



Neonatal Hypoglycemia: Clinical Management & Diagnostic Pathways

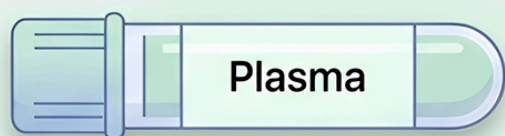
A visual protocol for neonatologists

Source Material:

Gomella, T. L. (2004). Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs (5th ed.). McGraw-Hill.

The Diagnostic Threshold & Screening Dilemma

The Clinical Threshold

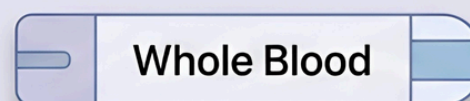


Definition: Plasma glucose $<40\text{--}45$ mg/dL in a term or premature infant.

Rule of Thumb:

Do not delay acute treatment for symptomatic infants while awaiting lab confirmation.

The Bedside Trap



Visual Alert: Whole blood glucose is 10–15% **lower** than plasma glucose.

Limitation: Glucose oxidase strips (Dextrostix/Chemstrip) are highly inaccurate at low ranges ($<40\text{--}50$ mg/dL).

Protocol: Never diagnose or treat solely based on screening strips. Always send a stat serum sample to the laboratory to confirm.



Clinical Presentation: The Symptom Spectrum

Neurologic

- Lethargy, hypotonia, and jitteriness
- Exaggerated Moro reflex
- Seizures, eye rolling, and coma

Cardiopulmonary

- Apnea and irregular respirations
- Cyanosis
- Tachycardia or bradycardia

Metabolic & Systemic

- Temperature instability (hypothermia)
- Poor feeding and inadequate sucking reflex
- Pallor



Profound hypoglycemia can be completely asymptomatic. Routine screening of high-risk infants is mandatory.

The Etiology Triad: Differential Diagnosis

Substrate Deficiency

Mechanism: Decreased glycogen stores and limited alternate fuels.

Populations: Premature, Postmature, IUGR / Small for Gestational Age (SGA).

Increased Utilization / Stress

Mechanism: Rapid depletion of energy reserves due to systemic stress.

Populations: Perinatal asphyxia, sepsis, hypothermia, polycythemia, shock.

Hormone Excess (Hyperinsulinism)

Mechanism: Increased fetal insulin production continuing post-delivery.

Populations: Infant of Diabetic Mother (IDM), Beckwith-Wiedemann syndrome, Islet cell adenoma / Nesidioblastosis.

High-Risk Profiles: Opposing Pathophysiologies

Infant of Diabetic Mother (IDM)

- **Incidence:** Hypoglycemia occurs in ~40% of IDMs.
- **Onset:** Typically presents 1–2 hours post-delivery.

1. Maternal hyperglycemia

2. Fetal hyperglycemia

3. Pancreatic beta-cell hyperplasia

4. Postnatal hyperinsulinism

Small for Gestational Age (SGA / IUGR)

- **Onset:** Typically presents 6–12 hours post-delivery.
- **Mechanism:** Diminished glycogen reserves + Decreased gluconeogenesis capacity + Reduced oxidation of free fatty acids (limited alternate fuels).
- **Note:** Glucagon therapy may be less effective due to poor glycogen stores.

Acute Triage & Initial Management

Point-of-care screen indicates
Dextrostix <25 mg/dL or Chemstrip <20 mg/dL

Path A: Asymptomatic
(No risk factors & clinically stable)

1. Action: Early feeding (D5W or formula).

2. Follow-up: Recheck glucose in 30–60 minutes.

3. Failure: If glucose remains low,
establish IV access.

Path B: Symptomatic
(or failure of oral feed)

1. Action: Immediate IV access is mandatory.
Proceed to IV Dextrose Protocol.

2. Alternative (No IV access): Administer
Glucagon 300 ug/kg (IM/SC) max 1.0 mg
while establishing access.

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Medicines

Glucagon

IM or subcutaneous

IV bolus

subcutaneous

1mg/mL Powder

Weight (kg)

