Underfeeding and overfeeding and their clinical consequences

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It is obvious that normal growth and development of any living organism requires adequate nutrition. It is the purpose of this paper to review the effects in the human species of a plane of nutrition which is higher, or lower, than this 'adequate' level. I cannot deal with deficiency or excess of individual nutrients such as vitamins or minerals, so the underfeeding or overfeeding in the title refers to variations in quantity of an otherwise well balanced diet. Having taken these steps to simplify my task, an inescapable difficulty still remains: one cannot sensibly discuss the effects of a deviation in quantity from an adequate diet unless that adequate diet is first defined. There is no absolute standard by which this can be done. It is now forty years since McCance & Widdowson showed that within any group of normal people, matched for age and sex, there is a twofold range of habitual food intake (Widdowson, 1936; Widdowson & McCance, 1936) and this range of individual variation has been confirmed since then by many investigators (Harries, Hobson & Hollingsworth, 1962). What is 'adequate' for one person, therefore, may be double or half what is adequate for another. I will therefore consider either the effects of diets which are grossly deficient or excessive, or alternatively the result of changing the plane of nutrition of a reasonably well-nourished person to a higher or lower level.

The most common example of underfeeding in the world concerns children in developing countries. The young child is particularly vulnerable, since he must meet the energy cost of growth (Millward, Garlick & Reeds, 1976), and also has a relatively higher protein requirement than the adult, at a stage of life when he is poorly equipped to forage for himself, and cannot take large quantities of food even if it is available. The clinical syndromes of kwashiorkor and marasmus are well documented (McCance, 1968). In malnourished children who die, the composition of the body is grossly distorted. It is difficult to express the alteration in body composition quantitatively, since a control child of the same weight as a malnourished one would be much younger, and one of the same age would be much heavier. A postmortem chemical analysis of the bodies of eleven children with varying types and degrees of malnutrition was made by Garrow, Fletcher & Halliday (1965) and Halliday (1967). The results are summarized in Table 1. Malnourished children show a gross deficit of protein, potassium and, in the case of marasmic children, fat. One-third of the total body fat in the body of a marasmic child may be found in the liver. The postmortem observation that there was a severe K deficiency was later confirmed in vivo (Garrow, 1965), and it was also

Table 1. *Whole-body analyses of well-nourished and malnourished children* (From Garrow *et al.* 1965, and Halliday, 1967)

Nutritional status	Age (months)	Oedema*	Fatty liver*	Body weight (kg)	TBW† (%)	Fat‡ (%)	Water[d] % FFW	Protein[e] % FFW	K/N ratio[f]
Expected normal	12	—	—	10·0	62·4	20·0	78·0	17·0	2·13
Recovered:									
(L.S.)	10	—	—	4·69	58·8	22·5	75·0	18·8	2·08
(C.W.W.)	14	—	—	4·17	64·7	19·2	80·2	16·7	2·30
Moderately malnourished:									
(R.H.)	9	—	+	7·50	61·3	25·5	82·4	14·3	2·07
(E.W.)	13	—	—	7·70	65·1	18·4	79·7	16·5	1·52
Marasmic kwashiorkor:									
(C.W.)	6	++	+	4·10	82·0	4·6	86·0	12·4	2·00
(S.D.)	16	++	—	6·70	83·1	3·9	86·5	10·7	1·82
Kwashiorkor:									
(C.B.)	14	+	++	5·67	69·1	16·0	82·3	13·8	1·40
(A.C.)	12	+	++	6·30	70·1	15·4	83·0	13·0	1·63
(A.S.)	12	++	++	6·30	81·9	6·4	87·5	9·3	1·65
(C.A.)	11	++	++	7·60	69·4	20·0	86·8	11·0	1·54
Marasmus: (G.S.)	14½	—	+	4·73	72·8	9·9	80·8	14·2	1·74

*Oedema or fatty liver absent —, moderate +, severe ++.
†TBW % = Total body water as % of total body-weight.
‡Fat % = Fat as % total body-weight.
[d]Water as % fat-free body-weight.
[e]Protein as % fat-free body-weight.
[f]Potassium (mmol) per g protein nitrogen.

possible to show that a large part of this K loss came from the brain, and could be replenished on recovery (Garrow, 1967). This K deficiency evidently is of clinical significance, since in an analysis of 248 malnourished children, 37 of whom died, the factor most highly correlated with death was electrolyte disturbance, rather than hypoproteinaemia or anaemia which might have been expected to reflect the severity of protein depletion (Garrow & Pike, 1967). Among the survivors, if malnutrition is prolonged there is certainly stunting of physical growth, and these stunted children fare worse in intelligence tests than their well-grown contemporaries. It is very difficult to distinguish in these children between the effects of undernutrition and those of a lack of intellectual stimulation (Birch, 1972). At one time it seemed that the lower intelligence score of twins, as compared with singleton children, might be a consequence of intra-uterine malnutrition, since during development they had to share accommodation and placental nutrition. Even this idea, however, must be revised in view of the observation by McKeown (1970) that the intelligence score of a surviving twin, when the other dies within the first month, is not different from that of singleton controls: in this study 962 twin pairs who took the verbal reasoning test in the 11 plus examination had an average score of 95·2, while in 148 pairs in which one twin died the survivor scored on average 98·8. This strongly suggests that the

poorer performance on average of twins is a consequence of postnatal events, and cannot be blamed on intra-uterine malnutrition.

