INTRAUTERINE GROWTH RESTRICTION
AN UPDATE ON CURRENT PRACTICES by Erin J. Dickert MD, MPH & Thomas Myles, MD

Intrauterine growth restriction (IUGR) is most often defined as fetal growth at less than the 10th percentile for gestational age. This corresponds to the postnatal small for gestational age (SGA) diagnosis defined as birth weight less than 10th percentile. These definitions do not distinguish between fetuses which are constitutionally small and those that are pathologically growth restricted. Fetal growth less than the 3rd percentile may be classified as severe IUGR.

Possible risk factors for IUGR include chronic maternal medical conditions, substance use (including tobacco), severe malnutrition, primary placental disorders, multiple gestation, fetal infection, genetic disorders, and teratogenic exposures. (ACOG Committee on Practice Bulletins, 2012) Associated maternal medical conditions include chronic hypertension, renal disease, restrictive lung disease, pre-gestational diabetes, (with evidence of vascular damage), cyanotic heart disease, antiphospholipid syndrome, collagen-vascular disease, and hemoglobinopathies.

Perinatal morbidity and mortality increase with decline in fetal weight. This is most apparent when fetal growth is less than 3rd percentile for age. (ACOG Committee on Practice Bulletins, 2012) This negative effect carries through intrapartum, neonatal and early childhood periods.

IUGR may be unrecognized in 1/3 of cases and up to 50% of cases are misdiagnosed. (ACOG Committee on Practice Bulletins, 2012) Screening for IUGR should be done at each prenatal visit with the measurement of fundal height. If there is clinical concern for IUGR based on risk factors and physical exam, it is important to obtain ultrasonographic evaluation of fetal weight. Asymmetric growth restriction, lagging of the abdominal circumference (AC) in comparison to the remainder of the fetus, is more concerning for pathologic etiology than symmetric growth restriction. AC growth lag is more predictive of fetal growth restriction than other ultrasonographic markers. (Chang TC, 1992)

Once IUGR has been recognized, it is important to identify any maternal or fetal risk factors which may have led to the condition. A detailed ultrasound exam for fetal anomalies (structural and functional) should take place. Fetal karyotype should be considered if growth restriction is early onset (second trimester), severe, or accompanied by structural anomalies or polyhydramnios. (Snijders RJ, 1993) If there is suspicion for viral (rubella, varicella, CMV) etiology, maternal serum IgG and IgM should be evaluated for seroconversion.

Bed rest has not been demonstrated to be efficacious to improve outcome. Supplementation of vitamins or minerals, maternal oxygen therapy, anti-hypertensive therapy, anti-coagulation therapy have not been shown to be effective for the treatment or prevention of IUGR. (ACOG Committee on Practice Bulletins, 2012)

HOW TO MONITOR?
Once IUGR is diagnosed, antenatal corticosteroid administration should be considered. Growth velocity should be followed with serial measures every 2-4 weeks beginning in the third trimester, as early as 26-28 weeks. (Society for Maternal-Fetal Medicine Publications Committee, 2012 Apr) A finding of severe IUGR should prompt consideration for inpatient management and maternal fetal medicine consultation.

Remote from term, there is not an established optimal monitoring regimen. The key is to identify the fetuses most at risk for in utero mortality or morbidity. Amniotic fluid volume should be followed, as oligohydramnios has value as a negative prognostic indicator. In addition, fetal well-being should be assessed weekly with BPP/modified BPP, NST, or contraction stress test. With a reactive NST, the fetus is less likely to suffer in utero demise. Nonreactive or abnormal NSTs are associated with acidosis, hypoxemia, or both.

(Continued on page 2)
Umbilical artery Doppler flow is not a screening tool for IUGR; however, it is useful in monitoring fetal well-being once the diagnosis is made and the fetus is viable. Weekly monitoring of umbilical artery Doppler flow reduces perinatal morbidity, as a normal flow pattern is reassuring for fetal well-being. In a recent review of 18 studies, a 29% reduction in perinatal deaths was demonstrated when Doppler flow was evaluated. (Grivell RM, 2012) Abnormal umbilical artery Doppler flow patterns (absent or reversed end-diastolic flow) are associated with poor perinatal outcome. Ongoing research is evaluating the utility of Doppler studies of the middle cerebral artery, ductus venosus, and maternal uterine arteries for the management of growth restriction. (Society for Maternal-Fetal Medicine Publications Committee, 2012 Apr)

WHEN TO DELIVER?

