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THE NUTRITION OF THE VERY YOUNG

Chairman : PROFESSOR J. L. HENDERSON, M.D., F.R.C.P.E., *Department of Child Health, University of St Andrews*

Nutrition in the neonatal period

By C. A. SMITH, *Departments of Pediatrics and Obstetrics, Harvard Medical School, and the Boston Lying-in Hospital, Boston, Massachusetts, U.S.A.*

My approach to my subject must seem a doubly provincial one to this audience. First, I necessarily look at nutritional science as a paediatrician interested in newborn infants and am incapable of inspecting neonatal life with the eyes of a nutritionist. A more serious handicap must be the limitation of my experience to that minor part of the world with a problem of food surplus rather than scarcity.

If I were allowed to define 'newborn' or 'neonatal', the words would apply only to the first 14 days after birth, in which time the decisive adjustments to extra-uterine circumstances are made. I would want another term to designate the time between birth and the establishment of some positive balances at 3-5 days. May I, then, here use 'newborn' or 'neonatal' for the first 2 weeks and 'early postnatal' for those first few days?

Even in that brief early postnatal period, certain characteristics emerge to differentiate newborn human beings as individuals, and one such characteristic is the general degree of their nutrition. Some infants are obviously undernourished at birth. They weigh much less than most others of their gestational ages, and appear thin rather than merely small. Some of the others are, of course, at the opposite end of the distribution curve and appear to be fat; some appear oedematous. By contrast with these, some have the appearance of dehydration; but thin infants and fat infants differ in another way. If attention is devoted to newborn infants for long enough, one must ultimately face the question of why human foetuses vary so widely in nutritional status even though one's primary concern with them is in their respiration, circulation, or metabolism.

Most attempts to relate the growth or weight of the foetus to the nutrition or diet of the mother, if pursued far enough, have shown some degree of association between the two. Among such investigations, the studies of Burke, Beal, Kirkwood & Stuart (1943) have been the best known to me, and these showed several associations between maternal diet and foetal nutrition. But I believe that Dr Thomson and others working under Professor Baird at Aberdeen found little if any evidence of such relationships (Thomson, 1957).

The belief that a more severe maternal undernutrition than occurs under any usual circumstances would regularly and inescapably affect foetal growth in man stimulated an investigation of the infants born to women living in the larger Dutch cities before, during, and after the 'hunger winter' of 1944-5 (Smith, 1947). Again, the broad general relationship demonstrated by Mrs Burke and her colleagues between ill-fed Boston mothers and their newborn infants (Burke *et al.* 1943) emerged as a tendency to lower average birth weight of infants born during the most severe deprivation, but no constant association between poor maternal and poor foetal nutrition.

My concern over this failure of the human foetus to reflect more consistently the maternal diet was much relieved in 1951 by the diagrams of McCance & Widdowson (1951) showing that whereas the human mother in 280 days has furnished material for growth to about 5% of the maternal weight, the ewe in half the period of human gestation produces a lamb nearly three times as big (with reference to maternal weight), and small laboratory animals provide nourishment far more rapidly for the relatively enormous growth of their gestating young.

Possibly many or all of the requirements for the relatively slow growth of the human foetus might be satisfied by a maternal organism on a marginal or submarginal diet. In that event, the variations of foetal weight commonly encountered suggest that foetal nutrition may be affected by other elements than the maternal diet or stores. We became interested in 'placental insufficiency' as an entity when Clifford (1954) described among the numerous infants who are postmature by dates a small group who appear to have been ill-supplied with food and—sometimes—with oxygen during foetal life. Indeed, we had already been interested in the relationship of presumable placental impairment associated with late foetal anoxia to post-term birth because of Walker's (1954) observations. It soon became apparent that not only the occasional post-term infants may be much too thin for gestational age and for length, but many infants born at term or prematurely have the same discrepancy between expected and actual birth weight. Twenty term infants with placental insufficiency of a considerable degree were described by Rumbolz & McGoogan (1953) who have kindly permitted the reproduction of Fig. 1 from their paper. We have recently seen an extraordinary infant of 41 (or possibly 45) weeks' gestation who weighed 1400 g at birth—a degree of undernutrition greater than any in this diagram. On the other hand, we believe that relatively milder grades of this syndrome of foetal undernutrition are common.

