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NUTRITION IN INFANCY

Chairman, Professor L. G. PARSONS

Nutritional Needs of Infancy

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Introduction

The views of babies on their own nutritional requirements are perhaps insufficiently considered, and the following quotation may, therefore, be of interest: "Dr. Clara Davis has allowed 12 babies from 6 to 8 months of age, who had never had any food other than breast milk, to select their own diet for a period of years from a large assortment of natural, uncombined, unsophisticated and unseasoned foods, such as we ordinarily associate only with an adult or an older child. Anorexia is here unknown except when sickness intervenes. Anyone who has experienced the thrill of seeing these perfect physical specimens sit down in silence to a meal, with an abandon that harks back to the primitive, must realize that the child has much to teach us about the dietetics of childhood. Anyone who sees one of these children eat ten eggs at one sitting, or seven helpings of potato, or, at six months of age, make a whole meal of chopped raw lettuce, or drink almost no milk for months (all actual experiences), and yet present an optimal state of nutrition as checked by all known methods, is less apt to allow 'one egg or two small patties of meat a week' at one year of age, or insist on 'a quart of milk or more a day' for all children, whether they want it or not. Dr. Davis has shown quite clearly that a consideration of food amounts, vitamins and calories alone is not sufficient for an understanding of all the factors governing nutrition." This quotation comes from a volume (White House Conference, 1932) mainly devoted to a scientific consideration of food amounts, vitamins and calories, and it certainly does not minimize their importance. The feeding of infants is still largely empirical. Certainly my own scientific knowledge of the minimum or optimum requirements during infancy of this or that constituent of the diet is very scanty, and both on that account and because this audience has such diverse interests, this subject has been a difficult one to tackle.

Some scientists in laboratories are inclined to ask why paediatricians have not filled in more of the gaps in our knowledge. May I put another question? "How many members of the Society would offer their babies as experimental animals for a study of the infant's requirements of protein or amino-acids?" Themselves, they might offer, but not their babies. The types of experiment permissible with babies are limited.

Three subjects of which our knowledge is almost entirely empirical loom large in infant feeding: (1) The baby's tolerance of different foodstuffs

and their varying digestibility. Sweetened condensed milk, for instance, which by scientific standards is an ill balanced diet, proves temporarily of great value because it is well tolerated; (2) The importance of gradualness, and the possibility of educating babies to tolerate many sorts of foods if started gradually; (3) The way in which psychological changes affect nutrition; for example, a baby of 5 to 7 months of age will take to "table food," as it is called, like a duck to water, whereas the baby kept on a milk diet till 10 or 12 months of age usually disapproves of nearly all solids, thereby causing acute family worries.

Individual Constituents of the Diet

During recent years two eminent official bodies have reported on the quantities of individual constituents of the diet needed by babies. Some of their data are shown in Table 1. The Technical Commission on Nutrition of the League of Nations in 1935 and 1937 attempted to define the actual requirements of babies (League of Nations, 1936, 1938). The blank spaces left in the table illustrate eloquently what was considered to be unsettled or unknown. In 1941, the Committee on Food and Nutrition of the U.S.A. National Research Council (Dann and Davison, 1942; Butler, 1942) laid down, not minimum or optimum requirements, but allowances which they would recommend in the present state of knowledge; from the standpoint of the clinician, this is a wise method of approach. The recommendations of these two bodies show certain differences. The League of Nations Commission gave smaller allowances of calories, vitamin D, vitamin C and, especially, vitamin B_1. A comparison of the allowances advised by the American Committee with the amount of, say, protein and calcium present in one pint of human milk (see Table 1), makes it clear that the Committee was thinking in terms of the artificially fed infant, or of one receiving mixed feeding.

Calories. Both bodies recommend for young babies an allowance of 45 Calories per lb. bodyweight, which is lower than that considered desirable for healthy young infants by most paediatricians. Perhaps this allowance was based on the needs of babies unable to move freely because subjected to the unnatural restrictions inherent in metabolic experiments, whereas our views as paediatricians are based on estimates, rough it is true, of the number of calories consumed by healthy active babies, growing normally, and out of doors daily. On such a basis, Holt and Fales (1921, 1) found that the Calorie intake per lb. decreased from about 55 for very young infants to about 45 towards the end of the first year. The American Committee allows 45 Calories per lb. throughout the first year. Thin and premature babies require a much larger calorie allowance per lb. bodyweight, even up to 75 Calories. Babies wholly bottle fed in the first week of life usually make satisfactory progress on an allowance of one-seventh of 50 Calories per lb. bodyweight on the first day, increasing by the same amount daily till the intake is 50 Calories per lb. bodyweight on the 7th day. The breast fed baby takes considerably less than this during the first two days of life (Mackay, 1941).

Protein. The protein allowance given by the American Committee, 3 to 4·g. per kg. bodyweight, or 1·4 to 1·8 g. per lb. bodyweight, is one very frequently allowed for babies on artificial feeds (Holt and Fales, 1921, 2). On this basis, about 15 per cent. of the baby's calorie intake

TABLE 1

NUTRITIONAL ALLOWANCES FOR INFANTS UNDER 12 MONTHS OF AGE

	Calories	Protein g.	Ca g.	Fe mg.	Vitamin A I.U.	Vitamin D I.U.	Ascorbic acid mg.	Vitamin B_1 mg.	Riboflavin mg.	Nicotinic acid mg.
Requirements: League of Nations (1936, 1938) Infant under 6 months	100 per kg. 45 „ lb.	—	—	—	—	400 to 500	5 to 15 as supplement to artif. food	0·045 per 100 Calories	—	—
Infant over 6 months	90 „ kg. 41 „ lb.									
Baby of 8 lb. ..	360					400 to 500	5 to 15 „	0·16		
Recommended allowance: Committee on Food and Nutrition, U.S.A. Nat. Res. Council (Dann and Davison, 1942; Butler, 1942) Infant up to 12 months	100 per kg. 45 per lb.	3 to 4 per kg. 1·4 to 1·8 per lb.	1·0	6	1500	400 to 800	30	0·4	0·6	4
Baby of 8 lb.	360	11 to 14	1·0	6	1500	400 to 800	30	0·4	0·6	4
Quantity in 1 pint human milk	390[1]	7·1[1]	0·2[1]	1·1[1]	1140 to 2850[2]	14 to 100[3]	7 to 62[2]	0·09[4]	0·08 to 0·3[1]	under 0·6 ?[1]
Quantity in 1 pint cow's milk	380[5]	18[5]	0·7[5]	0·4[5]	1436[2] (pasteurized)	0 to 57[2]	2 to 16[2]	0·2 to 0·4[2]	0·6 to 1·7[2]	0·6 to 1·8[1]

Authorities: [1] Marriott and Jeans (1942). [2] McCollum, Orent-Keiles and Day (1939). [3] Drummond, Gray and Richardson (1939). [4] Knott, Kleiger and Schlutz (1943). [5] McCance and Widdowson (1940).

would be supplied as protein. This is much more than a baby can get if breast fed. Only about 7 to 10 per cent. of the breast fed baby's calorie intake is derived from protein, and it receives perhaps about 1 g. protein per lb. bodyweight daily. The bottle fed baby usually gets all its protein from cow's milk, but part of the carbohydrate and sometimes some of the fat is derived from other sources. A protein allowance of 3 to 4 g. per kg. generally implies that in practice nearly two-thirds of the bottle fed baby's total calorie intake is derived from cow's milk. The casein of cow's milk is considered of lower biological value for the infant than the lactalbumin of human milk, because the amino-acid composition is different; for example, breast milk protein contains a higher proportion of the sulphur containing amino-acids, but whether a protein allowance 50 per cent. greater is necessary for the baby given cow's milk is doubtful. It should not, however, be forgotten that the clinician's custom of supplying a quantity of cow's milk which will provide a liberal allowance of protein to bottle fed babies is based in part on evidence of malnutrition found in babies given for a long period a food such as sweetened condensed milk, which supplies only a little over 1 g. of protein per lb. bodyweight. It is obvious that if only about half the energy value of the food is derived from milk and the rest from refined sugar, as in sweetened condensed milk, and no supplements of vitamins or minerals are given, the possibilities of deficiency are numerous, and malnutrition, if it is present, need not be the result of protein deficiency.

Recent knowledge of the association of the B vitamins with protein is an additional reason for insisting on a liberal protein intake for bottle fed babies and, as the protein content of cow's milk is high, this is easily supplied. The milk of many other species is much richer than human milk in protein (Powers, 1925), possibly because their young grow much more rapidly than human babies, and it has been suggested that the protein in human milk may prove inadequate for a baby gaining weight especially rapidly. A premature baby of $3\frac{1}{2}$ lb. birth weight, making satisfactory progress, gains weight in relation to its birth weight twice as quickly as a full term baby of 7 lb. at birth. If fed on breast milk, it consumes probably 40 per cent. more milk per lb. bodyweight than the full term baby, and derives from it nearly 1·5 g. protein per lb. bodyweight. The practice of giving a casein supplement to premature babies fed on breast milk to provide extra protein is very unusual in this country though it has been advocated abroad, and my own observations on the good progress of premature babies on breast milk, and their frequent intolerance of artificial feeds, convinces me that it is unnecessary and undesirable as a routine measure.