“The fetus should be delivered if the risk of fetal death exceeds that of neonatal death.” (ACOG Committee on Practice Bulletins, 2012)

The decision to deliver is often due to nonreassuring fetal status or cessation of fetal growth on ultrasound. At or near term, IUGR may be an indication for delivery based on clinical circumstances. However, in the recent DIGITAT study, minimal differences were noted in perinatal or neonatal outcomes between those pregnancies affected by IUGR at term based on differences were noted in perinatal or neonatal outcomes. (Boers KE, V. W. (2012). Neonatal Morbidity after Induction vs Expectant Monitoring in IUGR at Term: a Subanalysis of the DIGITAT RCT. American Journal of Obstetrics and Gynecology, 344.e1-7.


ABOUT THE AUTHORS

Dr. Erin Dickert is a Fellow at Saint Louis University in the Division of Maternal-Fetal Medicine and SSM St. Mary’s Health Center.

Dr. Thomas D. Myles is a Saint Louis University Professor of Obstetrics and Gynecology, Department of Obstetrics, Gynecology and Women’s Health, as well as the Director, Maternal-Fetal Medicine Outreach and Director, Fetal Evaluation and Treatment Center, SSM St. Mary’s Health Center, St Louis, MO.

WORKS CITED


FORMULARY FACTS

DRUG SHORTAGES BY LAURIE NIEWOEHNER, PHARM.D.

Now, more than ever, we’re seeing medication shortages in the United States posing challenges to the treatment we provide. These shortages can adversely affect drug therapy, delay medical procedures and result in medication errors. As health care professionals, we must develop aggressive strategies to address the shortages to provide safe, equivalent alternative therapy.

CONTRIBUTING FACTORS TO DRUG SHORTAGES:

• Raw materials unavailable – Many of the materials used in pharmaceuticals come from outside the U.S. If there are environmental or climatic changes, this can affect harvesting, storage, or transport of the product.

• Manufacturing difficulties- In the past, the sole manufacturer of a drug product has halted production in response to a FDA action concerning non-compliance with good manufacturing practices.

• Manufacturer discontinues production- One company may decide to halt production of medication due to profit margin. This places extra pressure on the remaining companies producing the medication.

• Natural disasters- Hurricanes and tornados can damage manufacturing facilities.

• Unexpected demand - Prescribing frequency of a medication can increase with new unlabeled uses, clinical practice changes, and disease outbreaks.

The following medications commonly used in obstetrics, have recently been affected by drug shortages:

Ketorolac (Toradol), butorphanol (Stadol), nalbuphine (Nubain), oxytocin, Duramorph, meperidine (Demerol), and ondansetron (Zofran).

DEVELOPING STRATEGIES FOR SHORTAGES:

1. Finding the cause of the shortage can provide clues to the duration.

2. Sequestering medication supply to pharmacy for more effective medication management. This may involve pharmacies drawing up the medication into a syringe to minimize wastage.

3. If shortage is likely to be long-term, alternative drug therapies need to be evaluated. See the ASHP website, listed below, for recommendations.

4. In some situations, pharmacies can utilize contract compounding firms to provide substitutions for drugs in short supply.

5. If alternative agents are used, complete staff education needs to take place discussing dosing, side effects, and monitoring. It is also important to discuss the differences between the unavailable product and alternate product.

RESOURCES FOR MANAGING DRUG SHORTAGES

FDA

www.fda.gov/cder/drug/shortages/default.html

Current and resolved drug shortages and drugs that are being discontinued.

ASHP

www.ashp.org/shortage

Product shortages, implications for patient care, alternate therapies, and estimated release dates.

CDC

www.cdc.gov

Vaccine shortages and recommendations for modifying immunization schedules.

