IN THIS ISSUE:
DIABETES AND PREGNANCY

The Perinatal Times

SSM Maternity Care
St. Mary’s Health Center

Cardinal Glennon
SSM Cardinal Glennon Children’s Medical Center

SLUCare
The Physicians of Saint Louis University
THE PERINATAL OUTREACH PROGRAM

The Perinatal Outreach Program is a collaborative effort between SSM Maternity Care at St. Mary’s Health Center, SSM Cardinal Glennon Children’s Medical Center and Saint Louis University School of Medicine.

It is designed to improve outcomes for mothers and babies through educational programs and quality improvement activities for regional perinatal care providers in eastern Missouri and Southern Illinois.

The Perinatal Outreach Program is the designated Administrative Perinatal Center for Southern Illinois.

PERINATAL TIMES EDITORIAL BOARD

Connie Thompson, RNC, BSN, Editor

SSM Cardinal Glennon Children’s Medical Center
Ayoob Ali, MD, MPH
Glenn Barber, RNC, BSN
Robyn Gude, RN, MSN
Mary Hope, RN, BSN
William Keenan, MD
Patricia Oberkirsch

SSM St. Mary’s Health Center
Erol Amon, MD, JD
Kathy Canchola, RN, BSN
Gilad A. Gross, MD
Katie Kulaitis, RN, BSN
Thomas Myles, MD
Pam Randazzo, RNC, BSN
Sharon Rector, RNC, MSN
Judy Wilson-Griffin

FUNDING

Financial support for the The Perinatal Times is provided by SSM Health Care - St. Louis and the Illinois Department of Public Health.

LETTERS

The Perinatal Times welcomes comments on any of its articles and will consider such letters for publication. Suggestions for future topics of interest or announcements are encouraged.

Please send correspondence to:

The Perinatal Times
Connie Thompson, Editor
SSM St. Mary’s Health Center
6420 Clayton Road, Richmond Heights, MO 63317
connie_thompson@ssmhc.com

IN THIS ISSUE:

1. GLUCOSE CONTROL DURING LABOR MADE EASY it’s not as formidable as it sounds

3. FORMULARY FACTS: LEVEMIR DURING PREGNANCY a step forward in managing diabetes

4. INFANT OF A DIABETIC MOTHER a unique set of challenges for the clinical practitioner

6. MONITOR CORNER cervical ripening & labor induction due to complex history
Controlling glucose in labor for women with diabetes is not as formidable as it sounds. Understanding three basic principles that underlie this practical approach to glucose management is the key to success:

1. Assure adequate hydration and nutrition
2. Avoid high glucose values and large boluses of dextrose
3. Frequently monitor and adjust fluids to achieve the goal of keeping the maternal glucose level between 70 and 110 mg/dl.

LABOR IS WORK

Labor is work - work that is performed most efficiently with adequate hydration and nutrition. Total fluids in the range of 200-250 cc/hr result in faster labors. The cells that do the work need fuel. Most women require 6-10 grams per hour of glucose to avoid starvation ketosis, when catabolism of fat and muscle kicks in. No calculator is required: “D5” means 5 grams of dextrose per 100 cc. It is easy to do the math from there. Avoid the temptation to use only fluids without dextrose. The cells of women with diabetes need as much fuel as anyone else. Monitor for ketones in the urine to see if the laboring woman is getting enough nutrition for her task. If she is, the ketones should be negative.

MORE THAN DEXTROSE

Giving dextrose is not enough. Glucose needs to get into the maternal cells to meet her needs. This job is accomplished by insulin, either endogenous or prescribed, and insulin keeps the circulating glucose levels appropriate. High circulating levels of glucose during labor easily cross the placenta and in some ways can undo months of even excellent control. The high levels can ramp up the baby’s insulin production and lead to hypoglycemia in the nursery. In addition, a large supply of glucose, either from high circulating levels or from a large bolus, can be detrimental to the fetus in times of distress by leading to build up of byproducts of anaerobic glucose metabolism when oxygen is limited. Keep the maternal glucose in the target range.

