SCREENING FOR HYPOGLYCEMIA IN HEALTHY TERM NEONATES: EFFECTS ON BREASTFEEDING

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ABSTRACT

Transient hypoglycemia in the early neonatal period is a common adaptive phenomenon as the newborn changes from the fetal state of continuous transplacental glucose consumption to intermittent nutrient supply following cessation of maternal nutrition at birth. Research has demonstrated that in the term, healthy newborn, this dynamic process is self-limiting and is not considered pathologic.

The American Academy of Pediatrics and the World Health Organization recommend that neonatal blood glucose screening be reserved for newborns who are at risk or symptomatic and conclude that universal hypoglycemia screening is inappropriate, unnecessary, and potentially harmful. Nevertheless, many hospital nurseries continue the clinical practice of routine early glucose screening on healthy, term newborns. This results in the misidentification of neonates captured while experiencing the normal, self-correcting physiologic blood glucose nadir who are then diagnosed with pathologic neonatal hypoglycemia.

Subsequent to this misdiagnosis, further surveillance and unnecessary, aggressive treatment interventions will follow that are potentially harmful to the successful establishment of positive maternal-infant interactions and the breastfeeding experience.

Research studies indicate that routine hypoglycemia screens, treatments, and interventions in the healthy infant are not evidence-based and result in a serious disruption of the initiation process and duration patterns of lactation.

Using the perspective of the theory of technology dependency, this inquiry explores the potential adverse sequelae of inappropriate glucose screening in the healthy breastfeeding newborn and describes selected outcome variables including: 1) the consequences of early maternal-infant separation, 2) the influence of early formula supplementation on breastfeeding discontinuance rates, 3) the effect of separation and supplementation on the onset of lactogenesis, and 4) the impact of hospital staff and provider recommendations of formula supplementation on maternal confidence to independently nurture her baby. J Midwifery Womens Health 2001;46:292–301

OVERVIEW OF NEONATAL HYPOGLYCEMIA

Generally, neonatal hypoglycemia is not a medical condition, but a manifestation of the newborn's ability to adapt from the fetal state of continuous transplacental glucose consumption to the extrauterine pattern of intermittent nutrient supply (1). With the abrupt cessation of maternal nutrition at birth, transient neonatal hypoglycemia in the first 2–4 postnatal hours is almost always universal in mammals (2).

Research has demonstrated that, even in the absence of enteral feeds, this phenomenon is self-limiting in healthy, term newborn humans as this initial postnatal nadir gradually increases over the subsequent days (3–6). In other words, the healthy, term neonate has the unique capacity to sustain normoglycemia and achieve successful metabolic adaptation, even in a fasting state, via the breakdown and mobilization of endogenous glycogen reserves found in the liver and kidney (glycogenolysis), hepatic synthesis of glucose from other substrates including glycerol, lactate, pyruvate, and glycogenic amino acid precursors (gluconeogenesis), and the production of alternative cerebral fuels such as ketone bodies through fatty acid mobilization (4,5,7–9). Moreover, as dietary carbohydrate intake from milk within the first days of life is low (providing approximately 20% to 50% of utilized glucose), the healthy neonate becomes largely dependent upon the process of gluconeogenesis and fatty acid mobilization within the first postnatal day for energy expenditure and the maintenance of glucose homeostasis (1,4). There is a need to consider the whole fuel milieu of the neonate not just glucose alone when evaluating and managing the hypoglycemic newborn. Researchers have concluded that neonatal hypoglycemia is a continuum, with an immediate and precipitous fall immediately after birth that is arrested before slowly returning to glucose levels that eventually are close to the adult reference range (2,5,7). Researchers have also concluded that "functional" or nonpathologic hypoglycemia may vary substantially from one baby to another, and no single blood glucose determination indicates functional hypoglycemia in every child (1,6–8).