ABOUT THE AUTHOR

Dr. Laurie Niewoehner is a clinical pharmacy specialist for maternal and neonatal medicine at SSM St. Mary’s Health Center, a member of SSM Health Care - St. Louis.
PULSE OXIMETRY SCREENING

For Critical Congenital Heart Defects by Ayoob Ali, MD, MPH, MBA, CPE, FAAP

Congenital heart disease is the most common birth defect and represents nearly 40% of all deaths caused by congenital anomalies. Critical congenital heart disease (CCHD), which encompasses the more severe forms, is present in 2.5 to 3 per 1000 live births.

Failure to diagnose CCHD early in life can result in high morbidity and mortality rates, because symptoms frequently present after closure of the pulmonary ductus arteriosus, after nursery discharge. Pulse-oximetry screening is a low-cost, painless, noninvasive test that increases the ability to identify newborns with CCHD before they clinically decompensate.

In September 2011, U.S. Department of Health and Human Services (HHS) approved adding Critical Congenital Heart Disease (CCHD) to the Recommended Uniform Screening Panel (RUSP).

The seven defects classified as critical congenital heart defects (CCHDs) are:
1. Hypoplastic left heart syndrome
2. Pulmonary atresia
3. Tetralogy of Fallot
4. Total anomalous pulmonary venous return
5. Transposition of the great arteries
6. Tricuspid atresia
7. Truncus arteriosus

The AAP has published strategies for the implementation of pulse oximetry screening. The highlights of screening implementation are as follows:

- The screening is targeted toward healthy newborn infants in the newborn nursery.
- Screening should be performed with motion-tolerant pulse oximeters.
- Screening should not be undertaken until 24 hours of life or as late as possible if early discharge is planned to reduce the number of false positive results.
- Oxygen saturations should be obtained in the right hand and one foot.
- Screening that has a pulse oximetry reading of ≥95% in either extremity with a ≤3% absolute difference between the upper and lower extremity would be considered a pass, and the screening would end.
- In effort to reduce false positives results, it is recommended that repeated measurements be performed in those cases in which the initial screening result was between 90-95% in either extremity or >3% difference between the right hand and foot.
- Infants with saturations <90% should receive immediate evaluation. It is important to note that the oxygen saturation thresholds for a positive screening result may vary at high altitude.
- In the event of a positive screening result, CCHD needs to be excluded with a diagnostic echocardiogram prior to discharge. This may warrant the use of telemedicine or transport of the infant to another institution for evaluation by a pediatric cardiologist. Infectious and pulmonary causes of hypoxemia should also be excluded.

References

http://www.cdc.gov/Features/CongenitalHeartDefects/


About the Author

Dr. Ali is an Associate Professor of Pediatrics at Saint Louis University and Neonatologist at SSM Cardinal Glennon Children’s Medical Center, St. Louis, MO.
A CASE STUDY BY SHARON RECTOR, RNC, MSN

BACKGROUND
The patient is a 39 year old, G5P2 2022 who presented to labor and delivery at 39 6/7 weeks gestation for cervical ripening and induction of labor due to gestational diabetes and a history of a deep vein thrombosis 5 years ago following a significant trauma. Her prenatal diabetes screen included a GCT result of 184 and was followed by a GTT with the results of 80/184/180/82. Her treatment included nutrition counseling and glyburide 5 mg every morning and 2.5 mg every evening. Anticoagulation therapy was recommended due to her history of DVT, but the patient declined. She reported smoking about ½ pack of cigarettes per day. Her obstetric history included two full term vaginal deliveries and two spontaneous miscarriages.

PRESENTATION
The patient’s admission vital signs included a BP of 132/78, HR 84, R 18 and T 98.4°. Her cervical exam on admission was 1 cm, long, floating, with medium consistency and posterior position resulting in a Bishop score of 2. Shortly after admission, electronic fetal monitoring revealed a fetal heart rate (FHR) of 140 bpm with moderate variability, occasional accelerations and no decelerations. The patient received 25 mcg of misoprostol (Cytotec) vaginally every 4 hours for 2 doses after which she was contracting regularly and gradually progressed into active labor. Her bedside glucose on admission was 165. An insulin infusion was initiated at 3 units/hour and the dose was titrated based on hourly glucose levels that ranged from 88-140.

