and will ensure that the quality of the hospital services will deteriorate long before any alternative adequate provisions can be created. The recent investigations into conditions at Ely and Farleigh Hospitals show how dangerous poor staff morale can be to the wellbeing of patients.

There is yet another danger which has been adumbrated by the Batchelor report on the staffing of mental deficiency hospitals in Scotland,² and that is that the care of the mentally handicapped will become divided between a number of specialists such as paediatricians and general and child psychiatrists, to whom the care of the mentally handicapped will present a fringe interest. Those who remember the dismally poor quality of the care for the aged before geriatrics was recognized to be an independent discipline will realize what a backward step such a fragmentation promises to be.—I am, etc.,

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¹ Department of Health and Social Security, *Better Services for the Mentally Handicapped*, London, H.M.S.O., 1971.

² Scottish Home and Health Department, *The Staffing of Mental Deficiency Hospitals*, Edinburgh, H.M.S.O., 1970.

SIR,—Miss Ann Shearer (4 December, p. 623) suggests that workers in the present hospitals are seeking to defend the existing pattern of residential care for the mentally handicapped in Britain by drawing false conclusions from Scandinavian practices. Without the co-operation and goodwill of the staff who care for the mentally handicapped the task of establishing new services will be harder, but the reasons for their scepticism are understandable.

This group of workers have suddenly found themselves spotlighted and made to feel scapegoats for years of parsimony and indifference towards the problems of the mentally handicapped by the community at large. It seems to them that the service which they have been giving for years has overnight become misplaced, misguided, misinformed, and has apparently been in the wrong hands. In these circumstances it is not surprising that the morale of staff in many hospitals for the mentally handicapped is poor.

Scandinavian provision has been advanced as a model, but there the equivalent of £60 per week per resident is spent on accommodation. This is three to four times as much as is spent on mentally handicapped patients in the hospitals in Britain, which constantly denied staff and resources, have had little chance to improve their effectiveness. In sparsely populated Sweden there are residential institutions which appear to be as remote and isolated as any in Britain, even though material conditions in them are undoubtedly better. Despite the emphasis on community care and small units Sweden (population 8,000,000) has nearly 2,000 places in so-called special hospitals which were excluded from the itinerary of a recent study tour,¹ but which, it was reported, housed the most severe and disturbed cases and where conditions, on the Swedes' admission, were not ideal. Hence the Swedish methods have not entirely eliminated hospitals, 7% of patients still being accommodated there.

It is these facts which prompt the suspicion that the protagonists of the Scandi-

navian system of provision are being disingenuous. The Swedes conceded that there is little voluntary service in their establishments for the mentally handicapped. Recent adverse reports about hospitals in Britain emphasized the need for a drastic overhaul of this provision for the mentally handicapped. Unfortunately this has trapped many well-intended people into supposing that some entirely different system copied perhaps from another country can be applied to all cases. In practice the apparent solution of one problem always raises others. The care of the mentally handicapped can be improved best by the application of a multi-disciplinary and eclectic approach. The relevant contributions of particular modes of provision—hospital, hostel, home, or community care—for the mentally handicapped can be determined only on a basis of evolution and experience in relation to the ethos and resources of each nation.—I am, etc.,

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¹ National Society for Mentally Handicapped Children. *Professional Study Tour 1971. Services for the Retarded in Sweden*. London, N.S.M.H.C. 1971.

Hospital-based Social Workers

SIR,—I was very interested in the letters from Drs. G. L. Davies and A. G. Chisholm and Dr. C. F. J. Cropper (1 January, p. 50). In adult psychiatry there is needed a hospital-based psychiatric visitor. These are a valuable if not essential part of a mental health team, and, indeed, their value is comparable in my view to that of a ward sister, charge nurse, or even a registrar.

Such workers have existed as part of the hospital staff, but in many areas they have also existed as part of the local authority staff by courtesy of the medical officer of health—I refer to the mental welfare officers. I consider their removal to the social services department to have been a retrograde step, taken in the teeth of expert opposition and advice. I think it arose through a semantic confusion through the words "welfare" and "social work" in the title.

What we really need is a psychiatric health visitor, and I think these should be recruited through a registered mental nurse training with a subsequent training in the methods and scope of the various other social services, and I think they should be employed by the area health boards to come. The new circular from the Department of Health and Social Security, "Hospital Services for the Mentally Ill," talks of teams, including a social worker. I think this post should be retitled psychiatric health visitor and the latter should be part of the team and not seconded from somebody else's team—that is, not from the director of social services.—I am, etc.,

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Hypoxic Newborn Infants

SIR,—Dr. J. S. Wigglesworth (1 January, p. 53) states that the burden of our paper (27 November 1971, p. 516) was to demon-

strate an association between asphyxia at birth and blood coagulation changes in the full-term newborn infant. He is quite right. Drs. Chessels and Wigglesworth's paper,¹ which appeared after our paper was accepted for publication, also gives evidence suggestive of such a relationship in babies of various gestational groups who were severely asphyxiated and required ventilation, though their series did not include results for matched control infants.

