

of feet, and similar topics. It is more difficult to use these visual aids in out-patient clinics. The film strip, however, has greater possibilities than the film. As many or as few frames of the strip can be shown as are appropriate to the occasion.

The number of film strips dealing with simple nutrition is limited, and generally the treatment of the subject is serious and rather formal. The film strip, with coloured cartoons, is in demand in Canada and in the United States of America to teach nutrition and hygiene of food. Such film strips have a great appeal but are costly to produce.

Many nutrition films made in this country are again rather formal. They are intended for the more scholastic approach of the classroom in a grammar school or training college. The material is often excellent but a fair knowledge of biology, of elementary physiology and of chemistry is assumed. A set of three films, recently produced by the Ministry of Food, has, however, been found to have a wide appeal for lay audiences. The films are called : *Body Building Foods*, *Protective Foods* and *Energy Foods*. They were originally intended for use in the housecraft room of a secondary modern school for boys and girls aged 13-15 years. The films have already been shown to groups of house mothers in charge of children in residential homes, to cooks in school canteens, to students of mental health and of social science, and have aroused new interest in a subject which, regrettably, is often considered dull. Films, however, are not ideal for teaching the fundamental points of nutrition. From the three films three film strips are being prepared. These will have much of the essential information of the films. They will be used for day-to-day teaching. The film may later animate and co-ordinate the principles of simple nutrition taught by the strip. These film strips and films will we hope also be of use in teaching out-patients.

It is of fundamental importance to present nutrition as an interesting part of everyday life. The opportunities of dietitians, doctors and nurses are great. They can be greatly helped by the use of visual aids. These aids, to be effective, must be used with care and forethought.

SUMMARY

1. Visual aids can be used to teach normal nutrition even when the dietitian is dealing with the deviations from the normal found in hospitals and in clinics.
2. An outline is given of the types of visual aid available for such teaching.
3. The wider use of visual aids is urged.

Diet and Obesity

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Obesity is unquestionably the commonest nutritional disorder in present-day Britain and gives rise to more ill-health than all the vitamin deficiencies put together. The skill of a trained dietitian is invaluable in the treatment of this disease. Even

were there no other practical problems in human nutrition, the dietitian would more than justify her training and knowledge by the help she can give to fat people. A proper nation-wide attack on obesity would employ more dietitians than are at present registered by the British Dietetic Association.

Importance of obesity

Life insurance companies know very well that obese people are poor life risks, but, unfortunately, there are no published data in Britain to indicate the seriousness of the risk. The statistics of the Metropolitan Life Insurance Co. (U.S.A.) show that an American male, 25 lb. over 'standard weight' at the age of 45, has his life expectancy (in years) reduced by 25% (Dublin & Lotka, 1936). Obesity impairs health in two ways. There are, first of all, the purely mechanical effects of too much weight-bearing: backache, flat-foot, arthritis of the knees and hips, varicose veins, abdominal hernia, bronchitis. Then there are certain metabolic defects that occur more commonly in the obese than in the normal population, though for reasons as yet obscure. These are arterial degeneration (atheroma), gallstones, diabetes mellitus and raised blood pressure. These complications, in their various combinations, make obese people poor subjects for surgery. Because their movements are slow they are unusually liable to accidents—at home, at work, or in the street. Though they may 'laugh and grow fat', their laughter is frequently hollow, for they know only too well that their youthful charms have gone.

Causes of obesity

Views about the aetiology of obesity have been subject to fluctuating opinion. In the rough-and-tumble of eighteenth century Britain a fat man was regarded as one who, by good living, had built up for himself a substantial reserve against the unpredictable dangers of famine and destitution. In the present century, the growth of the new science of endocrinology has given the doctor a useful escape from the distasteful task of telling his obese patients that they are greedy; it could all be blamed on 'the glands'. The problem of deciding just which glands to blame remained to be settled. Obesity is certainly not a characteristic feature either of hypothyroidism (myxoedema) or of hypopituitarism (Simmond's disease). Though farm-yard animals are fattened by castration, the same effect is not regularly seen in man.

The lack of any positive evidence for an endocrine cause has led to a return to the earlier belief that the vast majority of sufferers from obesity simply eat too much; the reasons for this may be straightforward gluttony or a disorder of the hypothalamus in the lower part of the brain. Social insecurity was a cause of gluttony in the eighteenth century and emotional insecurity may account for much of it in the twentieth. Many women, especially, overeat because they are unhappy. A dietitian has only to visit the homes of some obese patients during the preparation of a meal to appreciate the common cause of obesity. There are the plates stacked high with bread, scones, cake and cream cookies, some of which are often munched before the meal even begins.

