son (1950, 1951) in the stress he lays on their importance. Although they can appear in diverse clinical states, the available evidence suggests that they derive from an altered or excessive adrenal steroid metabolism, and they can certainly occur in the course of treatment with A.C.T.H. Simpson (1951) has described some of these cases of pubertal obesity under the names of adipose gynism and adipose gynandrism, and there is no doubt that they constitute a definite clinical picture which we have all seen, although Le Marquand (1951) may well be right in regarding them as extremes of the continuous spectrum of childhood obesity rather than as isolated entities. However this may be, it would appear to be impossible to understand them without regard to the adrenal cortex, and the same applies to maternal obesity, for when endocrine abnormalities occur in these women their trend is always in the direction of a Cushing syndrome and never in that of acromegaly. Indeed, pregnancy is a well-recognized precursor of the Cushing syndrome.

Our present knowledge, however, does not enable us to go much farther than the general statement that the adrenal cortex is undoubtedly concerned in the mechanism of obesity. Apart from the possibility that it may affect the site of the deposition of fat, and that Kendall's compound A (11-hydroxycorticosterone) and compond B (corticosterone) may cause a decrease in weight accompanied by an increase in the percentage of fat, there is some clinical evidence that the gland may have a direct effect on appetite, and, if so, this may provide a link with what has already been said concerning the hypothalamus. It is well known that the use of both cortisone and A.C.T.H. may occasionally be accompanied by a marked increase of appetite and gain of weight; in my experience this is particularly true of A.C.T.H., and I well remember one child, given it for nephrosis, who immediately developed an appetite so insatiable that it was like that described for rats after hypothalamic damage; for if food were not at hand she would consume her chalks, and so long as food was available she would go on eating. Simpson (1952) has described the case of a child with a tumour of the adrenal cortex who developed a similar appetite with resultant obesity. The link between the adrenal cortex and such appetite is still a mystery.

We have no understanding of how the hypothalamic regulation of appetite is itself controlled, but Kennedy (1953) has suggested that hypothalamic damage leads to an increased appetite by interference with the satiety mechanism, which may normally be actuated by a sensitivity in the appropriate hypothalamic cells to metabolites in the blood stream. No single metabolite has been proved to be capable of exerting this control, though glucose has been suggested. One cannot avoid the speculation that adrenal steroids may influence appetite by a direct effect on the hypothalamic cells concerned, and that an excess of certain steroids may provide the hypothalamus with entirely false information, causing it to legislate for a state of need in the presence of a state of plenty. For this is essentially what happens when the hypothalamus is injured or a child with an adrenal cortical tumour develops an insatiable appetite.

Speculation is hazardous; nevertheless it seems fitting to end on such a note, for the problem of obesity has not yet disclosed all its secrets.

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ASSESSMENT AND RESULTS OF OBESITY*

BY

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For my purpose I shall define obesity as the condition in which the amount of body fat is greater than normal; the tiresome question of normality I have discussed at length elsewhere (Sinclair, 1948). The term "overweight" merely means that the body weight is greater than normal, having regard to such factors as race, sex, somatotype, height, and age.

Assessment of Obesity

A common but highly erroneous method is to obtain the weight and height of a person and compare the former with so-called standards of weight for given sex, age (if not adult), and height. The basis of the standards is seldom considered. When nutritional surveys were conducted in Germany for the Control Commission immediately after the war considerable attention was paid to body weight, and especially to longitudinal studies of body weight in individuals. Some 100,000 persons in a wide variety of categories were weighed monthly, and these weights were compared by the Hollerith method with the previous weights; unfortunately the Control Commission had these monumental results pulped in a moment of lightheartedness. Such longitudinal studies on single individuals are very valuable, although it must be remembered that undernourished persons may adapt by doing less work, thereby conserving their body weight, and that extracellular fluid may increase and mask a decrease in the weight of the fat and tissues. But a single estimate of body weight in cross-sectional studies did not give much information.

The standard tables available were those of Hassing-Schall; it was very difficult to discover their origin, which in fact proved to be unimpressive. Since the standards were much higher than those in the use of the U.S. Army, it was not surprising that Germans appeared in many cases to have lost a considerable amount of weight, and the possibility that Hassing's subjects were obese had not been considered. Further, his male figures (Brugsch, 1922) contained the common error of being the average or arithmetic mean of a large number of observations (his female figures were obtained by subtracting an arbitrary 0.89 from the male figures): since body weights are not normally distributed about the mean but are skewed to the right, the average weight of a group of persons is more than the centre of the distribution (or mode).