Monitor glucose about every 2 to 3 hours during latent phase or cervical ripening; increase to hourly in active labor. An intravenous insulin infusion is the most common way to control glucose levels in labor. The correct rate of maintenance insulin infusion is the rate that keeps the glucose level in the same range from hour to hour. The correct incremental increase will allow an elevated glucose level to return to the target range within an hour. Fortunately, most women with diabetes in pregnancy can be maintained in the target range with one unit per hour, with incremental increases of one unit per hour for every 30 mg/dl above the target range. Some women with high insulin resistance will need 2 units per hour for maintenance and a tighter incremental increase. Very insulin-sensitive women (especially with type 1 diabetes) may only need 0.5 units/hr and a looser scale for increases. If those rates and incremental increases don’t quite do it, adjust the scales empirically to achieve glucose levels in the target range. For St. Louis area SSM hospitals, standard order sets are in the electronic record, which make ordering and altering these infusions easier. These order sets have recently been updated and will soon be released.

THERE ARE ALWAYS CAVEATS

1. An insulin infusion is not always required. Many diet-controlled gestational diabetics can maintain glucose values in labor on their own, but this cannot be assumed, so monitoring these patients is just as critical. Repeated subcutaneous injections of rapid-acting insulin may be an alternative to an intravenous infusion, but doses are difficult to guesstimate and absorption may be variable when sites of injection are exercised. Women using an insulin pump who present in active labor, may keep their glucoses stable and in the acceptable range throughout the entire labor using their pump basal rates. Similarly, women taking insulin who present in active labor may deliver before their insulin on board

(Continued on page 2)
dissipates and not require any more insulin during labor. Some may actually require extra glucose if their insulin peaks while they are NPO during active labor.

2. With long inductions and cervical ripening, meal coverage becomes an issue. Adjustments in an insulin infusion for the duration of the meal and the following hour can cover a meal, as will subcutaneous injections. Most clear liquids have calories, as well, that are quickly absorbed and cause a spike in the glucose level if not covered. Thinking ahead and common sense are the best allies in these atypical situations.

REFERENCES
References available on request.

ABOUT THE AUTHOR
Dr. Dorothea Mostello is a Saint Louis University Professor of Obstetrics and Gynecology, Department of Obstetrics, Gynecology and Women’s Health; a Maternal-Fetal Medicine specialist in the Maternal-Fetal Medicine Division and Director of the Diabetes-in-Pregnancy Program at SSM St. Mary’s Health Center, St. Louis, MO.

GLUCOSE CONTROL DURING LABOR - MADE EASY
(Continued from page 1)

NPH insulin, with or without rapid-acting insulin, had been considered standard of care in pregnancy when patients required insulin. Two basal insulins (glargine, Lantus® and detemir, Leveimir®) are widely used in non-pregnant patients as an alternative for NPH. Studies have shown that detemir does not differ significantly from regular human insulin with regard to activation of insulin-like growth factor-I (IGF-I). Stimulation of IGF-1 receptors increases the risk for excessive fetal growth. However, glargine has higher affinity to bind to IGF-I receptors than detemir thus decreasing the potential for glargine’s use in pregnancy. (1,2)

Detemir is a peakless, long-acting insulin with a duration of 24 hours, administered once or twice daily. Because of its peakless effects, fasting blood sugar (FBS) should be the primary target for adjustments. Detemir should not be mixed with other insulin and is stable at room temperature for 42 days after opening. (3) In contrast, NPH has a peak that can vary but occurs approximately around 4-12 hours and is generally split into two doses per day. (4)

Until recently, safety data was lacking for detemir during pregnancy; however, a recent randomized, open-label, controlled, multicenter trial published in October 2012 demonstrated similar effects of detemir and NPH during pregnancy. (5) This trial evaluated the efficacy and safety of insulin detemir compared to NPH in 310 pregnant women with Type 1 diabetes.

