Video Case

IUGR

Objectives:

- → Define intrauterine growth restriction IUGR
- → Describe maternal, placental, and fetal causes of fetal growth restriction.
- → List methods of detection for fetal growth restriction.
- → Describe the prevention and management of fetal growth restriction.

- → Slides
- → Important
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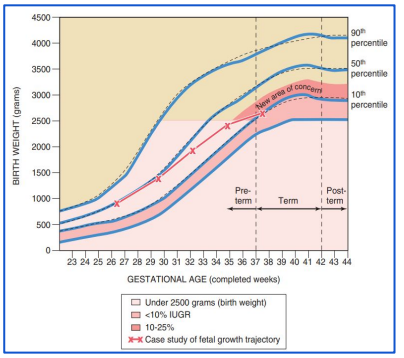
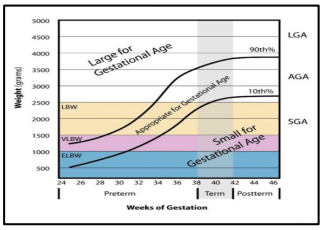
Female presentation

Video Case | Editing File

IUGR

Intrauterine Growth Restriction (IUGR) :





- **Common definition**: the birth weight of a newborn infant is below the 10th percentile for a given gestational age. This assumes the fetus is not growing to its genetic potential.
- **Low Birth Weight definition:** is <2,500 grams (5 lb, 8 oz). Clearly, neonatal morbidity and mortality are affected by lowering birth weight. However, 70% of these fetuses are constitutionally small.
- The terms small for gestational age (SGA), low birth weight (<2500 grams), and IUGR should not be used synonymously. The term SGA merely indicates that a fetus or neonate is below a defined reference range of weight for a gestational age, whereas IUGR (<10th percentile) refers to a small group of fetuses or neonates whose growth potential has been limited by pathologic processes in utero, with resultant increased perinatal morbidity and mortality
- **Significance**: is to identify infants who are at risk of implications "morbidity and mortality". Alteration of fetal growth may have "short-term" and "long-term" implications.
    - **Short-term** : Lack of adequate reserve to continue intrauterine or even undergo the stress of labor.
    - **Long-term** : Cardiovascular disease, insulin resistance, and Obesity.
- **Dating**: Accurate early pregnancy dating is essential for making the diagnosis. An early sonogram (<12 weeks) is most accurate if conception date is unknown, by measuring crown rump height. **Don't change gestational age based on a late sonogram.**

Types of Intrauterine Growth Restrictions

Early –Onset "symmetric" Growth Restriction (<32 w)	**Late-Onset "Asymmetric" Growth Restriction (>32 w)**
• Growth secondary to **hyperplasia**, irreversible, can cause a decrease in organ size and function. • Can lead to irreversible effect • Associated **maternal factors:** Infection, Smoking, multiple pregnancies, chronic maternal disease. • The commonest infection associated with IUGR is cytomegalovirus (CMV). Mother may have complained of flu-like illness. Fetus has sonographic findings compatible with CMV (e.g. microcephaly and cerebral calcification) • Etiology: Fetal **(decreased growth potential)**, i.e., aneuploidy, early intrauterine infection, gross anatomic anomaly.	• Growth secondary to **hypertrophy**, reversible. • More amenable to the restoration of fetal size with adequate nutrition. • better prognosis • Most likely cause is **uteroplacental insufficiency,** often associated with the development of **pre-eclampsia.** • Etiology: Maternal + Placental **(decreased placental perfusion)** due to **chronic maternal diseases** (hypertension, diabetes, SLE, cardiovascular disease) or **abnormal placentation** (abruption and infarction).

IUGR

Types of Intrauterine Growth Restrictions

Symmetrical IUGR	Asymmetrical IUGR
• **Head and abdomen both small** • All ultrasound parameters (HC, BPD, AC,FL) **are smaller than expected.** • head-to-abdominal circumference ratio may be normal, but the absolute growth rate is decreased, and estimated fetal weight is reduced. • Workup: detailed sonogram, karyotype, and screen for fetal infections. • Antepartum tests are usually **normal**.	• **Head normal; abdomen small** (the brain is preferentially spared at the expense of abdominal viscera especially the liver and pancreas) • When asymmetric growth restriction occurs, usually in the third trimester, the BPD is essentially normal, whereas the ratio of head to abdominal circumference is abnormal. • Amniotic fluid index is often decreased, especially if uteroplacental insufficiency is severe. • Monitoring is with serial sonograms, non-stress test, AFI, biophysical profile, and umbilical artery Dopplers

The principle differential diagnosis is:

