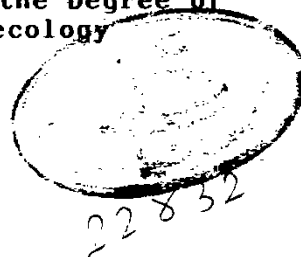


FETAL WEIGHT ESTIMATION BY ULTRASOUND IN
COMPARISON TO THE ACTUAL BIRTH WEIGHT

THESIS

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BY

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Introduction

INTRODUCTION

Many factors affect fetal growth. Growth and eventual birthweight are determined by intrinsic genetic information. This is modified by stimuli to growth that are probably largely hormonal and by constraints on growth external to the fetus. Maternal weight bears a close correlation to fetal birthweight, but maternal height has less of a correlation. Race plays a part, in that Asian infants are about 350g smaller than black infants who, in turn are about 150g smaller than Caucasian infants. Male fetuses are 150-200g bigger than female infants. Recognition of fetal weight is important because fetal growth and eventually birthweight, affect the infants post-natally. Many attempts have been made at methods of predicting fetal weight.

By clinical palpation and a knowledge of the expected weight, Loffler, (1967) was able to guess fetal weight to within 450g in 86% of cases. The error increased, however, when very small or very large babies were considered. Prediction of birthweight by ultrasonic means is mainly performed from measurement of the abdominal circumference (AC),

I FETAL GROWTH

General Aspects of Fetal Growth

Several comprehensive surveys of the growth in fetal weight and its different dimensions as pregnancy advances have been made on series of induced abortions, late abortions, premature deliveries and normal deliveries (Streeter, 1920, Scammon and Calkins, 1924, Gruenwald and Minh, 1961, Hendriks, 1964, Hendriks, 1967, Rantakallio, 1969, Usher and Mclean, 1969, Sterky, 1970, Brenner, et al., 1976). Although findings in the second trimester in particular, have largely been done on pathologic pregnancies, the consistent results obtained in these large series were taken to represent fetal growth in normal pregnancies too (Campbell, 1974(b), Brenner, et al., 1976). Fetal weight, length and head circumference follow a sigmoidal curve and fetal growth has therefore been divided into four phases (Suzuki, 1977):

1. a phase of slow growth up to the 15th-16th week,
2. a phase of accelerating growth from the 16th-27th week.
3. a phase of maximal growth from 28th-38th week
4. a phase of deceleration after the 38th week.

Review Of The Literature

Factors Influencing Normal Fetal Growth

- Genetic and Environmental Factors

A multitude of genetic factors as well as environmental ones significantly influence the growth of organisms as a whole, its cells and organs and the biochemical balance during the ante-natal period (Thomson, et al., 1968, Brenner, et al., 1976). The racial heritage of the mother is one of the most important genetic factors. Merideth (1970) comparing the birth weights in several geographically and ethnically different populations, has shown that the largest children in the American Indian Cheyenne tribe had an average birth weight of 3800g. At the same time the average birth weight in the Luni tribe of New Guinea was found to be 2400g. Among environmental factors it is worth emphasising life at high altitudes above sea level where there is a low partial oxygen pressure. This is well illustrated by data from Peru. In the mountainous regions of the Andes, the average weight at birth is 2950g, while in the Lima coastal area the average birth weight is a whole 1000g higher (Kruger and Arias-Stella, 1970).

Ounsted (1970) wrote about the genetic influence

of the Y-chromosome. He determined that the male fetus grows more rapidly than the female fetus in mixed multiple pregnancies and that the presence of the male embryo accelerates the growth of the female twin. The same author maintains that intra-uterine growth is controlled by the maternal system which acts through two variables. The first is the hypothetical maternal regulation which slows down fetal growth and the other affects the antigen reaction between the fetus and the mother (Ounsted, 1971).

In experiments with mice, Billington (1964) found that the intensity of the penetration of trophoblasts in the decidual tissues is determined by the size of the placenta and that this penetration is greater when the trophoblastic antigen is different from the decidual antigen.

James (1964) explained these phenomena being the maternal immunological response to the paternal antigens which then influences the size of the placenta. The more rapid intra-uterine growth of the male fetus could be explained by the following mechanism: the Y-chromosome produces stronger antigens than the X-chromosome and thus gives the male fetus

a greater antigenic difference from that of the mother which is not the case with the female embryo (Ounsted and Ounsted, 1973). Nevertheless, the problem of biological differences among different populations and the influence of genetic factors such as the Y-chromosome still remain important questions. The question of the mechanisms whereby the above-mentioned factors act is still unresolved. Do they exert their influence indirectly on fetal growth by increasing the utero-placental transport of nutritional substances or do they exert their influence by increasing the secretion of insulin?

