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.

Many local anaesthetics are known adversely to affect *in vitro* tests of platelet function,³ but it is unlikely that lignocaine in the concentrations present in donor blood will have any detrimental effect. At high concentrations (10 mg/ml) lignocaine inhibits clot retraction⁴ and platelet aggregation and adhesion, but at concentrations of about 1 mg/ml no effect was seen.³ The satisfactory clinical results obtained from many thousands of platelet transfusions would also support this optimism.

Hypersensitivity reactions to blood in the form of urticaria have been estimated to occur in 3% of all transfusions⁵ and are generally attributed to the presence in the recipient's plasma of antibodies to IgA⁶ or IgG allotypes,⁷ or to the presence of reagins.⁸ It is possible that some of these reactions are caused by prior sensitization of the recipient to local anaesthetics. The opportunity for sensitization of the general public is high through the widespread use of local anaesthesia for dental, radiographic, and minor surgical procedures as well as through blood donation and transfusion.

These findings emphasize the wisdom of using some form of deplasmatised red cell preparation (such as red cell concentrates or frozen red cells) whenever the patient's requirement is only that of increased oxygen-carrying capacity.—We are, etc.,

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¹ *Lancet*, 1972, **1**, 477.

² Prescott, L. F., and Nimmo, J., in *Lidocaine in the Treatment of Ventricular Arrhythmias*, ed. D. B. Scott and D. G. Julian, p. 168. Edinburgh, Livingstone, 1971.

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⁴ O'Brien, J. R., *Journal of Clinical Pathology*, 1961, **14**, 140.

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⁶ Schmidt, A. P., Taswell, F., and Gleich, G. J., *New England Journal of Medicine*, 1969, **280**, 188.

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⁸ Mounsell, K., *British Medical Journal*, 1944, **2**, 236.

Congenital Hemihypertrophy

SIR,—In their paper on congenital hemihypertrophy Dr. M. Henry and others (13 January, p. 87) state that there are three principal hypotheses of pathogenesis, one postulating a chromosomal abnormality, which was suggested by the finding of congenital asymmetry in patients with diplo/d/triploid mosaicism. These observations stimulated the late Professor Penrose and me to study the chromosomes of nine patients with congenital asymmetry.¹ Three had hemihypertrophy in the strict sense, three had hypertrophy affecting predominantly one limb, while in two hemiatrophy appeared to be a more accurate description. The final patient had hypertrophy of her right arm and her left leg. This was the only patient in whom we found any suggestion of a chromosomal abnormality. There were two or possibly three triploid cells in a skin biopsy specimen from the affected arm but none in that from the leg. We concluded that although diploid/triploid mosaicism can be

associated with marked asymmetry of the body, it seems to be rare in those selected on account of asymmetry. We also noted that there was no indication of a uniform aetiology for this malformation.—I am, etc.,

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¹ Johnston, A. W., and Penrose, L. S., *Journal of Medical Genetics*, 1966, **3**, 77.

Day Hospitals and Day Centres

SIR,—The recent report by Dr. D. H. Gath and others (13 January, p. 94) on psychiatric day hospitals in Birmingham draws attention to problems surrounding day patient care. These problems are not peculiar to psychiatric day hospitals; many also apply to local authority mental illness day centres and to both day hospitals and day centres for the elderly, the physically handicapped, the mentally handicapped, and psychogeriatric patients.

In preparatory work at the University of Kent and the London School of Hygiene and Tropical Medicine for a study of day hospitals and day centres, I have collected together the official guidelines for provision of day-care facilities for these five groups. The figures, taken from many sources, are given in the table, and this is, as far as I am aware, the first time these figures have been presented together.