A very important variable in considering the assessment of obesity from overweight is the somatotype. As W. H. Sheldon (1940) has shown, every person has a greater or less degree of endomorphy, mesomorphy, and ectomorphy. Those persons in whom one or other of the first two predominates will tend to be overweight, and the ectomorph will tend to be underweight. The endomorph has excessive fat, and his overweight indicates obesity; but the mesomorph has large muscles and bones, though he may not be tall, and the amount of fat is not necessarily excessive. Indeed, a mesomorph

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such as 163 would normally weigh much more and have much less body fat than an ectomorph such as 425 of the same height. So the measurement of body weight will not necessarily tell us much about obesity.

Methods of Measuring Body Fat

The easiest way of estimating the amount of fat in the body is to measure the amount in the subcutaneous tissue, where about half the total is located. This method has been used by clinicians for a long time. Quantitative studies have been made since the end of the last century; sometimes one site has been used, but Edwards (1950) tried 93 points and finally selected 53. In wartime studies in England, and subsequently in the famine in the Netherlands and in post-war Germany, we used a simple form of calliper and two sites—over the top of the biceps and midway down the back of the forearm. It was found that there was a correlation between the thickness of subcutaneous tissue and relative weight (as judged from standards for given sex, age, and height) of the order of 0.5; this, though highly significant and indicating that fat persons tend to be overweight, does not mean that overweight is necessarily an accurate measure of obesity. The measurement of subcutaneous fat from soft-tissue *x*-ray films (Stuart, Hill, and Shaw, 1940; Reynolds and Asakawa, 1948) does not seem to have any advantage over that of skin-fold thickness.

Professor McCance has mentioned methods of determining the composition of the body. The most accurate way of measuring the total body fat is by determining the density of the whole body (Kohlrausch, 1930; Behnke, 1942; Rathbun and Pace, 1945; Keys, 1953). Keys (1950), in a study of young and middle-aged men, found a correlation between abdominal skin-fold and relative weight of 0.475 (similar to our correlation); but the correlation between density and relative weight was only −0.24, indicating again that overweight does not necessarily afford a good index of obesity. His correlations between abdominal skin-fold and density were −0.84 for the young men and −0.60 for the middle-aged men (Keys, 1953), so we may reasonably be content to measure obesity by the thickness of subcutaneous tissue.

Assessment of Obesity in Children

The assessment of obesity in children is more complicated. There are two well-known spurts in growth that do not necessarily occur at the same ages, and the problem of what is normal growth is difficult to solve; further, the distribution of subcutaneous fat varies with age. There has been a tendency to regard maximum growth of children as the optimum. In lower animals it has been shown by various workers that overnourishment during growth leads to early maturity and a shortened life-span. A characteristic of the genus *Homo* is the relatively long time taken to reach maturity; there is no evidence that it is desirable to shorten this period of development and some evidence that it is harmful. Some children appear to put on weight much more readily than others, and endocrine glands are often blamed. We may perhaps agree with Rynearson, who is co-author of an excellent book on obesity (Rynearson and Gastineau, 1949), that "the only glands involved in obesity are the salivary glands"; yet some facts are not easily explained. For instance, Peckos (1953) claimed to have found that endomorphic children ate less than ectomorphic; but her dietary methods are not beyond criticism. It is a common observation that some endomorphs, whether children or adults, appear to eat relatively little and yet gain weight, whereas some ectomorphs appear to remain thin on large calorie intakes. The matter requires careful investigation. It must be remembered that endomorphs have relatively less surface exposed and therefore lose less heat, and they tend to be less active with slower movements and fewer gestures than the "lean and hungry" ectomorph; on the other hand, more work is done by the endomorph for a given movement.

Effects of Obesity

If obesity were unimportant there would be little need to measure it. Apart from mechanical inefficiency and aesthetic undesirability, it is the cause of disease and premature death. It is somewhat trite to mention that a person who is 40 lb. (18 kg.) overweight carries around an extra 40 lb.; this means that extra energy is expended, extra food is eaten, and there is some muscular hypertrophy (Keys, 1953). There is an increased incidence of degenerative arthritis of the lumbar spine, hips, and knees (Harmon, 1942; Danowski and Winkler, 1944; Faust, 1946), and of hernias, varicose veins (Dublin, Fisk, and Kopf, 1925), and fractures (Coombs, 1936); accidents are more frequent, presumably because the obese are slower in their movements and clumsy (Dublin and Marks, 1930). The postural changes cause respiratory and circulatory inefficiency, with emphysema (Kerr and Lagen, 1936) and decreased vital capacity (Short and Johnson, 1939).

