The Placenta and Low Birth Weight

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I. Introduction

In recent years the child with low birth weight (LBW) with or without signs of intrauterine malnutrition has attracted much attention. The condition has human as well as economic aspects since it scores high in perinatal deaths and brain damage (Potter and Davies, 1969; Fitzhardinge and Steven, 1972; Bjerre and Östberg, 1974). By gaining a better understanding of the extent to which placental changes could indicate the etiology of low birth weight, the pathologist can contribute to the prevention of this condition.

That the placenta, as a link between mother and child, can affect fetal development and postnatal adaption is certain. However, an infant with low birth weight and perhaps signs of fetal deprivation may be encountered without there being extensive lesions in the placental parenchyma. This often frustrates the pathologist, who inherits the task of looking for placental “insufficiency.” He or she must realize that the condition is multifactorial and may be the result of premature delivery, multiple pregnancy, maternal disease and malnutrition and, less commonly, a primary disturbance in placental function. It is practical to think in terms of “fetal supply line” used by Gruenwald (1975a) which, schematically, can be divided into maternal, fetal, and placental aspects. These could act independently or together. All studies of the placenta must thus be related to clinical information from mother and child. The placental lesion might then fit as a piece of puzzle into the clinical picture and help explain and prevent future fetal distress and wastage.

All infants with low birth weight used to be called “premature”. With the recognition of full-term infants having low birth weight and reflecting insufficient intrauterine growth, the terms “dysmaturity” (Sjöstedt et al., 1958), “pseudoprematurity” (Sönderling, 1953), and “small for dates” (Butler and Bonham, 1958) have come into use. Gruenwald (1963) used the terms “chronic” and “subacute” fetal distress, which are not invariably present in these cases. More recently, it has become apparent that infants born prematurely by date can also be retarded in growth.

Before an accurate definition of terms can be achieved, there are two basic problems to be solved. The first is determination of the duration of gestation; the second has been the lack of knowledge about what is normal or pathologic growth in relation to length of gestation. Length of gestation can now be determined objectively by applying knowledge about the development of the fetal nervous system in conjunction with visible external signs of maturity (Saint-Anne Dargassier, 1962; Koenigsberger, 1966; Usher et al., 1966; Harnack and v. Bernuth, 1971). Efforts have been made to gain a better understanding of the problem of normal or abnormal intrauterine growth by the construction of statistical diagrams based on large populations and relating length and weight of infants to the length of gestation. The problems inherent in this have been