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Diagnosis and management of fetal growth restriction: the role of fetal therapy

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Fetal growth restriction remains a major cause of perinatal morbidity and mortality in modern obstetric practice. Placental insufficiency is the most common association, but is often a diagnosis of exclusion. Currently, no treatment can ameliorate or reverse established growth restriction: maximising gestational age and judicious timing of steroid administration and delivery are the primary tasks for the obstetrician. Although comprehensive surveillance of the preterm fetus now includes ductus venosus Doppler studies, its effectiveness in timing delivery has yet to be confirmed in randomised controlled trials. More basic research on the regulation of fetal growth is needed before specific therapies for established growth restriction can be developed.

Key words: ductus venosus; fetal growth retardation; fetal monitoring; small-for-gestational-age; ultrasonography, prenatal; umbilical arteries.

INTRODUCTION

Fetal growth restriction (FGR) is a term used to describe a pathological limitation to the growth of a fetus, implying a failure to reach a given size potential due to adverse genetic or environmental factors. The clinical significance of FGR relates to the dramatically increased perinatal mortality, with mortality being eight times higher when weight is below the 10th percentile and nearly 20 times higher when weight is below

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the 3rd centile.[1] When associated with prematurity, outcomes are even worse with survival figures of less than 50% for gestations less than 28 weeks.[2,3]

In addition to increased mortality, both short- and long-term morbidity are increased in the growth-restricted fetus. Neonatal complications include birth hypoxia[4,5] and, when combined with prematurity, increased risks of respiratory distress syndrome, necrotising enterocolitis, retinopathy of prematurity, infection and hypoglycaemia.[5,6] Evidence is also accumulating to suggest that there are long-term metabolic consequences growth restriction, such as increased risks of insulin resistance, cardiovascular complications and psychiatric disorders.[7,8]

As a result of these effects on perinatal morbidity and mortality, accurate diagnosis and management of the growth-restricted fetus remains one of the most important goals of antenatal care.

DEFINITIONS

Various growth percentile thresholds for defining small-for-gestational-age (SGA) babies have been used; the most commonly accepted standard is the 10th centile.[9] However, babies below the 10th centile, which by definition make up about 10% of the population, are a heterogeneous group, including the constitutionally small normal babies and those with true growth restriction with the accompanying increase in morbidity and mortality. As the majority of SGA infants will have no increased morbidity or mortality, distinguishing between the healthy, normal $<$ 10th centile fetus and the truly growth-restricted compromised fetus is important if medical resources are to be targeted appropriately and if unnecessary interventions, such as iatrogenic prematurity, are to be avoided.

CAUSES OF FETAL GROWTH RESTRICTION

The causes of FGR can be divided into those two broad groups: (1) intrinsic fetal factors reducing growth potential, such as an euploidy, genetic syndromes and congenital infections; and (2) those affecting the transfer of adequate nutrients and oxygen to the fetus. These include processes originating in the placenta, such as the endothelial dysfunction associated with preeclampsia; and maternal factors such as severe undernutrition and maternal drug use and smoking (Box 1).[10] Of all the maternal risk factors,

Box 1. Risk factors for fetal growth restriction

Maternal factors

- Low ($<$ 50 kg) pre-pregnancy weight
- Cigarette smoking
- Substance abuse
- Severe anaemia
- Maternal hypoxia
- Previous growth-restricted baby
- Recurrent miscarriage