

Intensive Care Nursery House Staff Manual

Intrauterine Growth Retardation

CLINICAL SIGNIFICANCE: Intrauterine growth retardation (IUGR) affects 3-10% of pregnancies; 20% of stillborn infants have IUGR. Perinatal mortality rates are 4-8 times higher for growth retarded infants, and morbidity is present in 50% of surviving infants.

DEFINITIONS and CLASSIFICATION:

- AGA**, appropriate for gestational age: Birth weight is between 10th and 90th percentile for infant's gestational age (GA).
- LGA**, large for gestational age: Birth weight >90th percentile for GA.
- **SGA**, small for gestational age: Birth weight <10th percentile for GA. Other definitions are sometimes used for SGA, including <3rd percentile for GA or more than 2 S.D. below the mean.
- IUGR vs. SGA:** IUGR refers to deviation and reduction in expected fetal growth pattern. Multiple adverse conditions inhibit normal fetal growth potential. Not all IUGR infants are SGA.

ASYMMETRIC vs. SYMMETRIC GROWTH RETARDATION: Most growth retarded infants have asymmetric growth restriction. First there is restriction of weight and then length, with a relative "head sparing" effect. This asymmetric growth is more commonly due to extrinsic influences that affect the fetus later in gestation, such as pre-eclampsia, chronic hypertension, and uterine anomalies. Postnatal growth after IUGR depends on cause of growth retardation, postnatal nutritional intake, and social environment. Symmetric growth retardation affects all growth parameters. In the human brain, most neurons develop prior to the 18th week of gestation. Early gestational growth retardation would be expected to affect the fetus in a symmetric manner, and thus have permanent neurologic consequences for the infant. Examples of etiologies for symmetric growth retardation include genetic or chromosomal causes, early gestational intrauterine infections (TORCH) and maternal alcohol use.

CAUSAL FACTORS:

A. Maternal

- Before pregnancy:**
 - Prepregnancy weight influences fetal size
 - Periconceptual nutritional status can affect embryogenesis (*e.g.*, folate deficiency).
- During pregnancy:** Factors that may adversely affect fetal growth include:
 - Low pre-pregnancy weight and small maternal size
 - Recent pregnancy and/or high parity
 - Poor weight gain during pregnancy, especially in latter half
 - Chronic illness - such as malabsorption, diabetes, renal disease
 - Inadequate or poorly balanced intake associated with alcoholism, drug abuse, poverty, adolescence, anorexia nervosa, food faddism
 - Maternal drug and alcohol use also influence maternal nutrition.
 - Decreased O₂ availability to fetus (*e.g.*, high altitude, severe maternal anemia)

- B. Uterine and placental factors** that can adversely affect fetal growth include inadequate placental growth, uterine malformations, decreased utero-placental blood flow (*e.g.*, toxemias of pregnancy, diabetic vasculopathy) and multiple gestations
- C. Fetal causes** are unusual, include familial genetic and chromosomal abnormalities and intrauterine infections (*i.e.*, TORCH), and usually have a poor long term prognosis.

PATHOPHYSIOLOGY: With maternal or placental causes of IUGR, there is decreased placental transfer of nutrient (including oxygen) resulting in reduced fetal body stores of lipids and glycogen resulting in neonatal hypoglycemia; chronic hypoxemia stimulates erythropoietin production leading to polycythemia. These infants are also at increased risk for perinatal asphyxia. Other associated problems include hypocalcemia, pulmonary hemorrhage, hypothermia and, with IUGR associated with toxemia, thrombocytopenia and leukopenia. With fetal causes, decreased growth is constitutive (due to genetic factors) or secondary to infection.

ASSESSMENT and MANAGEMENT:

- Treat asphyxia if present.
- Measure weight, head circumference and length to categorize the type of IUGR.
- Careful physical examination for anomalies and dysmorphic features.
- Blood glucose and hematocrit** to detect hypoglycemia and polycythemia. See sections on Hypoglycemia (P. 153) and on Polycythemia (P. 112).
- Serum Ca^{++} , WBC count with differential and platelet count.
- Infants with IUGR due to placental factors have $\uparrow \text{O}_2$ consumption. This $\uparrow$ insensible water loss to a variable degree (as much as 20-30%). Compensate for this by increasing IV fluid intake. These infants may also need greater intake ($>150 \text{ mL/kg/d}$ and $>100 \text{ kcal/kg/d}$) to achieve adequate growth.
- Further workup and treatment depends on abnormalities identified on history and physical examination.

OUTCOME:

- Perinatal mortality** for IUGR infants is 5-20 times greater than for AGA, mainly due to intrauterine death, perinatal asphyxia, and congenital anomalies.
- Neurologic morbidity** is 5-10 times higher than for AGA infants, especially for infants with $\downarrow$ head circumference at birth. Intellectual and motor function (excluding those with congenital infections, chromosomal abnormalities) depends on adverse perinatal events and on the specific cause of growth restriction. Early identification and treatment of hypoglycemia and polycythemia improves outcome. Neurologic abnormalities are usual with genetic and infectious causes of IUGR.
- Retarded growth:** With placental causes of IUGR, catch-up growth occurs after birth, but these patients usually remain smaller than expected.
- Fetal “programming” of cardiovascular disease:** Recent studies implicate IUGR with adult onset of hypertension, coronary heart disease, hypercholesterolemia, and diabetes. These studies suggest that IUGR has long term effects on endocrine development and homeostasis.