Calcium. No minimum calcium requirements for infants can be given because the variables are so great. The baby's need of calcium depends on many factors, including the rate of growth, the amount of vitamin D available, and the quantity of calcium precipitated in the gut by other fractions of the diet, such as the phytic acid of cereals and the oxalic acid of vegetables and fruits. In a full term foetus the long bones have more cancellous bone and smaller marrow cavities than in the young child, and the baby can draw on some of this cancellous bone as a calcium store (Eliot and Park, quoted by Stearns, 1939). Stearns (1939) calculates that, with average calcium retention, the calcium content, reckoned

as a percentage of bodyweight, of a normal breast fed baby, diminishes during the first three or four months of life, and that, for the premature baby fed on breast milk, the fall in percentage calcium content is still more striking. The baby given cow's milk supplemented with vitamin D may retain an amount of calcium considerably in excess of that which could have been derived from breast milk; the result has been called "hypermineralization". Some have considered that an actual calcium deficiency is common in breast fed infants, and the difficulty of preventing rickets in many rapidly growing premature babies by administration of vitamin D certainly suggests that this may be the case.

The calcium allowance of 1 g. daily, advised by the Committee on Food and Nutrition, seems very liberal; it is perhaps 5 times as much as is contained in a pint of breast milk. About one and one-third to one and a half pints of cow's milk daily would supply this allowance.

Vitamin D. The vitamin D requirement in the diet varies from nil for the baby adequately exposed to ultraviolet light, to perhaps something in excess of 10,000 I.U. daily for some young premature infants growing rapidly (Park, 1940). Even in winter in our climate, outdoor life gives a measure of protection against rickets, and severe rickets is seldom seen in a baby taken out of doors regularly throughout the winter. The vitamin D content of human milk naturally gives no hint of the baby's total requirements, since the main natural source of this vitamin is that produced by the action of ultraviolet light on the child's skin. Professor Noah Morris who has investigated the relative efficacy of vitamins D_2 and D_3 for the cure of rickets in young children will, I hope, discuss some of the many factors concerned in the vitamin D requirement.

I should like to emphasize the need of starting vitamin D early, particularly for premature babies who often develop craniotabes by the 2nd month of life, and the importance, when dealing with large numbers of infants who are not under strict medical supervision, of giving as a routine a dose likely to protect under all circumstances, since a period of indoor life during an illness, followed by a spurt in growth, or the omission of the dose for some weeks, may cause a baby to be short of this vitamin. The Ministry of Health's recent rate of dosage which allowed a maximum of perhaps 200 I.U. daily for babies under 6 months of age was certainly dangerously low (Mackay, 1942) but, fortunately, in very many areas, infant welfare medical officers ignored the official dosage and gave much larger amounts from other sources.

As a result of the memorandum of the British Paediatric Association (1943), I understand that the vitamin D value of the official "cod liver oil compound" will be doubled, and the official doses will in future be 700 I.U. daily for full term babies, and 1400 I.U. for premature babies. When it is desired to give very large doses to premature babies, more concentrated preparations must be given. The vitamin D of very concentrated preparations is probably poorly utilized, and this must be borne in mind when such preparations are used.

Vitamin A. Estimates of the vitamin A value of human and cow's milk show wide variations, and little is definitely known concerning the infant's requirements. With the values found by Guilbert, Miller and Hughes (1937) for young animals as a basis and, in their own phrase, the "desirable minimum set for practical purposes" for a 10 lb. baby

lies between 450 and 1350 I.U. daily. Infections appear to increase the amount required. It will be interesting to hear from Dr. Kon how this estimate compares with the vitamin A values for the breast milk he has examined during the past year. I have given babies dried roller process milk as their sole source of vitamin A and compared them with controls getting liberal supplements of vitamin A (Mackay, 1934). As far as I could compute from figures kindly supplied me by Dr. Coward and by Dr. Carr, the vitamin A value of this dried milk averaged about 1000 I.U. per reconstituted pint. The controls getting extra vitamin A showed no superiority over the first group in any respect, except that they suffered on the average from a smaller incidence of minor skin infections, which I ascribed to a deficiency of vitamin A. However, on repeating the observations on other groups of babies, even this difference disappeared (Mackay, 1939). I imagine, therefore, that the baby's needs should be amply covered by an allowance of 1500 I.U. daily. The official cod liver oil compound in the new dosage will, I understand, provide about 3500 I.U. of vitamin A daily.

Vitamin B_1, Riboflavin and Nicotinic Acid. As far as I know, the evidence regarding the infant's requirements of vitamin B_1, riboflavin or nicotinic acid is scanty, and not much is known concerning the range of concentration of these substances in human milk. The values for cow's milk in Table 1 are quoted from McCollum, Orent-Keiles and Day (1939) and from Marriott and Jeans (1942), and those for human milk from Knott, Kleiger and Schlutz (1943), and from Marriott and Jeans (1942). Knott, Kleiger and Schlutz (1943) consider that the infant's needs of vitamin B_1 are not met by the quantities often found by them in breast milk, and the values for riboflavin and for nicotinic acid in breast milk given by Marriott and Jeans (1942) would not provide the infant with the allowance of this vitamin advised by the Committee on Food and Nutrition (Dann and Davison, 1942; Butler, 1942).

Ascorbic Acid. Thirty mg. of ascorbic acid daily, the quantity advised by the American Committee, sounds a liberal allowance for an infant. It would be contained in, say, 2 oz. of orange juice. I hope Dr. Harris will tell us how much of this, given daily, would be immediately excreted in the urine and, presumably, what spills over at once into the urine is in excess of normal needs. However, it seems that many illnesses increase the normal requirements. Hess (1920) pointed out, for example, that an attack of influenza would precipitate overt scurvy in a baby on a borderline diet. Recent work has shown that vitamin C is concerned in the metabolism of certain amino-acids in premature babies and probably in other infants also (Levine, Gordon and Marples, 1941). No doubt considerations such as these influenced the American Committee to recommend a liberal allowance of vitamin C.

Iron. A young baby has two sources of iron on which to draw besides the very small amount present in milk; these are the iron stored in the liver at birth, and that derived from the breakdown of superfluous red cells after birth. Nevertheless, a subnormal haemoglobin level which can be cured by liberal administration of iron develops during the first year of life in a majority of babies. The more we learn about iron metabolism the more puzzling the problem becomes. The presence of 6 mg. of iron daily in the food, as advocated by the American Committee, cannot be relied on

to prevent nutritional anaemia. By giving soya bean flour with dried milk I have supplied bottle fed babies with 6 to 12 mg. of food iron daily (Mackay, 1940). This quantity of iron, though it prevented anaemia in a majority of babies did not protect all, and a few developed severe anaemia which was cured by giving iron as a drug. Dr. McCance and Miss Widdowson tell me that there is a considerable amount of phytic acid in some soya flour. Elvehjem, Siemens and Mendenhall (1935) found that 12½ mg. daily of iron as ferric pyrophosphate given with copper did not give uniform protection but, when the dose of iron was increased to 25 mg., nutritional anaemia was prevented; 25 mg. daily is, however, a quantity it would be impossible to supply to babies as food iron. There is evidently some key we lack. What is it that will allow a baby to utilize properly such amounts of iron as can be supplied in the food? Till we know this, we should do well to give all babies medicinal iron and thus prevent this type of nutritional anaemia. In addition we should certainly supply food rich in iron during the second half year of life.

Clinical observation as well as metabolic work on infants (Oldham, Schlutz and Morse, 1937) suggests that meat is a valuable source of food iron. The babies who were allowed by Davis (1928) to choose their own food evidently approved highly of meat. The iron of egg is easily utilized, but one yolk supplies only about 0·5 mg. On the other hand, the iron of the much vaunted spinach is poorly utilized (Stearns and Stinger, 1937; Schlutz, Morse and Oldham, 1933; Oldham, Schlutz and Morse, 1937), and the iron of wholemeal bread, as shown by Widdowson and McCance (1942) is precipitated and not absorbed. There is only one foodstuff with which I have produced rapid cure of nutritional anaemia in babies and that is liver; I gave about a quarter of a pound daily, supplying perhaps 16 mg. iron, to a few babies of over a year with rapid cure of their anaemia (Mackay and Goodfellow, 1931).

Practical Methods of Assessing the Nutritional Needs of an Infant

A baby, particularly a young baby, suffers much more quickly and severely from shortage of water than an older person, and its fluid needs are large. The work of Young and McCance (1942) has once more focussed attention on this need. The young baby cannot concentrate its urine, it cannot eliminate a large quantity of acid or base in a small volume of water. A baby fed at the breast gets with its food during the first 5 months of life, about 2½ oz. of fluid per lb. bodyweight in 24 hours; thus a ten pound baby gets about 25 fluid oz. in the day, and most babies get and appreciate more than this allowance of water, especially in hot weather. A ten stone adult on the same basis would receive 17½ pints of fluid daily. After 6 months of age the need for fluid diminishes, and the infant's desire for water is a good indication of its needs.

Perhaps the commonest of all feeding errors is underfeeding. It is surprising how often symptoms of hunger go unrecognized in breast fed and bottle fed infants. Hence arises the great importance of comparing a baby's actual intake of calories with its theoretical requirements. In order to simplify the calculation for bottle feeds, I, personally, have them all made up to provide approximately 20 Calories per fluid ounce, which is the calorie concentration of breast milk or cow's milk. Though I constantly check a normal baby's daily calorie intake against its

theoretical requirements, I rely to a great extent on appetite and rate of gain in weight as the ultimate criteria of its quantitative needs and, after mixed feeding has started, these remain as a rule my sole criteria.

The term "nutritional needs" of infancy, besides covering the quantitative balance of the diet, also extends to cover digestibility, a matter of prime importance to the baby. In our present state of knowledge, I believe that the superiority of breast feeding to artificial feeding is due chiefly to two factors: greater digestibility of the milk and less risk of infection through the introduction of pathogenic organisms. I will not attempt to consider how more breast milk can be provided, for the causes of the unsatisfactorily low incidence of breast feeding are manifold, including many conditions interfering with the mother's serenity of mind and health of body. Propaganda in favour of breast feeding can achieve little unless fundamental causes are tackled.