1. Chromosomal abnormality or some other genetic problem.
2. Congenital infection.
3. Uteroplacental dysfunction.

Fetal Causes "Symmetric"	Placental Causes "Asymmetric"	Maternal Causes "Asymmetric"
• **Aneuploidy** (e.g., T21, T18, T13). • **Intrauterine Infection** (e.g., TORCH, listeriosis). • **Structural anomalies** (e.g., congenital heart disease, neural tube defects, ventral wall defects	• Primary placental disease (mosaicism, chorioangioma) • Infarction. • Abruption. • Win-twin transfusion syndrome (TTTS). • Velamentous cord insertion. • Essential or pregnancy-induced hypertension • Obesity	• Hypertension (e.g., chronic, preeclampsia). • Small vessel disease (e.g., SLE, long-standing type 1 diabetes). • Malnutrition (low BMI) or obesity. • **Tobacco smoking**, alcohol, street drugs. • Infections (viral, protozoal)

Risks on fetus:

- **Intrapartum** : fetal heart rate abnormalities, C-sec. , Low Apgar scores, Cord blood acidemia.
- **Neonatal**: Polycythemia, Hyperbilirubinemia, Hypoglycemia, Hypothermia, Apneic episodes.
- **Long term:** Largely dependent on the etiology of the IUGR and the gestational age at delivery, increased risk of cardiovascular disorders, insulin resistant, obesity.
- Fetal alcohol syndrome features: IUGR, microcephaly, microphthalmus, nasal hypoplasia

IUGR

Diagnosis:

- The key **screening tool** for fetal growth disorders in low-risk women is the assessment of **uterine size** by serial fundal height measurement and clinical palpation of estimated fetal weight Leopold maneuver.**Not accurate**
- **Ultrasonography** is the **gold standard to assess fetal weight** (by the growth parameters):
    - Biparietal diameter (BPD) "alone assess fetal size".
    - Head circumference (HC).
    - Femur length (FL) .
    - Abdominal circumference (AC): the single most effective parameter for predicting fetal weight because it is reduced in both symmetric and asymmetric IUGR.
    - Amniotic fluid volume
    - Calculated fetal weight

  All together are an estimate of fetal weight. Most formulas for estimating fetal weight incorporate two or more parameters.
  Indications: (1) the fundal height lags more than 3 cm behind expectations or (2) the mother has **high-risk** conditions such as preexisting hypertension, chronic renal disease, advanced diabetes etc..
- **Umbilical artery Doppler velocimetry** **very important in the management of IUGR** Abnormal umbilical Doppler (absent or reversed end diastolic flow) can help predict fetuses at increased risk of poor fetal outcome. Increase in S/D ratio.
- **Uterine artery systolic /diastolic (S/D):** evaluate the fetal-placental circulation, as placental resistance ↑ the diastolic flow ↓, therefore, there is an ↑ in S/D ratio.
    - **Absent / reversed end-diastolic flow predicts worse prenatal outcomes and it's usually an indicator for delivery.**
- **Middle cerebral artery (MCA Doppler):** It reflects fetal adaptation this is because the fetus always tries to spare the brain circulation. when there is a ↓ in placental perfusion, there is an ↑ in MCA flow. it's an indirect way to assess how much blood is going to the fetus. MCA normal = normal adaptation

437 note:
First we look at doppler of placental vessels then we look at doppler of MCA.Difference? MCA is a more advanced stage of fetal decompensation. We see what's happening at the placental level because if we have uteroplacental insufficiency that means there's resistance at placental level that's affecting blood flow to the fetus (1st stage), then the defensive mechanism is increased flow in MCA because the brain is spared (2nd stage).

Management:

Pre-pregnancy:

- An important part of **preventive medicine** is to anticipate the risk for women with a prior infant with IUGR, and to consider interventions before a woman plans her next pregnancy. Improving nutrition; stopping smoking, alcohol, and drugs; work stoppage; and bed rest may improve fetal growth.
- Assessment for evidence of **early cardiovascular disease** as a cause of the IUGR includes: hemoglobin A1c (HA1c), high-density lipoprotein (HDL), and C-reactive protein (CRP) (risk of diabetes, hypertension, and inflammation).
- For women with **antiphospholipid antibodies** and a past history of giving birth to an infant with IUGR, low-dose aspirin (81 mg/day) in early pregnancy may reduce the likelihood of recurrence.
- For patients with one of the **hereditary thrombophilias**, low-dose heparin (5000 U twice daily), with or without low-dose aspirin (81 mg/day), has also been shown to reduce the risk of recurrent IUGR.

