with the DF is able to separate thalassaemia trait from iron deficiency: 50 patients have an MCV of less than 75 fl; a high platelet count (more than 400 × 10^9/l) is found only in the cases with iron deficiency; when the platelet count is normal or low, the discriminant function differentiates the iron deficiency (positive DF) from the thalassaemia trait (negative DF). So we were able to identify 26 of 26 cases with iron deficiency and 17 of 19 cases with β thalassaemia trait.

In conclusion, the platelet count associated with the discriminant function may serve as a useful screening tool to detect the majority of patients heterozygous for thalassaemia or deficient in iron.

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A case of small-cell Sézary syndrome with null-cell features (Goldstone *et al.*, *Journal of Clinical Pathology*, 1976, **29**, 848)

This case was originally investigated and treated at the Royal West Sussex Hospital, Chichester. He presented in December 1974 with a history of a rash on the thighs. A skin biopsy was reported as the plaque stage of mycosis fungoides (Dr Knowles). He symptomatically improved on therapy with Dimotane and Dermovate (1 in 3) but soon relapsed and was admitted to hospital in February 1975. At this time the rash (which was itchy) was widespread over the trunk (back and front) and legs. A second biopsy was again reported as typical of mycosis fungoides. There was no splenomegaly nor lymphadenopathy. Haematological investigation showed the white count to be raised at 29 × 10^9/l with 70% small, mature-looking lymphocytes. Haemoglobin level and platelet counts were normal. Bone marrow aspiration did not show any lymphocytic marrow infiltration.

Histological examination of a normal sized lymph node, removed from the groin, showed the presence of a well-differentiated, diffuse, lymphocytic lymphoma.

The surface characteristics of the peripheral blood lymphocytes were studied in March 1975; 70% were null cells, 20% T cells, and 10% B cells. Transmission electron microscopy showed the cells to be Sézary cells. Over the next three months the skin condition remained much the same but the WBC count rose progressively until, by the end of May, it had reached 58 × 10^9/l with mature lymphocytes predominating. On 29 May 1975 treatment was started with chlorambucil, 6 mg daily. The total WBC dropped steadily over the next eight weeks and, towards the end of this time, the skin condition suddenly improved. Just before this occurred arrangements had been made for the patient to be transferred to Cambridge for consideration of whole body irradiation.

Although this additional information does not affect the conclusions of Goldstone *et al.*, it does illustrate the diagnostic problems associated with this bizarre disorder. We feel your readers will find it interesting.

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Occurrence of e antigen in acute hepatitis B

The recent paper by Gibson and Ruparelia (*Journal of Clinical Pathology*, 1977, **30**, 925-927) suggests conclusions which are quite unjustified by its evidence. Of 44 patients with hepatitis B, only six were found e antigen positive, and only two became HB_sAg carriers (why substitute 13·6% and 4·6% for integers in the summary?). If presence of e antigen and becoming a carrier of HB_sAg were independent, the probability of their coincidence in this group would be about 0·006, so the finding reported proves nothing. In addition, the authors acknowledge that they may have failed to detect e antigen in some of their patients and did not do so in two of three who eventually formed anti-e. Their conclusions about the duration of HB_s antigenaemia are also open to criticism for the same reasons.

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The author comments as follows:

In reply to Mr Davey's letter of 29 November 1977, I should like to make the following comments: the results in our paper are straightforward and although no firm conclusions are reached, those that are, are self-evident. No claim of independence is made. Of six cases where e antigen was found, none became a carrier of HB_sAg, indicating that the presence of e early in the course of acute HB_sAg is not necessarily of prognostic value. Unfortunately, blood samples from the early acute phase of illness are difficult to obtain, and of 90 cases of acute hepatitis studied, only 44 satisfied the necessary criteria to be included (ie, first serum sample taken within one month of jaundice onset and the final sample cleared of HB_sAg or when it became evident that the carrier state had developed). Those not included were patients whose serum samples were taken very late during the HBs antigenaemia and these were all e negative. The importance of this report is that in cases where blood samples were available from within one week of onset of jaundice, e was detected in these first specimens (but not in the following specimens) in 6 of 15 cases, while in the remaining 29 cases where the first specimen was obtained following one week of the onset of jaundice, e was not detected in any of the samples. The difference between these two groups is very significant.