Dr. Kon may perhaps give us some figures indicating how breast milk is influenced by the mother's diet, and show us, for example, how the vitamin C content has fallen to disturbingly low levels during the last winters.

Normal babies, fortunately, can be trained to tackle and tolerate many sorts of food, provided the introduction is gradual. Gradualness is a very important key to infant feeding. Many years ago, I remember Sir Robert Hutchison saying he would undertake to teach a baby to eat beefsteak, and his audience laughed at this startling statement. Nowadays many of us regularly train babies from $5\frac{1}{2}$ to $7\frac{1}{2}$ months of age onwards to eat beefsteak, duly minced, as well as fish, eggs, vegetables and fruit. The babies in the Davis experiment who chose their own food, ate much meat and fruit, but did not appreciate spinach, lettuce or turnip. Their milk intake seldom averaged more than a pint daily.

What is the physiological age for starting mixed food? There is no use studying the habits of African tribes or Pacific Islanders to find out what is normal or natural in this respect. I once had the hopeful idea of getting a lead from the apes, but a bibliographical hunt and enquiries from the Natural History Museum and the Zoological Society led to the conclusion that no one knew the age at which ape mothers start their babies on solids, or what these solids may be. Ten months is definitely too late to begin offering solid food to human babies, for by that age most infants will not readily adopt a mixed diet. Glazier (1933) has produced some interesting figures which bear out another experience of paediatricians. He showed that babies kept on a plain milk diet until late in the first year are retarded in development as compared with babies starting other foods at about 3 to 6 months of age. Possibly by 6 months of age the breast fed baby is running short of other substances besides iron. Personally I start babies on solids by $5\frac{1}{2}$ to $6\frac{1}{2}$ months of age, and by $7\frac{1}{2}$ to 8 months I aim at giving them about a pint of reconstituted, full cream, dried milk daily, meat, fish, egg or cheese at two meals in the day, as well as fruit or fruit juice as available, and small quantities of potato, vegetable puree and cereal. By 12 months of age I advise that they should have their entire meat ration, say 6 oz. a week. Many babies appreciate a little crisp bacon and one or two ounces of cheese in the week. During the first year of life special precautions are necessary to ensure a sufficient supply of vitamin C, vitamin D and iron. The

Government issue of concentrated fruit juice and cod liver oil compound, will, we hope, soon provide enough of the first two. Iron I habitually give in the form of iron and ammonium citrate, 4½ grains daily, providing about 100 mg. of iron. This I give regularly from 2 to 12 months of age to all babies whether breast fed or bottle fed, and the evidence that the babies are better in health as a result of an iron supplement is, I think, complete (Mackay and Goodfellow, 1931).

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Discussion

Professor N. Morris (Department of Materia Medica and Therapeutics, The University, Glasgow), opener: The assessment of optimum nutritional requirements is beset with innumerable difficulties clearly illustrated in the special problem of vitamin D. In actual fact the minimum requirement of this factor for infants and children is not known and there is considerable divergence of opinion as to what are the early indications of deficiency. Requirements vary according to growth activity, supply of calcium and phosphorus, exposure to ultraviolet light, and the immediate

past history of the patient with special reference to infection; nutrition is obviously much more than food.

The following questions should be considered: (1) What is the minimum dose of vitamin D which will protect the majority of infants? The answer is likely to be of some importance in view of the probable scarcity of supplies in the post-armistice world. (2) Is it possible to protect all infants? This can be answered in the negative. A committee of the British Paediatric Association (1943) recommends 700 I.U. daily, which will certainly protect the majority of full term infants, and 1400 I.U. which will suffice for many premature infants. It is probable that in time of scarcity much less will suffice for full term infants, especially if they are taken out of doors for sufficiently long periods. At the moment, however, with an ample supply of vitamin D it is not wise to make any allowance for seasonal variations as this tends merely to confuse mothers and health workers. It is doubtful whether there is any real danger of excess in the clinical use of vitamin D. Calcinosis, reputed to be produced by excess of vitamin D, was reported long before the vitamin was dreamt of and large doses of vitamin D have been used in the treatment of tuberculosis and rickets without any ill effects. Idiosyncrasy to vitamin D may exist but it then appears with minimum as well as large doses. There is little evidence of differences in potency between various forms of vitamin D. Comparison of the healing of rickets in infants of the same age and with the same degree of growth activity, treated with vitamin D_2 and vitamin D_3, showed no difference in the rate of healing judged by clinical, radiological and biochemical tests (Morris and Stevenson, 1939).

I should like to suggest that The Nutrition Society might make recommendations as to optimum and minimum requirements of vitamins, particularly vitamin D, in view of the fact that although optimal amounts are desirable, the world shortage may be such that only minimal amounts should be allowed.

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Sir Joseph Barcroft (Physiological Laboratory, Cambridge): The recently formed Committee on Nutrition Surveys of The Nutrition Society could possibly deal with Professor Morris's suggestion.

Dr. S. K. Kon (National Institute for Research in Dairying, University of Reading): Dr. Mackay asked about the vitamin A content of breast milk; we find that full lactation milk contains some 35 I.U. per g. fat, which is equivalent to about 700 I.U. per pint. In the last two years Dr. Mawson and I have examined several hundred samples of milk and have found for other vitamins the following mean values per pint: vitamin B_1 85 μg. (28 I.U.); vitamin C 14 mg. in winter and 26 mg. in summer; riboflavin 140 μg. It is of interest that human milk contains only about one-sixth of the quantity of riboflavin present in cow's milk.

Dr. L. J. Harris (Dunn Nutritional Laboratory, Cambridge): In reply to Dr. Mackay's inquiry about vitamin C, I agree that the proposed allowance of 30 mg. is quite generous and sufficient to keep an infant in a fairly high degree of saturation, so that if any more is given, as in a

test dose, most of it will be immediately excreted. To prevent possible misunderstanding, however, I should like to emphasize that the fact that *some* of the vitamin is being excreted daily in the urine does not necessarily mean that enough of it is being taken in the diet, as has sometimes been erroneously supposed. Even with very low intakes of a water soluble vitamin, or with none at all, small amounts may still continue to be excreted for some time in the urine; as more is given, relatively more is excreted and, with large doses most of it is excreted. It is not, therefore, a matter of the kidney threshold but rather of a gradually increasing measure of excretion as the tissues become more saturated with the increasing intake. It cannot be argued, therefore, that, because some of the vitamin overflows into the urine every day, the subject is necessarily receiving a surplus, any more than it can be argued that a continued excretion of water in the urine denotes an adequate water intake. What can be concluded is that when that level of intake has been reached at which *most* of the vitamin begins to be excreted, these being the circumstances in which the plateau of saturation is reached on the very first day of test dosing, then the optimum requirement has also been reached or passed. The optimum may be lower than this; one cannot say how much lower. It is certainly not possible, on the other hand, to argue about optimum requirements from minimal excretions in the urine.

On the more general question of requirements, I agree with Dr. Mackay that it is wise to allow a generous margin. There is growing evidence from experiments with animals and from human experience, that the optimum intake is higher than the mere minimum needed to prevent deficiency disease.

Professor A. C. Frazer (Department of Pharmacology, Hospitals Centre, Birmingham, 15): Professor Morris stated emphatically that hypervitaminosis D does not exist. If he means that such a condition does not readily occur with ordinary therapeutic doses, that is one point. There is no doubt, however, that in controlled animal experiments hypercalcaemia and death can be induced with large doses of vitamin D. It has been shown also that other vitamins may be toxic in large doses.

Professor H. P. Himsworth (University College Hospital Medical School, London, W.C.1): I can support Professor Morris in his criticism of the conception of hypervitaminosis D. I know of a child with hypoparathyroidism who for the last 10 years has never taken less than half a million I.U. of vitamin D a week, and often a million and a half. If vitamin D produces toxic symptoms *per se* then one would expect that such doses over such a period would elicit them. Actually no ill effects have occurred at any time; particularly there has been no evidence of metastatic calcification. It would appear that the toxic symptoms attributed to vitamin D are due to hypercalcaemia and differ in no way from those associated with hypercalcaemia arising from other causes. The inference is that it is safe to give any amount of vitamin D provided hypercalcaemia is not produced.

Mr. E. C. Wood (Virol, Ltd., Hanger Lane, London, W.5): More exact and detailed analyses of human milk should be obtained as the best available means of determining the nutritional needs of the infant,

provided we start, as I believe we should, from the hypothesis that the milk of mothers whose own diet is adequate in all respects is completely sufficient for the proper health and development of their young up to the normal age of weaning.

If analytical work on human milk is to be useful it should satisfy the following conditions: (1) The samples drawn must be truly representative of the entire contents of the breast in view of the known differences between foremilk and strippings. (2) The mother's diet must be investigated since it is important to know whether the normal concentration of a given factor in milk can be markedly raised or lowered by alterations in the maternal diet.

As one instance of the queries which await an answer, I need only refer to the amount of iron, about 1 mg. daily, which appears to be supplied in human milk, in comparison with the amounts, 50 to 100 mg. daily, which have been suggested as necessary in order to avoid anaemia in infants, A possible explanation is that this anaemia is physiological not pathological.

The Diagnosis of Nutritional Disorders in Infancy

Acting Wing Commander R. W. B. Ellis, R.A.F.V.R. (The Glebe House. Hawridge, Berkhamsted, Herts.)