Yet there are some obese patients who eat surprisingly little and still become fat. There is undoubtedly an endogenous or metabolic cause of obesity as well as an exogenous one of simple overeating. Realization of this has led to a recent denial that gluttony is a common cause of obesity (Spence, 1952). This view is surely mistaken, but none the less, the metabolic problems of obesity are worth considering.

Metabolism in obesity

There are two essential problems : where does the body get the energy which it stores as fat ? ; and by what metabolic means does it succeed in storing it ?

Energy metabolism. There can be no argument about one thing. The only possible origin of the fat in the adipose tissue is the food ; it does not come out of the air. So obesity, whatever its cause, is a problem for dietitians. But how does the obese body conserve energy as fat ? It does not, apparently, digest its food more efficiently than the normal body ; it is not a more efficient machine—in fact the contrary is the case. Obese people actually burn more fuel than normal subjects. Their oxygen uptake at rest may be as much as 25% above the normal for people of the same height, age and sex. Many politicians would dearly love to know this secret, of how to save and yet spend freely ! The high oxygen uptake of the resting, obese patient is probably due to the fact that they have more active muscle than a normal person. When a patient puts on weight, it is not all fat or water ; extra fat needs extra muscle to move it about, so the obese patient has an abnormally large muscular volume (Keys, 1952).

The most likely explanation of this paradox is that normal people have some way, as yet undefined, of disposing of surplus calories, probably by slow loss of heat from the body. This facility may be impaired in the obese subject. Certainly many normal people regularly ingest more calories than their bodies need, without becoming obese. This is illustrated by many diabetics who, once their disease is established, manage to keep in health and maintain their weight on a diet providing far fewer calories than they formerly consumed. This idea of *luxus Konsumption* (Grafe, 1933)—the burning of surplus calories—is not new, though temporarily out of fashion ; yet it provides the only present rational explanation of the way in which small eaters can become obese.

Metabolic control of fat storage. Endocrine factors. If it is agreed, then, that the obese body stores as fat energy that the normal body would eliminate as heat, it remains to be considered how this is accomplished. There is much indirect evidence to suggest that insulin is concerned in the conversion of glucose to fat in the body. Best (1952) has lately shown that hypophysectomized animals become obese when treated with small, but increasing, doses of insulin. A chronic excessive output of insulin by the islets of Langerhans of the pancreas would be expected to result in obesity ; that this may actually happen is suggested by Ogilvie's (1935) demonstration that the islets are frequently hypertrophied in patients who have become obese shortly before death. Chronic overactivity of the islets may lead eventually to failure and atrophy, with resulting diabetes. This may explain why

a small proportion of people, who have been obese for many years, ultimately develop diabetes.

It is probable that the foregoing chain of events may result from overactivity of the anterior lobe of the pituitary. Young (1948) showed that the same partly-purified extract of the gland that produced rapid growth in puppies, caused diabetes in their parents—presumably by overworking and exhausting the islets of Langerhans. The parallel to this in clinical practice is the 'large-baby, diabetes syndrome'; some obese mothers give birth to babies over 10 lb. in weight and, years later, themselves develop diabetes (Gilbert, 1949). In such cases it is to be presumed that the overactive anterior pituitary is excreting excessive amounts of growth hormone, resulting in a large baby, and that the same overactivity accounts for the mother's obesity and subsequent diabetes. But it seems unlikely that it is the growth hormone itself that causes obesity in the mother because obesity is not a characteristic feature of either gigantism or acromegaly. In the present state of our ignorance about the nature and mode of action of the various secretions of the anterior pituitary, it is perhaps dangerous to speculate too much on the mechanisms whereby they may contribute to the production of obesity. It is clear, however, that certain extracts of the gland may oppose the action of insulin in converting glucose to glycogen in the muscles (Colowick, Cori & Slein, 1947) as a prelude to its oxidation. This frustration of the normal role of insulin may lead to its greater production by the islets of the pancreas and to its alternative employment in the conversion of glucose to fat. Thus the glucose is not burnt up and lost as heat, as would happen in the normal body.

There is another secretion of the anterior pituitary, adrenocorticotrophic hormone (ACTH), which stimulates the adrenal cortex and encourages a particular type of obesity seen in Cushing's syndrome. But this is clinically distinct from the common 'simple' obesity, which cannot, apparently, be blamed on any disturbance of the pituitary-cortical mechanism. But this does not preclude the probability that the anterior pituitary contributes to obesity in other ways.

It is worth noting that obesity is especially liable to begin at three periods in life when there is other evidence that the anterior pituitary is unusually active, namely at puberty, during pregnancy and after the menopause. So perhaps there is more justification than we have liked to admit in recent years for blaming 'the glands' as a cause of obesity; but this is a secret that we had better keep from our patients!