IUGR

Antepartum care:

The goal is to expedite delivery before compromise and after lung maturation; This requires regular fetal monitoring with a twice-weekly non stress test (NST) and biophysical profile (modified: NST + AFI)

Fetal monitoring:

- Monitoring the growth-restricted fetus involves serial fetal measurement:

(Abdominal circumference - Amniotic fluid index - Cardiotocography - Doppler ultrasound)

- Fetuses with absent end-diastolic flow are **hypoxaemic**, these changes may appear up to 5 weeks before demise.
- Reversed end-diastolic flow is suggestive of **preterminal compromise**; the fetus may die within 1-2 days if not delivered.
- If fetal monitoring is normal do Ultrasonography. findings:
  - **Normal** growth → no clinical intervention.
  - **Abnormal**:
    - ➔ Strongly suggests IUGR → delivery is indicated at gestational ages of ≥34 weeks.
    - ➔ Assess pulmonary maturity by amniocentesis, but If severe oligohydramnios, delivery should be strongly considered without assessment of lung maturity.
    - ➔ These fetuses are at great risk of asphyxia, and the stress associated with IUGR usually accelerates fetal pulmonary maturity.
  - **Ambiguous**
    - ➔ Equivocal for IUGR → bed rest (w/ kick counting), fetal surveillance, and serial U/S measurements at 3-weekly intervals are indicated to avoid preterm delivery.

Amniotic Fluid Index:

Fetal urine production is significantly lower in the SGA fetus than in the AGA fetus. Decreased renal perfusion results in oligohydramnios AFI< 5

Biophysical exam:

-Breathing -Tone -Movement -Amniotic fluid volume -Cardiotocography

- Requires about 40 mins observation of fetal breathing movements. (Takes time not for every fetus only suspected IUGR)
- A persistently abnormal biophysical score is associated with the absence of end-diastolic flow.
- A score ≤ 4 indicates fetal hypoxia and/or placental insufficiency. In this case, labor should be induced.

437 note:

- Management depends on the GA.
- Once you confirm lung maturity and GR is progressive(baby not doing well) then management is delivery.
- **Preterm**: give dexamethasone first and continue to follow up the baby, once the baby is compromised(abnormal CTG, doppler or biophysical profile) then deliver. Choosing between c-section or induction of labor depends on OBY history, state of baby, cervical ripening and many other factors.
- **Imp: We don't give dexamethasone after 34 weeks.**

IUGR

Labor and after birth:

- The goal is to expedite delivery before compromise and after lung maturation.
- Our goal is to deliver the healthiest possible baby at the appropriate time.
- The timing of delivery should be based on the results of the antenatal testing, fetal growth pattern, dopplers, and gestational age.
- IUGR per se is not a contraindication to induction of labor , but there should be a low threshold to perform a cesarean delivery because of the poor capacity of the IUGR fetus to tolerate asphyxia.

After birth:

- **Examine**: the infant to rule out the possibility of congenital anomalies and chronic infections.
- **Monitor**:
  - ➔ **Blood glucose levels** because the fetuses do not have adequate hepatic glycogen stores, and hypoglycemia is a common finding.
  - ➔ **Hypothermia** as it is not uncommon in these infants.
  - ➔ **Respiratory distress syndrome** is more common in the presence of fetal distress because fetal acidosis reduces surfactant synthesis and release.

Macrosomia:

Fetus with estimated fetal weight (EFW) >90–95th percentile (Large for gestational age) Birth weight ≥4,000–4,500 grams.	
Causes	**Maternal**: History of macrosomia pregnancy, increase in pregnancy weight gain, multiparity, prolonged gestation >40 weeks , gestational diabetes, overt diabetes , obesity. **Fetal**: Male, beckwith-wiedemann syndrome, genetic
Risk factors	• Gestational diabetes mellitus. • Overt diabetes. • Prolonged gestation. • Increase in BMI (obesity). • Increase in pregnancy weight gain. • Multiparity. • Male fetus
Risks on mother and fetus	• **Maternal**: Postpartum hemorrhage (due to uterine atony), vaginal or perineal laceration, operative delivery, C-sec, Pelvic floor injury, puerperal infection. • **Fetal**:Shoulder dystocia, clavicular fracture,lower Apgar score, obesity later in life. • **Neonatal**: NICU (Neonatal intensive care) admission, hypoglycemia, Erb palsy.
Diagnosis	Fundal height measurement and clinical palpation of estimated fetal weight “however still poor”. U/S used to rule macrosomia out, “associated with a significant error in macrosomia”. Sonogram EFW. Accuracy in estimating birth weight is poor. Errors in prediction of EFW at term are ±400 grams.
Management	**Elective cesarean (if EFW >4,500 g in diabetic mother or >5,000 g in nondiabetic mother)** Or **Early induction**, but this may result in increased cesarean delivery rate due to failure of induction.

