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Guidelines for Hypoglycemia Screening and Intervention in At-Risk Infants

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EDITOR'S NOTE

In the 45 years that I have been a neonatal nurse, and the 26 years that I have been the co-editor of *Merenstein and Gardner's Handbook of Neonatal Intensive Care*, there has never been a clinical practice guideline from the American Academy of Pediatrics (AAP) covering postnatal hypoglycemia. Even the latest edition of *Guidelines for Perinatal Care*¹ published by the AAP and The American College of Obstetricians and Gynecologists has no direction for neonatal care providers about postnatal hypoglycemia.

Finally, the 2009–2010 Committee on Fetus and the Newborn of the AAP actually tackled this problem and in 2011 published its *Clinical Report—Postnatal Glucose Homeostasis in Late-Preterm and Term Infants*.² The report provides a practical guide and algorithm for all clinicians, medical, nursing and advance practice nurses, who screen and manage neonates at-risk for hypoglycemia. As a liaison member, Dr. Rosalie Mainous represented the National Association of Neonatal Nurses to the Committee on Fetus and Newborn in their extensive work on this topic. Dr Mainous and her doctoral student, Maryam Alaradi, graciously accepted my invitation to co-author this paper for *Nurse Currents*.

Nurses are the professionals who assess and care for neonates during transition and provide ongoing care and assessment throughout their hospitalization. Regardless of the geographic location of the newly born—the labor, delivery, recovery, postpartum (LDRP) area, a mother-baby unit, a transition nursery, Level I/normal newborn nursery, Level II/ medium risk/special care nursery, Level III/NICU/ICN nursery, on a pediatric unit or at home/office visit—all nurses caring for neonates must be able to assess, intervene, and re-evaluate for postnatal hypoglycemia.

Healthcare institutions also have an obligation to their clients to provide on-going education for professional staff and instituting policies/procedures/protocols that represent the national and state standard of care. During my 35 years of legal consultation on nursing negligence/malpractice cases I have seen some atrocious hypoglycemia policies/procedures /protocols. In one hypoglycemia case that I reviewed, the hospital's hypoglycemia policy was four sentences long! By reading the document, I could guess who wrote it, and when I persisted in querying the defense attorneys they finally "owned up" to the fact that "a doctor" had written the nursing policy! Needless to say, I declined to testify for the defense, recommended that nurses write nursing policies and procedures, and offered to share four Colorado hypoglycemia policies from the 1970's that were a higher standard of care than this hospital's 1990's document.

Now, with the new AAP guideline, creating institutional policies that reflect the national standard of care should be easier. Just as the AAP's 2004 document, *Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation*,³ provided guidelines for assessment and management of this potentially serious complication, the AAP report on postnatal hypoglycemia provides much-needed and long-overdue guidance.

LEARNING OBJECTIVES

Following reading this article, the reader will be able to:

- Describe the process used to develop a clinical report by the Committee on Fetus and Newborn of the AAP.
- Identify which infants are most at risk for hypoglycemia.
- Detail the process of glucose metabolism and insulin production.
- Describe a treatment plan for infants less than 4 hours of age and for infants 4–24 hours of age who are at-risk for hypoglycemia.

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Neonatal nurses (and the newborns we care for) are indebted to the Committee on Fetus and Newborn, its 2009–2010 chairperson, Lu-Ann Papile, MD, all the committee and liaison members and staff, and particularly David Adamkin, MD, the lead author, for their countless hours of literature review, deliberation and writing of the clinical report. Their work and this guideline make our job as neonatal-care providers more effective and evidence-based.

~ Sandra Gardner

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3. American Academy of Pediatrics, Subcommittee on Hyperbilirubinemia: Clinical practice guideline: Management of hyperbilirubinemia in the newborn infant 35 weeks or more weeks of gestation *Pediatrics* 2004; 114:297.

Hypoglycemia is the most common metabolic problem encountered in Neonatal Intensive Care Units (NICU) and newborn nurseries.¹⁻³ Low blood glucose level is considered a vital chemical indicator of an underlying disorder.