The careful statistics presented by Dublin and his colleagues of the Metropolitan Life Insurance Company, New York (Dublin and Marks, 1930; Armstrong, Dublin, Wheatley, and Marks, 1951), have shown that at all ages men and women who are overweight have a higher mortality than the average; and the greater the degree of obesity the greater the mortality. Obese men had higher death rates from degenerative cardiovascular and renal diseases, diabetes mellitus, and cirrhosis of the liver; obese women had relatively the greatest excess mortality from diabetes mellitus and diseases of the gall-bladder, especially gallstones. The mortality is relatively worst among those under 45: among both men and women the number of observed deaths from degenerative diseases of the circulatory system and kidneys is 70% more than the expected, and it is at least double the expected among those under 45 (Armstrong *et al.*, 1951). In the course of pregnancy and childbirth obesity is a hazard: there is a greater frequency of complications, deaths, toxaemia, and large babies (Matthews and Der Brucke, 1938; Odell and Mengert, 1945; J. H. Sheldon, 1949; Gilbert, 1949).

Biochemical Aspects

I wish to mention in particular two conditions—diabetes mellitus and atherosclerosis—in which there is an increased formation of cholesterol, and to make a few brief remarks about biochemical aspects. According to Joslin, obesity is a forerunner of diabetes from middle life onwards in fully 90% of the cases, and only 5% are underweight before the onset (Joslin, Root, White, and Markle, 1940). Further, after the age of 40 the mortality from diabetes among overweight women insured at substandard rates was eight times as high as that of average or underweight women. There are two distinct biochemical mechanisms to be considered.

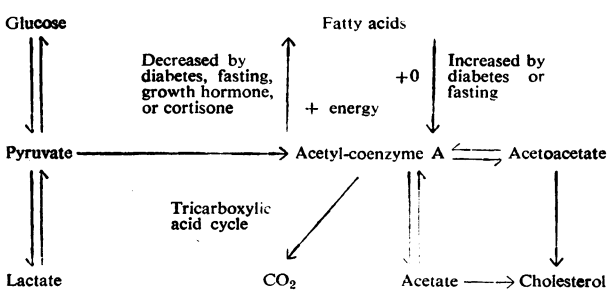

Chart showing process of formation of cholesterol.

First, fatty acids are formed in cells from 2-carbon ("active acetate") fragments arising from pyruvate and hence from glucose, or from other sources such as oxidation of fat (see Chart). Though insulin promotes the synthesis of fatty acids, probably by making energy available within the liver cells for this synthesis, it is known not to be essential: liver slices from depancreatized cats will

convert C^{14}-labelled acetate into fat at a normal rate if the hypophysis is removed (Brady, Lukens, and Gurin, 1951). These workers also found that pituitary growth hormone or cortisone decreases the synthesis of fat, and in the intact animal growth hormone or A.C.T.H. depresses the synthesis (Welt and Wilhelmi, 1950). But the reverse process —the breakdown of fatty acids into 2-carbon fragments—is increased in diabetes as it is in fasting, and from these fragments acetoacetic acid and hence cholesterol are formed (Brady and Gurin, 1951); this accounts for the ketosis and hypercholesterolaemia of diabetes. It in no way accounts for the association of obesity with diabetes; indeed, if there is decreased formation of fat from carbohydrate and increased oxidation of fat one might be tempted to expect diabetics to be thin.

Dr. Meiklejohn has suggested that chronic excessive output of insulin by the islets of Langerhans might produce obesity and lead eventually to failure and atrophy of the islets, with resulting diabetes. But the characteristic middle-aged obese diabetic belongs to the insulin-resistant type and shows no atrophy of the islets after death. When we cannot explain a mechanism in man it is useful to turn to rats and mice. This has been done with success by Jean Mayer, of Harvard, who has studied obese patients and hereditarily obese mice (Mayer, 1952). He believes that appetite is conditioned by the effective blood-glucose level, or the amount of sugar that can pass into and be used by cerebral cells in unit time. Food intake is regulated to keep the effective blood sugar constant.