Teaching case

A 20 year-old G2P1 African-American woman is referred to you from her family physician for an obstetrics consultation. She is currently 35 0/7 weeks based on a certain **LMP with regular 28 day cycles**. At her last prenatal visit, her **fundal height measured 30 cm**. In taking her history about her prior delivery, she tells you that she delivered 3 weeks before her due date, but that her baby was small, **weighing 2400 grams** (average is 3.2-3.3 kg) . She does not report any other pregnancy complications. She **smokes** 2 packs of cigarettes a day and has gained 8 pounds during this pregnancy. Physical Exam: **BP 110/70** ; fundal height is 30 cm. Fetal heart tones are present.

- **Obstetrical Ultrasonography Report:**
  - Fetal number: Single (imp to know bc if fundal height is high it could be multiple gestation)
  - Position: Cephalic (affects fundal height. Eg: transverse lie)
  - Placenta: Anterior
  - Amniotic fluid volume: Normal[1]
- **Fetal biometry:**
  - BPD: 82.9 mm = 33.3 ± 3.1 weeks
  - HC: 299.7 mm = 33.2 ± 3.0 weeks
  - AC: 274.0 mm = 31.5 ± 3.0 weeks
  - FL: 58.0 mm = 30.3 ± 3.0 weeks
  - Humerus: 51.2 mm = 29.9 ± 2.8 weeks
  - Estimated fetal weight = 1700 ± 308 grams, **less than the 10th percentile** at 34.9 weeks.
  - Fetal Anatomy: Normal (to rule out any chromosomal or structural abnormalities)
  - Umbilical artery Doppler Flow: S/D ratio = 2.66 (normal) (to rule out uteroplacental insufficiency)

Q1: How do you interpret the ultrasound?

Based on the sonographic findings of parameters, “smaller than anticipated” the fetus qualifies for a diagnosis of IUGR.

Q2: What can you tell the patient is the possible etiology of the IUGR?

Important for OSCE

Maternal Factors	- Medical conditions (hypertension, renal disease, diabetes, vascular/autoimmune disease) all affect the blood vessels - Substance use and abuse (tobacco, alcohol, cocaine) - Infections (viral, protozoal) - Teratogen exposure
Fetal Factors	- Small constitutional size (genetic from the mother or father) - Genetic & structural abnormalities - Multifetal gestation
Placental Factors	- Primary placental disease (chorioangioma, mosaicism) - Abnormal placentation (previa, abruption, hematoma)

1: When there's inconsistency in fundal height we assess 3 things: fetal size, fluid volume, and space occupying lesions (eg. fibroids)

Teaching case

Q3: The patient asks you why the fetal growth problem was not detected earlier. What are the methods to screen and diagnose fetal growth disorders?

- Because she's a smoker and that's related to uteroplacental insufficiency that appears **late** in the pregnancy. Make sure the LMP is accurate!
- A review of the pregnant patient's medical and **obstetrical & menstrual history** is needed to determine whether she is at risk for abnormal fetal growth. Fetal anatomic survey will also screen for fetal and placental findings.
- The key screening tool for fetal growth disorders in low risk women is **assessment of uterine size** by fundal height measurement. However, fundal height assessment is not accurate as a diagnostic tool.
- **Ultrasonography** is the **gold standard** to assess fetal weight. (to confirm)
- **Umbilical artery doppler velocimetry:** (absent or reversed end diastolic flow) can help predict fetuses at increased risk of poor fetal outcome.

Q4: What would you tell the patient are the potential consequences of IUGR?

The primary concern regarding IUGR is an increase in perinatal morbidity and mortality. The risk of stillbirth is dependent on gestational age and the primary etiology. In addition, both intrapartum and neonatal complications may increased, depending on the gestational age at delivery.	
Intrapartum	- Increased risk of fetal heart rate abnormalities - Cesarean delivery - Low Apgar scores prematurity → NICU → may get infections or die - Cord blood acidemia
Neonatal	- Polycythemia - Hyperbilirubinemia - Hypoglycemia - Hypothermia - Apneic episodes
Longterm	- Lifelong increased risk of cardiovascular disorders

Q5: How would you approach managing this patient?

