LONG TERM PROGNOSIS OF LOW BIRTH WEIGHT INFANTS

ESSAY

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INTRODUCTION

INTRODUCTION

Scattered references to the growth-retarded fetus have appeared in the literature for more than thirty years ago (Colman and Rienzo, 1962).

Until 1961, the World Health Organization (W.H.O.) defined as prematures all infants weighing 2500 g or less at birth. With the recognition that birth weight alone does not take cognizance of gestational age and maturity, WHO amended its earlier definition and, in 1961, suggested that infants weighing 2500 g or less at birth be termed "infants of low birth weight" (WHO Technical Report, 1961). Birth weight could then be related to independent assessments of gestational age and the intrauterine-growth-retarded fetus, or small-for-date infants.

Following these early reports, many terms have been used to designate the fetus whose growth is impaired: pseudopremature, small-for-dates, dysmature, fetal mal-nutrition syndrome, chronic fetal distress, intrauterine-growth retardation, and small-for-gestational age (SGA) baby (Cassady, 1981).

Studies in the United States and Great Britain show that about one-third of all infants whose birth weight is less than 2500 g (L.B.W.) are not truly premature, but are small-for-gestational age (Walker , 1967) . Recent evidence, however, suggests that in developing countries, more than eight in ten LBW infants are born at term (Cassady , 1981) .

Such observations have led some authors to speculate that much of the variance in LBW rates is due to population differences in the incidence of SGA (Belizan et al., 1978). The magnitude of this problem is second only to prematurity as a cause of perinatal mortality. While the preterm infant has an increased neonatal mortality, the SGA baby has a vastly increased fetal death rate (Lugo and Cassady, 1971). The overall neonatal mortality among SGA infants (3.2 %) is less than the appropriately grown prematures, but more than the appropriately grown term infants (Lugo and Cassady, 1971).

Congenital anomalies occur more frequently in SGA infants. The SGA term infant has a 5 % to 7 % incidence of congenital anomalies (Lugo and Cassady, 1971), and compromised physical as well as neurodevelopmental growth appears to be common in many of these babies (Fitzhardinge and Steven, 1972).



Definition

Lubchenco and coworkers (1963) defined any infant whose birth weight is at the tenth percentile or less for gestational age as SGA. Gruenwald (1966) preferred to define as SGA any infant whose birth weight is more than two standard deviations below the mean for any given week of gestation, corresponding approximately to third percentile on the intrauterine growth curves.

Attention has recently been directed toward evaluation of physical measurements other than weight in distinguishing impaired from normal fetal growth (Brans and Cassady, 1975).

The Academy of Pediatrics, through its committee on the Fetus and Newborn, recommended that all newborn infants be classified by birth weight, gestational age and some standard for intrauterine growth (Silverman, 1967). This recommendation followed years of experience, classifying infants into two categories: low birth weight (2500 g) and full birth weight (more than 2500 g).

It is also well known that the low-birth-weight designation included preterm and term infants (Lubchenco, 1981).

The World Health Organization, with support from European Pediatric Group, has set the dividing line between preterm and term birth at 37 weeks from the first day of the last menstrual cycle (Lubchenco, 1981).

From the aforementioned statements, we can conclude that:

- \star Low birth weight (LBW) is a birth weight (BW) of \angle 2500 gram .
- \pm Small-for-gestational age (SGA) is defined as WB \angle -2.0 standard deviations (SD) .
- * Preterm birth is that occurring before the 37 $\underline{\text{th}}$. week of gestation ($\underline{/}$ 259 days) .
- # Appropriate for gestational age (AGA):

 a BW more than 2.0 SD from the mean BW for

 gestational age (Hagherg et al., 1984) .

Incidence of Low Birth Weight:

The percentage of all births weighing less than 2500 g varied from 3.9 in Sweden to 10.8 in Cuba (World Health Organization , 1978) .

Diagnosis

Fetal:

Clinical assessment of risk factors in the maternal history may be useful in as few as one-third or as many as two-thirds of these cases (Galbtaith et al., 1979).

Use of such simple clinical methods as manual estimation of fetal weight, maternal assessment of fetal activity, or serial measurement of fundal height are helpful (Mathews, 1975).

Several biochemical indices of feto-placental function have been proposed as useful. Among these are diminished levels of crystine aminopeptidase, oxytocinase, human placental lactogen, B₁-glycoprotein and estriol, as well as elevated levels of —fetoprotein, heat-stable alkaline phosphatase, N-acetyl B-glucosaminidase and —aminonitrogen in maternal serum (Arias , 1977).

