the consultant should have the final say over who gets his job, but I feel he has a duty, having agreed to participate in the matching plan, to see all the students interested in his job before making his choice. If the number of consultants breaking the rules like this were to increase, then the whole scheme would break down.

Obviously there are advantages of the scheme which make it preferable to the previous state of affairs, such as the rules about no student holding more than one teaching hospital post and all jobs starting on standardized dates. However, the deficiencies mentioned above, must, I feel, be corrected if it is to be fair.—I am, etc.,

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Infectious Mononucleosis

SIR,—It was perhaps inevitable that Dr. H. G. Penman, a clinical pathologist who is joint editor of an excellent book on infectious mononucleosis,[1] should find much to criticize (2 June, p. 546) in my short article on this disorder (12 May, p. 350).

Much of the criticism is to do with the omission from my article of many aspects of infectious mononucleosis which Dr. Penman obviously feels should have been included. I should like to make it clear that I was requested by the Editor of the *B.M.J.* to write an article giving stress to advice on the diagnosis and management of infectious mononucleosis as it presents to the general practitioner, with particular mention of the role of antibiotics in treatment. My selection from the vast amount of information available on this disorder was in accord with this brief.

The article was written several months ago, when most of the data concerning the role of the Epstein-Barr (E.B.) virus in infectious mononucleosis was derived from serological studies. I admit to being sceptical of interpretation from serological studies in patients with a condition in which a wide variety of antibodies to human, animal, bacterial, and viral antigens have been described, and this scepticism accounted for my statement that "more research is needed to define the exact role, *if any*, of this organism in the causation of infectious mononucleosis." The more recent studies on isolation of E.B. virus from throat washings of patients with infectious mononucleosis[2 3] are much more convincing and I would certainly now acknowledge a definite role for this virus. I am, however, still hesitant about accepting the unequivocal view that E.B. virus is "without doubt the cause of infectious mononucleosis."[4]

As Dr. Penman states, infectious mononucleosis has been notifiable in some areas for several years, though it is not a nationally notifiable disease. The figures I gave for incidence in my article (2-6 per 10,000 each year) take into consideration the findings from local notification and, in fact, are the same as those quoted in the book in infectious mononucleosis edited by Dr. Penman (p. 67). In my experience, as in that of others, the preponderance of infectious mononucleosis in females in the 15-20-year age group mentioned by Dr. Penman is balanced by male preponderance in the 20-25-year age group.

The persistence of atypical cells in infectious mononucleosis for longer than two weeks is, I would agree, an important diagnostic point, but in general practice it is much less easy to collect and transport serial specimens of blood for laboratory examination, so I stressed numbers rather than duration in my article. Discussion on seronegative cases or timing of laboratory tests in relation to duration of illness would have further lengthened an article already over the 2,000 words requested by the Editor.

I did not feel that a discussion of the I/i blood group system and its relevance to infectious mononucleosis (anti-i would appear to be the commonest cause of the haemolytic anaemia which rarely complicates this condition) was appropriate for inclusion in this particular article.

Perhaps I should have mentioned the rapid slide "monospot test" as an alternative to the standard Paul-Bunnell-Davidsohn test, as many laboratories appear to have forsaken the latter in favour of the former, but my own opinion is that the monospot test is less specific than the standard test, false positives being not uncommon. I also feel more secure in a positive titre of at least 1/40 in the Paul-Bunnell-Davidsohn test though, as Dr. Penman suggests, this may be unnecessary.

The incidence of clinically obvious jaundice in my experience is about 8% but most such patients are only mildly jaundiced. I have not myself gained the impression of an increased incidence of rashes in jaundiced patients though both jaundice and rash occur together in the same patient on occasions.