The early neonatal condition of transient hypoglycemia in a healthy infant should not be viewed as a pathologic phenomenon, since the condition will promptly self-correct as blood glucose concentrations spontaneously rise to maintain normoglycemia (2). For this reason, the Committee on Fetus and Newborn of the American Academy of Pediatrics (AAP) and the World Health Organization (WHO) both consider universal glucose monitoring for hypoglycemia in healthy, neo-

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1526-9523/01/$20.00 • PII S1526-9523(01)00180-5

TABLE 1
Recommendations for Hypoglycemia Screening

American Academy of Pediatrics: "Universal neonatal screening of blood glucose for hypoglycemia is not warranted in most nurseries. Furthermore, there is no evidence that asymptomatic hypoglycemic infants will benefit from treatment" (10).
World Health Organization: "Healthy term newborns who are breastfeeding on demand need not have their blood glucose routinely checked and need no supplementary foods or fluids" (5).

nates to be an inappropriate procedure (5,10). See Table 1.

Glucose screening should be reserved for at-risk infants. At-risk infants are those associated with changes in maternal metabolism (diabetic mothers, mothers administered excessive intrapartum glucose, and mothers receiving drug therapy including terbutaline, ritodrine, propranolol, and oral hypoglycemic agents) (1). At-risk infants associated with neonatal problems include infants that are small for gestational age (SGA), infants with intrauterine growth restriction (IUGR), premies, discordant twin (weight 10% $<$ than larger twin), low birth weight ($<$2,500 g), ill and/or symptomatic babies suffering from sepsis, perinatal hypoxia-ischemia or perinatal stress (5 minute APGAR equal to or less than 7, or cord pH $<$7.2), Rhesus disease, polycythemia (venous hematocrit $>$70%), cold stress, hypothermia (temperature equal to or less than 35°C), respiratory distress, erythroblastosis fetalis, and Beckwith-Weidman Syndrome (1,5,8,11,12).

Healthy but large for gestational age infants are not considered at-risk unless known to be infants of diabetic mothers (5). Hypoglycemia, which recurs or persists at 48–72 hours of age, suggests an inborn error of metabolism or endocrine disorder and requires a medical consult, further assessment, and pediatric management (2,5,6,11).

Despite these recommendations, many hospitals continue the clinical practice of universal glucose screening upon admission to the nursery (1). While this routine most importantly serves to screen the at-risk neonate of concern, it also serves to identify the population of healthy newborns who are captured experiencing the normal, self-correcting physiologic blood glucose nadir. As a result, further hypoglycemia surveillance and treatment interventions follow that unfortunately include the provision of early formula supplementation to breastfed neonates and early maternal-infant separation. These unnecessary interventions result in potential harm to the successful establishment of positive maternal-infant interactions and the breastfeeding experience (13–16). Furthermore, there is no evidence that treatment of asymptomatic transient hypoglycemia offers any short-term or long-term benefit over no treatment (2,10).

CASE EXEMPLAR

Lisa (not her real name) is a 26-year-old primigravida at 39 weeks pregnant who presented in active labor to a busy tertiary care hospital. Her antepartal course had been uncomplicated. She progressed well in labor and experienced an uncomplicated birth of a 3,600-g healthy, term newborn female, Apgar score 9/9. To Lisa's delight, her newborn baby achieved latch-on and suckled well at 20 minutes of age. Lisa was thrilled at having initiated successful breastfeeding within the first postpartum hour.

Per hospital protocol, the baby was transported to the triage nursery for newborn evaluation at 1 hour of age; this included a routine glucose screen. The heelstick glucose level (via reagent strip Glucometer method) was 40 mg/dL. The baby was immediately given formula supplementation by the triage nurse as a routine intervention for hypoglycemia. Subsequent heelstick glucose measures were obtained every 30 minutes until the blood sugar level was greater than 45 mg/dL for 3 successive hours. When Lisa arrived at the nursery, she was greatly distressed to see the nurse formula-feeding her baby. Moreover, she was informed that her baby had a dangerously low blood sugar level that required further intensive testing and continued surveillance in the nursery. Lisa was overcome with acute fear and anxiety. How could this be? Just a few minutes earlier her baby was a healthy newborn; now, she was being treated for a pathologic condition.

