with his whole body in the lateral position), and with the attendant's four fingers of the more convenient hand slightly cupped, and applied to the under surface of the chin, pulling it gently forward in such a way that each breath can be felt on the palm of the hand. Very little pressure is needed, and the method is easily learned in a few minutes even by a beginner. Immediate warning is given should respiration cease or even become shallow, and only one hand is used. I always demonstrate to each new group of nurses how chest movement *per se* means only that the patient is attempting respiration, not necessarily breathing—a fact known to all anaesthetists but usually kept a carefully guarded secret. —I am, etc.,

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C. J. LEVY.

Treatment of Macroglobulinaemia

SIR,—We were interested to read Dr. D'A. Kok's letter (January 12, p. 120) and note his agreement with the merits of plasmapheresis in the relief of the effects of raised plasma viscosity in the rapidly progressive phase of Waldenström's macroglobulinaemia, and we would, in addition, re-emphasize its apparently beneficial effect in the control of the abnormal bleeding tendency.

There are now several reported cases in which this treatment has led to amelioration of symptoms, and we hope that the simplified form of plasmapheresis described by us will permit a wider use of this procedure.

The rarity of this disease makes the evaluation of treatment with drugs difficult, since it is impossible to assemble a large enough series of patients. Furthermore, the rate of progression may vary and we believe the possibility of natural remission exists. A further case, recorded below, illustrates the latter point. Here, credit for recovery might well have been erroneously ascribed to treatment had the patient, in fact, been on any drugs.

Miss A. B., aged 71, was first seen in June, 1957, with a three-months history of abdominal pain and discomfort. She was found to have hepatosplenomegaly and an anaemia (Hb 73%, W.B.C. 2,700 (polymorphs 33%, lymphocytes 63%, monocytes 4%)), but investigations, including bone-marrow and liver function tests, were inconclusive and the cause of her anaemia remained obscure. She was admitted to hospital on September 25, 1959, complaining of tiredness, lethargy, dyspnoea on exertion, and swelling of the ankles. There had been no complaint of visual disturbance. Examination showed a markedly anaemic old lady with gross oedema of both ankles. There was no abnormality in the central nervous system apart from slight bilateral deafness. The heart was enlarged, the apex beat being in the fifth left interspace in the anterior axillary line. Pulse 90 per minute, regular. The blood-pressure was 150/50. There was a short, soft systolic murmur heard all over the praecordium. In the respiratory system there were rales at both bases but no other abnormal signs. In the abdomen the liver was enlarged 3 cm. below the right costal margin and the spleen palpable just below the umbilicus. The optic fundi showed several haemorrhages but no papilloedema or venous congestion.

On investigation the patient's haemoglobin was 36%, red cells normocytic and showing slight anisocytosis and poikilocytosis; reticulocytes less than 1%; direct Coombs test negative; serum bilirubin 0.5 mg.; faecal urobilinogen normal (two estimations); E.S.R. (Westergren) 135 mm./one hour; W.B.C. 3,800 (44% polymorphs, 51% lymphocytes, 5% monocytes); total serum proteins 7 g./100 ml., paper electrophoresis showing a small amount of abnormal pattern in the gamma globulin position; there was also a slight increase in alpha globulins. Ultracentrifuge studies (Mill

Hill) showed a significant increase in serum macroglobulins. Bone-marrow: a small amount of marrow was obtained with difficulty. Erythropoiesis was normoblastic and the myeloid cells appeared normal. There was a small increase in the number of mature lymphocytes (approximately 30%).

No drugs were given, but over ten days the patient received the packed cells from 10 pints (5.7 litres) of blood by intravenous transfusion and was discharged on October 20, 1959, the haemoglobin having risen to 72%. When seen a month after discharge the haemoglobin was 62% and the E.S.R. 84 mm./one hour.

The patient continued to attend the out-patient department and remained well until February, 1961, when she was readmitted with pneumonia, involving the right middle and lower lobes, from which she made a rapid recovery after receiving antibiotic therapy.

Between October, 1959, and September, 1961, the haemoglobin ranged between 65% and 78%, whilst the E.S.R. gradually fell from 120 mm. on May 27, 1960, to 45 mm./one hour in September, 1961, when the total serum protein was 7 g./100 ml. and electrophoresis showed no abnormality, the abnormal band of protein being no longer visible.

The patient last attended the out-patient department on August 3, 1962, when the haemoglobin had risen to 90%, the E.S.R. was 35 mm./one hour, and W.B.C. 3,800 (61% polymorphs, 35% lymphocytes, 4% monocytes).

In this case it seems that spontaneous and lasting remission coincided with blood transfusion, but it would be unwise to assume that it was necessarily produced by this procedure.

We would like to thank Dr. A. L. Jacobs for permission to publish this further case.

—We are, etc.,

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N. CONWAY.
J. MILES WALKER.

Diabetes Mellitus and Pernicious Anaemia

SIR,—In their article on the association between diabetes mellitus and pernicious anaemia (January 19, p. 159) Dr. G. Arapakis and his colleagues wisely are cautious when speculating on an aetiological connexion.

Their suggestion that patients who need insulin have an increased risk of developing gastric atrophy would be of considerable interest if this association could be clearly demonstrated in the juvenile-onset diabetic. Crozier and her associates¹ pointed out that intrinsic gastro-intestinal disease in juvenile diabetics occurs much more frequently than hitherto recognized, and the occurrence of upper gastro-intestinal disturbance, with or without demonstrable gastric retention, in the diabetic is well known.²⁻⁴ Izzo, incidentally, added a possible association between the latter and advanced diabetic renal disease,⁵ one of the "complications" of diabetes which may affect, more selectively, the juvenile-onset diabetic.⁶

Since the co-existence of these two familial conditions—namely, diabetes mellitus and pernicious anaemia—was first described by Parkinson in 1910,⁷ several authors have compared them⁸ and noted the occasional occurrence together of anaemia, histamine-fast achlorhydria, and neurological symptoms in the diabetic. Accuracy in the diagnosis of the pernicious anaemia is essential in any future study, especially if this is to be retrospective, and in this context past experience points out the masking effect of infection on the bone-marrow picture.^{9 10} Any observation on their time of onset or on the duration or "complications" of the diabetes cannot be answered dogmatically when the overt

appearance of both conditions, as the authors point out, may well be delayed.