In infants with this appearance, the weight and morphology of the placenta are usually abnormal, especially when undernutrition is severe. But it is by no means always so. Gross and microscopic appearance of the placenta and injection preparations of its vessels are sometimes quite normal (and maternal diet quite satisfactory by history) yet, judged by appearance and measurements, the foetus has been kept from making the full growth expected. In such instances, the uterine blood supply and the vessels of the placental site, which may be called the 'maternal placenta', might be morphologically or functionally abnormal if they could be investigated. Evidence that the fault is not primarily in the foetus itself is furnished by the rapid

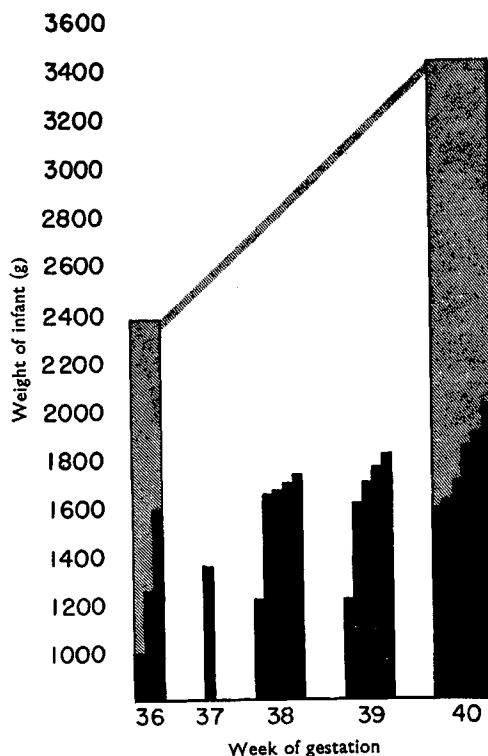


Fig. 1. Birth weights and gestational ages of twenty infants affected by placental insufficiency, as compared with expected normal. (Reproduced with permission of the authors (Rumbolz & McGoogan, 1953) and the Editor, *Obstetrics and Gynecology*.)

accumulation of weight in some of these infants once calories are available to them after birth.

Thus, the nutrition of the foetus must be the product of maternal nutritional circumstances, but is perhaps more directly controlled by uterine and placental factors, not all of which can be inspected at every delivery. This brief mention of the concept of 'placental insufficiency' has considered only the weight of the foetus. It should go without saying that many other and more specific aspects of the nutrition of such foetuses merit future investigation—including the question of whether the post-term foetus ever loses weight *in utero* because of this mechanism. Investigations of the total nutritional state of the foetus and of the size and condition of the foetal placenta may be less difficult than the necessary studies of morphology and function at the placental site. But more may soon be known about this inaccessible area through the enterprise of physiologically minded obstetricians. Until we know how to make all placental mechanisms 'sufficient' rather than insufficient, some well-nourished mothers will give birth to small and poorly nourished foetuses. And some of these will not only be of chronically undernourished appearance but also have signs of recent or current anoxia, so that the nutrition of the foetus is becoming of interest not only for its own sake but also for its implications.

A different, though sometimes related, aspect of nutrition during the neonatal period involves questions of when, how, and on what the newborn infant should be fed. Decisions in this area may have immediate importance, but, though they must be made in the newborn period, they often affect more the subsequent course of the infant.

Perhaps the warning is needed that one such question which could some day require a decision in some maternity hospitals, though it has not yet reached ours, is the spoon-feeding of semi-solid foods to extremely young infants. One school of thought in our country has now brought this practice into the 2nd (and even the 1st) week after birth. Since we appear to export the more unusual manifestations of our culture to other nations (perhaps because they are surplus commodities) people in Britain may find themselves faced with attempts to introduce cereals, pureed vegetables, and even meats, into the neonatal diet over here. The difficulty (as with so many aspects of the adaptable newborn organism) is to prove to its advocates that such early feeding of solids may do harm and can do no particular good. Unfortunately, the mere fact of an infant's doing at a few days an act previously expected at 3–6 months is evidence—to some—that a useful advance has been accomplished. But the significant questions are, of course, those concerning the composition, amounts, and times of the usual milk or formula feedings, and the requirement for supplementary vitamins and minerals.

The usual feeding of newborn infants in my part of the United States will probably today not be with human milk. This fact may simply be admitted and further argument about it be here omitted. Yet one should also mention an evident increase of interest in breast feeding among a rather large group of intelligent urban or suburban young people anxious to participate in the simpler functions of a life steadily becoming more complex. To people whose catchwords have become 'do-it-yourself' and 'togetherness', against powerful forces which cause things to be done by machines, and keep people apart, having babies and nursing them offer increasingly desirable satisfactions. I suspect that a good many of us provide less help in the introduction of breast feeding than young mothers would like, but perhaps we can dismiss that problem and other questions of human-milk feeding now, at least as regards infants born at term. Premature infants, in our hospital at least, have little direct contact with their mothers for some time after birth, so that provision of human milk to them usually means preservation of the milk in some form for gavage or bottle feeding until, at some weeks or months of age, the infant can be put to the breast. That is a rare eventuality with us; indeed in many—perhaps most—premature-infant nurseries in the United States, infants are fed on an artificial mixture of modified cow's milk from the first.