This experience is repeated in hospitals across the country on a daily basis. Is there evidence to support the

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practice of routine glucose screening, both in the short and in the long term? Is a mother's choice to exclusively breastfeed to be ignored under these circumstances? How does this practice affect neonatal health? The authors conducted a review of the literature on the phenomenon of neonatal hypoglycemia and examined such issues as newborn metabolic adaptation, neonatal glucose testing methods, the effects of formula supplementation of breastfed infants, and the consequences of early maternal-newborn separation. They found that many babies are being unnecessarily tested for hypoglycemia and inaccurate blood glucose methods are often used, leading to unnecessary and potentially harmful interventions from a non-research-based clinical practice.

REVIEW OF THE LITERATURE

The earliest reporting of neonatal hypoglycemia in infants of nondiabetic mothers was in 1937 by Hartmann and Jaudon, the researchers who first observed that neonatal hypoglycemia occurred quite regularly during the first 4 or 5 days of life in the normal newborn (8,17). Over the next 50 years, most of the research on neonatal hypoglycemia was conducted on fasting infants, infants at risk, or infants of diabetic mothers. Unfortunately, past research has rarely focused on neonatal hypoglycemia in the normal, exclusively breastfed newborn (5). However, the more recent literature is replete with research data that clearly validates the normalcy of the common neonatal physiologic adaptive metabolic response, the inappropriateness of glucose monitoring of healthy breastfed neonates in the early neonatal period, and the harmful effect of this practice on parental well-being and successful establishment of breastfeeding. See Appendix. The negative sequelae associated with universal neonatal glucose screening make the practice incongruous with the endorsements of breastfeeding promulgated by the American College of Nurse-Midwives (18), AAP (10,19), WHO (5), and the United States Department of Health and Human Services (20).

To summarize, the literature provides clear evidence of a postnatal metabolic adjustment period that, when experienced by the healthy neonate, includes a self-limiting blood glucose nadir occurring at 1–2 hours of age. Research evidence concludes that this transient hypoglycemic condition is self-correcting, with the glucose concentration gradually increasing from 3 hours after birth and continuing over the subsequent days as the neonate adjusts from an absolute dependence on placental nutrition to metabolic and nutritional independence via intermittent enteral feedings and the use of several adaptive extrauterine metabolic mechanisms. Early and exclusive breastfeeding has been demonstrated in the literature to safely meet the nutritional needs of the newborn. The unfortunate cascade of interventions (maternal-infant separation, supplemental feedings, decreased frequency and duration of breastfeeds, increased medical surveillance) that follows the erroneous pathologic diagnosis of neonatal hypoglycemia leads to several negative short- and long-term sequelae, including impaired breastfeeding initiation, delay in lactogenesis, insufficient milk supply, early termination of breastfeeding, and a decrease in maternal confidence to adequately nourish her baby.

CONCEPTUAL FRAMEWORK

Sandelowski (21), offers a provocative and illuminating theory that describes technology dependency in health care as a "complex, culturally inscribed, and self-perpetuating phenomenon that may appear as the cause, consequence, or intervening process that is exhibited in patients and their caregivers." The reliance on devices and techniques to evaluate, satisfy, or resolve health-related needs or problems, results in many short- and long-term consequences, either intended (positive) or unintended (negative). The unintended consequences include those effects that are viewed as undesirable, unforeseen, or by-products of the dependency that result in further morbidity and mortality or far-reaching social outcomes. Some examples of unintended outcomes of technology dependency include: technogenic syndromes (the treatment becomes the disease), contraction of capabilities and choices (limiting human capability or replacing human functions), and defensive medical practices (technology substantiates responsible and lawful behavior).

An inherent property of technology dependency is the manifestation of the paradigm of care in which human beings have become objects for technical manipulation; experience is anatomized and self is separated from body. This anatomization of body/experience is not only an outcome of technology dependency, but also a process by which technology dependency perpetuates the technologic model of reality. Additionally, although machine-generated information is popularly considered more objective (by virtue of its quantitative nature) than human-generated information, it often results in creating as much uncertainty as it reduces. The illusion of certainty (and sometimes blatant uncertainty) that machine-generated information creates leads to further technologic measures to validate the uncertain technologically generated information. In this case, the outcome of technology dependency (uncertain results and information) is the creation of an increased requirement for and further dependency upon technology. Still another effect of dependence on technology as the only, the best, or most promising solution to health problems is the masking of the existence of other viable solutions in the prevention and treatment domains.