Nutritional disorder accounts for, or plays a part in, the majority of diseases occurring in infancy. It may be due primarily to infection, enteral or parenteral, to metabolic disorders, such as coeliac disease and diabetes, to environmental factors, or to diet. The diet may be deficient in total calories; it may be deficient in more than one factor, or it may be excessive and, though many healthy babies have been known to eat with impunity coal or sawdust, or to drink vintage port, the diet may contain gastro-intestinal irritants which will precipitate profound nutritional disorder. Mechanical and environmental factors are almost equally important. Swallowing of air which causes distension and vomiting is likely to occur from prolonged sucking on an empty breast or the use of a bottle with a hole in the teat too small to allow a free flow of milk. The former will produce a "breast shy" baby who screams and refuses to suck when put to the breast. This condition has been ingeniously overcome by use of Russell's ingenious device for supplementary feeding by which a bottle is suspended from the mother's shoulder and a soft rubber catheter leads from this to the nipple; the baby thus stimulates lactation and obtains an adequate feed. Mental disturbance and restlessness in the mother will usually have a direct effect on the nutrition of the infant and, whilst the sudden loss of weight so often seen in breast fed babies after an emotional shock to the mother is largely due to diminished lactation, it is also increased by restlessness of both mother and infant during breast feeding.

The diagnosis of simple underfeeding rests on observation of the state of nutrition and behaviour of the baby, examination of the weight chart, the results of test feeding in breast fed babies, and calculation of the food formula in bottle fed babies. I should like to emphasize the order in which these three diagnostic factors have been placed. An acutely hungry baby will be fretful and the sleep rhythm of both the baby and its

parents will be interrupted; the baby will tend to gobble its feeds, with consequent air swallowing and often vomiting. If the feed is further diluted, as is often misguidedly done, all the symptoms will be increased. In more severe cases of malnutrition, there will be obvious loss of subcutaneous tissue and elasticity of the skin, and general apathy.

The keeping of a weight chart may easily do more harm than good if it is not intelligently done, since no baby will gain weight with mathematical regularity. Although the average gain is just over 4 oz. a week during the first year of life, the normal variation is considerable, depending to some extent on the initial weight loss after birth, and the activity of the baby. During the second year, the average gain is only approximately half this amount, and the variation greater. Thus a fat inactive baby who weighs 28 lb. at a year old, may be expected to remain almost stationary or even to lose weight for one or two months as it becomes more active and learns to crawl or toddle. Except with very small babies, or where there has been some definite feeding difficulty or illness, daily weighing in the home is to be strongly discouraged as likely to spread alarm and despondency. Weekly weighing is sufficient for healthy babies in the first year, and usually monthly weighing in the second year of life. Failure to gain weight or loss of weight for more than a week during the first year, in the absence of obvious cause such as weaning or illness, should raise the suspicion of underfeeding.

Test feeding by weighing the clothed infant immediately before and after a breast feed, will give the number of ounces of milk ingested at that particular feed. An isolated estimation, however, is of little value, since there may be a difference of at least 100 per cent. between the amounts taken at the first morning and the midday feed. Ideally, therefore, the test should be made at each feed for 24 hours to obtain an accurate estimate of the daily milk intake. Similarly, fat estimations on isolated samples of breast milk are apt to be most misleading, unless the breast is completely stripped and the pooled yield analysed.

It is extremely rare for a nutritional disorder to be caused by the nature of the fat, protein or carbohydrate content of human milk, though under urban conditions breast feeding cannot be regarded as a safe prophylactic against rickets, or, in premature infants and older babies, against iron deficiency anaemia. Infantile beriberi has been described in infants whose mother's diet was grossly deficient in the vitamin B complex but, apart from its vitamin and salt content, human milk shows little variation with changes in the maternal diet.

In calculating the calorie intake of bottle fed babies from the formula prescribed there is often a wide gulf between the theoretical result and reality. If the amounts have been prescribed in spoonsful, an error of 50 to 100 per cent. is to be expected, depending on the size of the spoon and the varying conceptions of fullness. Fortunately most manufacturers of dried milks provide a scoop which measures a drachm weight of the particular brand of milk, but these are not interchangeable for other brands, and many mothers gauge the approximate amount by eye. Further errors may be introduced by the residue left in the bottle if the infant is not taking the whole of every feed, and by post-prandial "posseting" which in a small infant may add up to a significant total, although not described by the mother as vomiting.

After this brief nursery preamble, I wish to consider in more detail the diagnosis of two relatively common deficiency diseases of infancy, rickets and scurvy, rather than attempt to cover a much wider field. These two conditions illustrate the methods of diagnosis applicable to infants, and some of the accompanying difficulties. In both diseases the diagnosis should be made on the results of clinical, radiological, and biochemical examinations; in both the clinical signs may be obvious or may be extremely difficult to interpret; in both the radiological picture is characteristic, and in both the biochemical investigations are limited by the practical difficulties of securing accurate measurements of urinary or faecal output, or of obtaining from small infants sufficient quantities of blood. It is possible to overcome some of these difficulties by use of a retention catheter or metabolism frame, but against the value of the result must be balanced the trauma and distress caused to the patient, and the constant attention necessary; I have for instance seen intractable and fatal diarrhoea and vomiting precipitated in a previously healthy infant kept for 36 hours on a metabolism frame. Similarly, the taking of 10 ml. of blood from a premature baby with anaemia might well do more harm than the biochemical confirmation of a state of rickets would do good. Furthermore, many mothers who are willing to bring their infants for clinical or radiological examination as out patients, will cease to do so if they find that repeated blood examinations are demanded.

Rickets

True rickets is seldom seem before the third month except in premature infants, and the great majority of cases diagnosed in this country without X-ray examination as foetal or neo-natal rickets have probably been examples of *osteogenesis imperfecta*, *achondroplasia*, or chondro-osteodystrophy. It is now generally taught that foetal rickets does not occur except in the presence of severe maternal osteomalacia, since the foetal skeleton will be calcified at the mother's expense. I have, however, seen one case which appears to refute this rule. After a normal delivery an infant was born at full term with multiple fractures and costochondral beading. Careful enquiry showed that the mother had received a normal, mixed diet throughout pregnancy, and had been in good health; radiological examination of her long bones showed no abnormality. X-rays of the infant's long bones showed osteoporosis and the typical appearances of infantile rickets; these were shown to Professor Parsons and Dr. J. Brailsford, who confirmed the radiological diagnosis. The urea content of the blood, the urine, and the stools were normal, but the value for blood phosphorus was reduced. Owing to the outbreak of hostilities it was unfortunately impossible to determine whether healing took place under vitamin D therapy.

At the other end of the age scale the incidence of rickets is less clearly demarcated and, though nowadays it is uncommon except in negro children living in temperate climates to see the disease in active form in children over 2½ years old, it may occur up to puberty, if conditions favouring its production are present. Cases of rickets resistant to treatment with vitamin D have been described in older children (Albright, Butler and Bloomberg, 1937; Bakwin, Bodansky and Schorr, 1940), requiring in one instance as much as 1,500,000 U.S.P. units of

vitamin D daily to promote healing. These older cases must of course be distinguished from renal and coeliac rickets.

In cases of suspected rickets, the case history should include not only details of feeding since birth and of any vitamin D supplements given, but also other particulars of housing conditions, family income, number of children in the family, and the provision made for care of the baby during the day if the mother goes out to work. Information about the birth weight and the possibility of prematurity should be obtained, as rickets is much more liable to occur in premature, than full term, infants. A history of recent infection is important, not only for its direct influence on nutrition but because a sick baby is unlikely to have been taken out of doors recently. General infections such as congenital syphilis are frequently associated with florid rickets, and it is possible that treatment with heavy metals such as bismuth may affect the incidence of the disease.

Diagnosis by Clinical Examination. As already indicated, it may be impossible to diagnose early rickets with certainty on clinical examination alone, and I propose to stress the difficulties of interpreting the clinical signs in doubtful cases rather than describe the fully developed syndrome. The skin should be examined for evidence of regular exposure of the face, hands and legs to sunlight, and the mucous membranes for anaemia. Muscle tone is best judged by observing the infant's activity, and whether it is backward in lifting the head, sitting up, crawling, or walking, it being remembered that the normal variation in reaching these milestones is very considerable and influenced by opportunity. The occurrence of abdominal distension will depend partly on muscle tone and partly on the presence of gastro-intestinal disorder which, though frequently associated with rickets, is by no means diagnostic.

Enlargement of the costochondral junctions is usually regarded as the earliest positive sign of rickets, but as these are always palpable, and are felt more readily in a thin than in a well covered infant, it may not be possible to say whether they are very slightly enlarged or normal. Hess has pointed out that after intensive antirachitic therapy has been given, a beading may remain which is not rachitic (Hess and Unger, 1920).

The size of the anterior fontanelle is of little value for diagnosis in young infants, since the normal variation is so great. The fontanelle should however be impalpable by 18 months and, if it persists after this time in the absence of raised intracranial pressure, rickets should be suspected. A patent posterior fontanelle is more likely to be due to raised intracranial pressure or to congenital abnormality than to rickets.

Craniotabes represents localized thinning of soft bone due to pressure. When it is due to rickets, it is developed in the occipital region from the weight of the head which is not lifted off the pillow. When seen, it is usually in young or premature infants, and it must be distinguished from a similar condition due to intracranial pressure such as fenestration associated with *spina bifida* and hydrocephalus, from *osteogenesis imperfecta*, and from congenital parietal foramina. These last are symmetrically placed in the parietal bones, and may be connected or separated by a median bar. They can almost always be found in one parent or in other members of the family. I have seen cases of typical craniotabes without any other evidence of rickets, and with the results of radiological and biochemical examinations normal.