Approximately 8 hours after the second dose of misoprostol, the patient was contracting every 2-3 minutes and was dilated 6-7 cm, 80% effaced at a 0 station. The FHR baseline was 145 bpm with moderate variability. A few FHR variable decelerations to 90-100 bpm lasting 30-40 seconds had been observed when the following tracings occurred. How would you interpret the tracings? What interventions are indicated? What do you think happened?

OUTCOME
In response to these tracings the patient was repositioned on her side, oxygen was administered at 10L/min per non-rebreather face mask, and a bolus of intravenous fluids was initiated.

The physician arrived at the bedside, and terbutaline 0.25 mg was administered IV. A vaginal exam was done, and the patient was found to be 6-7 cm, 80% effaced and 0 station. Three more similar decelerations occurred over the next 7-8 minutes, and the decision was made to perform an emergency cesarean section.

A baby girl was delivered and handed off to the pediatric team. Apgars were 8 and 9, and she weighed 3810 grams or 7 pounds 8 ounces. Venous cord blood gases included a pH of 7.20, CO2 56, BE -6.6 and HCO3 22.

Both mom and baby had a normal postpartum course and were discharged on day three.

DISCUSSION
The first strip demonstrates a prolonged deceleration to 60 bpm lasting approximately 3 minutes with a brief return to the 130-170 bpm range. This is followed by a variable deceleration to 75 bpm lasting approximately 90 seconds with a return to a baseline FHR of 145 bpm with moderate variability. Variable deceleration is defined as an abrupt (from onset to the beginning of the nadir ≤15 seconds) decrease in the FHR ≥15 bpm below the baseline lasting ≥15 seconds but <2 minutes (Macones et al., 2008). Intrauterine resuscitation measures were implemented as previously stated. The second strip includes two late decelerations defined as a gradual (onset to nadir ≥30 seconds) decrease in the fetal heart rate from the baseline associated with uterine contractions in which there is a delay in timing with the nadir of the deceleration occurring after the
peak of the contraction and in most cases the onset, nadir and recovery of the deceleration occur after the onset, peak and end of the contraction respectively (Macones et al., 2008). With the depth of these decelerations to approximately 70 bpm and the abrupt recovery to 145 bpm there was some debate as to whether these were late or variable decelerations. It was decided that there was a gradual onset with a delay in timing, and they were labeled as late decelerations. It is worth pointing out that whether they are late or variable, if they are recurrent with either minimal or moderate variability, it is a category II or indeterminate tracing. This is not predictive of abnormal fetal acid-base status. Because it cannot be classified as category I (normal) or category III (abnormal) it requires evaluation and continued surveillance and reevaluation, taking into account the entire associated clinical circumstances (Macones et al., 2008). In this particular situation the team felt that the interventions were not resulting in resolution of the pattern, and the decision was made to move to expeditious birth via a cesarean section.

REFERENCE

INTRODUCING THE ALL NEW RESOURCE ROUNDUP

The “Resource Roundup” is a new addition to “The Perinatal Times,” and one we’re really excited about. Here you’ll find major announcements from organizations that publish recommendations or guidelines which impact the care given to moms and babies. This includes organizations like AWHONN, ACOG, the FDA and more.

JANUARY 2012
AWHONN launches public campaign to encourage pregnant women to “Go the Full 40” in an effort to educate women on the importance of full-term pregnancies. Access AWHONN at www.awhonn.org. For patient education materials go to www.health4mom.org.

FEBRUARY 2012
ACOG releases Practice Bulletin #125 “Chronic Hypertension in Pregnancy.” Members, click to access ACOG online at www.acog.org.


MARCH 2012
AAP releases Policy Statement “Breastfeeding and the Use of Human Milk.”

FDA approves Surfaxin, a synthetic surfactant, for use in treatment of RDS. Go to FDA website at www.fda.gov.

JUNE 2012
ACOG releases Practice Bulletin #127 “Management of Preterm Labor.”

ANNOUNCEMENTS

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.

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If you would like future editions sent to you electronically, email Patricia Oberkirch at patricia_oberkirch@ssmhc.com to register with the Perinatal Outreach Program.