the reader is in a position to pursue further points in which he might be specially interested. This book will appeal to a wide variety of readers and certainly to all those interested in medical genetics.

H. LEHMANN

Clinical Pathology in Mental Retardation. By R. D. Eastham and J. Jancar. (Pp. viii + 356; 79 figures. 65s.) Bristol: John Wright & Sons. 1968.

Formerly mental deficiency practice was a placid backwater of psychiatry. Little was known about the causes of mental defect. The majority of cases were labelled primary amentia, and a single all-purpose gene determining mental deficiency was invoked as the cause.

A remarkable change in our concept of this subject has occurred in recent years. Many different underlying abnormalities have now been identified as producing the symptom of mental retardation. This has not only altered our attitude to the retarded but brought the subject back into the fields of general medicine, paediatrics, and genetics.

The main advances have been in metabolic and chromosomal studies. These subjects are admirably treated in this book which is the result of collaboration and exchange of ideas between a psychiatrist and a clinical pathologist, both consultants to the Stoke Park Hospital Group.

The chapters deal in turn with many disorders of amino acid, lipid, carbohydrate, endocrine, and connective tissue metabolism known to cause brain damage *in utero* or during the first two years of life. Each of the abnormalities or syndromes is described under clinical findings, mode of inheritance, laboratory findings, and nature of lesion. The often complicated biochemical changes are explained in clear metabolic diagrams and a very full bibliography follows each chapter.

There is a short chapter on chromosomal anomalies and a large section on the many other abnormalities causing mental retardation. The book concludes with a list of 78 syndromes associated with mental defect but of unknown aetiology. Future observation and careful investigation of these syndromes, many of which are rare, will lead to knowledge of their causation and eventually to their prevention.

The book is clearly and concisely written and has been carefully edited and produced. It is essentially a book of reference, being packed with facts, and will surely be welcomed by all those whose work, whether clinical or pathological, confronts them with the manifold aspects of mental retardation.

L. T. HILLIARD

An Inquiry Concerning Growth, Disease and Ageing. By Philip R. J. Burch. (Pp. vii + 213; illustrated. 63s.) Edinburgh: Oliver & Boyd.

This book presents 'a unified theory of many physiological and pathological phenomena that, hitherto, have not been clearly connected'. The author links 'central

growth control, cellular differentiation, immune mechanisms and some forms of genetic polymorphism to those many disease and ageing conditions that contribute to the ill-health, unhappiness and impoverishment of mankind'.

There seem to be two basic elements in Burch's theory. The first derives from work done by Professor Burwell and himself, and from ideas of Sir Macfarlane Burnet. According to Burwell, the function of the lymphoid system is to stimulate mitosis and to control growth by a positive self-recognition mechanism. This involves a two-way flow between the lymphoid tissue and the tissue it controls. This implies mutual recognition based on identity relations between interacting macromolecules which, the author believes, can be explained by London-van der Waals' forces. This seems surprising in that usually these forces are remarkably non-specific, and are not long range in the biological sense even though they are so called for some physical phenomena. His theory then claims that much disease is caused by a breakdown in this two-way flow which results in auto-aggressive (or 'auto-immune' in more general terminology) conditions, and that these are initiated by a small number of random events, namely a special form of somatic mutation in the genes of the lymphoid (or comparable) cells. Ageing, in the main, consists of a very large number of specific auto-aggressive disorders of this type.

The second element of this theory is derived by analogy with radioactivity. The decay of radioactivity in any given mass of radium depends on completely random, disintegrating events in the mass of atoms. The rate of disintegration (or the intensity of the radioactivity) decreases with time according to a particular negative exponential law. He finds a similar relation when he plots the relative age-specific initiation rate of certain diseases, on a logarithmic scale, against the estimated mean age at the initiation of the disease. From this he deduces that the initiation of the disease is caused by random events (in cell nuclei), very much as radioactivity is caused by random disintegrations of atomic nuclei.

The theory is developed carefully and honestly. For example, for this relation it is necessary to determine the age at which the disease was initiated; in fact the available statistics show only the age at which it was diagnosed. Guess-work (or 'trial-and-error procedures') plays no small part in estimating the age at which the disease began, but Dr. Burch makes this clear. And to get all his selected conditions to fit his theory he has to postulate as many as 18 random initiating events for one condition. In his eagerness to generalize his theory he even claims that the first fractures of the forearm are due to somatic mutations: 'Although trauma may often precipitate this type of fracture . . . the underlying pathogenesis of the condition is generally auto-aggressive.'

The theory is closely argued, and a considerable weight of evidence is offered in its support, though Dr. Burch admits that, 'unfortunately, the would-be synthesiser is obliged to survey fields in which he has no specialist training, and he cannot always distinguish,