clinically normal subsequently develop the disease. In a study by Betterle *et al*[5] four of nine adrenal autoantibody positive, non-Addisonian patients developed the disease within one to 31 months, and a fifth had reduced adrenocortical reserve.

Our results, therefore, suggest that the incidence of parathyroid autoantibodies in autoimmune adrenal disease is less than that originally observed.[1]

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Effect of BPL on haemoglobin electrophoresis

It is the practice of this department to add the compound B-propiolactone (BPL) to whole blood or plasma from patients who are HTLV-III positive. Previous workers,[1 2] have described the effect of BPL on several biochemical and haematological measurements.

During the laboratory investigation of a patient positive for HTLV-III with a sickle haemoglobin, a sample treated with BPL (Sigma Chemicals; final concentration 0·25%) gave a changed haemoglobin electrophoretic pattern, using cellulose acetate in Tris-edetic acid-borate (TEB) at pH 8·9 (Figure). This was also observed with treated normal samples and samples treated with another structural variant (Hb-C). Detection of abnormal haemoglobins was thus rendered impossible.

Further investigation showed that the Itano solubility test[3] for sickle haemoglobin and the sickle test (using sodium metabisulphite)[4] gave inconsistent results that were difficult to interpret. This could lead to false negative findings in patients with the sickle gene. Samples from such patients requiring investigation of a possible haemoglobinopathy should not be treated with BPL.

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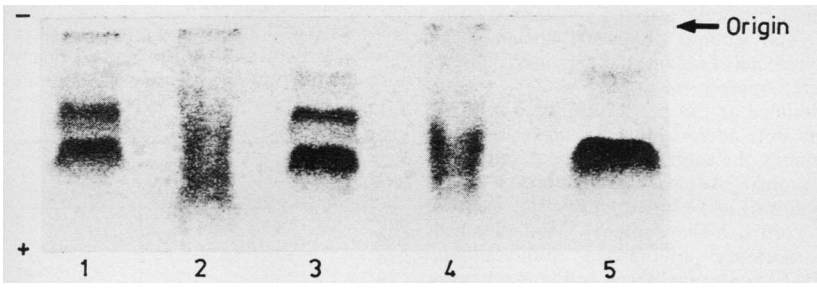

The effect of BPL on haemoglobin electrophoresis (1) sickle cell trait control; (2) sickle cell trait sample treated with BPL; (3) sickle cell trait sample untreated; (4) normal sample treated with BPL; (5) normal sample untreated.

Book reviews

Methods in Complement for Clinical Immunologists. Ed Keith Whaley. (Pp 330; £40.) Churchill Livingstone. 1985.

This is a laboratory workbook edited by a leading expert. Professor Whaley has written a substantial proportion himself; other contributions are predominantly from Glasgow. Following an introduction to the complement system, the book outlines in detail laboratory procedures for complement. These cover purification of the different components, their measurement, and immunochemical and related assays with complement components. There are some chapters describing the role of complement in specific disorders such as renal disease. The book most closely resembles a laboratory work book, and this is how it should be used. Methods are broken down into a series of simple stages. A novice in complement immunochemistry should have no difficulty in undertaking many laboratory techniques using complement by simply following the descriptions. Some may find that the book describes a few methods with which they are only too familiar in unnecessary detail. Nevertheless, I would recommend it as an essential practical handbook for laboratory workers in this field.

DL SCOTT

Brain's Diseases of the Nervous System. 9th ed. Sir John Walton. (Pp 701; £45.) Oxford University Press. 1985.

One of the more attractive aspects of specialisation is the close association between clinicians, radiologists, and pathologists. Perhaps none more so than in the neurosciences where the anatomical, functional, and biochemical complexities of the nervous system, including muscle, require that the neuropathologist has a considerable awareness and appreciation of related disciplines. Any text that helps in the acquisition and integration of large amounts of multidisciplinary knowledge is therefore to be greatly welcomed. The ninth edition of *Brain's Diseases of the Nervous System* fulfils this basic need, because it provides a comprehensive account of pathophysiological principles as they relate to the clinical features and investigation of disease and dysfunction of the nervous system. With its extensive modifications and many new refer-