Deformities of the thorax such as flaring of the ribs, Harrison's sulcus and pigeon breast may occur with any type of respiratory obstruction such as chronic bronchitis, asthma, or congenital *morbus cordis*, present in early infancy and, though frequently associated with rickets, are not diagnostic of it. Hess (1924, 1931) has described a not uncommon type of non-rachitic softening of the ribs, which may be present at birth and persist for several years.

Frontal bossing may give rise to a characteristic rachitic facies, but is seldom seen except in well marked cases, whilst a generalized prominence of the frontal region may occur in otherwise normal babies.

Delayed dentition is often, but not invariably, present in rickets, but here again the normal variation is so great that unless no teeth have erupted at 12 months little significance can be attached to it in diagnosis.

Enlargement of the epiphyses and deformities of the long bones occur in severe and long standing cases, but in early rickets may be so slight as to be impossible to detect clinically.

Radiological Diagnosis. Probably the earliest bony changes occur at the costochondral junctions, but as it is extremely difficult to get satisfactory X-ray pictures of these in infancy, the wrist is usually selected in preference. In active rickets the ends of the radius and ulna are cupped, and the growth line instead of being clear cut is irregular or feathery. There is also some general osteoporosis though, in order to be sure of this, it may be necessary to photograph the wrist of a normal baby of the same age on the same film. It is uncertain exactly how early in the disease these changes can be demonstrated, but healing can often be seen in from three to four weeks with antirachitic therapy. It is usually but not invariably possible to distinguish renal rickets by the radiological appearance alone (Parsons, 1927, 1, 2), but coeliac rickets, which is essentially of the same type as infantile rickets, gives a similar picture.

Biochemical Diagnosis. Owing to the difficulties of carrying out studies of calcium balance on infants, biochemical investigations are usually limited to estimations of serum content of calcium, phosphorus, and phosphatase. As a rule in rickets the calcium value is not significantly reduced, being usually about 8·5 or 9 mg. per cent.; if, however, tetany is associated with rickets, the value will be below 6 mg. per cent. The phosphorus value is more constantly, though not invariably, low and, though this finding is not in itself diagnostic, low phosphorus values being found for instance also in pneumonia, it is a confirmatory finding. It has been said that in active rickets the product of the calcium and phosphorus values is below thirty, and that if it is above forty active rickets is not present. This is a useful guide, but exceptions to both statements will be found. The most valuable single estimation is that of the plasma phosphatase. Morris, Stevenson, Peden and Small (1937) found the value for plasma phosphatase raised in 84·1 per cent. of 256 cases with rickets, as compared with 68 per cent. of the same group showing a low value for phosphorus. Variations of the plasma phosphatase value in the same individual appear to give an index of the activity of the rachitic process, the values falling during the course of successful treatment.

Scurvy

Acute infantile scurvy is a condition not necessarily associated with poverty and neglect. The last four cases I saw before the war all came

from good homes and had been having orange juice regularly. In one instance the batch of oranges used was apparently inactive, while in the other three cases an over zealous mother had taken the precaution of boiling or scalding the orange juice before use. All these infants appeared well nourished and no anaemia was present. The condition is usually seen in infants between 6 and 18 months of age, who have been reared on pasteurized, boiled, or dried milk. The outstanding feature is the acute pain, the infant screaming if it is touched or even approached. It lies with the thighs, which may be swollen, slightly flexed and abducted. Ecchymoses of the skin or orbit may be present, and the gums are typically swollen or show petechial haemorrhages. In one instance where they were gangrenous, I learnt that the gums had been lanced seven times by the father, a doctor, who thought that the symptoms were due to teething. A valuable confirmatory sign, which is constantly present in acute cases, is the occurrence of red cells in the urine. In more chronic cases, scorbutic beading of the ribs is characteristic. Unlike rachitic beading, where the costochondral junction is diffusely enlarged, the beading of scurvy shows a sharp line of demarcation between the rib and cartilage. Thus, if the finger is passed over the enlarged end of the rib, it descends a steep step as it reaches the cartilage.

While acute fulminating scurvy presents little difficulty in clinical diagnosis, milder, chronic, and sub-clinical cases of vitamin C deficiency in infancy may easily pass unrecognized. Since manifest scurvy probably takes from two to nine months to develop, it might be expected that a study of the early case history would give help in the diagnosis of latent scurvy. In fact, the previous symptoms are so vague and variable that no clear syndrome can be described; in many instances the infants have appeared well previously, and have gained weight normally. The first sign to appear is tenderness of the limbs and, when this is present, the case has passed from the stage of latent to that of manifest scurvy. It has been shown by Parsons and Smallwood (1935) that a true vitamin C deficiency anaemia may occur with manifest infantile scurvy, but it is not certain that this is present in the pre-scorbutic state.

Radiological Diagnosis. In their classical paper on the recognition of scurvy with especial reference to the early X-ray changes, Park, Guild, Jackson and Bond (1935) detailed the characteristic features, and correlated them with the pathological changes. The earliest radiological change is usually found at the ankle, involving the anterior corner of the tibia. The cortex in this area appears thinned and indistinct; subsequently a cleft of rarefaction appears immediately below a line of densely calcified matrix at the end of the shaft; in some cases the corner of the shaft appears to be torn away. In more advanced cases, the band of calcified matrix may extend beyond the contour of the shaft and become fragmented, while the area of rarefaction extends across the shaft. Fractures may occur through the latter, with displacement of the epiphysis. The epiphysis has a characteristic signet ring appearance, owing to general rarefaction and thinning of the cortex. In advanced and healing cases evidence is seen of subperiosteal haemorrhages and calcification along the shafts of the long bones. Early changes are most commonly seen at the ankles, but they also occur at the wrists, knees and shoulders. At the elbow, movement is usually full, and radiological changes are much less

constant and characteristic. The difficulties in obtaining a satisfactory X-ray photograph of the costochondral junctions makes examination of them of little value in the diagnosis of scurvy, as in rickets.

Biochemical Diagnosis. Estimation of the excretion of vitamin C in the urine has provided a valuable method of confirming the diagnosis. While it is by no means certain that saturation with vitamin C is the normal or even the optimum state of nutrition, saturation tests give a much better index of the degree of vitamin C deficiency than single estimations of vitamin C in the urine. Saturation tests are not as easily made in infants as in adults and older children, owing to the difficulty of obtaining 24 hour specimens of urine, or specimens voided at fixed hours. A modification of the method, described recently by Harris (1943), will probably prove the most practical, timed specimens being obtained by catheterization.

Incidence of Rickets and Scurvy at the Present Time

From what has been said, it is clearly difficult to give exact and incontrovertible figures for the incidence of deficiency disease in infancy at the present time. In a recent clinical and radiological survey undertaken by the British Paediatric Association (1944) on children between 3 and 18 months of age in various parts of the British Isles, it was found that there was considerable difficulty in assessing the significance of minor clinical signs, and that X-ray photographs of the wrist did not in most instances provide the earliest evidence of mild rickets. Thus in one rural area in Lincolnshire, where 200 infants were examined, clinical evidence of rickets was found in 5 per cent. while there was radiographic evidence of active rickets in 1 per cent. only. In some urban areas the discrepancy was much greater. While there is little doubt that of recent years the incidence of severe Glissonian rickets has decreased throughout the country, there is still considerable room for improvement in our clinical diagnosis of minor degrees of vitamin D deficiency.

In the same survey 4 cases of scurvy were noted in the first 1638 X-ray pictures examined. This is, however, probably an underestimate of the incidence, since only the right wrist was photographed in each case.

It has been possible to cover only a small part of what is essentially a major field of medicine, but an attempt has been made to indicate a few of the stumbling blocks which lie in the path in making a diagnosis of nutritional disorder in infancy.

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Discussion

Dr. S. G. Graham (Royal Hospital for Sick Children, Yorkhill, Glasgow, C.3), opener: There are four principal nutritional defects to be considered in infancy and, in order of frequency, they are: undernutrition, iron deficiency anaemia, rickets and scurvy. By far the most serious and most common one is undernutrition. Such defects predispose to infection and, conversely, infection can bring out clinical evidence of these defects so that a vicious circle is set up. The recognition of the severer forms causes no difficulty, but it is reasonable to suppose that for every severe defect in nutrition in a community of children there are several mild ones, some of them bordering on normality. It is impossible to draw a line between what one would call normal and what should be regarded as a nutritional defect. The experienced clinician can by a consideration of all factors, heredity, environment, diet, maternal care and so forth, anticipate the development of nutritional defects and by simple means take the necessary steps for their prevention.

Dr. J. Pemberton (Royal Hospital, Sheffield): The non-specific effects on infants of a bad diet should be emphasized. Deficiency diseases do not normally kill but non-specific complications often do. In childhood chronic inflammations of the mucous membranes such as bronchitis and *otitis media* are directly associated with too small expenditure on food; their complications are often fatal.

Professor H. D. Kay (National Institute for Research in Dairying, University of Reading): In using the plasma phosphatase level to assist in the diagnosis, and in the assessment of the rate of cure, of infantile rickets, it should be noted that the plasma phosphatase value of the normal infant is fairly high in early life and diminishes with increasing age. In plotting the change in phosphatase values against age in a case of suspected rickets it is desirable, therefore, to plot also on the same graph, as controls, the figures for the average normal infant within the same age range.

