Fetal malnutrition—the price of upright posture?

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Summary and conclusions

The pattern of preterm fetal growth faltering, normally seen in man, differs from that observed in animals. This type of fetal growth cannot be considered as an adaptation to facilitate birth but is more likely to be due to rapid evolution and imperfect adaptation to the upright posture. The pattern of posture and physical activity during pregnancy may therefore be an important determinant of fetal growth. Differences in intrauterine nutrition existing between social groups, usually ascribed to variations of maternal diet and nutrition, may well result from different patterns of maternal activity in the weeks preceding birth.

Introduction

In man the fetal growth curve in the second half of intrauterine life is unusual, if not unique. Initially growth is linear, but towards term there is a tendency to fall away from this early pattern. This picture has been derived from cross-sectional studies based on weights of normal live-born infants of varying gestational age.[1] It has been confirmed by longitudinal measurements of the fetal biparietal diameter using ultrasonic cephalometry.[2]

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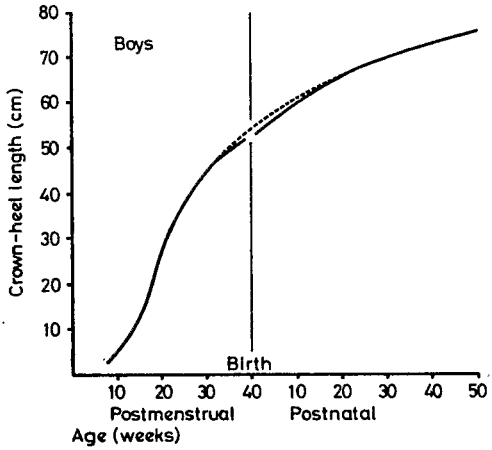
This faltering of growth just before birth is intriguing; no animal growth studies to date show a comparable period of growth faltering at the end of intrauterine life.[3] It seems to be provoked by the maternal environment in that soon after birth the infant shows increased growth and rapidly regains the size predicted by its original regular growth pattern (see figure).



Human perinatal growth (after Tanner[26]). The irregularity of human growth curve at time of birth cannot be considered adaptive. It is more likely to be an imperfection that may result from acquisition of upright posture and be outweighed by selective advantages of such a change.

Moreover, short-gestation babies do not show a dip in growth when they reach the time at which they should have been delivered.[4] On the basis of anatomopathological evidence, Gruenwald has suggested that this growth faltering results from inadequate fetal nutrition.[5] To explain this phenomenon he postulated that the transfer of nutrients across the placenta was limited at the end of pregnancy by some unknown maternal factor. Some clues as to the nature of this factor may lie in the consideration of the ways in which certain relevant human features differ from those of animals. I want to explore some of these differences and to offer an explanation for man's peculiar pattern of intrauterine growth.

Non-adaptive nature of preterm growth faltering

One might postulate that slight growth faltering occurring just before birth was an adaptation to facilitate delivery. This is a particularly difficult process in the human species; the pelvis was considerably modified by the acquisition of the upright

position,[6] and the fetal head is very big compared with other animals.[7] This adaptive interpretation, however, is contradicted by some epidemiological and anatomopathological data.

In most population studies the mean birth weight is slightly lower than the optimal birth weight—that is, the birth weight associated with the lowest perinatal mortality.[8] This was noted even before induced birth became commonplace.[9] This suggests that most newborns, even in well-off communities, do not achieve optimal growth in utero. Consequently, any growth faltering, however slight, that might be provoked by some degree of malnutrition, would be expected to increase perinatal mortality.

It may be argued that this epidemiological evidence is based on populations in which the possibility of caesarean section minimises the disadvantages of large body size. Mechanical problems arising during delivery, however, are mainly related to head volume, which is little affected by the level of intrauterine nutrition; growth-retarded babies have a head size very similar to normally developed babies.[10] On the other hand, their heart, thymus, and liver, which have a key role in supporting the stress of birth, are very underweight.[10] Indeed, morphological changes associated with intrauterine malnutrition are further evidence that the phenomenon is not adaptive; they are exactly the reverse of what would be expected if they had appeared under selective pressure to improve the outcome of delivery. More probably the appearance during evolution of this pattern of growth—that is, some degree of malnutrition just before birth—was associated with an increase of perinatal mortality.

Hypothesis

Darwin in his first edition of *The Origin of the Species* in 1859 advanced the idea that natural selection could not achieve absolute perfection. Even now it is agreed that adaptation by evolution must be and is imprecise and imperfect.[11,12] If growth faltering at the end of intrauterine life is not an adaptive phenomenon, and may have been associated with an increased perinatal mortality, it must be regarded as one of the imperfections that represent the cost of rapid evolution. This implies that this anomaly of fetal growth appeared relatively recently on the scale of evolution; otherwise it should have been progressively eliminated. Moreoever, we must postulate that this imperfection occurred because it was associated with the acquisition of some other human characteristic with sufficient selective advantages to outweigh any increase in perinatal mortality.

Undoubtedly, of the recently developed features that single us out from other species, the adoption of upright posture caused the greatest anatomical and physiological changes. Their

magnitude has been compared to that of the acquisition of flight in birds.[13] This characteristic may be sufficiently advantageous to outweigh several coincidentally arising drawbacks. One of these, unquestionably, is a relatively difficult birth; the pelvis transmitting the weight of the trunk to the legs is the part of the skeleton most affected by this change in posture, and the path traversed by the fetal head during birth is peculiarly complicated.[14]

The theory advanced here is that the repercussions of upright posture are manifest not only at the time of birth, as generally admitted, but also in the preceding weeks by impaired fetal growth. Supporting this is the fact that certain changes have occurred also in maternal cardiovascular physiology, which tend to reduce uterine blood flow and hence fetal nutrition, principally in the last weeks of pregnancy.