- Once IUGR is suspected/confirmed, **serial sonographic assessments** to monitor fetal growth is indicated every few weeks (10 days to 2 weeks) & CTG to look for decelerations & doppler.
- If the pregnancy is remote from term, periodic antenatal fetal testing is indicated (Biophysical profile [BPP], modified BPP, Non-stress test, are all acceptable).
- **The timing of delivery should be based on the results of the antenatal testing, fetal growth pattern, dopplers, and gestational age.**

441 doctor's notes

- IUGR nowadays called FGR = fetal growth restriction
- What is the difference between IUGR and low birth weight?
- IUGR: fetus inside uterus
- Low birth weight: after delivery
- We compare fetus weight to the chart that contain ideal number for each gestational week (make sure accurate GA and LMP)

- Why it's important? Lead to IUFD
- Two types :

1. Early onset <32w: permanent damage (كل سيء هنا اسوا)
2. Late onset>32: less severe, at the end of pregnancy

- What is the commonest cause for both types?
- uteroplacental insufficiency

- **Diagnosis:**
- fundal height
- Ultrasonography Is the gold standard looking mainly to Head circumference (HC) and Abdominal circumference (AC)
- Umbilical artery Doppler velocimetry: absent or reversed end diastolic flow
- uterine artery(not used nowadays)
- MCA Doppler: commonly use in isoimmunization(rare in IUGR)

- What is worst absent or reversed?

Reversed, we have to push her for delivery immediately regardless gestational week

- **Management:**

Give Low dose **aspirin** 81mg/day to any of the following: (now they give to any pregnant)

1. SLE
2. antiphospholipid syndrome
3. Prior history of IUGR proven due to placental insufficiency (سيء بعني ولدب وارسلوا المسبمه ولقوا السبب بحسر او اى سابى بعطوها)
4. HTN
5. placental insufficiency
6. No specific gestational age for delivery, any comprised to mother or fetus indicate delivery (الافصل بطل اكسر وقب بقدر بحلس فيه)

- **Macrosomia (big baby)**

Risk factor:

1. A chubby mother may deliver a chubby baby
2. Hx of macrosomia pregnancy(double)
3. gestational diabetes
4. Complication:
5. Maternal
6. Postpartum hemorrhage Due to uterine atony (لو احبك اسمى مبلك لبسب ملابسك بيوسع)
7. Laceration
8. Fetus: dystocia (nerve injury)
9. Management:
10. Council the mother give her option for c-section, if you deliver vaginally might be complications

Reference

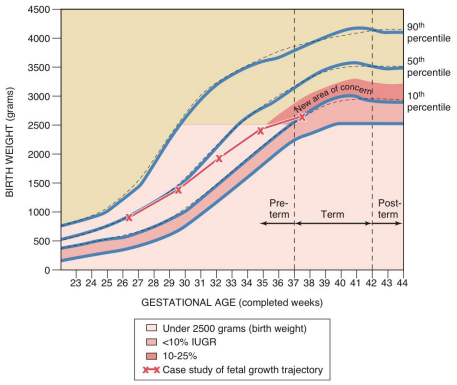


FIGURE 12-1 The birth weight/gestational age growth curve showing three different parameters that describe the characteristics of the fetus at birth. First, the *light pink color* shows that any birth weight below <2500 grams is defined as underweight (low birth weight [LBW]). The *medium pink color* defines those pregnancies <10th percentile and defines fetuses that are classified as intrauterine growth restricted (IUGR). The *dark pink color* defines a new area that defines a fetus that is not LBW but is IUGR based upon the birth weight/gestational aged growth curve. In addition it defines a group of fetuses who have had normal estimated fetal weight in early pregnancy, but because of complications during pregnancy (see case history in text) have poor fetal growth and become IUGR. This case is classic because it fits the criteria as defined by David Barker when he described the fetal origins for the risk of adult cardiovascular disease secondary to fetal programming in utero. (The birth weight-gestational curve modified using Oken E, Kleinman KP, Rich-Edwards J, et al: A nearly continuous measure of birth weight for gestational age using a United States national reference. *BMC Pediatr* 3:6, 2003.)

hormone, but many premature infants still develop RDS. Several human studies using instillation of surfactant into the pulmonary tree immediately postdelivery have shown dramatic improvements in lung mechanics and infant survival. A wide variety of surfactant preparations are now available, including synthetic surfactants and surfactants derived from animal sources.

Intrauterine Growth Restriction

Intrauterine growth restriction (IUGR) by definition occurs when the birth weight of a newborn infant is below the 10th percentile for a given gestational age. The terms small for gestational age (SGA), low birth weight (<2500 grams), and IUGR should not be used synonymously. The term SGA merely indicates that a fetus or neonate is below a defined reference range of weight for a gestational age, whereas IUGR (<10th percentile) refers to a small group of fetuses or neonates whose growth potential has been limited by pathologic processes in utero, with resultant increased perinatal morbidity and mortality (Figure 12-1 defines these parameters). **Growth-restricted fetuses are particularly prone to problems such as meconium aspiration, asphyxia, polycythemia, hypoglycemia, and mental retardation. They are at greater risk for developing adult onset conditions such as hypertension, diabetes, and atherosclerosis (see Barker hypothesis in Chapter 1).**

ETIOLOGY

The causes of IUGR can be grouped into three main categories: **maternal, placental, and fetal.** Combinations of these are frequently found in pregnancies with IUGR.