Photo by Fanfo

Hypoglycemia is the most common metabolic problem encountered in Neonatal Intensive Care Units (NICU) and newborn nurseries.¹⁻³ Low blood glucose level is considered a vital chemical indicator of an underlying disorder. Since glucose is the main source of cellular energy, any disturbance that

might influence its availability or the mechanism of utilization could result in hypoglycemia. As a result, assessment of blood glucose level is an important component of basic neonatal care in all neonatal nurseries.³ While the majority of hypoglycemia cases occur as the result of normal adaptation to extrauterine life,⁴ and are not

associated with significant problems, prolonged hypoglycemia, if not detected and treated early, could lead to adverse neurological sequelae and subsequently poor prognosis.

Much debate has taken place about the precise definition of neonatal hypoglycemia and its treatment. Currently, little evidence exists to

support a clear definition for hypoglycemia in neonates or a universal plasma glucose concentration range.⁵ Neither the duration of hypoglycemia nor a specific serum glucose level has been shown to be predictive of permanent central nervous system damage in at-risk infants.⁶⁻⁹ However, nurses are still challenged with the clinical care of neonates at risk for postnatal hypoglycemia and its potential complications.

This article presents the physiology of glucose metabolism, the importance of glucose in neonatal brain function, the signs and symptoms of morbidity and the screening and management algorithm recommended by the American Academy of Pediatrics (AAP).

A practical guide and algorithm for neonatal hypoglycemia screening and management has been developed by the AAP's Committee on Fetus and Newborn. The clinical report proposes early recognition of at-risk infants, initiation of blood glucose testing, and prophylactic measures to avert neonatal hypoglycemia.⁶

Since we are on the frontlines of neonatal care, nurses are the primary agents for early detection of neonatal hypoglycemia. Careful observation and a proper assessment are crucial, as well as an awareness of which infants are most at risk for negative outcomes. Therefore, the need for the algorithm is universal for all nurses caring for neonates.

Glucose Homeostasis

Glucose is the main energy source for the fetus. The oxidation of glucose generates energy and unused glucose is stored in the liver in the form of glycogen. Through facilitated diffusion, the fetus receives a continuous supply of glucose from the mother via the placenta. Fetal glucose levels depend on the maternal glucose levels rather than on the maternal insulin level, because insulin does not cross the maternal-fetal placental barrier.¹⁰ As early as 13 weeks of gestation, the fetus produces its own insulin.¹¹ Insulin, produced by beta cells in the pancreas, is essential in cellular uptake of glucose through the facilitated diffusion process.¹²

Beginning in the second trimester, glycogen synthesis increases steadily until 36 weeks of gestation when it increases rapidly until term and reaches 50 mg/g of tissue.¹¹ Although gluconeogenesis enzymes exist by

the third month of gestation fetal glucose production is very modest.^{13,14} During this period, if glucose supply to the fetus is decreased because of maternal hypoglycemia or any defect in the placenta, the fetus attempts to maintain glucose levels through fatty acid metabolism (ketogenesis). After prolonged periods of glucose deficiency, the fetus begins producing its own glucose by glycogenolysis and later by glyconeogenesis. Producing endogenous glucose, instead of having glucose provided in adequate amounts by the mother, takes a toll on fetal growth and development.¹⁴

At birth, clamping the umbilical cord abruptly discontinues maternal-fetal glucose transfer. At this time, umbilical venous plasma glucose levels are 60–80% of the maternal venous glucose concentration.^{13,15} “The aim of neonatal glucose homeostasis is to provide the brain and other vital organs with sufficient glucose as a key energy source.”¹¹ Before feeding is initiated the newborn begins its own glucose production, which is considered an intermittent source of energy supply.¹³

At birth, glycogenolysis is stimulated, as the decreasing levels of insulin coincide with the increasing levels of catecholamines and glucagon.¹³ Although glycogenolysis yields a rate of 4–6 mg/kg/min of hepatic glucose in a full-term infant,^{13,14} and 8–9 mg/kg/min in both the fetus and the preterm infant,¹⁴ this amount alone is not enough to supply the newborn with energy. Therefore, depot fat mobilization is essential for energy production. Because it is mainly formed during the third trimester of gestation, 15%¹³ to 16%¹¹ of a full-term newborn's body weight is composed of depot fat, compared to 2% in the preterm infant.¹¹ The liver is only capable of storing 50 to 75 grams of glycogen per kilogram of liver, which is equivalent to 200 to 300 calories.¹⁰ Therefore, when hepatic glycogen stores are depleted within 12 hours of birth,^{11,16} the newborn will then depend on either exogenous glucose supply or on an endogenous glucose production through gluconeogenesis.¹⁶

