ize and be retained within existing staff structures evidently does not appeal to those with commercial experience.

Specific criticisms of English policies are not spelt out in the report proper, and this was not its purpose. But there are certainly many implied criticisms of the policies of the Department of Health and Social Security, and some agree with those made in the B.M.A. report. For example, both studies disagree with the Department's policy of spending the major part of its development resources on systems relying on real-time input and output, the Scottish study mainly on grounds of the expense of the experiment and a judgement that success is unlikely, the B.M.A. report mainly because greater benefits were foreseen in other applications of computers and again because the cost of real-time processing would be too high for extended use in the health services.

But the chief criticism falls on methods of determining policies rather than on the details of the policies adopted. The B.M.A. report complained of this, and the foreword to the Scottish study declares that England seems "to move towards policies in a mysterious way which defies analysis." However, the most serious criticism arising from the Scottish report is an implied one. It shows that it is after all possible to begin by considering limitations and objectives and to proceed from there rather than to begin with the ways and means and then look for the premises later or avoid them altogether. The report shows too that it is possible to make policy recommendations on rational grounds and then expose both the objectives and the recommendations to expert examination and public discussion. This is a lesson that England has yet to learn.

Mitochondrial Antibodies

Antibodies to mitochondria were first found in human sera from patients with primary biliary cirrhosis.[1] They are most readily detected by the fluorescent-antibody method. Sections of rat kidney can be used in this technique, because the antibodies lack both organ and species specificity and the tubule cells of the kidney are conveniently rich in mitochondria. Complement-fixation tests may also be employed with purified suspensions of mitochondria as the antigen. The serum titres by the two methods show a high degree of correlation.

These antibodies have been reported in 79 to 94% of patients with primary biliary cirrhosis, and their virtual absence from patients with extrahepatic jaundice makes the test of considerable value in differential diagnosis.[2] But antimitochondrial antibodies are not common. They are apt to be found in association with other autoantibodies and with supposed autoimmune disease. Thus they are found in 8% of cases of systemic lupus, 2% of rheumatoid arthritis, and in 1-2% of patients with thyroid disease, pernicious anaemia. and idiopathic Addison's disease. In healthy middle-aged people their incidence is less than 1%. Thus they are to be expected mainly in a small group of patients with autoimmune disorders, particularly elderly women.

A valuable study of these antibodies has recently been reported by J. G. Walker, D. Doniach, and I. Doniach,[3] based on an examination of 2,500 sera from hospital patients suspected of some autoimmune disease. When all cases suspected on clinical grounds of some underlying hepatic disease were excluded, 77 still remained with demonstrable antimitochondrial antibodies. Only 35, however, were available for follow-up, and these were classified into three groups. The first group comprised eight cases with low-titre antibodies and no clinical or biochemical abnormalities suggestive of liver disease. These included four patients with connective-tissue disease and four with thyroid abnormalities associated with various antithyroid antibodies. In the second group were 17 cases with moderate or high-titre antibodies but no other evidence of liver disease. They included eight patients with connective-tissue disease, three with thyroid disease, and one with pernicious anaemia. The third group was of ten cases with moderate or high-titre antibodies and with demonstrable hepatic abnormalities. Five of these patients showed various forms of connective-tissue disease and two had thyroid disease associated with thyroid autoantibodies.

Hepatic abnormalities in the third group of ten patients were evident from the findings of a raised transaminase in five, a raised alkaline phosphatase in eight, abnormal retention of bromsulphthalein in seven, and a raised serum cholesterol in four. Though the serum albumin was within normal levels in all ten, a raised globulin due to increased gammaglobulin was present in nine. In addition antibodies to smooth muscle[4] were present in four cases. Biopsies of the liver were available from eight of the patients in this group, and all showed histological abnormality consisting of scattered single-cell necroses with accompanying mononuclear cell reaction, cellular infiltration of the portal tracts, and an increase of reticulin. In four the changes were mild and in four they were severe, with extension of reticulin into the lobules, proliferation of bile ductules, and more intense cellular infiltration of the portal tracts. None showed regeneration nodules or lymphoid follicles.

It is evident from this and previous studies that antimitochondrial antibodies are especially correlated with hepatic disorder, though they are also occasionally found associated with other autoantibodies when the liver is apparently normal. Even if other antibodies are detected or the patient is suffering from an identified autoimmune disease, the presence of antimitochondrial antibodies should direct attention to the liver. As Walker and colleagues show,[3] about one-third of such patients may have both biochemical and histological hepatic abnormalities. The precise nature of the lesions may be ill-defined. They seem most likely to be an early stage of primary biliary cirrhosis, but chronic active (lupoid) hepatitis and cryptogenic cirrhosis are also possibilities. In any event the wider use of tests for antimitochondrial antibodies should bring to light many cases of hepatic disease at a much earlier and perhaps more readily reversible stage than are diagnosed at present.

[1] Walker, J. G., Doniach, D., Roitt, I. M., and Sherlock, S., *Lancet*, 1965, **1**, 827.
[2] Doniach, D., Walker, J. G., and Roitt, I. M., *Acta Gastroenterologica Belgica*, 1968, **31**, 399.
[3] Walker, J. G., Doniach, D., and Doniach, I., *Quarterly Journal of Medicine*, 1970, **39**, 31.
[4] Johnson, G. D., Holborow, E. J., and Glynn, L. E., *Lancet*, 1965, **2**, 878.

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