- Hormones

The role of hormones in the regulation of fetal growth is also not entirely clear although the majority of published works have not confirmed a significant influence. Jost, (1962) and Remion, et al., (1968) found in experiments on rabbits that hypophysectomy of the mother and decapitation of the fetus does not significantly influence the birth weight. In human embryos without pituitary glands, the birth weights and lengths were within normal limits (Ried, 1960). Studies of the endocrine glands of anencephalic children, have provided us with valuable knowledge.

In these children the hypothalamus is missing as a rule and as a result there is a disturbance in the hypothalamic-hypophyseal integrity. The birth weights of such children are mostly below the 50th percentile (Honnebier and Swabb, 1973), but it is still insufficiently clear whether this should simply be attributed to the absence of the brain mass and cranial wall or whether it is the influence of the hypothalamic-hypophyseal axis on the intra-uterine growth of an anencephalic fetus. The actual existence of this influence has been confirmed by the works of Hennebier and Swabb (1973). The linear regression of the birth weights of 122 anencephalic fetuses showed a growth rate of 97g weekly in comparison with 143g weekly in the control group of healthy fetuses. In further experimental work the same authors found significant growth retardation in rabbit fetuses 21 days after the removal of the brain, performed between the 18th and 19th day of pregnancy. Kurjak, et al., (1976b) in a retrospective work reviewed 143,175 case histories of which 88 were found to be anencephalic fetuses. By statistical analysis they found that the weekly growth of the anencephalics was 105g while in the control group it was 147g. These works show that the data available on the in-

fluence of the embryo's own hormonal development has on fetal growth is quite inadequate and markedly contradictory. Additionally, there are still no data on the level of hormonal development in anencephalic newborns.

* Human Placental Lactogen (HPL)

The amount of HPL found in the body fluids of the embryo is especially small and it is therefore unlikely that this placental hormone directly affects fetal growth. However, it can indirectly affect fetal growth by accelerating maternal lipolysis (Grumbach, et al., 1968).

Because of the resulting increase in the amount of free fatty acids, less glucose will be used by the maternal organism leaving more for the utero-placental transport. In contrast to the high level of HPL in the maternal blood, Kaplan and Grumbach (1965) found an almost insignificant amount of this hormone in the blood of the embryo. The very low values of HPL in the blood of pregnant women carrying children with renal agenesis should not be erroneously interpreted because these values approach normal ones following correction in relation to the weight

of the placenta (Josimowich, 1971). It would seem necessary to obtain more reliable data regarding the levels of these peptides in the fetal fluids as well as data on a larger number of newborns from all gestational groups.

* Insulin

In the discussion of hormonal influences on fetal growth, a special place is occupied by insulin. It is common knowledge that a large baby is sometimes born even in a well-controlled insulin dependent pregnancy. Although there is still no direct evidence that this is the cause of accelerated fetal growth, it is justifiable to believe that this fact has a particular significance. It appears that this effect is strictly connected with the so-called critical period in pregnancy judging from the findings of Cardell (1953) that the fetuses of diabetic mothers show accelerated growth only after the 28th week of pregnancy. This was verified by the experiments of Foa and Guidotti (1961) in which insulin caused the transport of glucose in the myocardia of the chick embryo on the 7th and 8th day of development. Before this time, glucose enters the myocardial cells independently of insulin. Similarly, Clark, et al., (1968),

found that insulin does not influence the consumption of amino acids in the rat heart until the 16th day of development.

the exceptionally valuable work of Ashworth, et al., (1973) has led to recent success in measuring the amount of insulin secreted from pancreatic tissue obtained from human fetuses in the period from the 15th to the 24th week of gestation. The results were presented in linear relation between the secreted insulin and fetal weight.

* Cortisol

In 1971, Moog issued the warning that cortisol can influence the cellular and biochemical growth of the fetus accelerating early activity of the alkaline phosphatase in the intestine. These studies facilitated the first therapeutic results using cortisol in the acceleration of the maturation of fetal lungs (Liggins and Howie, 1972). Of the different influences of cortisol on growth, it is worth mentioning the induction of the activity of intestinal enzymes, alkaline phosphatase and disaccharides in particular (Doell and Kretchmer, 1973). This also causes more rapid maturation of the intestinal epi-

thelia. Jost and Picon, (1970) have found cortisol to have a very important role in the deposition of hepatic glycogen in rats. This effect must however be present during a particular period of gestation. The positive effect of cortisol on the maturation of the lungs and the synthesis of lecithin has been demonstrated biochemically (Farrel and Zaohman, 1973).

* Thyroxine

Thyroxine also affects fetal growth. Although a shortage of this hormone is not associated with a reduction in birth weight, this deficiency will cause retarded cellular and biochemical development, especially of the central nervous system cells (Walravens and Chase, 1969, Eayrs, 1953).

Growth at the cellular level has been studied by Enesco and Leblond, (1962) and Winick and Noble (1967). They describe three stages of growth:

1. cellular division with rapid increase in the amount of D.N.A. and constant cellular size.
2. A slower division of cells associated with a growth in cell size.
3. A growth in cellular size without any increase in the number of cells.