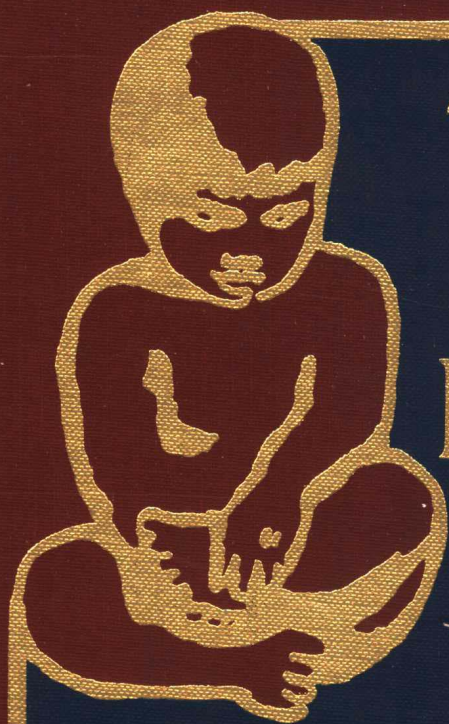


Second Edition



Textbook of Pediatric Nutrition

Editors

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Raven Press

TEXTBOOK OF PEDIATRIC NUTRITION

Second Edition

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Preface

The Textbook of Pediatric Nutrition is a carefully designed tool for medical professionals, including pediatricians, family physicians, surgeons, nutritionists, and dieticians. It is a compendium of the latest knowledge in pediatric nutrition geared to provide optimum nutritional care of normal children, prenatally through adolescence, as well as of children with a variety of disease states.

For the healthy child, it is often the quality of nutrition that determines the meeting of potential. The first two years are the ones of greatest growth, physically as well as developmentally. In extreme deprivation, these parameters can be severely influenced, affecting not only the child but adult potential as well. Although most children in industrialized countries do not experience the episodes of severe malnutrition common in third world countries, mild forms of deficiencies may also affect, more subtly, both growth and development. More common, however, in countries where day-long grazing has replaced mealtimes, and eating is more to assuage boredom than hunger, obesity and its long-term negative ramifications predominate.

For the child with primary disease states, including cancer, diabetes, and sickle-cell anemia, nutrition often makes the difference in prognosis and outcome. Awareness of this fact is an important factor in acknowledging nutrition's essential role in the management of children with these disease states. Ignoring this fact often results in a state of severe malnutrition, not unlike that found in third world countries, which impacts significantly on ultimate morbidity as well as mortality.

Research in nutrition has produced a body of knowledge both broadened and refined since the first edition of this book was published. The first edition highlighted not only primary nutritional deficiencies in children, but also the important role that nutrition plays as a secondary factor in the majority of disease states which effect children. This volume is comprised of the most recent findings in the world of pediatric nutrition, compiled by many of the world's leading experts. It is divided into 4 sections, *Infant Nutrition*, *Nutritional Deficiency States*, *Nutritional Support of the Hospitalized Child*, and *Clinical Nutrition*, totalling 45 chapters. Appendices with formulas for enteral and parenteral feeding are also included.

We hope this book successfully meets the needs of those medical professionals who understand the importance of optimal nutrition to the normal, healthy child, as well as to those children with secondary nutritional deficiencies. We also hope *The Textbook of Pediatric Nutrition* further establishes nutrition as an essential subspecialty in the practice of pediatrics and an essential adjunct to other pediatric subspecialties.

Robert M. Suskind
Leslie Lewinter-Suskind

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CHAPTER 1

Nutritional Factors in Pregnancy Affecting Fetal Growth and Subsequent Infant Development

Ronald A. Chez

The fundamental requirement of a species is the ability to reproduce. The female body is designed to reproduce; toward that end the physiological and biochemical aspects of a woman's life are appropriately modified to accommodate and support a pregnancy when it occurs.

The role of the health care team is to foster the health and well-being of the mother, fetus, infant, and family (1). Although this is done, in large part, during the perinatal period, optimally, care should extend from preconception through pregnancy, labor, delivery, and the postpartum period.

The components of perinatal care include early and continuous risk assessment to evaluate the need for special care, promotion of health via patient education, and medical and psychosocial interventions that support maternal and fetal tolerance to the pregnancy (1,2).

A dominant focus of each of these components of perinatal care is maternal nutrition. The normal-weight woman who enters pregnancy well nourished and then maintains good nutritional health throughout pregnancy and lactation has markedly improved her potential of experiencing a favorable outcome. Obtaining this goal is the shared responsibility of the patient and her health care team.