Day Hospitals and Day Centres: Guidelines on Provision per 100,000 Population

Need Group	N.H.S.	Local Authority	Total
Elderly	26 ¹	50 ⁵	76
Mentally ill	65 ²	60 ⁵	125
Psychogeriatric	52 ³	—	52
Mental handicap	10 ⁴	130 ⁴	140
Physical handicap	—	30 ⁵	30
	153	270	423

The sum of these guidelines on day provision is over 400 places per 100,000 population and is greater than the present acute bed provision. Clearly a major effort of expansion is required if these guidelines are to be met. More data need to be collected about the function of each type of day unit and much more thought given to the particular purpose of each kind of provision and their relationships to other branches of the health and social services.—I am, etc.,

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¹ Department of Health and Social Security, *Hospital Geriatrics Services*, letter to hospital authorities. London, D.H.S.S., 1971.

² D.H.S.S., *Hospital Services for the Mentally Ill*, HM(71)97. London, D.H.S.S., 1971.

³ D.H.S.S., *Services for Mental Illness Related to Old Age*, HM(72)71. London, D.H.S.S., 1972.

⁴ D.H.S.S., *Better Services for the Mentally Handicapped*, Cmnd. 4683. London, H.M.S.O., 1971.

⁵ D.H.S.S., *Local Authority Social Services 10-year Development Plans 1973-1983*, Circular 35/72. London, D.H.S.S., 1972.

Diseases of Western Civilization

SIR,—With regard to the article by Mr. Denis P. Burkitt (3 February, p. 274) I hope that, on the grounds of their being constructive, I may be forgiven if I add the reasons why I consider it so important,

with our refined Westernized diet, to distinguish the conditions directly due to fibre loss from those indirectly due to fibre loss, via over-consumption.

In the first place, though there is only a loose clinical association between all the manifestations of what I have termed the saccharine disease (pronounced like the river Rhine and meaning related to sugar(s), into which all carbohydrates are converted in the gut), there is a decidedly close clinical association between the conditions in the group caused by fibre loss, such as intestinal stasis, varicose veins, and diverticular disease; and likewise between the conditions in the group caused by over-consumption, such as obesity, diabetes, and gall stones. And these clinical associations can throw a penetrating light on aetiology. For example, I believe that perhaps the most vital clinical association of all is that between coronary disease and diabetes, for since, as I have tried to show, there are the strongest reasons for believing that diabetes is caused by the over-consumption of refined carbohydrates, especially sugar, it is clear that coronary disease may well have this basic causation, too, quite apart from other strong reasons.

But in the second place, and far more important, there is the question of treatment. Thus adding fibre like unprocessed bran to the diet, as set out in an earlier letter (13 May 1972, p. 408), will have a magical effect on the first group of conditions, caused by direct fibre loss, but almost none on the second group, caused by over-consumption. The reason is that though such bran will correct over-consumption arising from white flour, it will do nothing to correct over-consumption arising from sugar, which for many cogent reasons I consider far the more serious of the two.

Thus adding bran to the diet to prevent the advance of coronary disease would be as futile as adding it to prevent the advance of diabetes or, for a slightly different reason, the advance of dental decay. To cope with these conditions, not directly caused by loss of fibre, it will be essential for any fibre added to bear on sugar consumption, and bran neither supplies the bulk, nor the natural amount of sugar, needed to be successful over this. What is required here is the replacement of sugar by natural sweet things such as raw fruit, sweet vegetables, and dried fruits, as I have tried to set out.¹

It may be added here that the importance of over-consumption (especially of sugar), as distinct from direct loss of fibre, in the production of many manifestations of the saccharine disease is well seen in the explosive outbreak of this disease now taking place in the Canadian Eskimos since their sugar consumption passed 100 lb (45.4 kg) per head per year. This has recently been set out in a comprehensive manner.² Dental caries, appendicitis, diabetes, and gall stones are now rampant among these people, and in my opinion other manifestations (especially coronary disease) will certainly follow. But it is impossible to explain these events by much loss of fibre. For the natural diet of these Eskimos has always been conspicuously lacking in fibre, which has seldom exceeded the fibre found in the faeces of the reindeer, sometimes eaten by them as a vegetable during the summer months, and in bilberries, also eaten at that period. In fact, the Eskimos pursuing a sophisticated existence today consume far more fibre—