Sandelowski's theory (21) of technology dependency provides an appropriate conceptual framework for analyzing the inappropriate clinical practice of early glucose screening in term, healthy breastfed neonates (reliance on devices and techniques to resolve health-related needs). This practice captures the normal, self-limiting, self-correcting physiologic blood glucose nadir experienced by the healthy neonate within the first 3 postnatal hours; this, in turn, is often interpreted as pathologic neonatal hypoglycemia (the evaluation becomes the disease). The misdiagnosis of hypoglycemia leads to treatments and interventions that include formula supplementation, mother-baby separation, and further neonatal surveillance (unintended, undesirable outcomes of technology dependency). Early mother-baby separation, further newborn surveillance, and formula supplementation result in short- and long-term consequences including difficulties in breastfeeding initiation, delays in lactogenesis, impaired breastmilk production, early breastfeeding discontinuance rates, and lowered maternal confidence (by-products of technology dependency that result in further morbidity or far-reaching social outcomes).

While there are those care providers who would advocate liberal newborn screening policies to capture the rare child who does not fit the standard screening criteria, one must also consider the cardinal importance of "primum non nocere" ("first do no harm") to those thousands of normal newborns who will be screened under such liberal policies (22). Screening is a preliminary procedure, such as a test or examination, to detect the most characteristic signs of a disorder that may require further investigation (23). Glucose screening is a preliminary test of a large sample of the population to detect the concentration of glucose in a blood sample that may represent a disorder requiring further investigation (23). Screening tests applied to an entire population, in this case, all newborns, often produce a large number of false positive results, especially when performed without regard to risk factors. This leads to further testing and treatment, often unnecessary and potentially harmful. Care providers advocating for liberal screening practices in healthy newborns are basing their policies on a technology dependent rationale. Current evidence supports the time-honored practice of screening based on a thorough history, physical examination, and risk assessment of the newborn, along with a detailed discussion of findings and shared decision-making with the infant's mother.

DEFINITION OF NEONATAL HYPOGLYCEMIA

Current controversy and confusion exists over the definition of neonatal hypoglycemia or a "safe" level of blood glucose in the term, healthy newborn. See Table 2. At this time, there is no universally accepted biochemical definition of neonatal hypoglycemia and there have been no research studies conducted on healthy, appropriate for gestational age (AGA), breastfed newborns. AAP currently defines neonatal hypoglycemia as a blood glucose concentration, measured by Dextrostix, of less than 40 mg/dL (10).

TABLE 2
Comparison of Research Findings and Definition of Hypoglycemia (24,25)

Author	*Age of Neonate*	*Hypoglycemia*
Srinivasan (3) (1986)	0–3 hours	<35 mg/dL
	3–24 hours	<40 mg/dL
	>24 hours	<45 mg/dL
Heck (24) (1987)	0–24 hours	<30 mg/dL
	24–48 hours	<40 mg/dL
Tanzer (25) (1997)	0–24 hours	<30 mg/dL
	24–48 hours	<40 mg/dL
Cornblath and Schwartz (17) (1991)	0–6 hours	<25 mg/dL
	6–24 hours	<30 mg/dL
	>24 hours	<40 mg/dL

Other recent studies define hypoglycemia in term infants as a serum level (serum or plasma glucose levels are 10% to 15% higher than whole blood) of 40 to 45 mg/dL after the first 24 hours of life (26). Most management protocols for neonatal hypoglycemia implemented in hospital nurseries are based upon research conducted on sick, premature, or severely fasted newborns over 30 years ago (6). Today, the most frequent justification for universal routine glucose screening is the detection of hypoglycemia in an otherwise asymptomatic newborn. This approach stems from a concern over an association between neonatal hypoglycemia and neurodevelopmental sequelae. However, there is no evidence linking asymptomatic hypoglycemia with important neurodevelopmental abnormalities (5,17).