Clinical guidelines to follow derive from both scientific research and observational studies. Adaptation to pregnancy in the human has been studied primarily at the macroscopic and descriptive level. Changes at the cellular level are less well understood. This chapter provides an overview of what is, and what is not, known about the role of nutrition in determining pregnancy outcome.

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ENERGY REQUIREMENTS OF PREGNANCY

Maternal and Fetal Components

At term, the normal pregnant woman will have accumulated tissues and fluids sufficient for the development of a fetus of appropriate weight for the gestational age. She will also have made the necessary biologic adjustments required to sustain her through labor, delivery, and lactation.

The end results of these changes include a 3,500 g fetus, a 650 g placenta, 800 ml of amniotic fluid, a 970 g uterus, breasts that now weigh 400 g, and an increase in circulating blood volume of 1,200 ml. Retention of approximately 1,700 ml extravascular volume also occurs; this may appear as dependent edema. The presence of generalized edema can add as much as 6,600 ml more fluid (3–5).

The normal weight pregnant woman also stores fat. This is presumed to be for maternal caloric needs in the third trimester and lactation. Estimates of accumulated body fat, derived from measurements of lean tissue density and skinfold thickness, range from 1,300 to 5,400 g (6,7). The total weight gain of the woman who does not have generalized edema is approximately 13 kg (5).

Maternal physiological adaptation to pregnancy begins in the first trimester during fetal organogenesis (8). These changes are preparatory for the subsequent role of sustaining the growth and development of the fetus while preserving maternal well-being in this and any future pregnancies.

The extent of the specific changes in maternal body composition of fat, lean tissue mass, and other non-fat tissue components is difficult to discern (9). The clinician measures only the end results of any composition change as incremental weight gain and total weight gain.

It has been difficult to find a dependable clinical tool to measure body composition. The technologies available for measuring the distribution of fat are dependent on the accuracy of the determination of lean body mass. This in turn can be a function of lean tissue hydration with body water.

Changes in fat and lean tissue distribution occur early in the first trimester. Skinfold thicknesses can be considered only estimates of change in fat content because the technique is difficult to reproduce; results vary according to the site chosen and are influenced by water retention (10). In addition, a determination of how much body fat is accumulated during pregnancy will be inaccurate if a prepregnancy measurement has not been obtained. Further confounding variables are maternal utilization of fat as a caloric source and retention of body water, both of which are features of normal third trimester metabolism.

Caloric Needs

Energy is necessary to supply the caloric requirements of the pregnant woman's daily activity, her own weight gain, and fetal growth. Endogenous maternal fat stores and increased consumption of calorie-rich nutrients are required to provide this energy.

The main portions of the energy expenditure requirement include basal and resting metabolic rate of the mother, thermogenesis, and physical activity. Although there is wide variation in the range of reported basal and resting metabolic rates, these rates do increase on average. The increment appears to be between 20,000 and 30,000 kilocalories (Cal) during the course of the pregnancy (11). There does not appear to be a change in thermogenesis.

Physical activity was thought to decrease because of an increase in sedentary behaviors of pregnant women. This has not been found, however, either in developed or developing countries. The increase in body mass during pregnancy requires more energy to meet the requirements for the activities of daily living as most women continue to work in the home, factory, or field (11).

The increment in total energy expenditure and therefore the requirement for dietary supplementation was thought to be 85,000 Cal per pregnancy or 300 Cal per day (5). It appears more likely that the overall cost is in the range of 55,000 Cal, or an increased need of about 200 Cal per day (11).

DETERMINANTS OF NEWBORN WEIGHT

Normal Birth Weight

Maternal weight gain during pregnancy reflects the weight of the products of conception, the accumulation of maternal tissue and retained water, and the length of

the pregnancy. There are maternal characteristics that statistically influence the weight gain including food access and intake, and fundamental changes in physiology or some other mechanism (12).

Birth weight at term is directly and linearly related to maternal weight gain. It is similarly related to prepregnant weight. These relationships are both independent and additive. The risk of a low birth weight newborn at term decreases as prepregnant body mass index increases. It also decreases as total weight gain, net weight gain, and net weight gain per week after 20 weeks gestation increases. The highest incidence of normal birth weight infants occurs in women of normal prepregnant weight who gain between 25 and 35 lb during pregnancy (13–16).

In the United States, lesser weight gain during pregnancy occurs in unmarried women, black women, multiparas, women older than 30 years of age, those with an increased body mass index, those less than 67 inches tall, cigarette smokers, and women with less than a 12-year education. However, in each of these categories, birth weights between 3,000 and 4,000 g at 39 to 41 weeks gestation do occur, making it difficult to indict any one behavior or demographic feature (12).

