yields to the advances of a man, to the inevitable biological process of pregnancy, or to inner feelings or urges she loses control. I do not think it is easy for a woman to talk about losing control, and certainly these feelings would not be elicited by questions delivered briskly as a patellar hammer eliciting tendon jerks. With skilled interviewing some of these women might talk about drop attacks in the context of their psychosexual experience, but who knows if a woman will tell her doctor her secrets? Cryptogenic indeed!—I am, etc.,

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Cell-mediated Immunity in Thyroid Disease

SIR,—We were intrigued by the report of Professor S. Sherlock's group regarding the study of migration inhibition factor against liver antigen and hepatitis-associated antigen (H.A.A.) in patients positive for H.A.A. (30 December, p. 754). We have been studying cellular immune reactions,¹⁻³ including the migration inhibition factor, in thyroid disease and have made some observations that may be relevant.

We have now shown an increase in the population of peripheral T-lymphocytes^{4,5} (assayed by E-rosette^{6,7} counts) in patients with Hashimoto's thyroiditis or untreated Graves's disease. When patients with Graves's disease achieve an immunological remission as a result of treatment, the concentration of T-lymphocytes returns towards normal.⁴ We would like to suggest that additional observations of E-rosettes in patients with either chronic persistent or chronic aggressive hepatitis positive for H.A.A. may characterize them further than a study of migration inhibition factor alone may be able to achieve. It should be pointed out that we could establish a high degree of correlation between E-rosette counts and migration inhibition factor against thyroid antigen in individuals with Hashimoto's thyroiditis or Graves's disease.

Since it is probable that in aggressive hepatitis a population of killer T-lymphocytes emerges which is directed towards liver cells carrying the antigen, a model for the study of cytotoxicity should be useful. Lymphocyte cytotoxicity can be assayed by the release of ⁵¹Cr⁸ or ⁸⁶Rb⁹ from murine lymphoma cells that had been previously coated with specific antigen.¹⁰—We are, etc.,

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Teachers and Patients

SIR,—I welcome your critical comments (3 March, p. 503) on the circular on "Teaching on Patients" issued by the Department of Health, especially your emphasis on the fact that the relationship between the teaching doctor and his patient is more important than the presence or absence of students. May I take issue, however, with your suggestion that patients with psychiatric disorders and conditions "charged with emotional overtones" are best seen in non-teaching clinics. It is of the greatest importance that future doctors should become sensitive to and learn to handle the emotional problems of all their patients, whether they happen to present with a psychiatric or a physical disorder. If students were to be excluded from all such consultations their education in a large and vital area of medical and psychiatric practice would be seriously deficient and it would be their future patients who would suffer as a result.

Experience in psychiatric outpatient clinics and on general medical and psychiatric wards has shown that such problems can readily be dealt with in the presence of students, and if they themselves take an active part in the interview both patients and students benefit from the experience. In fact, the ability to create the right kind of relationship with a patient is best learnt if students get personally involved in interviews with patients presenting with psychiatric and emotional problems early on as part of their learning experience at their teaching hospital. Any suggestion that such patients should not be seen in teaching clinics should therefore be resisted.—I am, etc.,

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Serum Alkaline Phosphatase and Rickets in Urban Schoolchildren

SIR,—Dr. W. T. Cooke and his colleagues (10 February, p. 324) have made an important contribution by going out into the community in Birmingham to screen secondary schoolchildren for biochemical and radiological signs of subclinical vitamin D, iron, folate, and vitamin B₁₂ deficiencies. The iceberg analogy is presumably as applicable to vitamin D deficiency as it is to protein-calorie malnutrition or hypertension. For every florid case in the hospital there are likely to be several mild-to-moderate cases in the community which are difficult to recognize but which have some increased risk of morbidity.

It would be helpful in interpreting the Birmingham data if the authors would tell us at what season of the year the children were examined. Rickets is more likely to occur after children have been deprived of strong sunlight during the winter^{1,2} and there is some evidence that serum alkaline phosphatase levels in apparently healthy children are higher during the months when the sun is at a lower angle.³ Folic acid intake, too, is likely to vary with the seasonal availability of green vegetables. The implications of the Birmingham findings would be more serious if they were obtained in early autumn than if they were obtained in late winter or spring.

Dr. Cooke and his colleagues say that

although there were more radiological signs of rickets in Asian children there were no significant differences of alkaline phosphatase between the three groups (Asian, white, and West Indian) in either sex. Fortunately, they provided us with the frequency distributions in fig. 2, which by my count show, for example, that 10 out of 77 Asian girls had alkaline phosphatase levels above 30 K.A. units as against only one (slightly elevated) out of 44 white girls. This difference is significant by the χ^2 test ($\chi^2=3.88$; $P<0.05$).

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Lignocaine in Donor Blood

SIR,—Salicylates have recently been demonstrated in about 25% of blood donations,¹ and small amounts of other drugs such as hypnotics, tranquilizers, and purgatives are probably often present in donor blood.

We should like to draw attention to another group of drugs which is probably present in all donations. The concentrations of lignocaine in 25 samples of outdated donor blood (23-27 days old) were estimated using a gas chromatographic technique² which is able to detect as little as 0.01 μ g of lignocaine per ml. The concentrations found in each bottle are shown in the accompanying histogram.

Concentrations of lignocaine (μ g/ml) measured in the plasma of 25 separate donations of blood (in acid-citrate-dextrose) after 23 days' storage at 4°C.

The mean plasma concentration was 0.39 ± 0.16 (S.D.) μ g/ml, equivalent to 156 ± 64 μ g in the plasma transfused. This is likely to be a considerable underestimation of the total transfused, as large amounts of lignocaine are also present in the red cells. (A plasma : red cell ratio of 1.47 : 1 at a packed cell volume of 50% has been reported for lignocaine³). The lignocaine is undoubtedly derived from puncture of the site of intradermal injection of lignocaine (3 mg) at the time of donation.

The use of local anaesthesia at the time of donation is a universal pattern in Great Britain, although this is not true of the U.S.A. It seems likely that all blood used in this country contains lignocaine or another local anaesthetic as an unsuspected additive. The clinical significance of this finding is at present unknown.