Mathiesen and colleagues randomized patients to either detemir or NPH plus insulin aspart with meals. Patients were included if they had a singleton gestation between 8 and 12 weeks or if they intended to become pregnant, were on insulin for at least 12 months prior to enrollment, and had A1c <8.0 at the beginning of the pregnancy. Insulin doses were adjusted to a pre-prandial plasma glucose goal of 72-108 mg/dL and a 2 hour postprandial glucose goal of <126 mg/dL. Goal A1c was defined as <6.0%.

No differences exist between groups in respect to baseline characteristics. In the primary outcome of A1c at 36 weeks, detemir was found to be non-inferior to NPH. However, FBS was significantly lower at 24 and 36 weeks gestation in detemir patients. No difference occurred with hypoglycemia, mean insulin dose, or weight gain between groups. Pregnancy and
neonatal outcomes were not presented in this manuscript but should be published later.

The study was open-labeled, therefore it could have been influenced by possible confounding factors. It is also important to note that the study was performed including patients with Type 1 diabetes, limiting its external validity in extrapolating to patients with gestational diabetes or type 2. However, it is likely that similar results would be seen in patients with gestational diabetes.

Unfortunately, initial dosing strategies for detemir have not been published. However, in non-pregnant patients, detemir can be initiated at bedtime doses of 0.1-0.2 units/kg/day. When used in non-pregnant patients, the total daily insulin dose is divided into 50% detemir and 50% rapid-acting insulin. The rapid-acting insulin is further divided to give 1/3 with each meal.

Detemir can now be considered an effective option for management of diabetes in pregnancy. Based on these findings, the FDA has changed detemir to pregnancy category B. (1) This is a major step forward in the management of diabetes in pregnancy.

EFFECTIVE RESULTS

<table>
<thead>
<tr>
<th></th>
<th>Determin (N=152)</th>
<th>NPH (N=158)</th>
<th>Between treatment Differences [95% CI], p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1c (5) at 36 weeks gestation</td>
<td>6.27</td>
<td>6.33</td>
<td>-0.06 [-0.21, 0.08], p=NS</td>
</tr>
<tr>
<td>FBS at 24 weeks gestation (mg/dL)</td>
<td>96.8</td>
<td>113.8</td>
<td>-16.92 [-30.1, -3.78], p=0.012</td>
</tr>
<tr>
<td>FBS at 36 weeks gestation (mg/dL)</td>
<td>85.7</td>
<td>97.4</td>
<td>-11.7 [-21.4, -2.2], p=0.017</td>
</tr>
<tr>
<td>Change in body weight (lbs)</td>
<td>25.3</td>
<td>24.2</td>
<td>p=NS</td>
</tr>
</tbody>
</table>

REFERENCES


ABOUT THE AUTHORS

Alicia B. Forinash, Pharm.D., BCPS, BCACP is an Associate Professor of Pharmacy Practice at the Saint Louis College of Pharmacy and an OB-Gyn/Maternal-Fetal Clinical Pharmacy Specialist at SSM St. Mary’s Health Center, St. Louis, MO.

Arezo Noormohammadi, Pharm.D. is a PGY2 Ambulatory Care Resident at the Saint Louis College of Pharmacy at SSM St. Mary’s Health Center.
In our society, where diabetes and obesity problems are increasing (no pun intended), we are seeing more and more infants born to diabetic mothers. This presents a unique set of challenges to the practitioner caring for these infants after birth. Improved maternal care during pregnancy may reduce some neonatal morbidities (hypoglycemia, polycythemia, hyperviscosity, intrauterine growth restriction), as well as fetal macrosomia with consequent birth trauma. The following is a review of the care of the infant born to a diabetic mother (IDM).

Macrosomia is the hallmark of diabetic pregnancy. Despite continued advances to normalize glucose concentrations in mothers, fetal overgrowth occurs more than in normal pregnancies. As glucose passes across the placenta via facilitated diffusion, maternal insulin does not cross. This requires the infant to produce its own increased insulin supply to utilize the glucose. This increase in insulin primarily stimulates adipose tissue. Brain and head growth tend to be spared, while shoulder size increases due to intrascapular fat development. IDMs have 50% more total body fat compared with infants of mothers with normal metabolism making them at increased risk for shoulder dystocia. They also have an increased abdominal girth due to hepatomegaly from insulin-driving glycogen storage.