Maternal

Maternal causes include poor nutritional intake, cigarette smoking, drug abuse, early cardiovascular disease, hypertension, diabetes, obesity (associated with leptin resistance), alcoholism, cyanotic heart disease, and pulmonary insufficiency. In recent years, the **antiphospholipid syndrome** (autoantibody production) has been identified as a cause of IUGR in some women, both with and without hypertension. Antiphospholipid antibodies such as lupus-like anticoagulant and anticardiolipin contribute to the formation of vascular lesions in both the uterine and placental vasculature that may result in impaired fetal growth and demise. Recently, several **hereditary thrombophilias** have been identified, which have been associated with a greater risk for IUGR, abruption, and preeclampsia. These conditions result in vascular lesions within the spinal arteries supplying the placenta. Identification and treatment with low-dose heparin and low-dose aspirin have been shown to reduce the risk of IUGR.

Placental

This category is representative of circumstances in which there is inadequate substrate transfer because of placental insufficiency. **Conditions that lead to this state include essential hypertension, obesity (associated with leptin resistance which leads to placental dysfunction), chronic renal disease, and pregnancy-induced hypertension.** If the latter occurs late in pregnancy and is not accompanied by chronic vascular or renal disease, significant IUGR is unlikely to occur. A small fraction of cases may be attributable to placental or cord abnormalities (e.g., velamentous cord insertion).

Fetal

Examples of fetal causes include **intrauterine infection** (listeriosis and TORCH [toxoplasmosis, other infections, rubella, cytomegalovirus infection, and herpes simplex] agents) and **congenital anomalies. Chapter 22 reviews TORCH infections.**

CLINICAL MANIFESTATIONS

Two types of fetal growth restriction have been described: symmetric and asymmetric. In fetuses with symmetric growth restriction, growth of both the head and the body is inadequate. The head-to-abdominal circumference ratio may be normal, but the absolute growth rate is decreased. Symmetric growth restriction is most commonly seen in association with intrauterine infections or congenital fetal anomalies.

When asymmetric growth restriction occurs, usually late in pregnancy, the brain is preferentially spared at the expense of abdominal viscera. As a result, the head size is proportionally larger than the abdominal size. The liver and fetal pancreas undergoes the most dramatic anatomical and biochemical changes. When there is insufficient nutrition to the fetus, caused by either poor maternal nutrition or poor uterine blood flow, the fetal liver fails to store glycogen because of inadequate glucose from the mother. **Changes in the liver are now thought to play an important role in programming the fetus for a greater risk of obesity and diabetes later in life.** The fetal phenotype (small size) is known as the thrifty phenotype, but when born into an environment of plenty, there is increased risk of developing obesity, diabetes, and cardiovascular disease in later life (see Chapter 1).

BOX 12-2

FACTORS TO BE EVALUATED IN DATING A PREGNANCY

- Accuracy of the date of the last normal menstrual period.
- Evaluation of uterine size on pelvic examination in the first trimester.
- Evaluation of uterine size in relation to gestational age during subsequent antenatal visits (concordance or size-for-dates discrepancy).
- Gestational age when fetal heart tones are first heard using a Doppler ultrasonic device (usually at 12-14 weeks).
- Date of quickening (usually 18-20 weeks in a primigravida and 16-18 weeks in a multigravida).
- Sonographic measurement of fetal length (crown-rump) in the first trimester is most accurate.

DIAGNOSIS

Growth restriction may go undiagnosed unless the obstetrician establishes the correct gestational age of the fetus (Box 12-2), identifies high-risk factors from the obstetric data base, and serially assesses fetal growth by fundal height or ultrasonography. **Fetal or neonatal IUGR is usually defined as weight at or below the 10th percentile for gestational age.**

Serial uterine fundal height measurements should serve as the primary screening tool for IUGR. A more thorough sonographic assessment should be undertaken when (1) the fundal height lags more than 3 cm behind expectations or (2) the mother has high-risk conditions such as preexisting hypertension, chronic renal disease, advanced diabetes with vascular involvement, preeclampsia, viral disease, addiction to nicotine, alcohol, or hard drugs, or the presence of serum lupus anticoagulant or antiphospholipid antibodies.

Recently, interest has focused on the prediction of patients at risk for IUGR at mid-pregnancy. Patients with abnormal triple screens (α-fetoprotein [AFP], human chorionic gonadotropin [hCG], and unconjugated estriol [UE3]) who do not have abnormal fetuses by ultrasonography and amniocentesis may be at increased risk for IUGR. In addition, elevations of umbilical artery and uterine artery Doppler assessments (increased resistance) as early as mid-pregnancy have been associated with a greater risk of IUGR as pregnancy progresses.