Upright posture and maternal haemodynamics

The raising of the trunk to the upright position provoked a forward projection of the sacrum and of the lower lumbar vertebrae. This skeletal modification, specific to man, decreased the available space for the pregnant uterus, as a result of which it interferes near term with normal maternal haemodynamics. The aorta and the inferior vena cava that run along the lumbar spine are vulnerable to compression at the level of L4 and L5, particularly when the subject is supine. Only when lying in the lateral position is this effect relieved.[8]

Compression of the aorta results in a fall in arterial pressure distal to the lumbar lordosis,[15] which tends in turn to decrease uterine blood flow and impair fetal nutrition. Compression of the inferior vena cava provokes diminution of the blood volume and a drop in cardiac output, whereas these are normally considerably increased during pregnancy.[8] When standing, a similar compounding effect is provoked by changes in hydrostatic pressure in the venous system due to gravity.[16,17] The combined results of these two mechanisms on the cardiovascular system are most pronounced during the last weeks of pregnancy and are relieved only when the pregnant woman is lying in the lateral position. Any effect on uterine blood flow must be to reduce it. This is supported by the isotope studies of Suonio *et al*[18] on pregnant women near term, which showed statistically significant changes in uterine blood flow associated with the move from the left lateral to the vertical position.

These haemodynamic findings imply that the cardiovascular system of pregnant women is not perfectly adapted to the upright position, and during evolution its suboptimal efficiency may have become a limiting factor for fetal growth.

Clinical implications

This hypothesis offers a simple explanation of the frequency of intrauterine malnutrition and its social distribution. The faltering of fetal growth at the end of intrauterine life is a phenomenon observed in all populations, but is more pronounced in those of low socioeconomic level.[1] Newborn babies from deprived social groups have a low mean birth weight and have anatomical and histological characteristics similar to older children with malnutrition.[19] As the quality of the diet in a given population is related to its socioeconomic level[20] it has been deduced that fetal growth retardation in underprivileged social groups results from poor maternal nutrition.

To some extent this traditional explanation of the social distribution of intrauterine malnutrition must be true; the fetus cannot receive from the mother more nutrients than the difference between her food intake and what she uses for her own metabolism. Consequently, severe maternal nutritional deficiency will affect fetal growth. This explanation, however, does not cover all the observed facts. Bergner and Susser[21] have pointed out that no survey has ever succeeded in showing convincingly a clear relation between maternal food intake and birth weight, except in extreme conditions, such as in wartime famine[22] or in other grossly undernourished populations.[23] Moreover, this interpretation does not explain why fetal growth falters near term, apparently as a consequence of malnutrition, even in communities in which diets appear perfectly adequate. Finally, in many developing countries with a low mean birth weight breast-fed babies with no other source of food exhibit growth during the first three months of life and often succeed in catching up in relation to the international standards.[24] This phenomenon is difficult to explain if one accepts that poor quality of maternal diet alone limits fetal growth in these populations, particularly as nutritional requirements of breast-fed infants are greater than for the fetus in utero.[25] If an individual's diet is sufficient to allow good lactation, then it should be adequate to support fetal growth during pregnancy. In such communities some non-nutritional maternal factor must be invoked to explain preterm growth faltering.

The hypothesis that fetal growth is limited by the efficiency of the haemodynamics of normal pregnant women, imperfectly adapted to the orthograde position, has several implications. Firstly, it suggests that fetal growth near term is influenced by maternal posture. The uterus is maximally perfused when the mother is lying on her side. The amount of bed rest at the end of pregnancy could thus be a limiting factor in fetal nutrition and growth. Secondly, it suggests that any heavy demand on the cardiovascular system at the end of pregnancy might well further impair mothers' capacity to sustain fetal growth up until birth. The avoidance of heavy sustained physical activity would therefore be necessary to allow normal fetal development.

This vascular interpretation explains why even in populations

where women are not malnourished a small faltering of fetal growth is normally observed just before birth; in the natural course of events no pregnant women remains permanently in the position associated with optimal uterine blood flow.

This hypothesis also offers an alternative or additional explanation for the social distribution of intrauterine malnutrition, which may be explained also by differences in the way of life among the social groups. In the underprivileged social classes in the West, and commonly in developing countries, housework is heavier and opportunities for bed rest are low throughout pregnancy. The pronounced faltering of fetal growth observed in deprived social groups could therefore result from limitations of maternal cardiovascular function quite independent of her nutritional state. This mechanism is quite compatible with the good weight gain of breast-fed babies, which may be observed even when their intrauterine growth, assessed by the mean birth weight, is reduced.

This argument is compelling when considering populations in whom normal growth of totally breast-fed babies occurs. It is highly questionable whether a purely nutritional explanation of intrauterine growth retardation is compatible with successful lactation as judged by good growth in early infancy. Thus good maternal nutrition during pregnancy is not the only prerequisite for normal fetal growth: it is also important that the mother is not subjected to heavy physical demands and is allowed adequate periods of rest in the weeks before birth.

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