The inheritance of obesity in the mice is a Mendelian recessive; the adult animals weigh up to three to four times the normal, they become diabetic by the ninth or tenth week of life, and they die prematurely with very much enlarged islets of Langerhans. The diabetes is characterized by a high degree of insulin resistance, and it is caused by a blocking by pituitary growth hormone of the action of hexokinase upon glucose. In the pre-diabetic phase in these animals, as in hypothalamic obesity, increased lipogenesis causes overeating; in the diabetic phase, the blood glucose is raised but the impaired phosphorylation of glucose keeps the "effective" blood glucose from increasing correspondingly. Mayer concludes that there is a suggestive analogy between the obesity-diabetes syndrome in mice and the insulin-resistant diabetes often encountered in obese individuals. The obese diabetic mice, like insulin-resistant middle-aged obese diabetics, have hypercholesterolaemia, and it remains to consider the relation of overweight to cholesterol metabolism and atherosclerosis.

Correlation between Obesity and Atherosclerosis

The agreed facts about obesity and atherosclerosis seem to be as follows. There is a correlation between overweight and hyperpiesis: for instance, a study of 74,000 industrial workers showed at every age and in both sexes a steady progression in both systolic and diastolic pressures with increase in body weight for height (Master, Dublin, and Marks, 1950); at necropsy, advanced atherosclerosis is much commoner in the overweight (Wilens, 1947). The development of atherosclerosis is related to the serum cholesterol level (Davis, Stern, and Lesnick, 1937; Keys, 1951a; Katz, 1952). In general, there is a steady increase in the mean serum cholesterol level of well-fed men from adult age to the mid-50s, the average annual increment being about 2 mg. per 100 ml. (Keys, Fidanza, Scardi, and Bergami, 1952). There is no relation between the serum cholesterol level and the habitual dietary intake of cholesterol (Keys, 1949; Gertler, Garn, and White, 1950), but the serum cholesterol level is altered by substantial changes in the dietary fat (Keys, Mickelsen, Miller, and Chapman, 1950; Hildreth, Mellinkoff, Blair, and Hildreth, 1951). There is a correlation between the concentrations in serum of cholesterol and of Gofman's lipoprotein molecules (Keys, 1951b). Therefore Keys, whom we were fortunate to have working with us for a year in Oxford recently, has been led to suggest that increased dietary fat raises serum cholesterol and produces atherosclerosis: since increased dietary fat will tend to cause obesity it is not surprising that atherosclerosis is commoner in overweight persons.

In many of these studies it is important to avoid the *post hoc* fallacy and the danger of confusing the cart with the horse. Because degenerative arthritis is commoner in the obese, we cannot necessarily conclude that obesity causes it: it might be maintained that arthritis limits movement and so causes obesity. Perhaps arteriosclerosis diminishes the blood supply of the hypothalamus and thereby increases appetite, with consequent obesity. In this connexion the recent work of Dublin (1953) is pertinent: he has shown that there is a substantial lowering of mortality amongst overweight persons who reduce their weight, and various disorders that are commoner in the obese can be diminished by slimming.

Ninety years ago William Banting (1863) began his *Letter on Corpulence* with the following words: "Of all the parasites that affect humanity I do not know of, nor can I imagine, any more distressing than that of obesity, and, having just emerged from a very long probation in this affliction, I am desirous of circulating my humble knowledge and experience for the benefit of my fellow man, with an earnest hope it may lead to the same comfort and happiness I now feel under the extraordinary change,—which might almost be termed miraculous had it not been accomplished by the most simple common-sense means." We know that obesity is very common, causes discomfort and ill-health and early death, and can be corrected by simple common sense. Since it also represents a waste of food, renders its subjects more prone to accidents, and can be measured, perhaps the Welfare State will consider taxing it.

Summary

Overweight and obesity are not synonymous. The estimation of obesity by comparing the weight of a person with standards of weight for given sex, age (if not adult), and height is often fallacious for several reasons: first, the basis of the standard is often doubtful; secondly, body weights are skewed and the average weight in a group is higher than the central weight; thirdly, somatotype is very important, since mesomorphs (muscular persons) may be heavier than endomorphs (fat persons) of the same height, and yet have much less body fat.

Obesity is satisfactorily estimated by measuring the thickness of a skin-fold. This correlates highly with the density of the body.