Hypoglycemia occurs shortly after delivery, as the infant has been receiving a steady state of glucose from the placenta. Once the cord is clamped, the infant transitions to using its own glucose stored as glycogen or relying on glucose from oral intake. The insulin levels do not drop off immediately and the infant requires sufficient glucose to prevent hypoglycemia. The goal of management should be to deliver enough glucose to achieve normoglycemia while avoiding stimulation of the pancreas to produce more insulin.

Hypocalcemia has also been observed, and infants may be asymptomatic, jittery or rarely, develop seizures. Recent data may point to maternal diabetes causing urinary loss of magnesium, which blunts parathyroid hormone secretion causing neonatal hypocalcemia.

Respiratory distress syndrome (RDS) is another potential problem of IDMs. Fetal hyperinsulinemia impedes production and release of surfactant into the lungs. Previous inaccurate estimates of gestational age due to macrosomia resulted in preterm delivery with associated RDS.

IDMs are at increased risk for polycythemia and possible hyperviscosity syndrome in the neonatal period. Erythroid precursors tend to be sensitive to insulin which causes an increased number of RBCs in the circulation. Hyperviscosity resulting from polycythemia may cause renal vein thrombosis, stroke, and other organ damage.

Due to high red cell mass, hyperbilirubinemia is common in IDMs. In addition, macrosomic infants tend to be bruised at delivery, and resorption of that blood can lead to hyperbilirubinemia.

These infants are also at risk for cardiomyopathy, including thickened interventricular septum and left or right ventricular wall. Rarely, they present with aortic outflow obstruction sufficient enough to cause left ventricular failure. Most abnormalities resolve within the first year.

Finally, IDMs are at an increased risk for congenital malformations, such as cardiac anomalies, spinal agenesis-caudal regression syndrome, neural tube defects, and gastrointestinal and urinary tract anomalies. Neonatal small left colon is a transient finding in IDMs. They present with intestinal obstruction with a Hirschsprung-like radiograph; however, they have normal bowel innervation. Eventually, they develop normal function.

There should be a clear and organized plan for delivery. Standard NRP protocol should be initiated at every delivery. Practitioners should be prepared for a possible shoulder dystocia, with resultant birth injury, and/or asphyxia during a vaginal delivery. Care should be taken to properly document 1 and 5 minute APGAR scores and look at cord blood gases if asphyxia is a concern. Injury to the brachial plexus could cause problems from damage to the nerves of the arm to unilateral diaphragmatic paralysis if the phrenic nerve is affected. Respiratory distress syndrome (RDS) is a common complication so the practitioner should be ready to appropriately ventilate the patient with CPAP, PPV, or intubation as needed.

Signs and symptoms of hypoglycemia are nonspecific and infants may not be symptomatic. Symptoms include jitteriness, agitation, poor feeding, lethargy, seizures, apnea, grunting and sweating. A plasma glucose should be checked in infants of diabetic mothers, even in the absence of symptoms.

As the plasma glucose concentration drops rapidly after delivery, a glucose level should be obtained within 30 minutes after birth. Guidelines for the close glucose monitoring and
Screening and Management of Postnatal Glucose Homeostasis in Late Preterm and Term SGA, IDM/LGA Infants

[(LPT) Infants 34 - 36 6/7 weeks and SGA (screen 0 - 24 hours); IDM and LGA >34 weeks (screen 0 - 12 hours)]