Glucose and Neonatal Brain Function

Insulin regulates the uptake of glucose by cells. In the brain, glucose supply is dependent on systemic blood glucose concentration and not dependent on insulin levels.¹² Main-

tenance of a steady flow of glucose is crucial for all tissue and organ functions. However, since glucose is the only substrate used by the brain, maintenance of adequate glucose levels is even more critical during normal conditions and especially during high-energy-demand conditions (such as sepsis, seizures and severe neonatal encephalopathy).⁵ The newborn has a higher brain-to-bodyweight ratio than an adult. As a result, cerebral glucose demand is higher and may account for as much as 90% of total glucose consumption.¹⁷

The glucose level below which the neonate might develop long-term neurodevelopmental impairment remains debatable.^{18,19} Several studies have shown that prolonged and persistent neonatal hypoglycemia can lead to adverse neurological sequelae, such as epilepsy, and the psychomotor and visual disturbances known as hypoglycemia-occipital syndrome.¹⁹⁻²² Another study has shown that early (0–6 hours of life) hypoglycemia in 36 to 42 gestational age (GA) infants was linked to severe hypoxic-ischemic encephalopathy (HIE).²² Poor prognosis may follow prolonged hypoglycemia, with various disabilities such as learning difficulties, cerebral palsy, mental retardation, seizures,¹⁴ and visual impairment.²

Neonatal Hypoglycemia: Definition

The definition of hypoglycemia in the neonate is controversial. Current studies have not been successful in arriving at a consensus for what are normal or abnormal glucose levels.^{6,14,23} A clear definition of hypoglycemia is complicated by contributing factors to the development of low blood sugar in the neonate. Among these factors are GA,²⁴ birth weight,¹⁵ intrauterine growth restriction (IUGR) and small-for-gestational age (SGA). Various methods of glucose measurement,²⁴⁻²⁷ and maternal conditions^{28,29} must also be considered in the definition and treatment of hypoglycemia.

The AAP's new algorithm provides a method of screening infants, managing their low blood sugar and decreasing their risk of complications. Because hypoglycemia is defined as “the concentration of glucose in the blood or plasma at which the individual demonstrates a unique response to the abnormal milieu caused by inadequate delivery of glucose to a target organ” care of every infant must be individualized.¹⁵ Current

Table 1: Operational Thresholds for Hypoglycemia in the Neonate⁴

Age in hours	GA	**PGC (mg/dL)	**PGC (mmol/L)	Conditions
First 24 hours	Healthy full-term or preterm (34-37 GA)	*30-35	1.7-2	Formula fed infant
		*≥45-50	≥2.5-2.8	Sick, LBW, premature infants suspected of having increased glucose requirements due to sepsis, hypoxia, or other major systemic illnesses
Beyond 24 hours of age		*40-50	2.2-2.8	

*Values below this range are an indication to raise blood glucose level and do not imply neurological damage.

Infants at all ages and GA with repetitive, reliable PG values < 20–25 mg/dL (1.1–1.4 mmol/L) should be given parenteral glucose and monitored at regular intervals. A diagnostic workup is indicated if hypoglycemia recurs or persists.

**PGC: Plasma Glucose Concentration

GA: gestational age; LBW: low birth weight

Adapted from: Cornblath M, Ichord R: Hypoglycemia in the neonate *Seminars in Perinatology* 2000; 24:136.

literature reports that hypoglycemia occurs when blood glucose concentration reaches <47 mg/dL (<2.6mmol/L).^{6,23,26,30} The S.T.A.B.L.E program has adopted a slightly more conservative value of 50 mg/dL (2.8 mmol/L), below which an intervention is required.³¹

Another approach to defining hypoglycemia is called Whipple's Triad. This approach depends on three criteria: (1) an accurate measurement of low blood glucose level; (2) the presence of signs of hypoglycemia, and (3) resolution of signs of hypoglycemia after restoration of normal blood glucose level.^{4,15,31,32} Table 1 presents conservative approximations of operational thresholds for the lower levels of normal glucose that can be tolerated by specific infants, ages and under established conditions.⁴

Incidence

The incidence of neonatal hypoglycemia is dependent on the definition of hypoglycemia and the glucose measurement tool. Approximately 1 to 5 per 1,000 term live births,^{17,29} and 15 to 55 per 1,000 preterm births are at risk of developing neonatal hypoglycemia.³³ SGA neonates are also at higher risk, with some studies reporting an incidence of 26%

in SGA infants with blood glucose levels below the threshold of 2.6 mmol/L (or 46.8 mg/dl).³⁴ A Swedish national population-based study found that 16% of infants born at 30 to 34 weeks gestation developed hypoglycemia.³⁵ Another Swedish population-based cohort study (from 2004 and 2006) found that hypoglycemia is prevalent in 41% of extremely preterm infants (<27 weeks' gestation) during the first week of life.³⁶

Who is at Risk for Hypoglycemia?