Human placental lactogen proved to be the most useful of the pregnancy associated proteins (Sp_l and pregnancy-associated plasme protein) in predicting fetal growth retardation. It increases in the serum with advancing pregnancy reaching a peak at 36 - 38 weeks gestation, followed by a slight decrease (Pledger et al., 1984).

Pregnancy-specific B_1 -glycoprotein (Sp_1) is produced by the placenta and released into the maternal circulation in steadily increasing amounts with peak concentrations occurring at 38 gestational week . Low Sp_1 levels have been found in association with intrauterine growth disorders in the third trimester . It would be helpful to study serial Sp_1 levels to gain a better understanding of second trimester placental function in conjunction with low birth weight and reflect placental size and function . Elevated maternal serum \sim -fetoprotein (AFP) levels are also associated with low birth weight and with fetomaternal bleeding (Haddow et al., 1984) .

Studies by Metcoff and others have clearly shown biochemical changes in leucocytes obtained from maternal venous and umbilical cord blood obtained at delivery of SGA babies (Yoshida et al., 1972).

Ultrasonic cephalometry has been helpful in determining rate of growth of the biparietal diameter (BPD) of the head. When this diameter is less than optimal, 53 % to 82 % of such infants have been found to have birth weights below the tenth percentile for gestational age (Whetham et al., 1976).

Recently, the measurements of crown-rump length; chest, trunk, abdominal and head circumference; intrauterine volume; and a variety of ratios, products and other combinations of these measurements has improved the sensitivity of ultrasound as predictor of impaired fetal growth (Wittman et al., 1979).

Neonatal:

Reduced birth weight for gestational age (light-fordates) is the simplest and oldest method of diagnosis . Impaired fetal growth has recently led to more careful assessment of physical findings at birth (Cassady, 1981). Soft tissue westing, diminished skin-fold thickness, decreased breast tissue and reduced thigh circumference have been cited as evidence for recent wasting and have been suggested, by some, as useful measurements for neonatal diagnosis (Brans and Cassady, 1974) . Widened skull sutures and large fontanelles, diminished foot, femoral and crown-heel lengths and delayed development of epiphyses have been cited as failures in bone growth (Woods et al., 1979). Combinations of measurements such as weight/head circumference, crown-heel length/head circumference, and birth weight/crownheel length have been used with increasing frequency to assess disproportionate patterns of growth (Woods et al.,1979).

Causes Of Low Birth Weight

Normal intrauterine growth for body weight, length and organ weights progresses in a systematic fashion between 28 and 38 weeks of gestation. From about 38 weeks onward, growth of the fetus and placenta departs from this previous pattern (Brans and Cassady, 1974).

Two major factors influence fetal growth: The inherent growth potential of the fetus and the growth support it receives by way of the placenta from the mother (Ounsted and Ounsted, 1973).

The classic study of Meredith (1970) brings to the

Altered Growth Potential:

and Inglis , 1974) .

attention that mean birth weights may vary from 2400 g (in New Guinea) to 3880 g (in The West Indies)
(Ashcroft et al., 1966).

Of particular interest are certain data which demonstrate striking ethnic differences in birth weight, regardless of current socioeconomic status or geographic location (Salber and Bradshaw, 1951). Such observations are the consequence of genetic variation, however, there is racial, ethnic and population differences (Johnstone

Retardation of growth in utero may be caused by factors inherent in the fetus itself. Subnormal embryonic growth may then be viewed as a form of maldevelopment. Examples of causes include certain genetically determined dwarfs, fetal infections, chromosomal syndromes, several congenital anomalies and some inborn errors of metabolism (Levy et al., 1978). Retarded fetal growth in the infant with intestinal or central nervous system anomalies which preclude normal swallowing, may suggest that amniotic fluid is a more important source of nutrients than currently suspected (Cassady, 1981).

Of equal interest is the presence of profoundly impaired growth in infants with end-organ insensitivity to insulin (D'Ercole et al., 1977) as well as in infants with pancreatic agenesis (Hill, 1978) observations which provide the importance of insulin/cell relationships in the growth process.

Impaired support for growth:

Before the third trimester of pregnancy, supply far exceeds needs of the fetal growth is determined by inherent fetal potential. By the third trimester, the adequacy of the supply line becomes the limiting factor in fetal growth.