2

Desired dose (mg/kg/dose)

0.3

Suggested dose: 0.2 mg/kg/dose

Glucagon

IM or subcutaneous - 1mg/mL Powder

Dose0.6mg

IntervalAMD

Reconstitution

Reconstitute with

SWFI or own diluent

1mL

Concentration after

reconstitution

1mg/mL

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Concentration after

reconstitution

1mg/mL

Dose after

reconstitution

0.6mL

Maximum dose

Maximum daily dose

1mg

Note: the maximum daily dose is 1 mg.

Additional information

Bibliographical references

Acute IV Management Protocol

1 The Bolus

10% Dextrose in water (D10W).

Dose: 2–4 mL/kg (250–500 mg/kg) via slow IV push.

2 The Continuous Infusion

Follow bolus immediately with D10W infusion at 6–8 mg/kg/min.

3 Monitoring & Titration

Check levels every 30 mins until stable, then q4h.

Titrate infusion up by 0.5–1 mg/kg/min as needed to achieve normoglycemia.

Calculation: Dextrose Infusion Rate

Glucose req
(mg/kg/min) =

Amount of glucose/mL × Total fluids

Weight in kg × 60 min



**OR JUST FILL THE
CAMPS AND HAVE
THE RESULT IN
SECONDS!**



Refractory & Persistent Hypoglycemia

Definition of Persistent

- Hypoglycemia persisting >7 days
- Requiring escalation of IV glucose to >16–20 mg/kg/min