Fetal/neonatal and/or maternal conditions place newborns at risk for developing varying degrees of neonatal hypoglycemia.

1. Premature and IUGR infants

Hypoglycemia is a common problem in preterm infants due to their limited glucose stores. In the SGA infant, hypoglycemia is a result of inadequate substrate available for glycogen synthesis. Neonates with asymmetrical IUGR have larger head-to-bodyweight ratios that result in higher brain glucose requirements.¹⁷

Late preterm infants (34 0/7–36 6/7 weeks of gestation) are often treated as if

they were full-term infants. However, the most common problems encountered by late preterm infants are cold stress and hypoglycemia. The development of hypoglycemia in the late preterm occurs because of delays in hepatic glycogenolysis and gluconeogenesis, limited enteral intake due to immature central nervous and digestive systems, and uncoordinated suck/swallow/breathe.³⁷

Cold stress aggravates hypoglycemia because hypothermia increases metabolic rate, oxygen consumption and glucose demand and utilization.³¹ Nurses should be aware of the consequences of cold stress and prevent it.

2. Infants with a history of perinatal stress

After birth, newborns with a history of asphyxia and hypothermia or those with respiratory distress may suffer from hypoglycemia. Depleted glycogen stores used for the normal transition to postnatal life and increased demands of higher than normal glucose utilization are the reasons for hypoglycemia in stressed newborns.¹⁷



3. Infant of a Diabetic Mother (IDM)

In the United States, approximately 154,000 (4%) pregnancies resulting in live births each year are complicated by diabetes. Of these pregnancies, 88% of the live births were from mothers with gestational diabetes mellitus (GDM), 8% from mothers with non-insulin dependent diabetes mellitus (NIDDM), and 4% with insulin-dependent diabetes mellitus (IDDM).³⁸ Poor glycemic control prior to conception and during pregnancy may have profound consequences for the embryo, fetus and neonate.^{29,39,40} Uncontrolled maternal glycemia causes neonatal hypoglycemia as well as transient hyperinsulinemia.⁴⁰ Conversely, controlled maternal glycemia, with minimal microvascular disease, can result in a pregnancy outcome that is comparable to that of normal pregnancy.³⁹

IDM is a term used to refer to infants born to mothers with either preexisting diabetes or GDM. Either maternal condition leads to an increased risk for newborn mortality and morbidity.²⁹ In utero, maternal hyperglycemia increases placental glucose transport and results in fetal hyperglycemia, which stimulates fetal pancreatic insulin production. After delivery, maternal glucose supply ceases even though newborn

insulin production continues. A cascade ensues of high insulin-to-glycogen ratio, which inhibits induction of gluconeogenic enzymes, hinders hepatic glucose production and results in hypoglycemia. Hypoglycemia may continue for 24 to 72 hours until insulin secretion returns to normal.¹⁷

4. Other conditions

Erythroblastosis fetalis causes an abnormal increase in the number of pancreatic beta cells and increases in the secretion of insulin.¹⁷ Other conditions that increase the production of insulin include: Beckwith-Weidemann syndrome, insulin-producing tumors, maternal tocolytic therapy and exchange transfusion with blood high in glucose content.³² Some risk factors are transient; others like familial hyperinsulinism and inborn errors of metabolism are prolonged.¹⁷

Clinical Presentation

The clinical signs of hypoglycemia are non-specific (see Table 2) and mimic symptoms from a variety of conditions such as sepsis, hypocalcaemia and intracranial hemorrhage.²⁷ Hypoglycemia may precede distur-

bances in the central nervous system, but there is a lack of evidence about what level of blood glucose is necessary for brain injury. If the neonate exhibits one or more signs in Table 2, hypoglycemia must be considered.¹⁷

Screening for Hypoglycemia

Prevention is always better than cure. Preventing hypoglycemia begins with identification of maternal, and fetal risk factors and the care provider's vigilance in instituting hypoglycemia screening in at-risk neonates. Preventing complications of hypoglycemia depends on early recognition of clinical signs associated with hypoglycemia and prompt intervention.