**Symptomatic and <40 mg/dL --> IV glucose**

<table>
<thead>
<tr>
<th><strong>Birth to 4 hours of age</strong></th>
<th><strong>4 to 24 hours of age</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INITIAL FEED WITHIN 1 hour</strong></td>
<td><strong>Continue feeds q 2-3 hours</strong></td>
</tr>
<tr>
<td><strong>Screen glucose 30 minutes after 1st feed</strong></td>
<td><strong>Screen glucose prior to each feed</strong></td>
</tr>
<tr>
<td><strong>Initial screen &lt;25 mg/dL</strong></td>
<td><strong>Initial screen &lt;35 mg/dL</strong></td>
</tr>
<tr>
<td><strong>Feed and check in 1 hour</strong></td>
<td><strong>Feed and check in 1 hour</strong></td>
</tr>
<tr>
<td><strong>&lt;25 mg/dL</strong></td>
<td><strong>&lt;35 mg/dL</strong></td>
</tr>
<tr>
<td><strong>IV glucose</strong>*</td>
<td><strong>IV glucose</strong></td>
</tr>
<tr>
<td><strong>25-40 mg/dL</strong></td>
<td><em><em>Refeed/IV glucose</em> as needed</em>*</td>
</tr>
<tr>
<td><em><em>Refeed/IV glucose</em> as needed</em>*</td>
<td></td>
</tr>
</tbody>
</table>

**Target glucose screen ≥45 mg/dL prior to routine feeds**

*Glucose does = 200 mg/kg (dextrose 10% at 2 mL/kg) and/or IV infusion at 5-8 mg/kg per min (80 - 100 mL/kg per d). Achieve plasma glucose level of 40-50 mg/dL.

**ASYMPTOMATIC**

Symptoms of hypoglycemia include: Irritability, tremors, jitteriness, exaggerated Moro reflex, high-pitched cry, seizures, lethargy, floppiness, cyanosis, apnea, poor feeding.

Once the blood glucose has been stable for 24 hours, begin slowly decreasing the IV infusion by 1-2 mL/hr every 3-4 hours maintaining blood glucose concentrations > 60 mg/dL.

Of note, avoid higher concentrations of dextrose as this may cause worsening rebound hypoglycemia. It can also lead to dangerous increases in plasma osmolarity.

**REFERENCES**

Adamkin, David. AAP Clinical Report—Postnatal Glucose Homeostasis in Late-Preterm and Term Infants from Committee on Fetus and Newborn. 2011.


**ABOUT THE AUTHOR**

Dr. Jeffrey Cooper is a Fellow at Saint Louis University and SSM Cardinal Glennon Children’s Medical Center in the Perinatal and Neonatal Medicine Program.
A CASE STUDY BY SHARON RECTOR, RNC-OB, C-EFM, MSN

PRESENTATION
Ms. L is a 20 year old, G3P1011 who presented to labor and delivery at 39 weeks and 3 days gestation with a spontaneously ruptured bow of water, leaking green tinged amniotic fluid. Contractions on admission were mild and irregular occurring about every 4-8 minutes. The fetal heart rate (FHR) baseline was 140 bpm, with moderate variability, accelerations present and no decelerations (Category I). Her cervical exam on admission was 2 cm, 50% effaced, -3 station, moderate consistency and midposition with a cephalic presentation. She was admitted for expectant management of labor.

BACKGROUND
Ms. L was previously diagnosed with Bipolar II Disorder for which she had inconsistently taken her prescribed medications. She had a history of marijuana use with a positive urine drug screen for marijuana during the pregnancy. She was also diagnosed with GBS bacteriuria in the first trimester. Her labor progressed spontaneously, and she received an epidural for pain management. She was given penicillin G 5 million units IV followed by 2.5 million units IV every 4 hours. Approximately 1 hour after the epidural was placed strip #1 was observed. Her cervical exam was 4 cm, 50% effaced, -2 station, soft and midposition. Her blood pressure was 116/62 to 120/70 and her heart rate was 62-68 bpm. Strip #2 occurred approximately 15 minutes later. How would you interpret these tracings? What interventions are indicated?

