Rhorer, L. (1905). Pflüg. Arch. ges. Physiol. 109, 375. Roscoe, M. H. (1950). Quart. J. Med. 19, 359. Roscoe, M. H. (1952). Lancet, 262, 444. Thorn, G. W., Koepf, G. F. & Clinton, M. (1944). New Eng. J. Med. 231, 76.

Diet in Heart Disease

By R. M. Fulton, Department of Cardiology, University of Manchester

Undernutrition is not an important factor in the causation of heart disease in this country, although elsewhere severe deficiency states such as beriberi may result in heart failure. A similar picture is seen in chronic alcoholics in whom there is a conditioned deficiency of vitamin B_1 , but such cases seldom occur nowadays. Undernutrition may be a factor in the incidence of rheumatic fever, and a decline in incidence has been noted in countries like Sweden where standards of living have improved. It is probable, however, that better housing has been as important as improved nutrition in producing this reduction.

Diet has some part to play in the treatment of heart disease. It is important to remember, however, that a patient with heart trouble is of necessity limited in many ways, and it is unjustifiable to impose further restrictions unless there is good reason for them. Needless restriction, by producing introspection, is indeed often harmful. In a well-compensated ambulant patient there is no evidence that any particular kind of foodstuff is harmful, and we can advise as Sir James Mackenzie did, 'the kind of food the patient likes, so long as it does not disagree with him' (Mackenzie, 1908, p. 263).

When the heart fails, however, some modification of this attitude is necessary. Congestion of the liver and alimentary tract impairs the normal processes of digestion and absorption. The cardiac output is reduced, and the demands of the digestive tract on the limited amount of blood available must be curtailed. Finally, there is diminished excretion of sodium by the kidneys, with a tendency for the retention of salt and water in the tissues. In the presence of decompensation, therefore, one must advise small, frequent meals which can be easily digested and absorbed, with adequate rest after food. Restriction of salt is advised in order to avoid the retention of fluid in the tissues. A really salt-poor diet, containing less than 2 g daily, may be achieved in hospital, but it is unappetizing and difficult to prepare. At home, the most one can expect is for the patient to avoid salty foods, beer, and added salt at the table. This reduces the daily intake to approximately 5 g of salt and, when the salt intake has been previously high, may be sufficient to prevent the accumulation of oedema. In most cases, however, digitalis and mercurial diuretics are required as well.

Perhaps the most important contribution of dietetics to the treatment of heart disease is in the control of obesity, for the reduction of excess weight is a very valuable weapon in the treatment of all forms of heart disease, but particularly of coronary artery disease (angina pectoris and coronary thrombosis). Although obesity produces no actual structural damage to the heart, it may itself give rise to all the symptoms of

heart disease, and when an organic lesion is present the additional burden of excess weight aggravates the symptoms and still further restricts activity. The basis of most reducing diets is a drastic reduction of fat and carbohydrate. The patient's co-operation is all-important and the ideal diet, therefore, should be easy to understand, not too difficult to adhere to and not too expensive. Reduction in weight is, at present, the single most effective measure we can employ in the treatment of coronary artery disease. It has been used on the assumption that a partly damaged heart will be more efficient if it has less weight to carry, but recent work raises the question whether dietary restriction of fat may not be of more fundamental importance.

Since the clinical syndrome of coronary thrombosis was first described (Herrick, 1912), it has steadily increased in frequency until it is now one of the most important causes of death in this country. This cannot be wholly explained by improved methods of diagnosis or by an ageing population. Any theory of aetiology must explain this increase, and must explain also why the disease is more common in men than women, in some races and countries than in others, and even in different occupations in the same country.

That coronary atheroma is an inevitable consequence of growing old is not borne out by the facts, and the influence of the 'stress and strain' of modern life is too vague a concept to be satisfactory. Atheroma is particularly common in conditions, such as diabetes and myxoedema, which are associated with a high blood cholesterol, and in familial hypercholesterolaemia, atheroma and sudden death from coronary thrombosis are common. Workers in America (Katz, 1952) have fed animals with a high-cholesterol diet and have succeeded in producing experimental atheroma. The proportion of cholesterol in these experimental diets, however, is far higher than is ever found in human diets, coronary atheroma is not always associated with a high blood cholesterol, and even when it is there is no evidence that the diet has contained an excessive amount of cholesterol. Despite the fact that coronary artery disease is frequently associated with obesity, there is little to suggest that an increased dietary intake of fat or cholesterol is important in producing atheroma. It is more probable that the metabolism of fat is at fault in some way, and recent work (Gofman, Jones, Lindgren, Lyon, Elliott & Strisower, 1950; Gofman, Graham, Lyon, Jones, Yankley, Simonton & White, 1951; Gofman, Jones, Lyon, Lindgren, Strisower, Colman & Herring, 1952) has shown that there is an increase in certain fractions of the serum lipoproteins in patients who have had a coronary thrombosis.

Further than this, one can only speculate, but environmental factors must play some part, and one is tempted to associate the increasing incidence of coronary artery disease with increasing mechanization and lack of physical activity. It is very probable that further information on the causation of coronary atheroma will be available shortly.

REFERENCES

Gofman, J. W., Graham, D. M., Lyon, T. P., Jones, H. B., Yankley, A., Simonton, J. & White, S. (1951). Circulation, 4, 666.

Gofman, J. W., Jones, H. B., Lindgren, F. T., Lyon, T. P., Elliott, H. A. & Strisower, B. (1950). Circulation, 2, 161.

Gofman, J. W., Jones, H. B., Lyon, T. P., Lindgren, F. T., Strisower, B., Colman, D. & Herring, V. (1952). Circulation, 5, 119.

Herrick, J. B. (1912). J. Amer. med. Ass. 59, 2015. Katz, L. N. (1952). Circulation, 5, 101.

Mackenzie, J. (1908). Diseases of the Heart. London: Oxford Medical Publications.

Clinical Aspects of Nutritional Problems in Obliterative Vascular Disease

By A. M. BOYD, Department of Surgery, University of Manchester Text not received for publication.

Physiological Aspects of Nutritional Problems in Obliterative Vascular Disease

By A. H. RATCLIFFE, Department of Surgery, University of Manchester Text not received for publication.