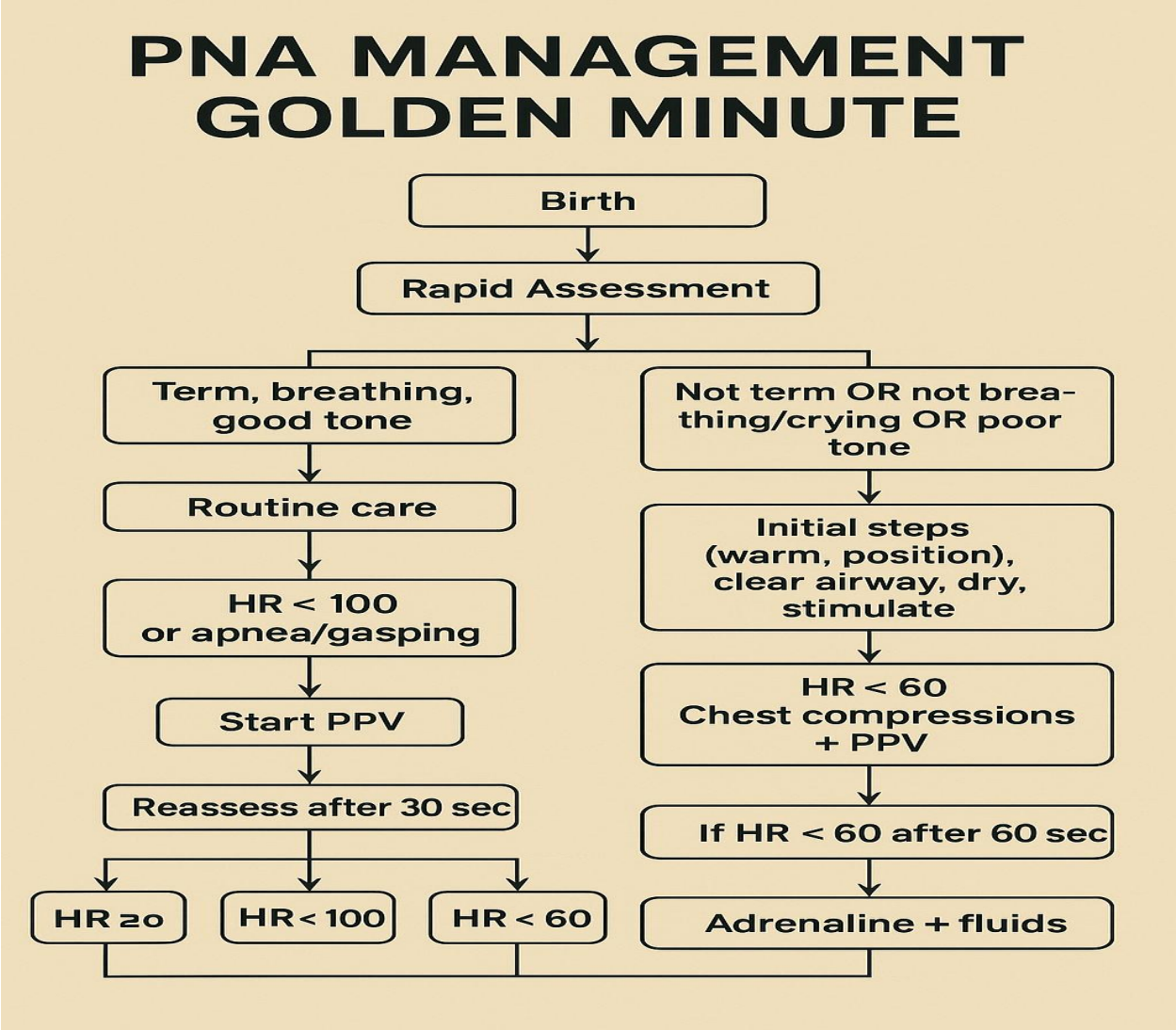
Step 5 – If HR still < 60 bpm after 60 seconds of coordinated PPV + compressions

- Give Adrenaline (0.01–0.03 mg/kg IV or via umbilical venous catheter).
- Consider volume expansion (10 mL/kg NS) if hypovolemia suspected.

Step 6 – Post-Resuscitation Care

- Transfer to NICU for monitoring, even if stable.
- Maintain normothermia (36.5–37.5°C).
- Monitor oxygen saturation (target 90–95%).
- Start glucose monitoring within 30 minutes.

Figure 2: ***PNA Management Golden Minute" Flowchart (for wall poster in delivery room)"***



6.4. Key Delivery Room Notes

- **Suction is NOT routine** — only if airway is obstructed.
- **Over-ventilation** may cause lung injury and hypocarbia.
- Keep **O_2 saturation targets** in mind to avoid hyperoxia.
- **Effective PPV** is the single most important intervention in PNA.

7. LABORATORY EVALUATION AND MANAGEMENT OF PNA

7.1. Laboratory evaluation

A. CBC
B. RBS
C. Urine analysis
D. Renal function test
E. Liver function test
F. Echocardiography as needed
G. Serum electrolyte
H. Stool for blood- if NEC is suspected
I. EEG
J. CXR
K. Brain imaging- transfontanel ultrasound, MRI

7.2. Management of newborn with asphyxia

A. **Keep NPO for severe PNA:** (because of risk of necrotizing enterocolitis, it can be for48 hours). Start feeding (with 5 to 10ml)when the neonate is passing meconium, clear gastric content, normo-active bowel sound and then advance as tolerated
B. **Fluid Management:** day 1 stat with 60ml/kg of 10% dextrose reduce to two third of the maintenance fluid in subsequent days if retaining fluid (avoid both overload and inadequate circulating volume), add calcium gluconate in the maintenance fluid
C. **Oxygenation:** it should be maintained in the normal range (Saturation between90-95%)
D. **Maintain normal temperature**: Cooling therapy is the standard treatment for hypoxic ischemic encephalopathy. However, it is not available in Ethiopian setup.See below for further information

E. **Correction of metabolic states**: Blood glucose has to be kept in the normal range. Hypoglycaemia is often seen in asphyxiated newborns. It increases energy deficit. It has to be treated based on hypoglycaemia protocol

- Hypocalcaemia (can cause seizure and decreased cardiac contractility) administer1- 2ml/kg of 10% calcium gluconate QID or add in to the maintenance fluid.

F. **Antibiotics** should be started and discontinue when sepsis is safely ruled out – Ampicillin with gentamycine or ampicilline with cefotaxime is the first line of antibiotic

G. **Seizure Treatment** (refer to the guideline on seizure treatment)

H. **Management of other organ system dysfunctions**

a. **Congestive heart failure** – diuretics , dopamine or dobutamine

b. Acute renal failure –dopamine Gastrointestinal – delay oral feeding

c. Hematologic failure – blood component replacement

I. Parent counselling has to be the integral part of management

Annex: Perinatal Asphyxia Follow up chart

The very important thing is to anticipate complications and act accordingly.

Table 7.2 Perinatal Asphyxia Follow up chart

Parameters	Day1		Day2		Day 3	
PR						
RR						
To						
SO2						
Wt						
HC						
Input						
Output						
Capillary refill						
RBS						
Urine analysis						
Serum electrolyte						
RFT						
LFT						
Gastric content						
Bowel sound						
Bloody stool						
Level of consciousness						
Neonatal reflexes						
Motor tone						
Seizure						
Progressive patient						
Treatment plan						

8. References

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