Another study which throws light on the effect of maternal undernutrition on the subsequent mental development of the child is that of Stein, Susser, Saenger & Marolla (1975). This is a most painstaking analysis of the mental competence of 19-year-old men who were born at the time of the Dutch Hunger winter of 1944–5. This short but severe period of undernutrition for a large population caused no detectable impairment of mental competence among the survivors when they were tested at the time of induction for military service.

Severe postnatal malnutrition in a child who is otherwise well cared for does not cause obvious mental handicap. In a series of eight children who had large resections of gut in infancy Valman (1974) found no obvious mental deficit 7–12 years later.

In this brief review of the clinical consequences of perinatal undernutrition I have concentrated on the subsequent mental development of the child, since this is the aspect which rightly concerns most nutritionists today. There are animal studies which clearly show that perinatal nutrition may decrease the number of brain cells in the foetus. It should be stated, however, that in the human species intelligence seems to be determined mainly by upbringing and genetic endowment, and there is no strong evidence that it is affected by perinatal nutrition.

The consequences of undernutrition in human adults may be studied in the tragic circumstances of famine or war, in a small number of experiments in which volunteers were undernourished in the laboratory, in the curious syndrome of anorexia nervosa, or in patients who are undergoing treatment for obesity. Famine and war are not conditions conducive to careful and dispassionate scientific enquiry, but the valuable knowledge gained in the aftermath of these disasters is available to nutritionists in publications such as the MRC Special Report 375 which records the investigations on undernutrition in Wuppertal in 1946–9. There are also records of the effects of severe privation in explorers and those who have been shipwrecked or isolated without food for long periods by aircrash in inhospitable and remote regions. In all these accounts the effects on physiology and body composition are analogous to those seen in malnourished children, but the adult is better equipped to withstand the starvation than the young child, since he does not need to grow, and has greater energy reserves as fat on which he can draw. The psychological effects of severe undernutrition in these natural disasters are difficult to categorize, since explorers and those who go on hunger strike for some idealistic reason are a highly self-selected group, and are probably of different psychological make-up from the involuntary victims of war, famine or shipwreck. In general, however, undernutrition in adults seems to produce weakness, apathy, lack of concentration, irritability and a decrease in sexual drive. These observations have been confirmed in the laboratory studies of undernourished volunteers. There have been two large studies of this sort: one by Benedict, Miles, Roth & Smith (1919) and the other by Keys, Brozek, Henschel, Mickelson & Taylor (1950). Both of these studies were conducted in America after world wars

had caused severe malnutrition in other continents, and so there was a need to understand the physiology of undernutrition. In Benedict's experiment normal young men were fed about 8 MJ (1900 kcal) daily for 3 months in order to produce a decrease in body-weight of about 10%. The later study in Minnesota is entitled 'The biology of human starvation', and in this case a loss of 25% in body-weight was produced by feeding a diet which supplied on average 6·53 MJ (1570 kcal) daily for 24 weeks. These figures are worth bearing in mind. A doctor may suggest to his obese patient that a diet of 1500 kcal per day (or often less than this) should be adhered to for six months, and this may be excellent advice, but he should realize that he is asking his patient to follow a programme which is more severe than that of the heroic volunteers in the Minnesota experiment.

Anorexia nervosa is a strange condition which affects girls in their late teens. Estimates of prevalence vary with the diagnostic criteria used, but by any standards it is not rare. Crisp, Palmer & Kalucy (1976) found 16 cases which fulfilled the most stringent criteria in a survey of 4107 schoolgirls aged 16 or over, and 11 cases among 8274 girls aged 15 or under. It is rare in boys. The interest of this condition for our purposes is that girls so affected are certainly grossly undernourished, but they do not show the features associated with the usual forms of adult undernutrition: although emaciated they remain remarkably alert and active, but menstruation ceases at a body-weight of about 45 kg. Apathy and irritability are therefore not invariable consequences of starvation.