At present, a number of sonographic parameters are used to diagnose IUGR: (1) biparietal diameter (BPD), (2) head circumference, (3) abdominal circumference, (4) femoral length, (5) amniotic fluid volume (6) calculated fetal weight, and (7) umbilical and uterine artery Doppler. Of these, **the abdominal circumference is the single most effective parameter for predicting fetal weight because it is reduced in both symmetric and asymmetric IUGR.** Most formulas for estimating fetal weight incorporate two or more parameters to reduce the variance of measurements.

Clinical Example of Fetal Growth Restriction

Using the standard fetal birth weight (vertical axis) and gestational age (horizontal axis) the fetal growth profile in a 34-year-old woman having her second pregnancy is illustrated in Figure 12-1. In this case the patient's first pregnancy was complicated by a fetus with IUGR at term. For the current pregnancy, her weight and blood pressure were normal with a body mass index of 30. At 20 weeks, the estimated fetal weight by ultrasound was at the 50th percentile and genetic markers were normal. Follow-up ultrasound at 26 weeks and 4 days showed a normal fetal weight at the 50th percentile, but by 29 weeks and 5 days, the estimated fetal weight decreased to the 25th percentile, at which time the maternal uterine artery vascular resistance was significantly increased and the fetal umbilical vascular resistance was normal. By 32 weeks, the fetal weight continued to decrease and both maternal uterine artery and the umbilical artery vascular resistance were abnormal, suggesting that this fetus was also going to develop IUGR. Ultrasonic measurements at 34 weeks and 3 days showed continued poor growth and by 37 weeks 3 days, the fetus was at the 10th percentile. Antenatal testing revealed an abnormal heart rate pattern (loss of fetal heart rate variability and a nonreactive fetal heart rate response to stimulation) and a reduced amniotic fluid index. The fetus was delivered by cesarean delivery with a birth weight of 2652 grams. Figure 12-1 illustrates the three different parameters that define how birth weight and gestational age are used to detect abnormalities of fetal growth.

As pregnancy advances, the head circumference remains greater than the abdominal circumference until approximately 34 weeks, at which point the ratio approaches one (see Figure 12-1). **After 34 weeks, the normal pregnancy is associated with an abdominal circumference that is greater than the head circumference.** When asymmetric growth restriction occurs, usually in the third trimester, the BPD is essentially normal, whereas the ratio of head to abdominal circumference is abnormal. **With symmetric growth restriction, the head-to-abdominal circumference ratio may be normal,** but the absolute growth rate is decreased, and estimated fetal weight is reduced (see Figure 12-1).

From 50-90% of infants with manifestations of IUGR at birth can be identified with serial prenatal ultrasonography. The accuracy depends on the quality of the assessments, the criteria used for diagnosis, and the effect of interventions applied when this diagnosis is made. For example, an improvement may be observed in fetal growth after interventions such as work stoppage, bed rest, dietary modification, and curtailment of the use of tobacco, hard drugs, and alcohol.

It is worthwhile to plot out each serial measurement on a standard growth curve. For example, a fetus measuring near the 10th percentile in mid-gestation may continue to grow along that curve (SGA) or, conversely, may fall well below the 10th percentile (IUGR) later in pregnancy (see Figure 12-1).

MANAGEMENT

Prepregnancy

An important part of preventive medicine is to anticipate the risk for women with a prior infant with IUGR, and to consider interventions before a woman plans her next pregnancy. **Improving nutrition and stopping smoking** are two approaches that should improve fetal growth. The patient should be assessed for evidence of early cardiovascular disease as a cause of the IUGR. The assessment should include measurement of biomarkers that define risk for cardiovascular disease such as hemoglobin A1c (HA1c), high-density lipoprotein (HDL), and C-reactive protein (CRP) (risk of diabetes, hypertension, and inflammation). **For women with antiphospholipid antibodies and a past history of giving birth to an infant with IUGR, low-dose aspirin (81 mg/day) in early pregnancy may reduce the likelihood of recurrence.** For patients with one of the hereditary thrombophilias, low-dose heparin (5000 U twice daily), with or without low-dose aspirin (81 mg/day), has also been shown to reduce the risk of recurrent IUGR.

Antepartum

Once a fetus has been identified as having decreased growth, attention should be directed toward modifying any associated factors that can be changed. Because poor nutrition and smoking exert their main effects on birth weight in the latter half of pregnancy, **cessation of smoking and improved nutrition can have a positive impact.** The working woman who becomes fatigued is more likely to have a low-birth-weight infant. **Work leave, or in some cases of maternal disease, hospitalization, will increase uterine blood flow** and may improve the nutrition of the fetus at risk.