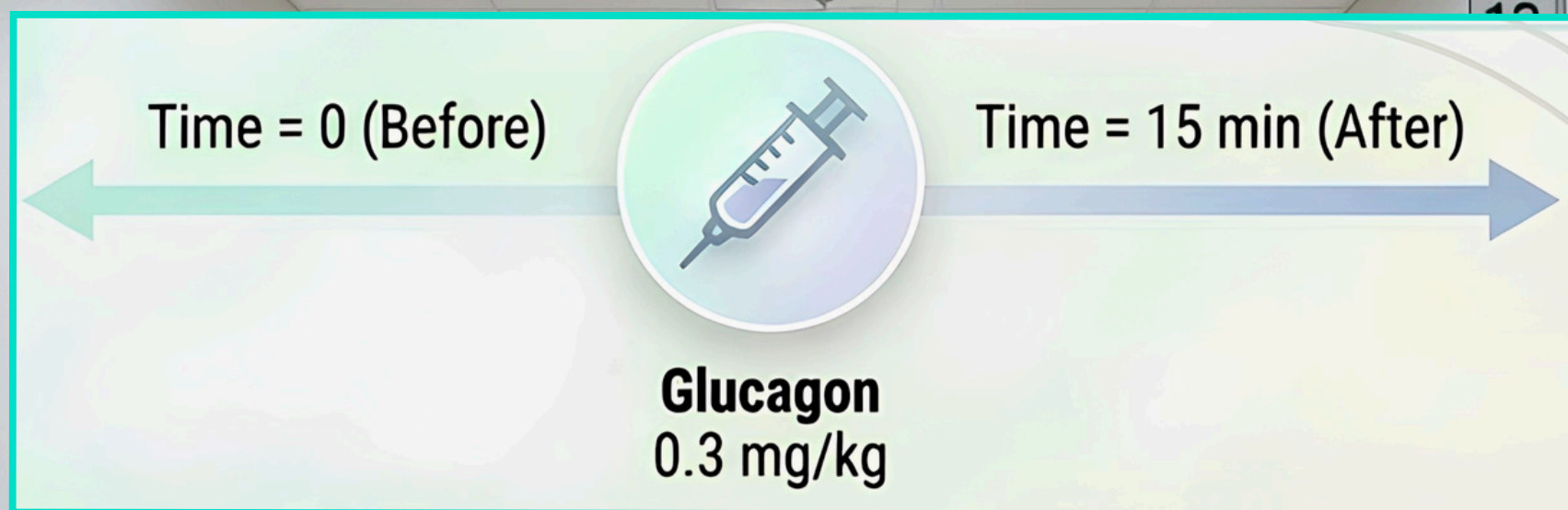
Clinical Actions

- **Escalation limit:** Rates >20 mg/kg/min are rarely helpful and risk fluid overload.
- **Intervention:** Endocrine consultation required.
- **Next Step:** Maintain normoglycemia and prepare for definitive diagnostic workup before administering empirical medical therapy.

Crucial Rule: Do not wean IV glucose blindly. Attempting to wean without a definitive diagnosis can mask underlying endocrine or metabolic errors.

The Definitive Diagnostic Workup

The Strategy: Obtain critical labs immediately before and 15 minutes after parenteral administration of Glucagon (0.3 mg/kg/dose).



The Critical Draw (Serum/Plasma)



Metabolites

Glucose, Ketones, Free Fatty Acids (FFA), Lactate, Alanine, Uric Acid.



Hormones

Insulin, Growth Hormone (GH), Cortisol, Glucagon, T4, TSH.

Urine Collection

Check for: Catecholamines, organic acids, specific reducing sugars.

Diagnostic Interpretation Matrix (The Glucagon Test)

Biomarker	Hyperinsulinism	Hypopituitarism	Metabolic Defect
Glucose (After Glucagon)	↑↑↑	Normal/↑	↓/Normal
Insulin	↑↑	Normal/↑	Normal
Ketones	↓	Normal/↓	↑
Free Fatty Acids (FFA)	↓	Normal/↓	↑
Lactate	Normal	Normal	↑↑
Cortisol & GH	Normal/↓	↓↓	↑

Inborn Errors of Metabolism (Acute Onset)



Type I Glycogen Storage Disease

Presentation: Profound hypoglycemia, hepatomegaly, lactic acidosis.
Immediate Action: Avoid fructose/galactose; institute frequent small feeds.



Galactosemia

Presentation: Jaundice, hepatomegaly, non-glucose reducing substances in urine.
Immediate Action: Initiate a galactose-free diet immediately on suspicion.



Amino Acid / Organic Acidemias

Presentation: Severe metabolic acidosis, lethargy, encephalopathy.
Examples: Maple syrup urine disease, propionic acidemia.

Pharmacologic & Long-Term Management

Hyperinsulinism



Drug: Diazoxide (10–15 mg/kg/day PO in 3-4 divided doses).

Mechanism: Directly inhibits pancreatic insulin release.

Hypopituitarism / Adrenal Insufficiency



Drug: Hydrocortisone (10–20 mg/m²/day) + continuous IV glucose.

Mechanism: Replaces absent counter-regulatory cortisol.

Acute Glycogen Mobilization (Rescue)



Drug: Glucagon (300 ug/kg IM/SC, max 1.0 mg).

Note: Less effective in SGA/IUGR infants with depleted stores.

The Neonatal Glucose Homeostasis Framework

Phase 1: Recognition & Triage

Screening (<45 mg/dL) → Symptoms vs. No Symptoms → D10W Bolus or Oral Feed.

Phase 2: The Fork in the Road

Transient (Resolves with ≤ 15 mg/kg/min IV glucose) vs. Persistent (>7 days or >15 mg/kg/min).

Phase 3: The Definitive Diagnosis

Glucagon Challenge → Hormone and Metabolite Panel.