INTERPRETATION AND DISCUSSION
In strip #1 the baseline FHR is 145 bpm, the variability is moderate, there are no accelerations and there are late decelerations (Category II). A late deceleration is defined as a gradual (onset to nadir ≥ 30 seconds) decrease of the FHR from the baseline associated with a uterine contraction. In most cases the onset, nadir and recovery of the deceleration occur after the beginning, peak and ending of the contraction, respectively. A late deceleration reflects disruption of maternal-fetal oxygen transfer from the environment to the fetus resulting in transient fetal hypoxemia (Tucker, Miller & Miller, 2009). Interventions that will maximize fetal oxygenation are indicated and might include maternal position change, intravenous fluid administration, reduction of uterine activity and oxygen administration. If the disruption of oxygen transfer is recurrent or sustained, it may progress to the stage of metabolic acidemia. The FHR during the decelerations is irregular, ranging from 80-90 bpm to 150-155 bpm, similar to the appearance of marked variability. Variability is a
characteristic of the FHR baseline, and as such it should not be used to describe decelerations. According to Tucker, Miller & Miller (2009) the appearance of variability during a deceleration has not been studied, and they discourage assigning undue significance to its occurrence. The moderate baseline variability in this strip is highly predictive of the absence of fetal metabolic acidemia.

In strip #2 the baseline FHR is 155 bpm, the variability is minimal, there is one acceleration near the end of the strip and there are recurrent late decelerations (Category II). Some might question the presence of late decelerations. They are subtle, the onset to nadir is about 25 seconds and the timing appears to be delayed. This doesn’t completely fit the definition of a late deceleration, but these characteristics don’t completely fit any of the definitions of the four types of decelerations. What do you do when a deceleration pattern doesn’t quite fit any of the definitions perfectly? Use your clinical judgment to most accurately label the deceleration pattern, continue appropriate interventions and observe closely. It is imperative that this type of tracing be reviewed and discussed by all members of the care team. Communication among care providers is paramount especially when there may be variation in the assessment, interpretation and plan of care.

OUTCOME

Interventions in this situation included lateral positioning, IV fluid bolus and the administration of oxygen. Within about 30 minutes the FHR returned to a baseline of 140 bpm, with moderate variability, occasional accelerations and no additional late decelerations. The contractions became more irregular with minimal change in her cervical exam. Oxytocin was administered and approximately 5 hours after initiation Ms. L was completely dilated. Shortly thereafter a baby boy was delivered vaginally with Apgar scores of 8 and 9 at 1 and 5 minutes respectively. He weighed 4200 grams. Mom and baby had a normal postpartum course and were discharged on day 2.

REFERENCE


ABOUT THE AUTHOR

Sharon Rector is an Education Consultant specializing in perinatal nursing at SSM Health Care in St. Louis.
NUTRITIONAL MANAGEMENT OF DIABETES & PREGNANCY
BY RITA A. CHRIVIA, RD, CSP, LD

With the rising rates of obesity in the United States and the resulting rise in diabetes, chances are you are seeing more mothers and infants affected by diabetes in pregnancy. Reported rates of diabetes in pregnancy range from 2-10% of all pregnancies with 88% of those due to gestational diabetes, another 8% due to Type 2 and 4% due to Type 1. (1) Poorly controlled diabetes before conception and during the first trimester can cause major birth defects in 5-10% of pregnancies and can result in spontaneous abortions in 15-20% of pregnancies. Poorly controlled diabetes in the second and third trimesters of pregnancy can result in excessively large babies, posing a risk to both mother and child during labor and delivery. Gestational diabetes is a risk factor for Type 2 diabetes in the mother; 5-10% of women diagnosed with gestational diabetes are found to have diabetes immediately after the pregnancy. An estimated 15-50% will develop diabetes in the decades following the affected pregnancy. (2)

The good news is that intensive treatment of diabetes in pregnancy is associated with outcomes similar to those in control populations. (3) For all types of diabetes in pregnancy, this involves medical nutrition therapy. Taking into account the total calories per day, the number of meals per day and the composition of those meals is important for reaching the goals of euglycemia, appropriate weight gain, and a healthy outcome for mother and child.