The results of our observations strongly suggested that the hypoxic babies had disseminated intravascular coagulation. Evidence for this includes lowered platelet counts, raised levels of fibrin degradation products, and prolonged thrombin clotting times. Hence we believe that babies who have sufficient asphyxia to cause a severe consumption coagulopathy are at serious risk of spontaneous haemorrhage, as the process of intravascular coagulation is accompanied by a reduction in certain coagulation factors.

An association between disseminated intravascular coagulation in the newborn and certain complications during pregnancy has been reported.^{2,3} It is therefore relevant that in our series antenatal complications (either pre-eclamptic toxæmia or antepartum haemorrhage) had occurred with 46% of the anoxic infants but with only 17% of the control babies. Two infants in our series who had severe asphyxia died. Both had Apgar scores of 0 at birth and required ventilation. One of these showed intracranial haemorrhage at postmortem. The parents of the other baby refused permission for necropsy, but as it had a falling haematocrit and a full fontanelle at death the diagnosis of intracranial haemorrhage seems likely.

We are grateful to Dr. Wigglesworth for drawing attention to the errors in Table I of our paper. The gestation in both groups was 39 and not 35 weeks as shown. The data for rectal temperatures in the two groups were inadvertently transposed: the percentage of babies who had had a rectal temperature of less than 35°C was 25% in the hypoxic group and 4% in the control group.—We are, etc.,

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¹ Chessels, J. M., and Wigglesworth, J. S., *Archives of Disease in Childhood*, 1971, 46, 253.

² Leissring, J. C., and Vorlicky, L. N., *American Journal of Diseases of Children*, 1968, 115, 100.

³ Edson, J. R., Blaise, R. M., White, J. G., and Krivit, W., *Journal of Paediatrics*, 1968, 72, 342.

Clotting Factor Concentrates

SIR,—Your leading article (8 January, p. 66) on the need for concentrates of the prothrombin complex, particularly for treatment of liver disease, prompts us to report our experience in this field.

We are just completing a pilot study on the feasibility of preparing such a concentrate at a regional blood transfusion centre. We chose the method of Bruning and Loeliger,¹ with slight modifications, for the following reasons: (1) supernatant ACD

plasma remaining after the removal of cryoprecipitate was used as the starting material, (2) the method was relatively simple for a laboratory without a fractionation plant, (3) production could be carried out on a batch basis by one trained person, and (4) the yield of clotting factors was high.

The method has proved satisfactory. The yield of clotting factors over 25 batches was as follows: factor II, 47%; factor VII, 93%; factor IX, 60%; factor X, 40%. The concentrates have been stored in the frozen state at -35°C . While this is adequate for short periods freeze-drying would be preferable. The concentrate was entirely satisfactory for the correction of clotting defects in patients with a congenital defect of factor IX and those with acquired defects of the prothrombin complex. Results to date show that a dose of concentrate, prepared from nine to ten donor-units, produces rises in factors II, VII, IX, and X of between 20 and 40%. No side effects have been observed.

Though we started to make this concentrate for patients with a congenital deficiency of factor IX (Christmas disease) adequate supplies of freeze-dried concentrate for the treatment of this condition are now available from the Plasma Fractionation Laboratory, Oxford. As mentioned in your leader, the main need for this material now lies in the management of patients with acquired deficiencies of the prothrombin complex. In an attempt to assess this demand haematologists at the six teaching hospitals in our area were recently asked to estimate the number of patients who might require the material. The estimates varied between three and 36 patients a year, with an overall suggested total of 85. While some of these patients would need only one dose (for example, to cover liver biopsy) others might need several doses for therapy.—We are, etc.,

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¹ Bruning, P. F., and Loeliger, E. A., *British Journal of Haematology*, 1971, 21, 377.

Diagnosis of Established Deep Vein Thrombosis

SIR,—We were interested to read the letters of Professor J. G. Murray and Mr. P. A. Griffiths and Dr. S. Talbot (4 December 1971, p. 624), which essentially criticized the value of the ^{125}I -fibrinogen uptake test for the diagnosis of established venous thrombosis. One of the more difficult problems of clinical medicine is the patient who presents with symptoms suggestive of deep vein thrombosis with or without symptoms suggesting a pulmonary embolus. This presentation is common and causes anxiety to both patient and doctor, especially if the patient is young. In this situation a negative ^{125}I -fibrinogen uptake test is, in our opinion, helpful.

While agreeing with Professor Murray that phlebography will give more information about the thrombus, the ^{125}I -fibrinogen uptake test is simpler and after an intravenous injection can be done by a technician.

In spite of the general view that phlebography is the most reliable technique it is well known that it cannot display every small calf vein, and we believe that the ^{125}I -fibrinogen uptake test is the more accurate test in this area.

Both letters point out, as we did, that there are known causes of false positives and, where possible, we excluded these patients from the test. This does not mean that we did not have proper controls. One studies a test in defined clinical circumstances. Our's was a study of the use of the ^{125}I -fibrinogen uptake test in legs where a diagnosis of deep vein thrombosis was being considered and other clinically detectable causes of similar symptoms excluded.