The hypothalamus. This area of the brain-stem is undoubtedly concerned with the regulation of appetite; damage to it experimentally in animals or, rarely, as a result of disease in man, produces a depraved, excessive appetite and consequent obesity. It seems likely that overeating from psychogenic causes is mediated through stimuli from the cerebral cortex directed to the hypothalamus which, in turn, controls the numerous neural and humoral mechanisms which may affect appetite and the utilization of ingested food.

Comment on metabolism in obesity. The complicated interrelationships between the mind, the cerebral cortex, the hypothalamus, the anterior pituitary, the islets

of Langerhans and, no doubt, other regulatory mechanisms are imperfectly understood as yet ; but we already have sufficient insight into them to be justified in regarding many cases of obesity as suffering from a true ' psychosomatic disease '.

Treatment

Fortunately, from the strictly practical standpoint of treating obesity, it matters little whether the particular patient is an ' over-eater ' (for psychogenic reasons or owing to some damage to the hypothalamus) or adjusts the regulatory mechanisms of the body to save energy where others, on the same diet, would lose it as heat. Obesity is always due to eating more food than the particular patient requires. The essence of treatment is therefore simple—cut the calories. The obese, ambulant housewife with few domestic duties will usually do well at first on a diet providing about 1000 Cal. made up of 100 g carbohydrate, 60 g protein and 40 g fat daily. Under conditions of bed rest in hospital an even lower caloric intake may be desirable, whereas an obese man engaged in active work will usually lose weight satisfactorily on 1500 Cal. or so. It is best that all foods should be weighed, at least at first.

A careful inquiry into the patient's previous dietary history is necessary before any diet is prescribed. The patient is unlikely to adhere to a diet completely strange to his notions of normal eating ; at the same time these notions may well cause a dietetic shudder. The previous diet, though quantitatively excessive, may have been grossly deficient in some qualitative respect, *e.g.* in ascorbic acid or in iron. The planning of a proper diet for an individual case of obesity is part of the dietitian's skill—to change only so much as is necessary to correct the errors, quantitative and qualitative, of the previous diet.

The doctor who lacks the inestimable advantage of the assistance of a trained dietitian is usually reduced to the unsatisfactory resource of handing the patient a diet sheet copied from a textbook and telling him to get on with it ; he rarely gets very far. Probably the best hope in such a predicament is to make use of a system of ' do's-and-dont's ' such as that proposed by Marriott (1949). Certain fattening foods are banned entirely from the diet in exchange for the thin compensation of being allowed half a pint of milk, 3 oz. of bread and an unlimited quantity from a limited list of non-fattening foods.

Once dietetic treatment is begun the patient needs to be seen at regular intervals for encouragement, to prevent relapses and to make such changes in the diet as will ensure a steady loss of weight of between 2 and 3 lb. weekly. Here again the skill of the dietitian is invaluable ; not only does she save the doctor an immense amount of time in discussing culinary details (in which he, in any case, is usually out of his depth), but she also, by her skilful management of the patient's problem, is in every sense the true treater of the case. For this reason it is imperative that the training of a dietitian should continue to include much more than an academic study of food and of food values. She must understand the patient in relation to his environment and be able to handle his problem with tact and sympathy. In a well-organized diet department, staffed by good dietitians, there should be

few defaulting, back-sliding patients whose defence is—' I did not come back because I was afraid of a scolding '. Even so, there are, inevitably, some defaulters ; a district visitor trained in dietetics is invaluable for rounding them up periodically and bringing them back to the clinic. Finally, the dietitian must be able to tell when the patient is not making progress or is developing trouble from one of the complications of obesity ; like the district nurse, she must know when the patient ought to be sent back to the doctor.

There are no contra-indications to dietetic treatment. Nearly all obese patients, whatever else their trouble, will benefit from a proper limitation of their caloric intake. Likewise there is no alternative treatment to dieting. Amphetamine sulphate by mouth is sometimes a temporary help in reducing excessive appetite ; dried thyroid gland by mouth can be tried in selected cases as a stimulant to metabolism ; restriction of salt intake may reduce a retention of water which sometimes occurs in the obese ; but these provide no satisfactory substitute for dietetic treatment.

Conclusion

Obesity is a common and most important psychosomatic disease. Its satisfactory treatment usually requires the skill of an experienced dietitian trained not only in the science of dietetics but also in the art of handling patients.

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Hospital Diet Departments in the United States of America

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My American experience was confined to 20 months in one hospital. However, I was able to see and hear enough of methods in other American hospitals to know that the practice in the hospital where I was stationed was somewhat better than the average. While I was there we entirely revised the hospital diet manual, using as our principal references the most recent editions of the Mayo Clinic Manual (Committee on Dietetics of the Mayo Clinic, 1949) and Proudfit and Robinson's (1950) *Nutrition and Diet Therapy*, and adapting the diets to our own needs.