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SUPPLEMENT

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Letters to the Editor should not exceed 500 words.

Hodgkin's Disease of the Thymus

SIR,—Your leading article (21 June, p. 713) deals with some of the interesting associations between the thymus and Hodgkin's disease. It is probable that granulomatous thymoma (as Ackerman[1] maintained) is indeed part of Hodgkin's disease, but definition is difficult, as pathologists must find Sternberg–Reed cells before they make their diagnosis. So far, while accepting the probability, we have kept granulomatous thymomas without demonstrable Sternberg–Reed cells in a separate category. To discuss this problem, as your leading article does, without reference to the work of Ackerman[1] or Lowenhaupt[2] is unusual.

Hodgkin's disease is primarily a disorder of lymph nodes spreading predominantly to other nodes. It may arise in a number of extranodal sites, one of which is the thymus. However, even if all granulomatous thymomas are included with those showing obvious Hodgkin's disease of the thymus, such an origin is still rare. I challenged A. D. Thomson's[3 4] theory that Hodgkin's disease was a primary carcinoma of the thymus at the time it was put forward,[5] while stressing the many associations between the thymus and the lymphomas. More evidence has come to light since then, particularly through their common relationships with immune disorder. As your leading article says, granulomatous thymoma is not known to be associated with myasthenia gravis, but we have recently reported[6] a case of Hodgkin's disease of the thymus which was associated with a pure red cell aplasia.

It may be that secondary involvement of the thymus is even less common than primary, though this is another distinction not easily made. In my experience[7] thymic Hodgkin's disease has a rather good prognosis favouring localized involvement. I have suggested[8] that a possible explanation of the infrequency of secondary involvement of the thymus in Hodgkin's disease may be due to the fact that recirculating lymphoid cells "home" to the lymph nodes and spleen but not to the thymus.

We require a critical review of the diagnosis of Hodgkin's disease as made by pathologists with reference to its possible recognition without the confirming presence of Sternberg–Reed cells, which might provide a more secure classification for granulomatous thymoma. We are further in need not only of a better account of those immune defects which may arise in the course of Hodgkin's disease but of those which may be concerned in its initiation.—I am, etc.,

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Hepatic Sensitization to Halothane

SIR,—Your leading article (21 June, p. 714) suggests that the jaundice which may follow halothane anaesthesia may be due to the sensitization of the liver to halothane. The suggestion would be more convincing if it were supported by more direct evidence of the assumed antigenicity of halothane. Allergic liver necrosis is readily reproducible in animals sensitized to a specific antigen.[1]

It is interesting to note that the incidence of the post-anaesthetic jaundice—1 in 10,000 administrations—is exactly similar to the predicted incidence of coincidental viral hepatitis in surgical patients one or more weeks after anaesthesia.[2] It has recently been observed in rats that halothane reduces the number of antibody-producing splenic lymphocytes.[3] There is therefore the possibility that halothane may upset the antigen–antibody ratio in patients with latent or chronic viral hepatitis, thereby precipitating an acute attack of the disease.

It is obvious that the problem of unexplained post-anaesthetic jaundice will not be solved until physicians provide themselves with the ability to diagnose viral hepatitis with serological and virological accuracy.—I am, etc.,

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Neutropenia after Trimethoprim/Sulphamethoxazole for Bronchitis

SIR,—It has been shown that there is strong synergy between trimethoprim with a sulphonamide and that the combined action is bactericidal.[1] Recently there have been several favourable reports on its use in chronic bronchitis[2] and urinary tract infections[3 4] without major adverse effects. Thirty-two of our patients with acute or chronic bronchitis were given 36 courses of trimethoprim sulphamethoxazole. In four patients significant neutropenia occurred, without clinical illness.

Their treatment regimen was either trimethoprim 320 mg. with sulphamethoxazole