ONE HUNDRED AND TWENTY-FOURTH SCIENTIFIC MEETING
FIFTY-FIFTH SCOTTISH MEETING
COLLEGE OF DOMESTIC SCIENCE, PARK DRIVE, GLASGOW

4 APRIL 1959

OBESITY

Chairman: DR A. P. MEIKLEJOHN, *Department of Medicine, University of Edinburgh*

Obesity and disease: Chairman's opening remarks

By A. P. MEIKLEJOHN, *Department of Medicine, University of Edinburgh*

In introducing this Symposium it seems appropriate to indicate briefly the importance of the subject. Obesity is unquestionably the most common nutritional disease in Britain and gives rise to more ill-health than all the vitamin deficiencies put together. It can also be an indirect cause of untimely death; life-insurance companies do not like fat people—and with good reason. Obesity predisposes to a variety of complications, some of which can shorten life.

Mechanical complications. First there are the effects on joints and ligaments of having too much weight to carry: backache, arthritis of the knees and hips, flat foot. An excess of fat round the trunk and in the abdomen is an obstruction to free breathing; chronic bronchitis in winter is a common association. Abdominal surgery becomes more difficult when the operational field is obscured by fat. Ventral hernia is a further complication of abdominal obesity. Varicose veins occur more commonly in fat than in thin people, perhaps because the normal pumping action of the underlying leg muscles—driving the blood heartwards—is impeded. Obese people are often slow in their movements and therefore prone to accidents, at home, at work or in the street.

Metabolic complications. The majority of people who contract diabetes mellitus in middle life are obese; yet they seldom develop ketosis and seem to have a remarkable facility for oxidizing acetoacetic acid. Obesity is often associated with chronic lipaemia and a raised level of cholesterol in the blood. Perhaps for this reason, obese people have an unusually high incidence of cholesterol stones in the gall-bladder and may develop widespread deposits of cholesterol in the arteries (atheroma), sometimes leading to coronary thrombosis, renal failure or gangrene. Incidentally, they frequently suffer from high blood pressure, though usually of a benign kind. The extra load on the heart of a large body and hypertension is liable to result in angina of effort.

Clearly, much has still to be learnt about metabolism in obesity—and especially the metabolism of lipids. The paper by Dr Pawan will help us in this. Any proper study of the subject requires precise methods for measuring the degree of the disorder; Dr Tanner's paper is therefore a valuable contribution.

The manifold causes of obesity are at present ill-defined, but one thing is clear: the body assimilates more calories than it needs. One cannot make fat out of the air,

its only possible origin is off the plate. It is a question of imbalance between food intake and energy expenditure. Since food intake is regulated by appetite, Dr Morrison's paper is pertinent.

The treatment of obesity is largely a problem for dietetic practice, and it is fitting that Miss Rose should conclude this Symposium with an account of her unrivalled experience in this field. Obesity is a very prevalent disease; a proper nation-wide attack on it would employ far more dietitians than are at present registered with the British Dietetic Association.

Obesity and the control of food intake in experimental animals

By S. D. Morrison, *Institute of Physiology, University of Glasgow*

Obesity of any sort, in man or in animals, is immediately the result of a positive energy balance. A positive energy balance can occur from an excessive food (energy) intake, from a deficient energy output as heat or work, from a diminished ability to utilize absorbed energetic materials, or from any combination of these causes. This fact, being stated, does not advance the solution of the physiological problem which obesity poses. It does, however, underline the primary thermodynamic nature of the physical problem, and points the way to some of the possible experimental approaches to the study of obesity.

Over the past 20 years various techniques have been developed for the induction of obesity in experimental animals. Such obesity may be used to find what might be described as experimental analogues of human obesity syndromes. Ultimately a more rewarding approach would probably be to use these obesities not so much for the study of obesity as such, but as tools for the investigation of the mechanisms by which the normal individual maintains body-weight, and particularly the energetic part of body-weight, within very narrow limits without apparent effort or consciousness.

The many types of experimental obesity may be classified in a variety of ways: according to method of induction, according to the predominant origin of energy imbalance, or according to a variety of associated metabolic criteria such as glucose tolerance or insulin resistance. The main types of obesity are listed in Table 1 along with a qualitative estimate of the changes in energy intake and output which contribute to the obesity. Whatever their origin and detailed causation, all these obesities have in common that the source of the energy imbalance lies at least partly and often predominantly in an increased intake of food.

Mayer (1953) has proposed a dichotomy of experimental and of clinical obesities into 'regulatory' and 'metabolic' types. The basis of this separation is the proposition that some of the obesities, the 'regulatory' group, are the result of a primary breakdown in control of food intake, and others are the consequence primarily of an abnormality in the utilization of absorbed materials, the alteration in food intake being merely a reflection of the metabolic abnormality. This distinction is valuable,