Low Birth Weight

Low birth weight is a dominant factor in newborn and infant morbidity and mortality (16,17). It also has a direct, adverse impact on subsequent childhood somatic growth, neuropsychological development, infant mortality, and infant morbidity. *Low birth weight is a result of premature birth and of intrauterine growth retardation.*

The single and most prominent direct determinant of premature birth is cigarette smoking (18). Cigarette smoking also increases the risk of spontaneous abortion, third trimester placental bleeding, and premature rupture of the membranes. Other direct determinants include low prepregnancy weight and a history of prior premature birth and spontaneous abortions. Maternal age and socioeconomic status are indirect established determinants (19).

The direct determinants of intrauterine growth retardation include maternal height less than 62 inches, low prepregnant weight, low maternal weight gain, cigarette smoking, alcohol consumption, and general maternal morbidity. Indirect determinants include race, parity, maternal age, and socioeconomic status (19).

Adolescence per se does not have an adverse impact on pregnancy or birth weight outcome if the young woman is of normal weight and gains appropriately. Thus, adolescents and mature women share the same determinants for birth weight (20).

The risk of preterm birth increases with poor weight gain. This is particularly true when the decreased weight

gain occurs after the 20th week of gestation. It is not clear if the etiology is a direct function of maternal dietary intake, incomplete changes in maternal body composition or physiology, increased energy expenditure, and/or psychosocial stress (21). Even when specific causes have not been ascertained, decreased weight gain during pregnancy does alert the provider to the need for intervention. Some interventional strategies may be introduced by the health professional, whereas others, more societal in scope, may be more difficult to initiate.

The appropriate solution for achieving satisfactory weight gain in an underfed pregnant woman would seem to be increasing the daily caloric intake. However, a relationship between an increase in energy intake and maternal weight gain and/or birth weight has not been consistently found. This may be a function of maternal nutritional vulnerability. Undernourished women who receive supplements can demonstrate an increase in birth weight, whereas women with less of a deficit between energy intake and requirements do not (9). Explanations for the inconclusive data predominantly relate to flaws in scientific method (22). Regardless, food as the therapy for malnutrition and undernutrition makes clinical sense.

Interestingly, mothers with a high body mass index have an increase in low birth weight with weight gains in excess of 20 lb. This may result from the increased incidence of illnesses in women with moderate and marked obesity. In women with normal or low body mass index, the risk of low birth weight begins to increase after a total weight gain of 45 pounds (12).

Women, Infants, and Children (WIC), a program of the United States Department of Agriculture, provides nutritional supplementation and counseling for pregnant and breast-feeding women and children up to age 5. Eligibility is based on nutritional status and financial need. Women who use the program are less likely to deliver low birth weight infants (23).

The WIC program is essentially supplementation with dairy products and cereals. This type of supplementation should be differentiated from supplementation with a high protein-to-calorie ratio diet, which may result in a negative effect on fetal growth and an increase in premature delivery (24).

High Birth Weight

Macrosomia is arbitrarily defined as a birth weight greater than 4,000 g. The rate of high birth weights has been increasing for the past decade. Most of the increase reflects a shift to the right of the entire curve of birth weights over time. Most high birth weights, therefore, may be considered secondary to normal biologic variation. Associations that result in a relatively higher proportion of macrosomic newborns include multiparity,

history of a previous macrosomic baby, large maternal stature, and postdatism. Direct determinants include prepregnant obesity, excess weight gain in pregnancy, and poorly controlled gestational and preexisting diabetes mellitus.

Macrosomia results in an increased incidence of dysfunctional labor, fetopelvic disproportion, shoulder dystocia, instrument delivery, birth trauma, and cesarean section delivery. Consequently, macrosomic infants have an increased perinatal mortality and morbidity with adverse sequelae into childhood. Obese infants of diabetic mothers tend to remain obese into adolescence and also have a higher risk of developing diabetes mellitus.

Between 15% and 40% of reproductive-age women are obese. Fecundity is not impaired by obesity; the incidence of twins actually increases. The primary antepartum complications associated with maternal obesity are hypertension and gestational diabetes. Intrapartum complications in the obese woman are those associated with delivery of a macrosomic infant (25).

Macrosomia secondary to maternal glucose intolerance has been recognized for several decades (26). The shift in the birth weight curve to the right in infants of diabetic women directly relates to the mean circulating maternal plasma glucose level during pregnancy. Maintenance of fasting and postprandial levels in the normal nonglucose intolerant range, by a regime consisting of daily, split doses of insulin, a proportioned diet, and exercise, will result in an infant of normal birth weight.

Twins

Twin gestation occurs in approximately 1.2% of births in the United States. Twin pregnancy is at increased risk of intrauterine growth retardation, premature delivery, discordance, congenital anomalies, gestational diabetes, pregnancy-induced hypertension, and perinatal acute and chronic asphyxia. Because of these associations, there is an increased incidence of perinatal mortality.