Though depletion of cellular marrow elements is extremely rare in infectious mononucleosis, I am sure Dr. Penman will not deny that it occurs.[5]—I am, etc.,

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1 Carter, R. L., and Penman, H. G., eds., *Infectious Mononucleosis*. Oxford, Blackwell, 1969.
2 Gerber, P., Nonoyama, M., Lucase, S., Perlin, E., and Goldstein, L. L., *Lancet*, 1973, 2, 988.
3 Miller, G., Niederman, J. C., and Andrews, L. L., *New England Journal of Medicine*, 1973, 288, 229.
4 Henle, W., and Henle, G., *New England Journal of Medicine*, 1973, 288, 263.
5 Worlledge, S. M., and Dacie, J. V., in *Infectious Mononucleosis*, ed. R. L. Carter, and H. G. Penman. Oxford, Blackwell, 1969.

Euglycaemic Diabetic Ketoacidosis

SIR,—In identifying a group of young diabetics presenting in ketoacidosis without significant hyperglycaemia, Dr. J. F. Munro and his colleagues (9 June, p. 578) have enhanced the panorama of diabetic metabolic upsets. With the exception of vomiting, however, there was difficulty in explaining the features.

In our experience such patients are characteristically youngsters with good renal function and in some we have identified a massive urinary loss of sugar and a greater tendency to a low renal threshold to glucose than in others. Since ketoacidosis is generally regarded as the metabolic outcome of excessive gluconeogenesis coupled with increased fatty acid release, it seems difficult to postulate that the relative euglycaemia is due to a lesser glucose formation in such cases. A greater urinary loss of glucose seems to us more likely. Whether this is a consequence of increased growth hormone secretion affecting renal function, related to the enhanced clearance reported in early diabetic renal involvement,[1] or simply represents one end of the spectrum in terms of the renal threshold to glucose, remains to be determined. Whatever the explanation, perhaps the clue lies in the suggestion by Dr. Munro and his colleagues that there is an ability to grow out of the tendency.—We are, etc.,

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1 Ditzel, J., and Schwartz, M., *Lancet*, 1967, 1, 276.

Remission of Hyperthyroidism

SIR,—The remission of hyperthyroidism in five of the cases treated by Dr. D. G. McLarty and his colleagues (12 May, p. 332), was interesting and presumably spontaneous, since propranolol did not alter the thyroid function. Carbimazole treatment may accelerate the remission, however, in some cases.[1] Why certain cases remit while others need orthodox treatment is perplexing. Is the immunity more transient and less active, perhaps, in cases which remit? Two of the five cases which recovered without treatment had unilateral proptosis, and another showed periorbital puffiness. These facts may hide an important clue.

Lymphocyte sensitization to thyroglobulin (and to long-acting thyroid stimulator) was demonstrated in both normal subjects, and occurs in patients with Graves's disease.[2] Winand and Mahieu[3] were recently able to separate two types of hyperthyroidism by a leucocyte migration test, using thyroid and retrobulbar extracts. They found positive tests in nine out of 10 cases with progressive exophthalmos, and also in six out of 10 cases without proptosis.[4] None of 109 positive patients given azathioprine with the antithyroid drugs developed exophthalmos.[3] Early immunosuppression thus seemed to prevent the eye signs, which otherwise would have been expected in about 40% of cases.

Malignant exophthalmos probably consists of more than periorbital oedema, lymphorrhages, and fibrosis, complicating overaction of the thyroid.[5] Biopsy of an external orbital muscle is rarely possible, however, in this condition, and necessarily the histological changes are advanced even when this is done. For obvious reasons a muscle biopsy was not obtained in a patient reported in 1972 with hyperthyroidism, asymmetric proptosis, and myasthenia gravis.[6] The woman at this stage had a negative Kveim test, though her lymphocytes showed strong sensitization in vitro.

Sarcoidosis may be far more common than is realized,[7] and nine cases of sarcoid exophthalmos were already recorded six years ago.[8] The "granulomatous exophthalmos" occasionally described,[9] may therefore be simply sarcoidosis. Sarcoid hyperthyroidism[10] is now being diagnosed more often, in children[11 12] as well as in adults.[13 14] In one case the hyperthyroidism resolved spontaneously in five months[10] and in another it remitted after 10 mg of prednisone was given daily for eight months.[15] Possibly a number of patients with progressive exophthalmos may have undiagnosed subclinical sarcoidosis. The occurrence of unilateral and euthyroid