Miss E. M. M. Hume (Lister Institute, Roebuck House, Old Chesterton, Cambridge): The possible effect of nutrition on respiratory infection has just been mentioned, and the use of single dose therapy with vitamin D has been referred to by several speakers. It is worth remembering that there are records in the literature of the dramatic relief of acute respiratory disease in children by very large single doses of vitamin D. It would be interesting to know if any of the speakers have had personal experience of this method of preventing or curing rickets; it seems to have been neglected in this country, but its worth is attested by innumerable workers in the United States and numerous European countries. It has great practical advantages and would appear particularly applicable to relief work in post-war Europe. It has the slight disadvantage of being rather extravagant of the vitamin.

Professor C. A. Bentley (Town Hall, Wallington, Surrey): Of the three types of malnutrition mentioned by Wing Commander Ellis, I am particularly interested in the deficiency of total calories, or starvation, misnamed marasmus. Constipation is the first sign of insufficient feeding of breast and bottle fed infants. This can be almost invariably corrected

by increasing the total quantity of food but there is widespread ignorance of this fact among medical practitioners, nurses, health visitors and mothers. When, therefore, an infant begins to suffer from insufficient feeding it usually becomes more and more constipated and, instead of being given more food, is dosed with purgatives of one sort or another. I have heard of mothers who administer liquid paraffin or milk of magnesia in every bottle of food which they give to their infant. It is not surprising that in such circumstances a state of extreme malnutrition easily results and the unfortunate child either dies of some intercurrent infection or is eventually sent to a children's hospital as a case of marasmus.

The Treatment of Nutritional Disorders in Infancy

Dr. A. A. Moncrieff (Hospital for Sick Children, Great Ormond Street, London, W.C.1)

Marasmus. Wasting is the commonest of all the gross nutritional disorders of infancy and, sheltering behind a Greek label, the condition is badly understood and still worse treated. Again and again I have babies referred to my out patient department with this label who are suffering only from chronic starvation. Sometimes it is a question of underfeeding at the breast, in others of feeding on a too weak milk mixture, but most usually I find merely that the infant is receiving the correct food but not enough of it. The quantities are correct for the infant's actual weight, but that actual weight is several pounds under what it ought to be for the age and birthweight, and hence we have the perpetuation of slow starvation, unrecognized because the very gradualness of the process means that the usual dramatic symptoms, sudden hunger, sleeplessness and crying are absent. A little knowledge about calories is a dangerous thing if it leads to calculations based upon an incorrect weight, and the essential measure required to manage these babies is to ascertain at once what weight they ought to be and start to feed up towards it rather than down to the actual weight.

Chronic Infection. Second in importance with the wasted baby comes the examination for chronic infection. At a certain hospital where I used to take my students for an afternoon visit the medical superintendent always furnished, at my request, a list of babies said to be suffering from marasmus. We spent a profitable afternoon allotting the correct label, *otitis media* in one, pyelitis in another, chronic skin infections in a third, lung infections in another. It is most important to realize that an increase in katabolism occasioned by infection will produce wasting despite a thoroughly satisfactory food intake. I well remember a difficult infant I once admitted to my ward from my infant welfare department because I could not get the baby to gain weight despite a high calorie intake. Investigation of the urine showed an infection of the urinary tract and the administration of potassium citrate produced at once an upward turn in a previously horizontal weight curve. More than once I have regarded as a nutritional problem an infant in fact suffering from chronic meningococcal infection, revealed at last by a suspicious stiffness of the neck. This aspect of nutritional disorders is insufficiently recognized; recurrent droplet infection in many households means wasted babies

that no amount of dietary supplements will improve, and this state of affairs has nothing directly to do with income limits. It may be that there is a vicious circle of chronic starvation and chronic infection, but if so it is easier to break by dealing with the infection than by relying on calories or vitamins.

Organic Disorder. Another important cause of wasting may be mentioned briefly, the mere existence of some organic disorder, such as congenital heart disease or even mental deficiency; the latter opens up difficult problems of metabolic control by the central nervous system which would take us far away from the subject of nutrition.

True Marasmus. I will also pass briefly over the babies with what may be called true marasmus. After a period of chronic starvation or after long drawn out intestinal disturbance, there not infrequently comes a stage in which the infant's tissues seem to have lost the power of assimilating the food brought to them. Like the cells of a diabetic which cannot burn up sugar owing to the absence of insulin, the cells of the marasmic infant seem to lack some essential enzyme. Some have claimed to overcome this cellular failure with adrenal cortical hormone, some with thyroid, some with insulin, some with blood transfusion and some with old fashioned fresh air and a change of scenery. The very multitude of remedies suggests that a true solution has not been found, and I suggest that there is a fertile field here for investigation by physiologists and biochemists which would not only yield a solution urgently required by paediatricians but also throw light on unsolved mysteries of cellular metabolism.

These problems, however, lie too far away from the subject of nutritional disorders in the narrower sense. In normal times these are in fact less common than those disturbances which I have already discussed on the border line of the subject.

Protein Deficiency. Specific knowledge of protein deficiency is lacking but many of us suspect its existence in babies fed on cow's milk mixtures modified in a slavish attempt to imitate the gross percentages of human milk. To dilute cow's milk so that its percentage protein content is the same as that of human milk is to lose sight of the fact that there is a biological difference in the relatively higher proportion of lactalbumin in human milk with all that this means in the content of essential amino-acids. It is surely wiser to recognize that, good as cow's milk protein is in the biological scale, it is inferior to that of human milk for the human infant, and such inferiority can to some extent, if not entirely, be remedied by giving more of it. No scientific evidence has ever been adduced to show that this does harm, but a deficiency of protein, pushed too far, may result in "famine oedema" and, may it not be, that one result of protein deficiency is the fat, flabby, hydrolabile type of child with a water metabolism easily upset by relatively mild infections?

With the other deficiency disorders we come to more familiar ground. The treatment of rickets, scurvy and nutritional anaemia is well known to you, and, in general, it may be summed up by saying that it is necessary to give the missing or deficient substance in as big a dose as is safely possible to put the trouble right as quickly as possible. Vitamin A deficiency I do not propose to discuss as it is relatively rare and relatively

easy to treat. I am not, as yet, entirely familiar with nutritional disorders uncommon in this country due to deficiencies of the vitamin B complex, although Bray's observations on the island of Nauru (Bray, 1928-29) suggest that perhaps I have been missing something. I will proceed to offer some comments on the modern methods of treatment of the three deficiency diseases which commonly come under observation.

Rickets. Rickets yields readily to a vitamin concentrate and it would be academic to argue about the relative values of the different varieties of vitamin D. The main thing is to get the rachitic process under control as quickly as possible and for this a dose somewhere in the region of 2000 to 3000 I.U. daily is appropriate. In terms of cod liver oil this may mean as much as 5 to $7\frac{1}{2}$ teaspoonsful daily and, for an infant who has hitherto not been taking oil, such a dose will frequently upset digestion. Cod liver oil and malt has no place at all in the treatment or prevention of rickets. Indeed before the war it was a common cause of rickets because it gave a sense of false security with little prophylactic value, a 10 per cent. emulsion yielding only 30 to 40 I.U. in a teaspoonful. Yet quite recently an orthopaedic surgeon attached to a children's hospital spoke of treating rickets, in relation to knock knee, with cod liver oil and malt. For immediate treatment vitamin D in concentrated form is the method of choice. The only question really is how much? Should we be content with the usual dosage already mentioned or is there something to be said for "Stoss" therapy with the intramuscular injection of 500,000 I.U. as a slowly utilized reserve? I have no first hand experience of this except in an example of vitamin resistant rickets, when I gave this dose weekly by injection for six months without any effect.

It has been stated by Hess and others that the natural vitamin is better absorbed than the artificially produced substitute, but if the dosage is adequate I doubt if this matters much in getting rickets under control. Later, no doubt, a change to cod liver oil or the National Cod Liver Oil Compound is desirable on various grounds, including those of cost, safety and availability. Finally I do not think that ultraviolet light should nowadays be relied on for the treatment of rickets. Its effects are slower than those of the vitamin D concentrate and speed has to be considered in this connexion. A fatal attack of *laryngismus stridulus* may occur in a rickety child during a period of therapeutic experiment. Treatment must also include a more gradual change of diet on to correct lines and the securing of an adequate milk intake, but these are secondary considerations.

Scurvy. Speed also comes into the story with scurvy, for a scorbutic child may die of a cerebral haemorrhage before the vitamin C deficiency has been corrected. Again, the argument about natural versus synthetic products is largely academic since ascorbic acid readily cures scurvy except in the most unusual examples of vitamin resistance, of which I have had to deal with one example, cured by intravenous injection of ascorbic acid. The dosage is simple, since excess will eventually leak out and the only problem is to pack in as much ascorbic acid as possible as quickly as possible. A dose of 200 to 300 mg. or even more daily is a good average for the infant at the usual age of nine months. Here again treatment must include correction of diet and the introduction of satisfactory mixed feeding. In this respect I would like to make a point

about the potato. There has been a recent tendency to recommend that use of the potato in infant feeding should be postponed until after the first year in order to secure familiarity with other vegetables, green and root. Experts point to the larger vitamin C content of watercress, cabbage and so forth. The point is that the bulk of vegetable required is most easily supplied as potato all the year round and, desirable as green stuff may be, there is a serious risk of vitamin C deficiency if potatoes do not form the main vegetable dish for the infant promoted to mixed feeding.

Nutritional Anaemia is easily treated by iron, and Helen Mackay's mixture with ferrous sulphate is eminently satisfactory (Mackay and Jacob, 1937). I have seen no digestive disturbances with dosage up to 12 grains a day for the infant of one year. Mixed feeding must be introduced also as soon as possible, and the suggestion of Widdowson and McCance (1943) should not be forgotten that some iron may be contributed by the cooking vessels.