In 1995, an international satellite symposium of the 40th Annual Meeting of the Japan Society for Premature and Newborn Medicine met to conduct a 30-year follow-up discussion of neonatal hypoglycemia in light of the advances in basic knowledge in the neurosciences, and neonatal physiology and metabolism throughout the past three decades (27). The symposium participants concluded that there is no current agreement on the definition of neonatal hypoglycemia. In this review, Dr. Kiyoko Yamaguchi (Maternal & Perinatal Center, Tokyo Women's Medical College, Tokyo, Japan) reported that 11% of the hypoglycemic babies that were studied at this center (diabetic mothers comprised 29% of the cases) developed long-term neurodevelopmental sequelae. However, these newborns were also complicated by conditions including very low birth weight (VLBW), intrauterine growth retardation, perinatal asphyxia, and

mothers with hypertension. Furthermore, of those VLBW babies who were followed and reported in this review, the average developmental quotient at 1.5 and 2.5 years and IQ at 4 and 6 years revealed no significant difference between the control and hypoglycemic infants (27). WHO has concluded that symptomatic hypoglycemia is associated with neurodevelopmental sequelae; evidence for a causative link is weak (5). A long-term prospective controlled study is necessary to resolve the question of prognosis and relative risk for neurodevelopmental abnormality in neonatal hypoglycemia. At this time, no such study exists and only fragments of this objective have been reported (8).

CLINICAL APPLICATIONS

Diagnostic Methods

WHO recommends the glucose electrode system (YSI 2300 State Plus; YSI Inc., Yellow Springs, Ohio) as the preferred method of bedside neonatal glucose measurement (5). The most important advantages of this type of glucose analysis system are: 1) it provides a rapid assessment and accurate diagnosis, particularly in the "low end" glucose values of interest in the screening and diagnosis of hypoglycemia, 2) it contributes to a reduction in the amount of unnecessary treatments and interventions, and 3) it requires no further confirmatory laboratory analysis (however, posttreatment follow-up tests would still be done) (28,29).

There are additional methods of blood glucose measurement, including reductiometric, glucose oxidase, hexokinase, paper reagent strips, and other glucose electrode systems. Problems and limitations exist in many of these methods. When used for newborn glucose screening, the reagent strip methods (Glucometer, Chemstrip, Dextrostix, Glucostix) are prone to many errors (do not accurately measure "low end" glucose values), detecting only 85% of true cases of hypoglycemia and 75% of babies truly normoglycemic (2,5,8,17). In the absence of reliable bedside methods, accurate laboratory blood glucose measurements should be made for neonatal blood glucose monitoring and for the diagnosis and management of neonatal glucose hypoglycemia (2,8).

Who Should Be Screened?

Equally as important as employing an accurate diagnostic method is the close and careful observation of the infant for evidence of clinical manifestations of hypoglycemia. These symptoms include; tremors, irritability, jitteriness, abnormal high pitch cry, exaggerated Moro reflex, seizures, lethargy, limpness, hypotonia, cyanosis, apnea, irregular rapid respiration, hypothermia, temperature instability, vasomotor instability, poor suck, and feeding poorly or refusal to feed with previous history of feeding well (1,5,8,12,30). A careful history and risk assessment should be obtained to identify the presence of risk factors for neonatal hypoglycemia.

The healthy, term, AGA neonate should never be diagnosed and treated for hypoglycemia on the basis of a reagent strip method, but should be diagnosed based on a reliable point-of-care or laboratory assay (5). When glucose assessment is indicated for the symptomatic neonate, measurements should be made immediately and a medical consult obtained (1). For at-risk, asymptomatic neonates, measurements should be taken at 4–6 hours of age, preferably before a feed (5). Term, healthy neonates without symptoms should not be screened at all (1,5,10,12). Neonates more often are universally screened at 1–2 hours of age, serving to detect the normal, physiologic glucose nadir that is self-limiting, self-correcting, and requires no intervention. This early screen results in many false positives and the misdiagnosis of pathologic neonatal hypoglycemia (5).