We are fortunate in having a pioneer human-milk bank, the Directory for Mother's Milk, in the Boston Lying-in Hospital, and we feed milk from that source to most of our premature infants for 1–2 months after their birth. In the present day of clean, sterile and standardized dairy milk and other cow's-milk products, and of absence of any considerable danger from diarrhoeal disorders, our leading arguments for it are the reliable digestibility of human milk and the relatively reduced osmolar

load of its electrolytes for renal excretion. The advantages to the infant of the greater nitrogen retentions and somewhat more rapid weight gains apparently afforded by artificial feedings of higher protein content are much debated and require evaluation by long-continued study of growing children rather than short-term balance investigations.

Short-term balances do give interesting suggestions as to the composition of sudden changes of weight occurring when the food of a newborn and, especially, of a premature infant is changed from human milk to an isocaloric formula, or vice versa. Some years ago, when Dr Marc Beem of Chicago was working in our nurseries, he showed that the gratifyingly large and prompt gains in weight frequently noted with introduction of artificial feedings were often accompanied by relative overretentions of sodium, chloride and water. An example from his data—which still await full publication—is given in Table 1. Conversely, after renal adjustment to the increased electrolyte content of formula feeding, return to an isocaloric human-milk intake was often followed by temporary weight loss with negative sodium and chloride balances.

Table 1. *Change in weight and in retention of minerals and nitrogen with change of food of a premature infant weighing 1800 g (data of Dr Marc Beem)*

Age (days)	Food	Intake (m-moles/kg/day)				Retention* (m-moles/day)				Weight gain (g/day)
		Sodium	Chloride	Potassium	Nitrogen	Sodium	Chloride	Potassium	Nitrogen	
26-37	Human milk	1.2	2.3	2.7	0.4	0.66	0.92	1.2	0.33	19
38-40	Half-skim milk mixture	4.3	6.6	8.9	1.0	6.3	6.6	5.5	1.2	78
38-46						3.3	4.5	3.4	1.05	55
47-49	Human milk	1.0	2.0	2.5	0.33	Loss	Loss	Loss	0.27	Loss
47-56						Loss	Loss	0.7	0.35	16

* Retention = intake - excretion in urine and stool.

Such data can neither be quite disregarded because of some inconsistencies of sodium and nitrogen retentions, nor can they be confidently extrapolated as evidence of differences which will alter tissue composition. The error of mistaking what is largely water retention for gain of solid weight usually becomes apparent as readjustments occur in the course of time. But potentially more dangerous to our thinking about growth of infants is the error of assuming from changes of mineral or nitrogen retentions in short balance periods, that the changed retentions will be continued indefinitely, so that what was called 'supermineralization' by Rominger & Meyer (1927) may be forced upon the growing infant, as by high intakes of calcium and phosphorus with vitamin D. Wallace & Taylor (1956) have recently found that widely different food intakes do not build differences of composition into the lean body mass of young and growing rats. They believe that neglect of skin losses in balance periods and failure to recognize changes of fat content as the body grows are the major reasons for misinterpretations. Admittedly, some unmeasured factors of insensible losses are lacking from Table 1.

Scepticism as to differences of mineralization during growth does not, of course,

allow us to disregard the iron intake of infants. However, we have not given supplements of iron to the term infants under our care during a week or two after birth, most of whom will be receiving an iron-containing cereal or other solid food by 3 months later. And we now rarely give iron to premature infants even if they stay 2 months in the nursery. These were our principles even before we had the opportunity to follow the iron received transplacentally by a group of normal infants (Smith, Cherry, Maletskos, Gibson, Roby, Caton & Reid, 1955), when our colleagues employed ^{55}Fe for measurement of red-cell volumes and hematocrits of women during pregnancy. The total haemoglobin of the sixteen infants born to those

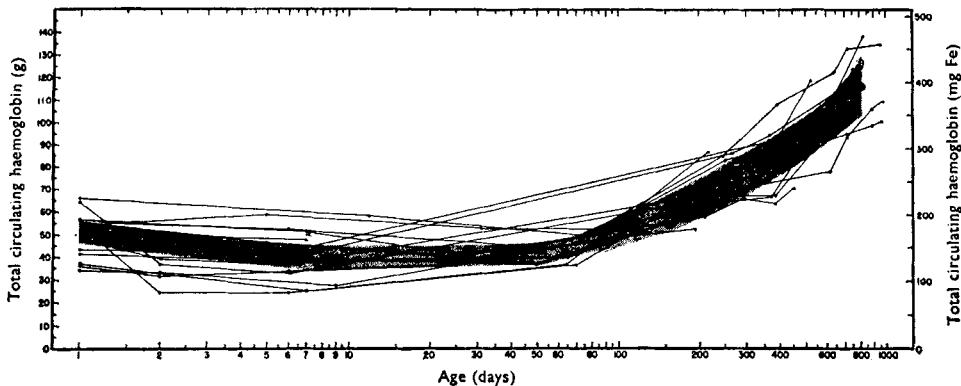


Fig. 2. Total circulating haemoglobin of sixteen infants up to 32 months after birth. The lines connect consecutive values for each infant. Shading indicates area bounded by two standard errors above and below the best curve through the mean values. (Reproduced with permission of the authors (Smith *et al.* 1955) and the Editor, *Journal of Clinical Investigation.*)