There is one important point which must be made even if it takes us away again from the strictly nutritional aspect. Anaemia may well be the result of chronic infection and such infection will not clear up while anaemia is present, nor will such anaemia yield readily to iron. I have had anaemic infants referred to my out patient department with running ears and urinary infections which required primary treatment before iron proved effective, but these could not strictly be classed as nutritional disorders.

In time to come, preventive measures may lead to a complete disappearance of the nutritional disorders which have been here discussed. We have the necessary knowledge, and speakers this afternoon will show us how it can be applied. It is the function of this Society to reduce the time lag between the laboratory and the domestic kitchen, and to provide evidence so strong that even politicians are forced to take action, and the elimination of undernutrition becomes a matter of cabinet policy.

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Discussion

Dr. J. V. C. Braithwaite (Paediatric Department, Royal Infirmary, Leicester), opener: There is a tendency nowadays to devote too much attention to deficiency diseases. Most nutritional disturbances in infancy result from infection or from wrong feeding. The mechanism of the disorders may be pictured as a disturbance of balance between putrefaction and fermentation in the bowel. Excessive putrefaction calls for a diet high in sugar, and low in protein and fat, while excessive fermentation may be corrected by a diet high in protein and low in fat and sugar. Loss of tolerance for food may be due to constitutional faults and may be caused by starvation or excessive heat. From any of the above conditions serious food intoxication may arise. Treatment must be by the withdrawal of milk and the parenteral, if possible intraperitoneal, administration of fluid. The apple treatment for diarrhoea and the administration of sulphaguanidine are two useful additional methods.

Miss R. M. Simmonds (Hammersmith Hospital, Ducane Road, London, W.12): Could parents attending infant welfare centres and nurseries be taught that little babies are very often thirsty and should be given boiled water to drink between feeds, especially in hot weather? It has recently come to my notice that babies have been given comforters to suck when they were crying only because they were thirsty. In each case the person in charge had not thought of giving drinks of water, and two women needed a lot of persuasion before they would consider doing so. Could we not legislate against the manufacture of comforters, especially during the shortage of rubber?

Professor A. St. G. Huggett (St. Mary's Hospital Medical School, Praed Street, London, W.2): It is regrettable that no emphasis has been laid on the fact that breast feeding is more natural and desirable than artificial feeding. This is important in view of the economic and social pressure on mothers to wean early, possibly too early. The optimum date for weaning has not been mentioned. It would be of interest to know how the nutrition of the pregnant woman influences her breast development and subsequent milk supply. Previous speakers have laid great stress on vitamins but, with the exception of Dr. Mackay, have mentioned proteins and fats much more briefly. To what extent is the excessive maternal katabolism after confinement able to provide protein for the milk supply, and how is this source of animal protein affected by the maternal nutrition before confinement?

Dr. E. H. Wilkins (11 Vesey Road, Wylde Green, Birmingham): The Government should be urged, quite apart from special war time restrictions, to make illegal the manufacture and sale of comforters or dummies.

Dr. D. H. Paterson (Hospital for Sick Children, Great Ormond Street, London, W.C.1): The incidence of coeliac disease has decreased lately. Thus, during the 15 years from 1924 to 1938, out of 45,771 new cases seen at the Hospital for Sick Children, Great Ormond Street, 73, or 0·16 per cent., were suffering from this disease; in the $3\frac{1}{4}$ years between September 1939 and December 1942 41,000 cases were seen, but only 41, or 0·1 per cent., had coeliac disease. The patients are now treated with some success with parenteral injections, on alternate days, of crude liver extract and of vitamin B complex after the method of May, McCreary and Blackfan (1942).

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Public Health Aspects of Nutrition in Infancy

Professor J. C. Spence (Royal Victoria Infirmary, Newcastle upon Tyne)

Text not received for publication.

Public Health Aspects of Nutrition in Infancy

Dr. H. E. Magee (Ministry of Health, Whitehall, London, S.W.1)

Previous speakers have dealt with nutritional needs, and with nutritional disorders and their treatment. The public health ideal is to establish

conditions which would result in the abolition not only of nutritional, but of all other, disorders in so far as the science and art of medicine make it possible. It is one thing to state the ideal and another to achieve it. We can be certain that we shall never achieve our ideal in its entirety. To do so would be to assume that this generation will achieve finality in knowledge and that individual men and women should become so unhuman as never to make mistakes or do foolish things. It would indeed be a dull world if mankind were to become so transformed. Fortunately there is no likelihood of individual men and women losing their individuality. We shall continue to do foolish things and make mistakes at times, to differ for the sake of differing and to have our conflicts and disputations. Some of us will stress in disproportionate fashion the importance of this or that factor, iron, calcium, iodine, vitamin A, B, C, D or others, to the neglect of other factors of equal importance. Undue emphasis of certain factors to the exclusion of others is an inevitable consequence of specialization, and I regard the correction of unbalanced enthusiasms of this sort as one of the most important functions of this Society. A quotation from a recent leader in the *British Medical Journal* (Editorial, 1943) is here appropriate "Though it is often alleged that the medical profession are slow to adopt new ideas, it is certaintly true that they allow a new idea, once adopted, to deflect the stream of medical thought too violently. Of this, bacteria, hormones, and deficiency diseases are outstanding examples. Each in turn diverted attention from the constitutional factor in disease".

In addition to foolish things being done and mistakes made, accidents occur and will continue to occur. These of themselves would prevent a number of individuals from attaining that standard of physical and mental well being which is the objective of those in control of public health. Nor must we neglect other factors than the purely nutritional which can have a profound influence on the nutritional state. I will mention only two: the amount of clothing put on to babies and the risk of infection. It can be frequently observed, even at the present day, that infants are overburdened with clothes to such an extent as to interfere gravely with heat dissipation. It must not be forgotten that the heat regulating functions of the human body are not fully developed during the first year of life.

It is important to remember that organisms are not of standard pathogenicity and virulence; they change in these respects. The number of different types of streptococci, for instance, is now, I believe, well over the 50 mark, and immunity against one type does not necessarily convey immunity against another. We should not, therefore, allow our zeal for nutritional science to lead us to lose sight of other factors which determine the growth and health of infants. Notwithstanding what I have said, an ideal is essential if we are to make any progress in knowledge. This ideal is that every infant should be so nurtured and nourished as to attain in adult life, and in full measure, that perfection of physical and intellectual stature which is its natural heritage. We must pursue this ideal not by mere defensive measures only, that is by purely preventive measures, but by positively going out to attain it.

We must begin with the mother, even before conception. The truth of this has been amply demonstrated by the results of the Toronto

(Ebbs, Tisdall and Scott, 1941) and London (People's League of Health, 1942) feeding tests, which have provided practical proof of the truth of the principles elucidated by nutritional research during the past thirty years.

Then we must also apply the principles of nutrition to the infant. These have been laid down by several authoritative bodies in recent years, and notably by the Technical Commission and other expert groups called by the League of Nations (League of Nations Health Committee. Technical Commission for the Study of Nutrition, 1937). These principles have been referred to by earlier speakers. There is no need for me to go into them in detail or to lay further emphasis on them, least of all perhaps on the great importance of breast feeding. I would like, however, to draw your attention to what Professor Fleming said at the meeting of the Society in Glasgow in February: "The qualitative alteration in breast milk caused by diet or other factors is of far less importance in infant feeding than the quantitative. It is better for a baby to receive the milk of an undernourished mother, even though in quality it does not reach the highest standard, than to be fed with cow's milk" (Fleming, 1944). I wonder how many here are prepared to agree with this. On general principles I am inclined to agree. Old Mother Nature has more wisdom than many imagine her to have. We are, for example, now learning that very much of the operative interference in the treatment of peptic ulcers, and in the conduct of labour, which was popular not many years ago was not always in the best interests of the patients.

Application of modern knowledge of nutrition has been made easier by the National Milk and Vitamin Schemes. These schemes I regard as the most important nutritional measures ever taken by this country. It would, however, be a great mistake to regard them as final. They will unquestionably need modification, and new schemes may have to be added as our knowledge of nutrition advances. Additions to our knowledge will not be made merely by laboratory experiments. Practical feeding tests and surveys will also be required. The new arrangements made by the Society for the co-ordination of nutritional field work is to be welcomed.

There is a very strong case for repetition at frequent intervals, of feeding tests of the type begun in Scotland in 1926 (Orr, 1928; Leighton and Clark, 1929), and repeated on a larger scale in 1935 (Milk Nutrition Committee, 1939), where milk was used as the test foodstuff. Those who have read the reports on these feeding tests will recall that the improvement brought about by the milk supplements in 1935 was nothing like as large as in 1926. The difference we may regard to a great extent as a measure of the general improvement in the national dietary which took place during the intervening years.

These and other feeding tests have shown us that we may expect with some confidence from the application of the knowledge we already possess, better health, better physique, greater vigour and less disease than existed before this new knowledge was brought to its present stage. The experience of this country since the war has given ample proof of the accuracy and importance of the new knowledge of nutrition. For the first time it has been put to a thoroughly searching test, and it has come out with flying colours. It would be wrong to undervalue this

achievement, and it would be an injustice to science and scientists to ignore, for there is a disposition in some quarters to ignore, the great contribution of scientists to this achievement. The credit belongs primarily to workers in the various fields of nutrition who have made contributions to knowledge and who, amongst our own allies and friends, have insisted in season and out of season on the fundamental significance to the prosecution of the war of applying the principles of nutrition. They have done this at the risk of being called cranks, faddists and queer people. Let us, therefore, give credit where credit is due, that is primarily and principally to scientists.