Overweight is particularly difficult to estimate in children because of the two characteristic growth-spurts and because there is doubt about what represents optimum nutriture: the maximum rate of growth is not the optimum, since overnourishment causes early maturity and probably early death.

Obesity shortens life and is accompanied by an increased incidence of accidents and of various disorders, including atherosclerosis, hyperpiesis, diabetes mellitus, chronic nephritis, arthritis, gall-stones, and complications of pregnancy and childbirth.

Hereditarily obese mice have increased lipogenesis, with consequent overeating; they are analogous to patients with hypothalamic obesity. Later they develop diabetes and are analogous to the middle-aged obese insulin-resistant diabetic.

Increased dietary fat (but not increased dietary cholesterol) tends to raise the serum cholesterol level and to produce atherosclerosis.

Reduction of body weight lowers mortality in overweight persons, and various disorders that are commoner in the obese can be diminished by slimming.

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"Health in Hot Countries" is the title of a British Council study booklet, edited and introduced by Professor J. M. Mackintosh. It is based on a series of broadcasts given in the Overseas Service of the B.B.C., and is intended for the use of medical and auxiliary staff training for public health work. The booklet presents British Commonwealth experience and achievement in the field of tropical medicine. The opening chapter by Professor G. Macdonald, director of the Ross Institute of Tropical Hygiene, is concerned with the collection and interpretation of community health statistics. Then follows a chapter by the professor of physiology at Khartum, Professor Dean Smith, on nutrition and health, which discusses the causes of malnutrition and their conquest. Dr. C. G. Pandit, secretary of the Medical Research Council for India, contributes a chapter on tropical diseases in which he gives a brief account of the treatment of malaria, plague, filariasis, kala-azar, and leprosy. The health services of Pakistan and Nigeria are the subject of the fourth chapter, the former being described by Lieutenant-Colonel Muhammed Jafar, director-general of health for Pakistan, and the latter by Dr. Oladéle Ajosé, professor of preventive and social medicine at Ibadan. Finally there is a chapter on "health engineering," written by Mr. J. W. Wright, a sanitary engineer, and Dr. J. A. Logan, director of medicine and public health, the Rockefeller Foundation, London. This chapter deals with the control of the environment and the vital problem of enlisting local co-operation in such work.

THE MAYLARD-SONNENBURG METHOD OF INTESTINAL ANASTOMOSIS

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In 1951 Kimbarovskij proposed "a new method of anastomosis of the small and large intestine." We believe that this method was devised and practised many years ago by Professor Sonnenburg, of Berlin, possibly in the late 'nineties, as told to us in a personal communication by the late W. D. Macfarlane, who was with Sonnenburg at that time.

Unaware of Sonnenburg's work, Maylard (1913) practised and developed the technique independently in Glasgow in the early part of the present century, probably about 1910. He introduced the method on account of its simplicity, safety, rapidity of execution, and immunity from leakage at the line of suture. The possibility of the establishment of a valve action comparable to the ileo-caecal valve of Talpius was regarded as a physiological possibility. Early sealing of the suture line was anticipated. His views were confirmed by post-mortem examinations. He found, in a patient who died of cardiac complications, that as early as six hours after operation the line of junction was sealed, no leakage had occurred, and regenerative processes had already begun.

His interest in this form of anastomosis was originally aroused by the difficulty he had experienced in dealing with faecal fistulae, especially those associated with the caecum and lower third of the ileum. He extended its use to cases in which he hoped to perform a partial or complete colectomy at a later date. In cases where there was an irremovable colonic tumour he performed an ileo-colic anastomosis in preference to any type of colostomy. He preferred it to a lateral or end-to-side anastomosis, the main reason being that by this method the possibility of leakage was substantially reduced.

With the exception of Bickham (1924), who gave a relatively accurate description of the method, no one appears to have described the technique.

The method is applicable to any condition in which the receiving tube is of greater calibre than the entering one. It may be practised as the first stage to colectomy, the colon being removed either in part or as a whole at a subsequent operation. It has been found useful in the treatment of ileo-caecal fistula, where it is necessary to re-route the intestinal flow from the ileo-caecal junction, as also in the treatment of obstruction of the alimentary canal proximal to the pelvic colon. It may be employed with advantage as a preliminary to subsequent operative procedures.

Principles

Technical simplicity, speed in execution, and immunity from peritoneal contamination owing to rapid sealing of the cut edges are the special attributes of this method. The