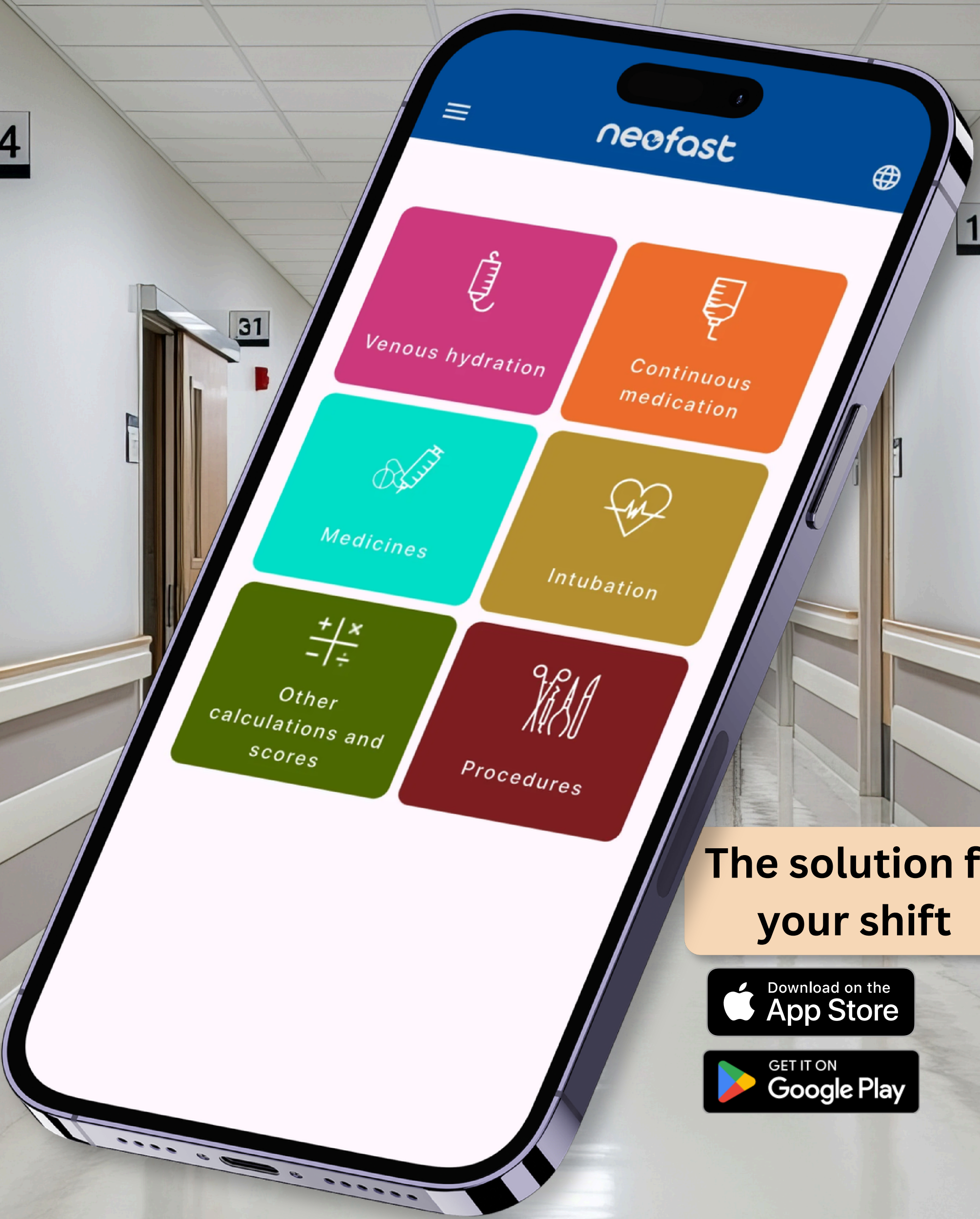
Phase 4: Targeted Therapy

Hyperinsulinism (Diazoxide) | Endocrine Defect (Hydrocortisone) | Metabolic Defect (Specific Diet).

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Venous hydration



Continuous medication



Medicines



Intubation



Other calculations and scores



Procedures

The solution for your shift



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