CORRESPONDENCE

Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are now being received that the omission of some is inevitable. Letters should be signed personally by all their authors.

Circulating Immune Complexes in Schistosomiasis

SIR,—We read with interest the report by Drs. M. A. Madwar and A. Voller (22 February, p. 435) of the demonstration of circulating soluble antigens and antibody in schistosomiasis and the authors' suggestion that their demonstration strongly supports the association of soluble immune complexes in the aetiology of the disease. To date, however, the demonstration of circulating immune complexes has been difficult, mainly owing to the lack of sensitive methods for their detection. Immune complexes demonstrated by the inhibition of complement-dependent lymphocyte rosette formation have been shown to be present in, for example, Crohn's disease[1] and steroid-sensitive nephrotic syndrome.[2] The demonstration in this way of immune complexes in diseases where by other methods the results have proved negative indicates that this technique is very sensitive.

We have applied this technique and another method available for the detection of immune complexes, precipitation by radiolabelled C1q and polyethylene glyco,[3] to the serum of 21 patients with schistosomiasis, either *Schistosoma haematobium* or *S. mansoni*, and nine patients with schistosomiasis and malaria. Control subjects consisted of 15 Africans in whom investigations for parasitic diseases were negative. A value of inhibition over 30%—that is, twice the mean value obtained for the control African sera—was considered to be indicative of the presence of immune complexes. Thirteen of the 21 patients with *S. mansoni* or *S. haematobium* infections and four of the nine with malaria and schistosomiasis had values over 30%. The presence of an active infection—that is, the demonstration of viable eggs in the urine or faeces—appeared to correlate positively with the demonstration of immune complexes. Precipitation by C1q and polyethylene glycol gave positive results in only one serum.

These preliminary results suggest that circulating immune complexes are present in the serum of some patients with schistosomiasis. The application of the rosette inhibition test for detecting immune complexes in the serum of patients with tropical diseases is being further studied.—We are, etc.,

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1 Ezer, G., and Hayward, A. R., *European Journal of Immunology*, 1974, **4**, 148.
2 Smith, M. D., *et al.*, *Clinical and Experimental Immunology*. In press.
3 Nydegger, U., *et al.*, *Schweizerische medizinische Wochenschrift*, 1974, **104**, 126.

Myasthenia Associated with Penicillamine Treatment

SIR,—The four cases of reversible muscle weakness associated with penicillamine described by Dr. R. C. Bucknall and his colleagues (15 March, p. 600) are of great interest. However, on the data given there is insufficient justification to apply the title of myasthenia gravis to the syndrome, however suggestive the clinical picture may have been. Though myasthenia gravis has not yet been given a totally satisfactory definition, there are electrophysiological characteristics by which it can be distinguished from other forms of muscular weakness which show a positive response to cholinesterase inhibitors, such as metabolic myopathy, polymyositis, and even motor neurone disease. In true myasthenia gravis the electromyogram (E.M.G.) should show specific abnormalities to enable the diagnosis to be made, and these tests could include the response to prolonged tetanization at different rates, the sensitivity to decamethonium, single-fibre studies of jitter and blocking,[1] and the quantity and size of miniature end-plate potentials.[2] The fact that only two of the four patients are stated to have had an E.M.G. performed and that in these it was "normal" is unsatisfactory. Either the tests used were not sensitive enough or the patients were receiving cholinesterase inhibitors at the time, in which case the tests should be repeated after withdrawal.

The implication in the discussion that the myasthenic state in these patients may be akin to other forms of antibiotic-induced neuromuscular blockade is confusing. The electrophysiological findings reported with weakness after neomycin treatment are similar to those in the myasthenic syndrome[3] and distinct from those in myasthenia gravis. This distinction is of more than academic interest, since the weakness of the myasthenic syndrome can be successfully treated with guanidine.

These cases are of great interest, but to avoid confusion the condition would be better described as a toxic myasthenia associated with penicillamine. Further cases will undoubtedly be encountered and further electrophysiological studies will be necessary.—I am, etc.,

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1 Stålberg, E., and Ekstedt, J., in *New Developments in Electromyography and Clinical Neurophysiology*, ed. J. E. Desmedt, vol. 1, p. 113. Basel, Karger, 1973.
2 Elmqvist, D., in *New Developments in Electromyography and Clinical Neurophysiology*, ed. J. E. Desmedt, vol. 1, p. 229. Basel, Karger, 1973.
3 Lambert, E. H., and Elmqvist, D., *Annals of the New York Academy of Sciences*, 1971, **183**, 183.