The AAP recommends screening at-risk newborns such as SGA, LGA, IDM and late-preterm infants. Healthy term newborns born after a normal, healthy pregnancy need not be screened, but term infants who have symptoms of hypoglycemia require blood glucose measurements as soon as possible.⁶

Point of care (POC) glucose measurement is the most common method used for glucose screening in the term nursery and the NICU.^{25,27} However, this testing is not completely reliable as a basis to diagnose hypoglycemia,^{4,24} particularly when glucose values drop below 40 to

Table 2: Clinical Presentation of Hypoglycemia in Newborn

Jitteriness, irritability, or tremors	Poor suck/coordination/feeding
Lethargy	Vomiting
Hypotonia	Tachycardia or bradycardia
Temperature instability	Cyanosis
High-pitched or weak cry	Eye rolling
Tachypnea	*Seizures
Apnea	*Coma
*May occur with prolonged and repetitive hypoglycemia ⁶	



50 mg/dL.²⁷ Laboratory confirmation should always follow low blood glucose concentration values obtained by POC.

Serum glucose measurement in the laboratory (using either the glucose electrode or the glucose oxidase method) is considered the gold standard approach for diagnosis of hypoglycemia.³⁰

Variations in glucose values may occur as a result of type of blood used, sample site, presence of polycythemia, timing between sample collection and analysis,⁹ and contamination of the sample with isopropyl alcohol.¹⁷ When sampling plasma rather than whole blood, plasma glucose values are generally 10–15% higher.^{24,27} Laboratory glucose values that are reported in mmol/L can be converted to mg/dL by multiplying by 18.²⁴

Recently, continuous interstitial glucose monitoring systems have been tested in newborns at risk for hypoglycemia. Continuous monitoring detects more hypoglycemic episodes than sporadic blood glucose measurement.^{30,41}

Management

Asymptomatic hypoglycemia

A recent survey about the management of neonatal hypoglycemia found that 19% of respondents treat asymptomatic hypoglycemia with either formula or intravenous dextrose.²³ Feeding formula to the asymptomatic newborn was reported as the most frequently used treatment. The same survey also found that 55% of asymptomatic IDM were less likely to receive treatment compared to 85% of preterm infants.²³

Healthcare professionals, particularly nurses, must understand the differences between glucose levels in breast-fed and formula-fed neonates. After birth, in all neonates, blood glucose concentrations drop rapidly to as low as 30 mg/dL in the first 1–2 hours of age, rebounding to >45 mg/dL by 12 hours of age.⁶ When compared to formula-fed newborns, breast-fed infants have lower glucose levels and higher ketone levels. Gluconeogenesis is enhanced with early breast-feeding. Nurses

caring for breast-fed newborns and their mothers must ensure that the infant latches on properly and breast-feeds adequately to avoid hypoglycemia during hospitalization and after discharge.³²

Symptomatic hypoglycemia

Intravenous dextrose infusion should be initiated immediately for infants with symptomatic hypoglycemia.^{6,12,42} An Australian survey found that 93% of respondents reported treating symptomatic hypoglycemia with intravenous glucose, either with bolus (48%) or increasing dextrose concentration (45%).²³ Few (6%) reported the use of medication, such as hydrocortisone or phenobarbitone, as the first treatment choice.²³

To achieve a plasma glucose level of 40–50 mg/dL, the AAP recommends a glucose dose = 200 mg/kg (dextrose 10% at 2 mL/kg), and/or IV infusion of 5–8 mg/kg per minute (80–100 mL/kg per day).⁶ Blood glucose should be checked every 30–60 minutes after the start

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of therapy and should be confirmed by a laboratory sample. Glucose therapy should be decreased gradually as enteral feeding is introduced and advanced.¹² Some infants might require higher glucose concentrations (at a rate of 12 to 15 mg dextrose/kg/minute [15% or 20% dextrose]).³² Neonates requiring higher glucose concentrations should have a central line placed to avoid extravasation of hyperosmolar glucose solution.¹²

If hypoglycemia (glucose levels below 40mg/dL) persists despite adequate treatment, hyperinsulinemic hypoglycemia should be suspected. A blood sample to measure the insulin level should be obtained and an endocrinologist consulted.⁶