Mr. Griffiths and Dr. Talbot state that we were measuring only pooling of blood in the legs. There is no evidence for such a statement and much to dispute it. In the 46 legs positive by one or both methods of investigation where serial measurements were made, five of 11 changed from false negative to positive, and one of three from false positive to negative. Thus repeating the examination produced only a slight improvement in the accuracy of the test. In numerous animal studies we have data showing a high concentration of isotope within six hours of administration in thrombi known to be 14 days old. Furthermore, in our clinical study there was no evidence, phlebographically or manometrically, that any of the patients without swelling had venous obstruction. As the legs were examined in an elevated position Mr. Griffiths's and Dr. Talbot's theoretical notion that the high count rate was due to pooling is unacceptable.

This test is not meant to replace phlebography. We use phlebography to confirm the diagnosis in most cases and as the initial method of investigation when there are local conditions in the legs such as superficial phlebitis, gross oedema, and trauma; when there is evidence of thrombosis above the mid-thigh clinically and ultrasonically; and when serial scans show the thrombus to be extending. However, for a large number of patients an initial quick screening with the ^{125}I -fibrinogen uptake test enables us to exclude a diagnosis of venous thrombosis and avoid further investigation.—We are, etc.,

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Physiology of Adrenal Cortical Function

SIR,—Dr. Bertha Singer's article "Adrenal Corticosteroids — Physiological Considerations" (1 January, p. 36) was clear and concise. However, it seemed to contain some inaccuracies which might lead to misunderstanding of important aspects of pituitary-adrenal function, particularly in the clinical situation.

Firstly, it was suggested that the pigmentation of Addison's disease may result from the high plasma ACTH activity. As the study of Abe and his colleagues clearly showed,^{1,2} βMSH is secreted whenever ACTH is secreted and is subject to the same three basic control mechanisms as ACTH—the nyctohemeral rhythm, cortisol feedback, and stress responsiveness. This is true under normal conditions and also in

Addison's disease, Cushing's disease, both before and after adrenalectomy, and in the ectopic ACTH syndrome. Since both are secreted in excess and in approximately equal amounts in these conditions and ACTH has only 4% of the melanotropic activity of βMSH it seems unlikely that ACTH contributes much to the pigmentation.

Earlier in the article it was stated that the immunological reactivity of ACTH resides in the C-terminal part of the molecule, which contains amino-acids 25-39, rather than in the N-terminal α^{1-24} portion responsible for steroidogenesis. Since antibody formation against tetracosactrin (α^{1-24} ACTH) can be demonstrated in occasional patients on long treatment with tetracosactrin-depot (Synacthen-depot, Cortrosyn-depot), it is evident that the N-terminal portion of ACTH may be antigenic as well as the C-terminal portion. Indeed if it were not so it would be impossible to produce antisera for N-terminal ACTH radioimmunoassay.^{3,4}—I am, etc.,

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- 1 Abe, K., Nicholson, W. E., Liddle, G. W., Island, D. P., and Orth, D. N., *Journal of Clinical Investigation*, 1967, 46, 1609.
- 2 Abe, K., Nicholson, W. E., Liddle, G. W., Orth, D. N., and Island, D. P., *Journal of Clinical Investigation*, 1969, 48, 1580.
- 3 Besser, G. M., Cullen, D. R., Irvine, W. J., Ratcliffe, J. G., and Landon, J., *British Medical Journal*, 1971, 1, 374.
- 4 Besser, G. M., Orth, D. N., Nicholson, W. E., Byyny, R. L., Abe, K., and Woodham, J. P., *Journal of Clinical Endocrinology*, 1971, 32, 595.

Crohn's Disease of the Large Bowel

SIR,—Your otherwise excellent leading article on Crohn's disease of the large intestine (15 January, p. 125) contains one important error. It states that toxic dilation does not occur. Schachter, Goldstein, and Kirsner reported¹ a case, and a further five cases were reported by Javett and Brooke.² We have seen three patients with toxic dilation of the colon treated by emergency colectomy in whom the histology was typical of Crohn's disease.—We are, etc.,

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- 1 Schachter, H., Goldstein, M. J., and Kirsner, J. B., *Gastroenterology*, 1967, 53, 136.
- 2 Javett, S. L., and Brooke, B. N., *Lancet*, 1970, 2, 126.

SIR,—A misconception in your leading article (15 January, p. 125) could have serious consequences if not corrected. The categorical statement was made that toxic dilation does not occur in Crohn's disease of the large bowel. If this becomes *obiter dicta* any case presenting with acute dilation will be regarded as one of ulcerative colitis without question. The prognosis and management of ulcerative colitis differ considerably from those of Crohn's disease. It is therefore important to know that acute dilation can complicate any form of so-called "non-specific" inflammatory disease of the large intestine—ulcerative colitis, Crohn's disease, and ischaemic colitis. Very recently we have had a patient with toxic dilation proved histologically to be Crohn's disease—