The objective of clinical management is to expedite delivery before the occurrence of fetal compromise, but after fetal lung maturation has been achieved. This requires regular fetal monitoring with a twice-weekly nonstress test (NST) and biophysical profile. Most institutions use a modified biophysical profile that includes an NST and AFI. The oxytocin challenge test (OCT) is rarely used because its false-positive rate approaches 50%.

Fetuses clinically suspected of IUGR could be approached as follows:

1. For cases in which results of fetal monitoring are **normal** and ultrasonic findings strongly suggest normal growth, **no clinical intervention** is warranted.
2. **When ultrasonic findings strongly suggest IUGR, delivery is indicated at gestational ages of 34 weeks or later only if abnormal fetal surveillance indicates an increased risk of fetal death. Pulmonary maturity should normally be documented by amniocentesis.** In the presence of severe oligohydramnios, amniocentesis may not be feasible, so delivery should be strongly considered without assessment of lung maturity. These fetuses are at great risk of asphyxia, and the stress associated with IUGR usually accelerates fetal pulmonary maturity.
3. For those cases in which ultrasonic findings are **equivocal** for IUGR, **bed rest, fetal surveillance, and serial ultrasonic measurements at 3-weekly intervals** are indicated to avoid preterm delivery

Assessment of fetal movements (kick counts) each evening while resting comfortably on the left side is a simple technique whereby a pregnant woman can help in the assessment of fetal well-being. If 10 movements are not perceived in 1 hour, a biophysical assessment should be arranged. Some providers instruct their patients, irrespective of their risk, to begin a fetal kick count chart at 28 weeks.

Doppler-derived umbilical artery systolic-to-diastolic ratios are abnormal in IUGR fetuses. Fetuses with growth restriction tend to have increased vascular resistance and to demonstrate low, absent, or reversal of diastolic flow. This noninvasive technique can be used to evaluate high-risk patients, and may help in the timing of delivery when used in conjunction with the modified biophysical profile (see Chapter 7 for more information about Doppler assessment of fetal well-being).

LABOR AND DELIVERY

IUGR per se is not a contraindication to induction of labor, but there should be a low threshold to perform a cesarean delivery because of the poor capacity of the IUGR fetus to tolerate asphyxia. As a result, **during labor, these high-risk patients must be electronically monitored to detect the earliest evidence of fetal distress.**

A combined obstetric-neonatal team approach to delivery is mandatory because of the likelihood of neonatal asphyxia.

After birth, the infant should be carefully examined to rule out the possibility of congenital anomalies and chronic infections. **The monitoring of blood glucose levels is important, because the fetuses do not have adequate hepatic glycogen stores,** and hypoglycemia is a common finding. Furthermore, **hypothermia is not uncommon** in these infants. **Respiratory distress syndrome is more common in the presence of fetal distress,** because fetal acidosis reduces surfactant synthesis and release.

PROGNOSIS

The long-term prognosis for infants with IUGR must be assessed according to the varied etiologies of the growth restriction. If infants with chromosomal abnormalities, autoimmune disease, congenital anomalies, and infection are excluded, **the short-term outlook for these newborns is generally good.** However, poor fetal growth in utero increases the risk for chronic conditions such as hypertension and diabetes later in life (see Chapter 1).

Postterm Pregnancy

The prolonged or postterm pregnancy is one that persists beyond 42 weeks (294 days) from the onset of the last normal menstrual period. **Estimates of the incidence of postterm pregnancy range from 6-12% of all pregnancies.** The incidence of postterm pregnancies has been reduced significantly in the past 10 years because induction before 42 weeks has significantly reduced fetal morbidity secondary to prolonged gestation.

Perinatal mortality is two to three times higher in these prolonged gestations. Much of the increased risk to the fetus and neonate can be attributed to development of the **fetal postmaturity (dysmaturity) syndrome, which occurs when a growth restricted fetus remains in utero beyond term.** Occurring in 20-30% of postterm pregnancies, **this syndrome is related to the aging and infarction of the placenta,** with resulting placental insufficiency. Some of these fetuses meet the criteria for having IUGR and should not have been allowed to advance to term. If there is any evidence of intrauterine hypoxia (such as meconium staining of the umbilical cord, fetal membranes, skin, and nails), perinatal mortality is even further increased.

The fetus with postmaturity syndrome typically has loss of subcutaneous fat, long fingernails, dry, peeling skin, and abundant hair. The 70-80% of postdate fetuses not affected by placental insufficiency continue to grow in utero, many to the point of **macrosomia (birth weight greater than 4000 g). Macrosomia often results in abnormal labor, shoulder dystocia, birth trauma, and an increased incidence of cesarean delivery.**

ETIOLOGY

The cause of postterm pregnancy is unknown in most instances. Prolonged gestation is common in association with an anencephalic fetus. This is probably

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