A Uniform Management Approach to Optimize Outcome in Fetal Growth Restriction

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KEYWORDS

• Fetal growth restriction • Fetal acidemia • Fetal Doppler • Umbilical artery
• Middle cerebral artery • Biophysical profile score • Neonatal outcome • Fetal testing

KEY POINTS

- A uniform approach to diagnosis and management of fetal growth restriction (FGR) produces better outcomes, prevents unanticipated stillbirth, and allows appropriate timing of delivery.
- An estimated fetal weight less than the tenth percentile in association with either an elevated umbilical artery Doppler index, a decreased middle cerebral artery Doppler index, or a decreased cerebroplacental ratio should be considered evidence of FGR. Early-onset and late-onset FGR represent two distinct clinical phenotypes of placental dysfunction.
- Integration of different testing modalities allows adjustment of monitoring intervals based on Doppler parameters and a more precise prediction of acid-base status based on biophysical variables.
- Antenatal surveillance of the growth-restricted fetus requires adjustment of monitoring intervals based on signs of disease acceleration, when delivery is not yet indicated.
- Thresholds for interventions are defined by the balance of fetal risks of continuation of pregnancy versus the neonatal risks that follow delivery and depend on gestational age.

INTRODUCTION

The main challenges in the management of pregnancies complicated by fetal growth restriction (FGR) are accurate identification of the small fetus at risk for adverse outcome, prevention of unanticipated stillbirth, and appropriate timing of delivery. A

Authors declare no relationship with a commercial company that has a direct financial interest in the subject matter or materials discussed in the article or with a company making a competing product.
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Obstet Gynecol Clin N Am 42 (2015) 275–288
http://dx.doi.org/10.1016/j.ogc.2015.01.005 **obgyn.theclinics.com**
0889-8545/15/$ – see front matter

uniform management approach to diagnosis and management of FGR consistently produces better outcome than is reported in observational studies that rely on a range of diagnostic, surveillance, and delivery criteria.[1–5] Once the diagnosis of FGR has been made, surveillance tests need to be applied at appropriate intervals until the relative risks of delivery outweigh the benefits of ongoing monitoring. These factors are determined by the clinical phenotype of FGR across gestational ages.

CLINICAL PHENOTYPE OF FETAL GROWTH RESTRICTION IN RELATION TO GESTATIONAL AGE

FGR evolves from a preclinical phase to clinically apparent growth delay and may eventually lead to fetal deterioration before the spontaneous onset of labor. Growth delay due to decreased nutrient delivery affects liver size and therefore the abdominal circumference (AC) first, and then growth of the head and entire body.[6] Abnormal placental perfusion in the maternal compartment results in increased blood flow resistance in the uterine artery flow-velocity waveform.[7] Abnormal perfusion of the fetal villous vascular tree is associated with decreased umbilical artery (UA) end-diastolic velocity proportional to the degree of flow impairment.[8] Abnormal oxygen diffusion across the villous membrane leading to lower fetal arterial PaO_2 is associated with a decrease in middle cerebral artery (MCA) blood flow resistance,[9] whereas decreased CO_2 clearance additionally increases the MCA peak systolic velocity (**Fig. 1**).[10] The relative predominance of these mechanisms determines the clinical picture of FGR.[11–16]

FGR that is established by the second trimester is associated with a greater degree of vascular abnormality in the maternal and fetal compartments of the placenta. In the mother, high-resistance uterine artery flow velocity waveforms and a 40% to 70% rate of associated pre-eclampsia are characteristic. In the fetal compartment, an elevation

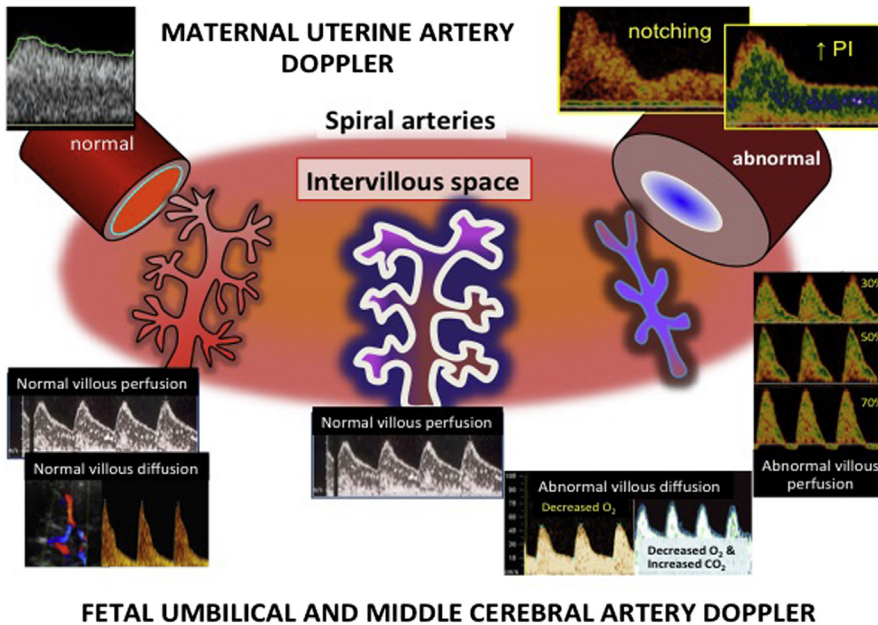

Fig. 1. Clinical correlates of maternal and fetal aspects of placental function.