The commonest example of undernutrition in adults in this country at present is the obese patient under treatment. It is difficult to estimate how many people are trying to lose weight by one means or another, but the following statistics give an indication of the prevalence of the problem. In 1973 the cost to the National Health Service of drugs promoted for the treatment of obesity was £2 478 926. From a survey by the Consumers' Association it appears that about half the people who answered a questionnaire about their efforts to lose weight had at some time consulted their doctors, and about half of those who saw the doctor had pills of some sort prescribed (Ashwell, 1973). On this basis there are probably about four million people seeking medical advice about obesity in a year. The three largest commercial slimming clubs claim an average weekly attendance of about 70 000 among them (Craddock, 1973) and a magazine concerned principally with slimming has a circulation of about 380 000 copies. Butterfield (1974) estimates that about 20% of the population of this country attempts to lose weight each year.

Overweight people have good reason to lose weight: obesity tends to reduce life expectancy, but weight loss restores it to normal (Dublin, 1953). Apart from the increased risk of early death, especially among young people (Blair & Haines, 1966) there are many other medical consequences of obesity. Among the more important disadvantages of being substantially overweight are decreased exercise tolerance, an increased liability to osteoarthritis of weight-bearing joints, greatly increased morbidity with any surgical procedure and especially abdominal surgery, usually a social disability (although some commediennes may make a better livelihood

because they are fat), and often a lower standard of medical care. This is not the list of disadvantages which is usually quoted, in which such factors as gallstones and flat feet or the risk of heart disease are prominent features. I have now been trying to provide medical care for a large number of very obese patients for some years, and have been impressed how much more difficult it is to make a sensible diagnosis in a fat patient than in a thin one. In a thin patient who complains of aches or pains or tiredness or shortness of breath or dizziness or change in bowel habit a reasonably thorough physical examination, and perhaps a few simple tests, will indicate if there is serious underlying disease. In a very obese patient effective palpation of the abdomen, for example, is virtually impossible, and symptoms which are subsequently found to be due to malignant disease, leukaemia, renal failure or autoimmune disease may be dismissed at first because these same symptoms could equally well be a result of obesity. So far as I know this suggestion has not been made before, but it seems quite likely that the most serious clinical consequence of obesity is that it prevents the obese patient from having any other disease diagnosed early and treated well. Surgeons, obstetricians and anaesthetists are, of course, well aware of the difficulties of operating on obese patients (Fisher, Waterhouse & Adams, 1975). In childbirth overweight women have more complications (Efiong, 1975) and obesity is an important risk factor for death of the mother herself (Maeder, Barno & Mecklenburg, 1975).

All this raises an important and controversial point, on which I hope there will be discussion. What is the correct dietary management of women who present at an antenatal clinic already substantially overweight? Some obstetricians base their ideas of the nutritional requirements of pregnant women on the diet which farmers have found to be profitable in pregnant sows (Brewer, 1967): this approach leads to a recommended intake far in excess of physiological requirements. Even the most open-minded obstetricians react with alarm to a suggestion that a pregnant woman should actually try to lose weight during pregnancy, since this may impair the growth of the foetus. On the other hand, if pregnancy is a closed season for the treatment of obesity, it is naïve to suppose that the mother will, immediately after delivery, start on a reducing diet, and if she is breast-feeding it can be argued that this is another closed season. Thus after several pregnancies the situation may well arise that severe obesity has developed. This is not merely a hypothetical danger: in the maternal mortality study of Maeder *et al.* (1975) out of a series of 250 maternal deaths 41% of the mothers had a non-pregnant weight over 150 lb (68·2 kg) and 12% were over 200 lb (90·9 kg). In a control group of 200 private obstetric patients the percentages above these weight thresholds were 15% and 2% respectively. I suggest that in our present state of knowledge it would be reasonable to advise a pregnant woman who was already overweight that she should aim to maintain constant weight during her pregnancy, and try to get down to normal weight between pregnancies.

Overnutrition in children is bad for them, but we have the same difficulty in defining what is meant by overnutrition in children as we had in adults. A recent survey in Sweden indicates that overweight or obese infants probably were having

on average about 10% higher energy intake than normal weight children (Sveger, Lindberg, Weibull & Olsson, 1975), but these averages conceal a large range of variation. Typically the coefficient of variation on the measured intake, whether it is per child or per kg body-weight, is about 15%, so the range of two S.D. from the mean goes from 70% to 130%, which is roughly the same twofold range which Widdowson and McCance have already noted, and the intake of the overweight and obese infants, although higher on average than that of the controls, falls well within the normal range.

This paper has been about the clinical consequences of underfeeding and overfeeding, and I have so far referred only to observations on the human species. There is a tendency to assume that somehow, in man as in other species, there must be some mechanism which, if only we let it work, would guide us to take the amount of food which is optimum for our health. Elsewhere I have discussed the control of energy balance in man, and the great technical difficulties in studying how such a mechanism might operate (Garrow, 1974). It might be fitting to conclude this review by reminding you that even the laboratory rat, that paragon of dietary regulation, apparently eats more than is good for it if it is given the chance (Ross & Bras, 1975). I suspect that the same holds true for man.

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