A pharmacological approach to correct hypoglycemia is only begun when all initial measures fail to increase blood glucose concentration above 40 mg/dL. The most commonly used medications are corticosteroids, glucagon, and diazoxide. Other agents like epinephrine and growth hormones are rarely used.^{12,32} Surgical intervention, focal/subtotal or near-total

pancreatectomy, may be considered as the last treatment resort for persistent hyperinsulinemic hypoglycemia that is unresponsive to pharmacological treatment.⁴³

Every neonatal nursery and NICU must have a written protocol for neonatal hypoglycemia management. This protocol must represent the current national standard of care for hypoglycemia management as outlined by the AAP. (See section below.) In the Australian survey, 97% of nurseries reported having a hypoglycemia protocol and 94% reported using it.²³ In this 2009 survey there was one level III nursery that reported no protocol for the management of hypoglycemia.²³

AAP Algorithm for Hypoglycemia

In 2011, the AAP published its clinical guideline on postnatal glucose homeostasis in late-preterm and term infants.⁶ The lead author of the clinical guideline by the Committee on Fetus and Newborn was David Adamkin, chief of the division of Neonatal Medicine in the Department of Pediatrics at the University

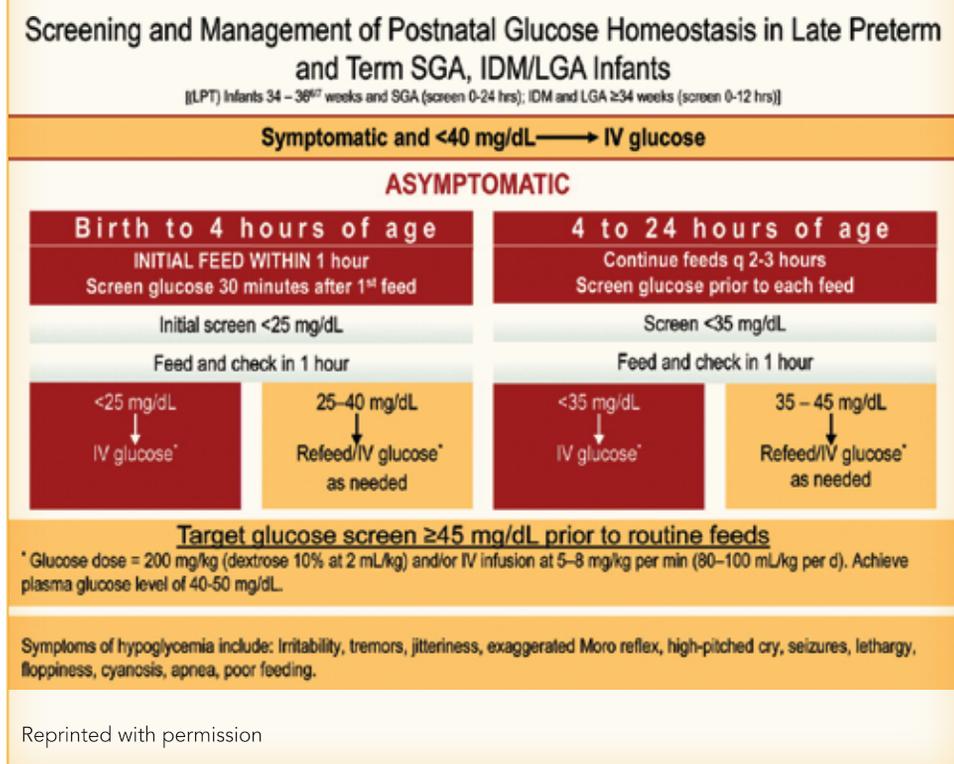
of Louisville. An expert in the area of neonatal nutrition, Dr. Adamkin initiated an extensive process of discussion, debate, literature review, comparison of clinical practices, as well as consultations with experts (personal communication, August 9, 2011), to develop the hypoglycemia screening instrument.

As a practical guide the AAP algorithm assists clinicians in the identification of newborns at risk for hypoglycemia and subsequent neurovascular damage.

