INTRODUCTION

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During the past two decades, remarkably intimate knowledge of the human fetus and his or her immediate environment has accumulated. It is now possible not only to identify but also to quantify with some precision physical abnormalities and functional derangement that affect the fetus (Pritchard et al., 1985) .

Growth and development are a continuum. Specific events take place at specific times along this continuum. From conception to birth, the weight of the human conceptus increases 6 billion times. Cell number multiplies to 2000 billion. Water, protein, fat and mineral content of the fetal body increase 1-2 billion fold each. Early in fetal life, growth is by hyperplasia, later by hypertrophy.

The endocrinological control of fetal growth is very important late in gestation. Insulin (as reflected by C-peptide level) and insulin-like growth factors play an important role in the regulation of fetal growth and weight gain. Insulin is secreted by fetal pancreatic beta cells primarily during the second half of gestation and is believed to primarily stimulate somatic growth and adiposity through classic endocrine modes of action (Hill and Milner, 1985) .

Insulin has been implicated as the primary growth hormone for intra-uterine growth. Insulin-like growth factors are believed also to be growth hormone dependent and have insulin-like

metabolic effects. They can promote fetal growth and cellular differentiation. The insulin-like growth factors (Somatomedins) are produced by virtually all fetal organs from early development onward and are potent stimulators of cell-division and differentiation acting probably in an autocrine and paracrine manner (D'Ercole et al., 1991) .