On a different tack, and starting another hare, the recent interest in the association of pernicious anaemia and primary hypothyroidism¹¹ is perhaps pertinent, despite the rare association of this thyroid disorder with diabetes in Joslin's series. With the similarities in the pathological progress of Hashimoto's disease and pernicious anaemia¹² one cannot resist the temptation to recall the initial "insulinitis" of the juvenile-onset diabetic,¹³ with his known progression to islet atrophy.—I am, etc.,

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A. G. STEWART.

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Infective Hepatitis and Marrow Aplasia

SIR,—Since Dr. C. S. Pitcher and Dr. E. M. Spence have drawn attention to the rare association of infective hepatitis and marrow aplasia (January 19, p. 171), I should like to record briefly a further case.

On March 29, 1962, a girl aged 8½ years was admitted with marked jaundice of three weeks' duration, her illness starting with nausea, vomiting, and anorexia. The liver was moderately enlarged, bile pigment was present in the urine and absent from the stools. The following results were obtained from investigations done soon after admission. Serum: Total proteins 6.8 g./100 ml., Kunkel's test negative, thymol turbidity 1 unit, bilirubin 29.6 mg./100 ml., van den Bergh positive, alkaline phosphatase 35 units/100 ml., S.G.O.T. 590 units/100 ml., S.G.P.T. >700 units/100 ml. Blood: Hb 99%, macrocytosis, target cells, no L.E. cells, W.B.C. 6,400/c.mm. (polymorphs 52%, lymphocytes 43%, eosinophils 2%, monocytes 3%). Prothrombin concentration 47%. Prednisolone was given for 38 days, and during this time jaundice and liver enlargement lessened, although the former persisted for approximately 11 weeks from the onset.

On August 21 she returned with spontaneous bruising of two weeks' duration. At this time the blood prothrombin concentration was 100%, Hb 45%, W.B.C. 3,000/c.mm. (polymorphs 24%, lymphocytes 72%, eosinophils 1%, monocytes 3%), platelet count 8,000/c.mm. Bone marrow (iliac crest): generalized hypoplasia of all cellular elements; the erythroblasts were mostly late normoblasts, and the majority of granulocytes were myelocytes. Inquiry was made into the possibility that marrow hypoplasia was the result of drug toxicity or of contact with domestic poisons, with negative results. Treatment included further prednisolone, testosterone propionate, and transfusions of platelet-rich blood and of foetal marrow.

Until her death from extreme gastro-intestinal bleeding five months after the onset of marrow aplasia, the patient suffered repeated bleeding from several sites, and treatment, apart from blood transfusions, had no discernible effect.—I am, etc.,

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KENNETH SIMPSON.

This Island Now

SIR,—I read with very considerable interest Professor G. M. Carstairs's "Reith Lectures" (*The Listener*, November–December, 1962), and also the summary of these lectures and your leading article thereon (January 19, p. 141).

I think Professor Carstairs has put us completely in his debt by his many fascinating observations on sociological trends in this country. If there is a weakness in his presentation, it is, I think, entirely due to his almost pure-Freudian psychological approach. (I think your leader has also this implicit criticism.)

Freudian psychological theory may satisfy some people as an "interpretation," but if so they must face the fact (acknowledged by Professor Carstairs) that it is deterministic in its philosophy, and that therefore it is, in this writer's opinion, almost barren as a guide to treatment ("... psycho-analysis has undermined our confidence in the reality of free will."—Fifth Reith Lecture). It is, I fear, of no great help to discover that a pessimistic patient was bottle-fed in his infancy (Second Reith Lecture). We cannot put the clock back and breast-feed him. Nor again, I fear, is it much help to be told that "much of the delinquency shown by working-class youths can be viewed as protest behaviour" ... about the "good things in life which they would like to have, but which are still beyond their reach." (Third Reith Lecture.) Professor Carstairs points out that the prison population has increased threefold since 1938 (Fifth Reith Lecture). But there is no comparison between the material things our adolescents can enjoy now and what were available to them in the thirties.

This exclusive reliance on Freudian theory has, in my opinion, been a disastrous development for British psychiatry. To give one simple example: Professor Carstairs nowhere mentions the enormous rôle of the sentiments (first enunciated by McDougall^{1 2 3}) in the formation of character and therefore in all the subsequent choices of life. All except feeble-minded delinquents know perfectly well that stealing is wrong and that they will be punished if caught. But very few of them have built up (largely due to parental neglect, ignorance, or lack of effort) any "sentiments" about stealing. They do not associate stealing with anything mean or vicious or shameful, and hence they are liable to repeat the offence at the first opportunity, but with perhaps greater cunning. The non-Freudian child psychiatrist has here a definite aim. He knows that if he has to succeed, he must by some means build up sentiments at, say, the age of 16 that should largely have been formed at the age of 6. This can be difficult, but is often enough surprisingly easy. Nor is it even impossible with hardened criminals. And sentiments, unlike beliefs, as McDougall has pointed out, are part of the enduring superstructure of the mind, and will therefore affect conduct throughout life. Nor does Professor Carstairs make any reference, in his many controversial statements on sex, to the disharmony of the sentiments which much of this conduct ("sex