I am convinced that a continuance of feeding trials will be essential after the war, partly to perfect our knowledge and partly to determine to what extent the diets of the people are in conformity with the knowledge we possess. From the point of view of the subject of this discussion, and more so because of the significance of the well being of the expectant mother, feeding tests of the type of the Toronto and London tests should, I suggest, be given first place. We have still much to learn about the diet of the expectant mother, particularly about its relationship to the disorders and accidents of pregnancy and labour, and to the health and vigour of the infant. There is, of course, room also for feeding tests on school children and adolescents, but tests on expectant mothers are unquestionably of first importance in the present state of knowledge.

If it be assumed that the mother has been properly fed and looked after during gestation and that the infant is being nourished and nurtured as modern knowledge would decree, how is the doctor to say that all is indeed going as it should? What in fact are the means whereby he is to judge?

He will observe the child and, having seen that it is free from any of the obvious defects mentioned by earlier speakers, he will form an impression as to the infant's progress and state of health. The accuracy of his conclusion will naturally depend on his skill and experience. He can get no help from the infant such as he could from an adult. The infant cannot tell him that it is very sick. Still less can it tell him that it is just a little below par but that there is nothing serious the matter. All the infant can do is to howl, and it will howl just as much for a sore toe as for a sore belly. The only other thing the physician has to guide him is the rate of growth of the infant. Repeated measurements of weight can tell him a lot. If the infant is not gaining then something serious is wrong, but how much should it gain? For answer the doctor may refer to Holt's tables or to information about the rate of growth picked up from his own experience. The relevancy of Holt's tables to British children is very much open to question, since they are derived from the weights of babies in the United States. The only growth curves of British infants under one year that I know of are those published by Hill and myself in the *Medical Officer* in 1938 (Hill and Magee, 1938). These curves are based on the weights and lengths of nearly 1700 healthy babies of both sexes from Newcastle upon Tyne, Leeds and Glossop. All possible precautions were taken to ensure accuracy in these measurements. The data are unfortunately too few to give completely satisfactory curves which could be applied with confidence throughout the length and breadth of this island. Naturally we should have preferred

to have measurements from ten or even twenty times as many babies in rural as well as urban areas scattered up and down the country. We had, however, to be content with what we could get and, unsatisfactory as they are, I venture to suggest that these curves are more suitable as a yardstick by which to measure the progress of infants than the United States tables which have been used heretofore for this purpose.

I think that records of the length should also be taken, naturally at less frequent intervals than weight measurements. The length can be measured with a fair degree of accuracy and, although it is in general of less value than weight, the length is nevertheless useful together with the weight as a guide to the rate of growth. I should like to hear the opinions of those who have had more practical experience in this matter than I have.

I should like to see in the annual reports of Medical Officers of Health tables or curves showing the rates of growth of babies and toddlers similar to the tables in school medical reports. From the point of view of the Ministry of Health, at any rate, such information from all over the country would indeed be of very considerable value.

There are some other matters to which I would like to draw attention. The neonatal mortality rate, for instance, is higher than we should like it to be, and attempts to elucidate the cause or causes are obviously desirable. The results of the Toronto experiment (Ebbs, Tisdall and Scott, 1941) suggest that the nutrition of the mother during pregnancy may be an important factor in its causation, but that there are other factors requiring investigation is practically certain. Then there is the infant mortality rate which, if we take as guide certain other countries, such as pre-war Holland and New Zealand, should be much lower than it is. The fact that the rate is higher among poorer than among better off classes in this country, as has been shown by Tietze (1939), is a matter which demands attention. For example, in 1930-32 the rate was 77 for unskilled workers and only 35 for the professions.

It seems to be now a well established custom that children should receive solid food at six months. Not so long ago nine or ten months was considered time enough. Do we really know what age is the most physiologically suitable? Some precise information on this matter would, I think, be helpful.

Another thing which has puzzled me for some time is how completely the cane sugar given to the artificially fed baby is absorbed. The answer turns on the question whether the *succus entericus* of the baby contains invertase, because it is known that cane sugar is always inverted before absorption. Moreover, even if it were absorbed in the uninverted form, it could not be metabolized as such. With its amazing adaptability, the human organism is, I think, quite capable of producing invertase ahead of what we may call the normal physiological time. It seems to me, however, that it would be more in accordance with general physiological principles to educate the body to secrete invertase by gradually increasing the doses of cane sugar rather than to subject it to the full dose of cane sugar from the commencement of artificial feeding, even if this gradual education were to involve a slight temporary deficiency in the intake of energy.

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Discussion

Mr. F. Le Gros Clark (6 East Common, Harpenden, Herts.), opener: Several aspects of the present nutritional services are worthy of scientific attention. In the past most schemes of this kind were designed only for certain income categories; the new schemes recognize physiological categories and give only secondary consideration to income levels. The establishment of this principle is an important stage in the public health services; the principle is clearly capable of extension and should be studied by research workers in its relation to the problem of population. Since the machinery of distribution of special foods is, in the main, part of the machinery of the Ministry of Food, its post-war character will have to be discussed. Ministries presumably already have figures of distribution that might, when analysed, be useful for other public health purposes. The percentage of actual beneficiaries, for example, varies considerably from one administrative area to another, especially in the advantage taken of the vitamin supplement schemes. A combination of local surveys with the comparative figures from area to area ought to give the administrator an indication how and where to direct his maximum publicity and persuasion.

Dr. E. H. Wilkins (11 Vesey Road, Wylde Green, Birmingham): Certain defects are much commoner and more severe in the poorer classes and tend to disappear in the well to do. Food undoubtedly is the chief factor contributing to growth and healthy development, but in the production of specific defects other factors have their influence. The physical condition of an individual can only be accounted for in terms of the entire complex of his nutrition and environment, interacting with his inherent resistance. Differences in nutrition and environment exercise their influence roughly as follows: (1) *Stature.* Class for class, in a community of fairly uniform racial composition, superior stature always goes with better nutrition despite obvious hereditary influences in individual families. (2) *Musculo-skeletal form and function.* The children of the higher economic classes have less parietal bossing, better shaped chests, and straighter legs; they have better muscular tone and therefore less *pes valgus,* more erect posture, better respiratory capacity, better sense of balance, and they walk and run better. Those of the poorer classes show greater prominence of the abdomen, abnormal tilt of the pelvis and lumbar concavity, more kyphosis, flatness of the chest and outward and forward displacement of the scapulae and shoulders. (3) *Teeth.* I can

only speak for the temporary teeth. At 5 years of age children of the professional class show about 6 times the freedom from caries, treated or untreated, shown by elementary school children. (4) *Skin.* The texture of the skin and its resistance to infective lesions follow the same general curve of economic and nutritional well being. I have seen striking improvement in the health of the skin of a group of schoolchildren over an 11 months' period of better feeding without any change being made in the home environment. (5) *General health.* Variations in general health, as shown by death and morbidity rates, especially of the respiratory system, also tend to follow the curve of socio-economic well being.

There is thus a general correspondence of physical condition with nutrition and environment. I would specially urge that, in so called nutritional surveys of children, account should be taken of the entire objective physical condition and functional health; there has been too much slipshod assessment of nutritional state in terms of mere fatness or thinness. I should like to recommend the holding by The Nutrition Society of a conference on the parts played by nutrition and by other factors in the production of common defects of physique and health.

Dr. V. M. Crosse (Maternity and Child Welfare Department, The Council House, Birmingham): In Birmingham 54 per cent. of neonatal deaths occur in premature babies. Forty per cent. of all premature babies born die in the first month of life; 25 per cent. of all premature babies born die of prematurity, and feeding difficulties are one of the greatest causes of mortality in this group. Premature babies after the end of the second week require per lb. bodyweight 60 to 70 Calories and 3 to 3½ oz. fluid. The chief difficulty is to get the infant to take sufficient food, but with larger babies there may be also risk of overfeeding during the first two days. Premature babies are very liable to rickets and anaemia. Rickets can generally be prevented by treatment with ultra-violet light and sufficient vitamin D. The "physiological" anaemia cannot be prevented by administration of iron or by liver therapy, but return to normal, usually greatly delayed in premature infants, may be accelerated by giving iron from the beginning of the second month. Breast feeding is very important for premature babies. The mortality at one year among infants discharged from the Birmingham Premature Infants' Ward in good condition and weighing over 5 lb. was 4 per cent. for those completely breast fed, 6 per cent. for those partly breast fed and 10 per cent. for those artificially fed.

Dr. D. H. Geffen (Council Offices, Enfield, Middlesex): The major duty of a local authority is to promote the welfare of babies and not their treatment when sick. In this respect there is a slight difference of outlook between the medical officer of health and the paediatrician. The welfare of children is a question involving normal routine social services, housing and various other matters, but a first class welfare scheme would certainly benefit from the advice of a paediatrician. I cannot persuade myself, however, that the hospital is a centre from which all welfare work should emanate. Such a suggestion from Professor Spence shows, in my opinion, a wrong conception of child welfare, one which those interested in child hygiene would do well to abandon. Surely Professor Spence was not serious in his suggestion that one could divide the antenatal care of the mother from that of the new born baby.

Professor A. C. Frazer (Department of Pharmacology, Hospitals Centre, Birmingham, 15): The role of universities and teaching hospitals in a national nutrition organization should be emphasized. Workers in these institutions have the advantage of help from other scientific departments, of close association with hospitals which will increase after the war, and of the broadening influence of teaching duties.

Nutrition is not concerned only with diet but also with the physiological processes of absorption, metabolism and utilization of foods. Limitations of time have prevented us from considering at the present meeting this aspect of the problem of infant nutrition. Hardly any mention has been made of preventive measures. It is to be hoped that these matters may be the subject of a future meeting.

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