histological picture was characterized by a massive, almost syncytial growth of reticulum cells with many Reed-Sternberg cells, severe lymphocyte depletion, and absence of eosinophils, plasma cells, or fibrinoid necrosis. This picture was completely different from the entity described as Hodgkin's sarcoma.

Deelman[3] and I concluded that this disease could be described, both on clinical and on pathological grounds, as a separate entity. I had the opportunity to discuss these cases personally in 1955 with both Dr. F. Parker and Dr. R. J. Lukes, who were at that time rather sceptical about my proposal to mark off this disease from classic Hodgkin's disease as a separate subgroup. However, it should be borne in mind that in 1955 discussions about the immunological nature of Hodgkin's disease were still unheard of. A good account of identical cases had already been given in 1953 by Miss A. M. Hippchen.[4]—I am, etc.,

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SIR,—Immunity may vary widely in individuals and particularly during different stages of a disease. Patients with lymphocyte-depleted Hodgkin's disease die rapidly, presumably because the illness is advanced by then and the immunity is almost spent. Untreated cases showing thrombocytopenia were also found in one series at Hammersmith Hospital (unpublished) to have died unusually promptly. The immune changes in this disease may originate some time before the symptoms and signs appear. Until the natural history of the illness is completed, however, the significance of the clinical and histological findings must remain doubtful. The pathological mechanism has to be traced to its source before it can become meaningful.

Both Hodgkin's disease and sarcoidosis sometimes show a familial incidence and blood eosinophilia, and occasionally they are combined.[1,2] Moreover, sarcoidosis is more likely to develop into Hodgkin's disease after the infiltration has healed, considering the time factor. Patients with Hodgkin's disease may have the same immune patterns as late-stage sarcoidosis, but they do not exhibit the early complications, such as sarcoid hyperthyroidism. During the course of sarcoidosis the immunity may often alter considerably, but usually the aggression is directed only defensively. The humoral response may lead rarely to early and inappropriate endocrine stimulation, and sometimes later to gland failure.[3] Cell-mediated responses are both active and prolonged in this disorder,[4] and when they are overactive one result perhaps may be Hodgkin's disease. The four types of Hodgkin's disease[5] probably arise because the degree of immunity is eventually different in each form. The scars of the immune contest are obvious, but the reason for them is still obscure. Sometimes the sarcoid agent[6] is possibly responsible for all types of Hodgkin's disease.—I am, etc.,

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American Medicine

SIR,—We enjoyed Dr. I. L. Gregory's visit to this hospital as a locum consultant surgeon in 1970 but did not realize how unobservant he was. He states (7 July, p. 50) that in none of the five provincial hospitals where he worked did he find an intermittent positive-pressure breathing machine. May I assure him that at that time we had in the operating theatres of this hospital four such machines and in the intensive care unit four ventilating machines for adults and two for babies and children. For 10 years we have had a 24-hour blood gas analysis service. I cannot, of course, make observations on his other comments about British or American medicine.—I am, etc.,

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Serum Immunoglobulins in Ankylosing Spondylitis

SIR,—We were interested to read that Dr. D. N. Golding (16 June, p. 663) found that one or more of the serum immunoglobulins was abnormal in five of his 10 patients with ankylosing spondylitis. We are currently studying this subject and would like to report our preliminary findings in view of the current interest in the aetiology of ankylosing spondylitis with its high incidence of HL-A W27[1] and the familial and clinical links with other seronegative arthropathies.[2]

Blood samples have been obtained from 25 patients with definite ankylosing spondylitis. Abnormally high results were found for IgG in 10 (40%) and for IgA in 12 (48%), while none had raised IgM or IgD values. Kriegel *et al.*[3] also found high levels of IgA and slight but insignificantly raised IgG levels in patients with progressive forms of the disease. Even more striking were the markedly raised immunoglobulin levels which we have found in the synovial fluid of patients with peripheral joint involvement.[4] These observations do not prove anything but suggest that immunological mechanisms may be playing a part in the aetiology of ankylosing spondylitis and are a stimulus for further research in this field.—We are, etc.,

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Hepatitis-associated Antigen in V.D. Clinic Patients

SIR,—The data of Dr. D. J. Jeffries and others (26 May, p. 455) raise some interesting points in relation to the changing views on the mode of transmission of hepatitis B.[1-4]

The increased hepatitis B antigen carrier rate in European homosexuals supports the proposed possibility of venereal transmission of the disease in homosexuals.[5] Skin and mucous membrane lesions usually present in homosexuals may indeed play some role in transmission. Since such lesions are also present in female prostitutes we should expect that they would also have an increased hepatitis B antigen carrier rate. However, the data presented by Dr. Jeffries and his colleagues argue against venereal spread in the heterosexual sense.

The results of a study to be published soon are in agreement with these findings. Thus hepatitis B antigen was found in nine (3·6%) out of 247 prostitutes who are regularly referred to us for check-up. A similar frequency (3·4%) was found in a sample of 379 pregnant women of similar age and of relatively low socioeconomic level. In view of the above results it would seem appropriate to look for additional reasons for the increased carrier rate among homosexuals.—I am, etc.,

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Hepatitis B Antigen in Ascitic Fluid in Cirrhosis

SIR,—Hepatitis B antigen (HBAg) is considered to be either the aetiological agent of type B hepatitis or at least closely related to it.[1-4] This antigen has been demonstrated in the serum of subjects with persistent hepatitis, chronic aggressive hepatitis, and cirrhosis.[5-7]

We have investigated the possible presence of HBAg in ascitic fluid from patients with cirrhosis whose serum was positive for the antigen. Samples of serum and heparinized ascitic fluid were tested for the presence of HBAg by an immunoreoelectrophoretic method,[8,9] using kits supplied by Farmitalia Laboratories. The specimens were collected simultaneously from subjects who had not previously undergone paracentesis and care was taken to ensure that the ascitic fluid did not contain blood. This study was performed on 12 patients with hepatic cirrhosis in the ascitic phase, eight of whom were HBAg-positive and four negative. The diagnosis of liver cirrhosis was always confirmed by laparoscopy and liver biopsy carried out after collection of the ascitic fluid.

HBAg was found in the ascitic fluid of all eight HBAg-positive subjects at a titre similar to that in the blood. The antigen was not detected in the ascitic fluid of the four HBAg-negative subjects, indicating that the