The lowest perinatal mortality of twins occurs in the 3,000 to 3,500 g range; this is associated with a total weight gain of 44 lb in the pregnant underweight woman and 41 lb in the normal-weight woman. The birth weights of twins increase linearly with the prenatal weight gain of women who were either underweight or normal weight prior to conception. Also, and independently, the proportion of low birth weight twin infants decreases as prepregnant weight increases. The lowest incidence of low birth weight occurs in women who were obese and very obese in their prepregnant state (27).

As a result of the increasing use of diagnostic ultrasound, multiple gestations are more frequently being identified in the first half of pregnancy. This should result in guidelines specifying greater incremental and to-

tal weight gain for multiple gestations over those for singleton pregnancies.

BIRTH DEFECTS

Minerals

Fetal exposure to high maternal blood lead levels coupled with an increase in postnatal blood levels can result in a decline in linear growth rates in the second year of life as well as slowed cognitive development (28,29). Food contaminated with methyl mercury ingested during pregnancy has resulted in infants with cerebral palsy, mental retardation, and systemic organ damage (30).

Iodine deficiency in pregnancy is not found in the United States. It is associated with cretinism, stillbirth, congenital anomalies, and hearing loss.

There is a lack of consistency in the studies that link zinc deficiency with adverse pregnancy outcome. It is, therefore, difficult to associate zinc intake and maternal serum levels of zinc with congenital anomalies. However, the possibility of nutrient-zinc interactions and associated changes in cell and circulating zinc levels cannot be readily dismissed (31).

Vitamins

There are potential risks from large overdoses of some vitamins during pregnancy. Excess vitamin D has been associated with infantile hypercalcemia syndrome. Vitamin A overdosage has been related to anomalies of bone, the urinary tract, and central nervous system. However, there is little evidence that vitamin C overdose during pregnancy will induce a relative deficiency in the newborn by increasing the metabolic clearance of the vitamin (9).

Nutritional deficiencies during the first half of organogenesis, particularly of folic acid, have been linked to the occurrence of neural tube defects. A number of studies have affirmed that the use of multivitamins containing folic acid during the first 6 weeks of pregnancy will prevent by more than half the recurrence of neural tube defects in the woman at risk (32).

Diabetes Mellitus

There is an increased incidence of congenital anomalies in the offspring of diabetic women. This occurrence has been directly related to the degree of control of blood sugar levels at the time of conception and during organogenesis (33). Maintaining euglycemia in the preconception period and then during the first trimester results in rates of congenital anomalies similar to those found in the normal population. Other potential adverse out-

comes that are increased in a poorly controlled diabetic pregnancy include stillbirth, neonatal death, neonatal morbidity, birth injury, intellectual delay and impairment, and childhood obesity. As manifestations of a metabolic derangement, all are preventable by supervised control of blood glucose levels (34).

Ketonuria has been associated statistically with lower mental, motor, and intelligence quotient scores in the children of both normal and diabetic pregnancies analyzed in the Perinatal Collaborative Study (35). However, additional analyses of these data, controlling for nonnutritional factors, have not confirmed these associations (36). Further, ketonuria occurs sporadically in most normal pregnant women (37).

CLINICAL IMPLICATIONS FOR PRENATAL CARE

Preconception Care

Prepregnant weight is an important clue to nutritional health. Body mass indices are independent of adult stature and permit reference points for the determination of under-, normal, and overweight categories. The Quetelet index for body mass is determined by dividing weight in kilograms by height in meters squared. The body mass index cutoffs are <17.9 for extreme underweight, <19.8 for underweight, >26.0 for overweight, and >29.1 for extreme overweight. Weight or weight-for-height cannot determine proportion or distribution of fat content and lean body mass; physical examination is more helpful.

Weight and nutrition do not necessarily relate. Historical factors and physical examination provide more specific information about nutritional status. Particular attention must be paid to certain women who are at higher risk for nutritional deficits. This category includes women who take antiseizure medication, have used combination oral contraceptives for a number of years, are food faddists, have had three or more pregnancies in the past 2 years, are smokers, chronically use weight loss or therapeutic diets for systemic medical illnesses, and athletes who engage in sports requiring prolonged aerobic expenditures (38). These patients will be better prepared for conception with purposeful attempts to achieve normal body mass index combined with the prophylactic daily use of vitamin and mineral supplementation.

Prenatal Care

A primary goal of prenatal care is to deliver a baby of appropriate weight for gestational age at term. This is more likely to happen in the normal body mass index woman who gains a total of 25 to 35 lb with an increment of about 4 lb a month after the first trimester. This can be accomplished with a diet of 35 Cal per kilogram