Berquist *et al.* (1976) and Frösner *et al.* (1977) have reported similar results and reached similar conclusions. Therefore,

if any relationship exists between the presence of e and the eventual progression to the HB_sAg carrier state, it is probably related to the persistence of e but is not necessarily related to the detection of e antigen early in the acute phase of hepatitis B.

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IgA localisation in glomerular diseases

We read with interest the article by Lawler *et al.* from Manchester in the October issue of the *Journal of Clinical Pathology*, in which they described the structural changes on light and electron microscopical study of 25 renal biopsy specimens that showed significant IgA deposition on immunofluorescence and correlated their findings with relevant clinical data.

Nephrologists differ considerably in the use and indications for renal biopsy in the management of their patients. For this reason analysis of renal biopsy material may vary widely between different centres. In Bristol, the use of biopsies in renal medicine has increased rapidly with the introduction of immunofluorescent techniques, and we are now using repeat biopsies both to study the natural history of certain types of renal disease and to assess response to treatment. With this background we thought your readers would be interested in our results on a larger series of biopsies.

A total of 500 percutaneous renal biopsy specimens have been studied by direct immunofluorescence. Of these 31 were strongly positive and 46 were moderately positive for IgA. These 77 biopsies came from 60 patients (48 with one biopsy, 7 with two biopsies, and 5 with three biopsies) and formed 15·4% of the total.

IgA, usually with the C_3 component of complement, was the sole localising immunoglobulin in only five cases; in 25 it was present with IgM; in 36 with both IgM and IgG; and in 11 cases with IgG. In 52 cases IgA was the predominant localising immunoglobulin, in 14 there was no predominant immunoglobulin, and in 11 cases IgG predominated.

Our morphological findings were in general agreement with those described in the Manchester series, and on light microscopy a similar proportion of patients could be designated as mesangial proliferative glomerulonephritis (*viz*, 35 (58%) of our 60 patients in comparison with 15 (60%) of the 25 Manchester cases).

It is not necessary in this letter to analyse further the morphology of our cases. However, there are significant differences when considering the clinical data and clinicopathological correlations. Irrespective of the morphological findings, we excluded from the diffuse mesangial proliferation group those cases which had been diagnosed on clinical or immunological grounds as suffering from Henoch-Schönlein nephritis or lupus nephritis. Eight patients came into each of these two groups, and in Henoch-Schönlein nephritis IgA was always present as the predominant immunoglobulin situated in the mesangium. In contrast, out of 23 patients (36 biopsies) with immunologically proven lupus nephritis, only eight had moderate or severe IgA and in only one of these was it the predominant immunoglobulin.

The clinical presentation of the 35 cases with diffuse mesangial proliferation is also of interest:

Recurrent haematuria	17 cases
Proteinuria	9 cases
Haematuria and proteinuria	5 cases
Loin pain	1 case
Haematuria and loin pain	1 case
Nephrotic syndrome	2 cases

The sex ratio in these patients was 27 male to 8 female. We agree that the prognosis in some of these patients was unfavourable and, although this was usually associated with increasing glomerular sclerosis, crescents were unusual in our series, and the presence of IgA, which was persistent in serial biopsies, did not seem to be related to prognosis. More important in assessing prognosis seems to be the development of hypertension and/or proteinuria occurring between attacks of haematuria. In some cases renal failure developed in an alarmingly short period of time.

It is not possible to analyse our results further in this short communication but, without detracting from the Manchester authors' findings, study of a larger series widens the spectrum of glomerular disease in which IgA may be found. Analysis and publication of renal biopsy material is essential, but the material must be interpreted in the light of local conditions. Only by close co-operation between pathologists and nephrologists at different centres can sufficient material be accumulated to present a true overall picture of renal disease. The correspondence column of this journal might act as a forum to accumulate more data from centres where time and personnel are limited by heavy workloads.

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