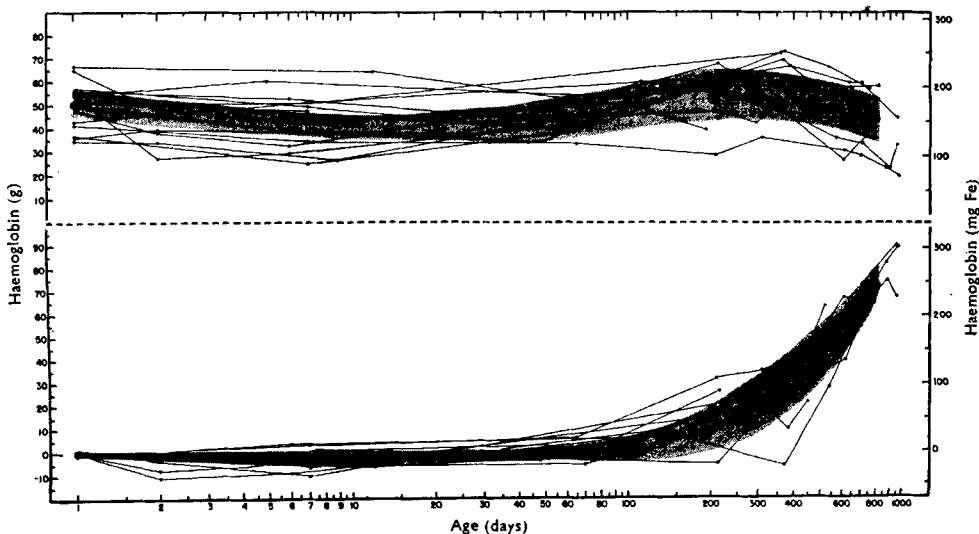


Fig. 3. Upper: haemoglobin from transplacental iron of infants described in Fig. 2. Lower: haemoglobin from dietary iron. Meaning of lines and shading as in Fig. 2. (Reproduced with permission of the authors (Smith *et al.* 1955) and the Editor, *Journal of Clinical Investigation.*)

mothers (and its iron content) took the course shown in Fig. 2, but, as shown in Fig. 3, the data indicated the first appearance of iron from the infants' feeding in their haemoglobin at about 100 days after birth. After that, of course, dietary iron continued to be used for haemoglobin, but the relatively steady conservation of what might be called 'birthright iron' during the nearly 3 years we could follow some of these infants interestingly substantiated what might have been predicted. Although these data indicate that deficiency of iron stores should not occur by 3-4 months, they do not deny a possible haematopoietic effect of iron administered to infants before their transplacental stores are at all depleted. Gairdner, Marks, & Roscoe (1955) have published evidence that supplemental iron given from 3 or 4 weeks after premature birth may cause such an effect at 50 days, long before transplacental iron stores of premature infants would be inadequate for manufacture of their haemoglobin.

Our experiences with the vitamin requirements of newborn life may be inferred from the fact that we give rather smaller doses of a smaller number of supplemental vitamins than we did 15 or 20 years ago. Late in the 2nd, or sometimes in the 3rd, week daily dosage is begun with a concentrate providing 50 mg ascorbic acid and 1000 i.u. vitamin D. The preparation currently used also contains 5000 i.u. vitamin A, reportedly because the manufacturers find it as easy to leave it in as to take it out.

These practices seem, in our favoured socio-economic circumstances, to be matters of nutrition unlikely to decide whether the newborn infant will survive or to alter the composition of his growth very greatly if he does so. If one may consider anything regularly given to an organism—newborn or not—as nutrition, our greatest concern with nutrition of the newborn has been with the earliest postnatal feedings, which are essentially water. For years we have thought that a delay of 3 days—or even longer—before giving fluids to a premature infant might involve less risk than the danger of vomiting and aspirating an earlier and probably unnecessary intake.

In a recent publication (Smith, 1957) I have taken up various challenges to this practice. We ourselves are beginning to wonder whether the dangers of aspiration of feedings given by skilled nurses are as great as we once thought. The question of the harm, if any, involved in postnatal water loss to the extent of temporary haemoconcentration interests the paediatrician so much that he hopes for any information the nutritionist—human or veterinary—can bring to its solution.

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