Hypoglycemia Prevention

In addition to making an accurate clinical assessment and diagnosis of neonatal hypoglycemia, the midwife is in a pivotal position to assist in preventing or minimizing hypoglycemia in the healthy, term breastfed newborn. To begin, the midwife* must carefully assess the relative indications of administering intravenous glucose solutions to laboring women as research has correlated the administration of intravenous glucose to laboring women with neonatal hypoglycemia (25). WHO recommends limiting intravenous glucose to <10 g/hour to avoid the development of neonatal hyperinsulinism and subsequent iatrogenic neonatal hypoglycemia (5).

A simple but critically important protocol for immediate care of the newborn should be the mainstay of midwifery* practice. Preventive measures and management strategies for newborn hypoglycemia should include the following:

1) Early and exclusive breastfeeding is safe to meet the nutritional needs of the term, healthy, AGA neonate (5,12).
2) Healthy, term, AGA newborns who are breastfed early and on demand do not require blood glucose screening and need no supplementary food or fluids (12,19).

* Midwife as used herein refers to certified nurse-midwives (CNMs) and certified midwives (CMs) who are certified by the American College of Nurse-Midwives (ACNM) or the ACNM Certification Council, Inc. (ACC); midwifery refers to the profession as practiced by ACNM/ACC-certified midwives.

3) Thermal protection of the newborn: Dry newborn well and place directly against mother's skin (5,31).
4) Initiate breastfeeding as soon as the infant is ready: Put the infant to breast, if possible within 30 to 60 minutes (12,19); research has identified a "stage of readiness" to breastfeed at around 20 minutes following birth (32). Avoid any maternal-infant separation until successful latch-on has been achieved during this critical window of opportunity (33–35).
5) Encourage frequent suckling and exclusive breastfeeding: A 5% glucose solution (20 calories/dL) is a poor nutritional substitute for human colostrum, which contains 6.4% lactose, 3% fat, 2–3% protein, and 55 calories/dL (36). In addition, feeding glucose water in the immediate postnatal period provokes negative metabolic effects including increased insulin secretion, decreased glucagon secretion, and the delay of the natural gluconeogenesis and ketogenic homeostatic processes (12).
6) Feed baby at the earliest sign of hunger: Crying is a *late* sign of hunger (19).
7) Encourage cuddling: Holding an infant restricts infant crying and perspiring; it also preserves neonate's stored energy of glucose and fat, thus reducing the risk of developing hypoglycemia (37).
8) Careful history-taking, risk assessment, clinical observation, and physical examination are more important than glucose screening alone in expediting the appropriate management of the child (30).
9) Healthy, term, AGA breastfeeding neonates do not develop symptomatic hypoglycemia as a consequence of intermittent enteral feeds. These babies must be evaluated for underlying disease; an infant who does not feed or is lethargic may be ill (5).
10) Reagent paper strip methods of glucose determination should not be used for newborns as they have poor sensitivity and specificity in neonates (8).
11) When glucose screening is warranted, the sample should be obtained at 4–6 hours of age, before a feed, using reliable bedside or laboratory methods. The current recommendation is to maintain neonatal blood glucose levels above 40 to 45 mg/dL; values below 40 to 45 mg/dL require careful review of the neonate's condition and pediatric consult (may reflect an underlying illness rather than underfeeding) (5).
12) Immediate glucose measurements should be obtained in symptomatic, premature, SGA, infants of diabetic methods, or ill newborns and the foregoing guidelines do not apply (5).

IMPLICATIONS FOR FUTURE RESEARCH

Despite there being more than 800 research papers in the world literature on the subject of neonatal hypoglycemia, there remains a critical need for further research on the best method of hypoglycemia screening and the proper definition and diagnosis of neonatal hypoglycemia. In fact, there are few studies of healthy, term, AGA breastfed newborns. Although most authorities currently recommend maintaining the infant's serum blood glucose above 40 to 45 mg/dL after 24 hours of age (26), there is no current definition of a normal or "safe" level of blood glucose for the term, healthy neonate who is breastfed early and frequently in the postnatal period without glucose water or formula supplementation and who maintains normal thermoregulation. Furthermore, there is the suggestion in the research literature that glycemic adaptation in the neonate is actually a dynamic continuum, varying according to the availability of alternate fuels and protective metabolic substrates such as ketone bodies for brain metabolism. Breastfed babies have higher ketone body concentrations, enabling them to mount a greater ketogenic response to hypoglycemia (1,2,5,38).