Although the carbohydrate content of the diet is the first priority, total nutrient content and total calorie intake cannot be ignored. A well balanced diet containing important sources of vitamins and minerals, and promoting appropriate weight gain, is the goal for any pregnancy.

Total calories per day should be based on the pre-pregnancy weight as a percent of ideal body weight. Women who are 80-120% of their ideal body weight are recommended to have intakes of 30 calories per kg or 14 calories per pound. For obese women (120-150% of ideal body weight or with a BMI greater than 27 kg/m2), 25 calories per kg or 11 calories per pound is the recommended intake. Women who are greater than 150% of their ideal weight are recommended to have intakes of 12-15 calories per kg and limit weight gain to no more than 8kg (17-18 pounds). (3) While weight loss during pregnancy is not recommended, a modest energy and carbohydrate restriction may be appropriate. (4) Avoidance of ketosis is important to allow adequate provision of glucose to the fetal brain. The diet should be culturally appropriate, containing 1-1.2 g protein per kg and at least 175 g carbohydrates per day. Total carbohydrates should be less than 45% of energy in women with gestational diabetes to prevent hyperglycemia. (5) The carbohydrates should be from complex sources with simple sugars and high glycemic index foods limited. Fiber intake of 25-30 g per day is recommended as is a low saturated fat intake of less than 10% total calories. (6)
Normal hormonal changes in pregnancy lead to decreased insulin sensitivity and increased insulin resistance. This can result in transient hyperglycemia after meals. Hypoglycemia between meals and at night can occur due to the continuous fetal draw of glucose. Women with preexisting diabetes should be encouraged to maintain consistency in the timing of their meals and to include at least a bedtime snack to prevent hypoglycemia. The recommended distribution of carbohydrates to prevent hyperglycemia or hypoglycemia is 10-20% at breakfast, 20-30% at lunch, 30-40% at dinner with up to 30% for snacks. Insulin resistance is highest in the morning due to increased cortisol levels. Adding protein to the morning meals and snacks may help combat hunger. Smaller, more frequent meals may also help combat hunger, reduce the mealtime carbohydrate load, reduce nausea and heartburn, and maintain a more stable postprandial blood glucose level. Unless contraindicated, the pregnant woman should be encouraged to participate in physical activity 30 minutes per day for a minimum of 3 times per week to improve glycemic control and general health. (5,6)

Involving a registered dietitian who is familiar with the components of medical nutrition therapy of diabetes during pregnancy will allow for an individualized meal plan that reflects treatment goals and takes the woman’s lifestyle into account. Barriers to compliance can also be addressed and the meal plan modified as needed for the best outcome for mother and infant.

Breastfeeding is recommended for women with preexisting diabetes and some data suggests it may decrease the risk of Type 1 diabetes in the infant. The woman with Type 1 diabetes who breastfeeds may experience hypoglycemia and should be encouraged to have a meal or snack available before or during feeds. Women diagnosed with gestational diabetes who breastfeed appear to have a lesser chance of developing Type 2 diabetes. Their children also have a decreased risk of childhood obesity and impaired glucose tolerance. (6) Postnatal follow up should include adapting the meal plan for breastfeeding and for achieving a more optimal weight if needed to minimize the risk of developing gestational diabetes in future pregnancies.

**REFERENCES**


**ABOUT THE AUTHOR**

Rita Chrivia is a Clinical Dietician at SSM Cardinal Glennon Children’s Medical Center in St. Louis, MO.
CONTINUING EDUCATION OPPORTUNITIES

Many continuing education opportunities, including traditional lectures, hands-on practice as well as online presentations, are available for perinatal professionals in eastern Missouri and southern Illinois. These are offered through SSM St. Mary’s Health Center, SSM Cardinal Glennon Children’s Medical Center, Saint Louis University School of Medicine and the Perinatal Outreach Program. Most programs offer nursing contact hours and/or CMEs.

For course calendars or more specific information on programs, please go to www.cardinalglennon.com and click on the “For Professionals” tab or call the Perinatal Outreach Program at 314-577-5317.