FETAL MALNUTRITION, BRAIN GROWTH AND MENTAL DEVELOPMENT

by

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INTRODUCTION

In the United States during 1972, there were 3,258,411 live births and 41,380 fetal deaths (1). Usher (2) concluded that 30% of the fetal deaths that he reviewed could be attributed to malnutrition of the fetus. Using that figure, 12,414 of the fetal deaths in 1972 could be ascribed to fetal malnutrition. Gruenwald (3), Urrusti et al. (4) and Scott and Usher (5) estimated that 33, 40 and 50% of low-birth weight infants are not "premature" but have suffered from fetal malnutrition or fetal growth retardation. Using an average figure of 41%, 205,736 of the 501,795 low-birth weight infants presumably were infants with fetal malnutrition. Therefore the estimated total incidence in the United States of babies either dying in utero from, or born with, fetal malnutrition would be approximately 218,150 infants per year.

In the past, any infant who weighed 2500 g or less was defined as premature. However, this definition did not take into consideration gestational age of the infant. Recently, "premature" infants have been divided into two groups: a) the true premature who is the normal size for his gestational age but is born too soon, and b) the small-for-date or small-for-gestational age infant who is full-term but has not grown properly in utero.

Small-for-date infants have been further divided into those with "intrinsic" growth failure and those with "extrinsic" growth failure (figure 1). Intrinsic growth failure results from congential malformations, inborn errors of metabolism and other genetic diseases. Whatever caused the growth failure is intrinsic to the fetus and does not involve

the placenta (6). Extrinsic growth failure is caused by abnormalities in the maternal and fetal environment.

Two types of extrinsic growth failure have been described (6). Type 1 generally is caused by maternal vascular disease. There is asymmetrical growth failure in the fetus. The brain seems to be of normal size and weight, but the liver is reduced in size and depleted in glycogen. Type 2 is caused by maternal malnutrition. Fetal growth failure is symmetrical in all organs. The brain and liver are reduced in size in proportion to body size.

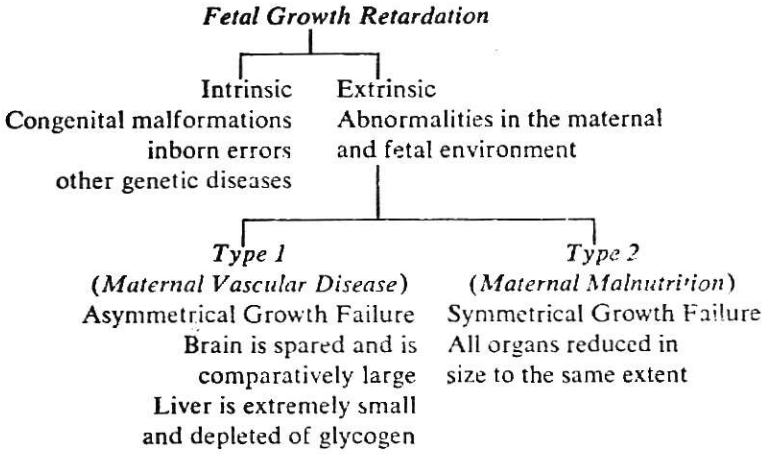

Fig. 1 Classification of fetal growth retardation (6).

Winick (6) stated that Type 1 fetal growth failure characterizes the vast majority of small-for-gestational age infants in developed societies; while Type 2 is the more prevalent form in developing countries and probably within the poorer segments of the United States. The purpose of this paper was to review current research concerning the relationship of maternal malnutrition to cellular brain growth and mental development of the offspring. The paper is divided into three main sections: normal growth and development of the brain, influence of malnutrition on brain development, and relationship of fetal malnutrition and mental development.

NORMAL DEVELOPMENT OF THE NERVOUS SYSTEM

Structural Development of the Nervous System

The human nervous system develops from the neural plate, a thickened area of embryonic ectoderm which appears during the third week of gestation. This plate becomes infolded to form a neural groove and neural folds. The neural folds fuse to form the neural tube which differentiates into the central nervous system, consisting of the brain and spinal cord. During the fourth week, the neural tube grows rapidly and forms the three primary brain vesicles: the forebrain, the midbrain and the hindbrain. The development of the adult shape of the brain is accomplished very early although the brain is still very immature. The forebrain gives rise to the cerebrum; the midbrain becomes the adult midbrain; and the hindbrain gives rise to the pons, cerebellum and medulla oblongata. The walls of the neural tube become thickened by proliferation of neuroepithelial cells which give rise to all nerve and macrogilial cells in the central nervous system. The neural crest cells differentiate into the posterior root ganglia, the sensory ganglia of the cranial nerves, autonomic ganglia and the Schwann cells. During this early period of organogenesis, the central nervous system is acquiring its general adult shape through a process of differential growth accomplished by cell division and migration within the tissue. The basic stages of nervous system development are essentially the same in all mammals (7,8,9).

Principles of Cellular Growth

Enlargement of any organ during the growing period may result from an increase in the number of cells (hyperplasia), an increase in the size of