The healthy, term breastfed baby *must represent the biologic norm.* However, there is little data on blood glucose and other metabolic substrates in these neonates. Therefore, a detailed study of the relationship between feeding patterns, breastmilk intake, substrate concentrations (including glucose), and patterns of neurodevelopment is urgently needed to identify the normal pattern of neonatal metabolic adaptation and define the normal and/or "safe" glucose level in the breastfed newborn.

CONCLUSION

Routine glucose screening of the term, healthy neonate is not an evidence-based clinical practice, and serves as a significant detriment to successful breastfeeding behaviors. As an important facilitator of labor, birth, and postpartum events, the midwife is placed in a unique position to routinely offer critical hypoglycemia prevention strategies, to identify and prevent unnecessary glucose screening, to educate other health care professionals on appropriate methods of glucose surveillance, to identify and seek proper medical evaluation for the at-risk neonate, and to defend the right of every term, healthy newborn to be exclusively breastfed.

This article was written in partial fulfillment of the requirements for the degree of Science with a Major in Midwifery in the Graduate Midwifery Program of the Philadelphia University. The authors gratefully acknowledge the contributions and skillful peer-editing provided by Carol Pugh, CNM, Julie Rice-Brewer, CNM, and Beth Grob, CNM of the on-line writer's academy. The authors also thank Bob Gleason of Yellow Springs Inc. for the tour of the facility and the

demonstration of the YSI 2300 Stat Plus glucose analyzer.

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APPENDIX

REVIEW OF THE LITERATURE

Authors	*Study Group*	*Study Focus*	*Findings*
Strinivasan, Pildes, Cattamanchi, Voora, and Lilien (3)	n = 60 (235 cord blood samples from fasted group) n = 284 (329 blood samples from fed infants)	redefine neonatal hypoglycemia in normal neonates	1) lowest glucose levels occurred 1–2 hours after birth 2) significant increase in glucose after 3 hours 3) early glucose nadir is self-limiting and self-correcting
Heck and Erenberg (24)	n = 64 (breastfed) n = 50 (bottle-fed)	define normal glucose values in first 48 hours of life; included comparison between breastfed and bottle-fed babies	1) defined hypoglycemia as serum glucose <30 mg/dL 1st day 2) defined hypoglycemia as serum glucose <40 mg/dL 2nd day 3) lower glucose levels in bottle-fed babies at 5–6 hours of age
Tanzer, Yazar, Yazar and Icagasioglu (25)	n = 35 (blood samples obtained at 1, 2, and 3 hours of age and before feed at 6, 14, 24, 36 hours of age)	define normal glucose values in first 48 hours of life; first research on neonates exclusively breastfed before 3 hours of age; examined the effect of IV dextrose administration to laboring mothers	1) defined hypoglycemia as serum glucose <30 mg/dL 1st postnatal day; <40 mg/dL 2nd postnatal day 2) confirmed expected glucose stabilization after 3 hours of age in exclusively breastfed neonates 3) correlated IV dextrose administration to laboring women with neonatal hypoglycemia
Yamauchi (37)	n = 38 (see below) n = 28 vaginal births; breast-fed 12 times on day 1 n = 10 cesarean section births; breastfed 6 times on day 1	first study to investigate the incidence of symptomatic and asymptomatic hypoglycemia in breastfed babies cared for by rooming-in; breastfed within 30 minutes after birth with early, frequent suckling	1) no reported case of symptomatic hypoglycemia (<40 mg/dL) in infants breastfed early, fed frequently, and cared for by rooming-in 2) lower blood glucose levels and greater weight loss in c/section babies (not rooming-in; supplemented with 5% glucose water)
Durand, Hodges, La Rock, Lund, Schmid, Swick, Yates and Perez (31)	n = 50 experimental group (n = 25); infants exclusively breastfed with skin-to-skin contact) control group (n = 25); infants removed from mother, placed under radiant warmer, bottle-fed	study relationship between breastfeeding, skin-to-skin contact, newborn thermoregulation, and glucose values	1) all infants maintained glucose levels >40 mg/dL 2) skin-to-skin contact resulted in higher neonatal temperatures than radiant warmer method 3) implied safety and efficacy of early,

APPENDIX
REVIEW OF THE LITERATURE (cont.)