The algorithm in Figure 1 is now the recommended method for screening and management of neonatal hypoglycemia in the late preterm and term infant at risk. The target glucose value prior to routine feeds in the algorithm is >45mg/dL.⁴ Figure 1 provides the bedside clinician with clear guidelines for management:

1. The symptomatic infant (glucose level less than 40mg/dl) is quickly treated with intravenous glucose: a bolus of 200mg/kg of 10% dextrose given at the rate of 2ml/kg. This is followed by a continuous infusion of 5–8 mg/kg per minute, with the goal of a glucose level of 40–50 mg/dl.⁶
2. Treatment depends on the age of the infant at screening and is divided into two groups, <4 hours of age and those >4–24 hours of age. Neonates less than 4 hours of age will not be treated unless the initial screen is <25mg/dl. Newborns >4 hours of age will be treated if their glucose level is less than 35 mg/dl.
3. The first feeding for at-risk infants should occur within 1 hour of birth. Thirty minutes after the first feeding blood sugar level is screened; if the value is <25mg/dL, the infant is re-fed and blood glucose is checked again 1 hour after feeding. Depending on the second glucose value, the infant may receive IV glucose (<25 mg/dl) or be re-fed (25–40mg/dL) and IV glucose administered as needed.
4. Neonates who are 4–24 hours of age are fed every 2–3 hours and the glucose level should be checked prior to each feed. If the screen is <35 mg/dl, the infant should be fed and the glucose rechecked in one hour. If the blood glucose level remains at 35–45 mg/dl, the infant is re-fed and given IV glucose as needed.

Figure 1: American Academy of Pediatrics Algorithm.





Conclusion

Neonatal healthcare professionals must be educated about fetal and neonatal glucose

Terminology

Hypoglycemia operational threshold: concentration of plasma or whole blood glucose at which clinicians should consider intervention, based on the evidence currently available in literature;¹⁵ an indication for action and not diagnostic of disease.⁴

Gluconeogenesis: synthesis of glucose from non-carbohydrate sources, such as amino acids and glycerol, that occurs primarily in the liver and kidneys whenever the supply of carbohydrates is insufficient to meet the body's energy needs.⁴⁴

Glycogenolysis: release of glucose by breakdown of glycogen stores in the liver.⁴⁴

Homeostasis: The tendency of biological systems to maintain relatively constant conditions in the internal environment while continuously interacting with and adjusting to changes originating within or outside the system.⁴⁴

Ketogenesis: production of ketone bodies⁴⁴ as a result of fatty acid breakdown.

homeostasis. Neonatal nurses must be able to properly and quickly assess at-risk infants and identify signs of hypoglycemia. *All* nurses caring for neonates must be aware of the current treatment modalities for neonatal hypoglycemia and advocate that newborns receive this standard of care. As “Partners in Care,” parents should be informed that their infant is at-risk or shows signs of hypoglycemia, and that frequent blood sampling will be needed.²⁷ Further research to identify safe neonatal glucose thresholds is also warranted.

About the Authors

Maryam Alaradi, RN, MSN, neonatal nurse practitioner, is a PhD student at the University of Louisville, School of Nursing and received a MSN from the University of Pennsylvania. She is currently a Fulbright Scholar. Maryam was born and raised in the Kingdom of Bahrain, an archipelago in the middle of the Persian Gulf, Middle East. After receiving her undergraduate nursing degree from College of Health Sciences in Bahrain, she worked in Labor and Delivery as a Midwife, in NICU as a supervisor and then as a neonatal nurse educator. Arriving from her native Bahrain to work with Dr.

Mainous, she is currently involved in research that identifies the impact of umbilical artery placement on neonatal cerebral and renal oxygenation. She has been accepted to present her work at the NINR 25th Anniversary Research Day. Her dissertation work is in the area of parental response and coping to pain in the newborn.

Rosalie O. Mainous, PhD, APRN, NNP-BC is currently Dean and Professor of Wright State University-Miami Valley College of Nursing and Health. She was a practicing Neonatal Nurse Practitioner for 8 years and currently serves on the American Academy of Pediatrics Committee on Fetus and Newborn, the only nurse on this committee. She will be presenting at the NINR 25th Anniversary Research Day on her study, *Impact of Umbilical Artery Catheters on Cerebral and Renal Tissue Oxygenation*. Dr. Mainous' work has been centered on pain in the newborn and the development of a prediction model for Intraventricular Hemorrhage. She also contributed to a manuscript that was part of the landmark report from the Institute of Medicine on the Future of Nursing, “Advancing Health, Leading Change.”

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