Authors	*Study Group*	*Study Focus*	*Findings*
			exclusive breastfeeding, skin-to-skin contact on neonatal thermal and glycemic control
Righard and Alade (32)	n = 72 (all breastfed) separation group (n = 34); infant removed from mother's abdomen at 15–20 minutes after birth; returned to abdomen after 20 minutes of "routine" procedures contact group (n = 38); infant remained uninterrupted on mother's abdomen for 1 hour/or until breastfeeding achieved	study the effect of delivery room procedures (early maternal-newborn separation) on the success of the first breastfeeding encounter; proposed correct vs incorrect suckling patterns are dependent on delivery room routines and analgesic medication (meperidine) administered to mother in labor	1) all unmedicated infants in contact group sucked correctly 2) only 7 infants in separation group sucked correctly; 11 sucked incorrectly; 16 refused to suck 3) none of the sedated (meperidine) infants in either group sucked correctly 4) early, uninterrupted contact (until breastfeeding is achieved) plus avoidance of analgesic sedation (meperidine) = strong predictor of successful breastfeeding initiation
Elander and Lindberg (39)	n = 150 (see below) separated group n = 30 non-separated group n = 120	study the effect of maternal-infant separation during the first week of life for mild newborn illness on breastfeeding rates at 1, 2, and 3 months	1) significant decrease in breastfeeding rates of separated infants seen at 1 and 2 months; 50% decrease in rates of breastfeeding by 3 months of age in separated infants
Schutzman, Hervada, and Branca (40)	n = 136 (see below) exclusive breastfeeding n = 78 supplemented breastfeeding n = 58	examine the effect of supplementation of breastfeeding on onset of lactogenesis	1) delay of lactogenesis noted in supplemented group 2) onset of lactogenesis correlates with volume of supplementation
De Carvalho, Robertson, Friedman, and Klaus (41)	n = 44 (see below) control group n = 24; breastfed every 3–4 hours experimental group n = 20; breastfed frequently	study the effects of early, frequent, and unrestricted breastfeeding on infant milk intake and growth during 1st month	1) significant increase in milk production and infant weight gain in experimental group 2) newborn weight gain provides reassurance and raises maternal confidence level (fear of insufficient milk supply leading cause of early lactation termination)
Loughlin, Clapp-Channing, Gelback, Pollard, and McCutchen (42)	n = 94 (mother/baby couple) assessment at 2, 4, and 8 week office visit; babies evaluated (weighed and examined); mothers complete self-administered questionnaire	study characteristics and frequency of early breastfeeding termination (first 2 months after birth); identify indicators found	1) risk factors for early breastfeeding termination: formula supplementation, use of hospital formula discharge "gift" packs,

APPENDIX
REVIEW OF THE LITERATURE (cont.)

Authors	*Study Group*	*Study Focus*	*Findings*
		in at-risk mothers for early breastfeeding cessation	low maternal confidence, failure to contact provider prior to switching to formula feeding 2) most frequently cited reasons for formula supplementation: insufficient milk supply, infant hunger, poor infant weight gain
Reiff and Essock-Vitale (15)	n = 50 (hospital nursing staff questionnaire) n = 77 (interview of mothers at 2 weeks postpartum)	study influence of hospital nursing staff attitudes toward bottle and breastfeeding, hospital factors, and mother's perceptions of hospital routines on early breastfeeding behavior	1) hospital staff routines and nonverbal "modeling" of formula products (posters, gift packs, occasional formula supplement) were more influential than positive verbal counseling, teaching, and support provided by nursing staff in shaping early feeding choices 2) first time mothers most vulnerable in the breast-to-bottle group