

tion of moderate severity and  $\alpha$ -haemolytic streptococci were cultured from the blood. Six hundred mg. (1 mega unit) of benzylpenicillin was given six-hourly by intramuscular injection. This was increased to 1,200 mg. six-hourly when the temperature did not settle quite to normal. Even so, low-grade fever persisted, and during the seventh week of treatment he developed a sore throat and generalized muscular pains. There was pronounced neutropenia (total white cells 2,300/c.mm. with 2% neutrophil polymorphs), slight anaemia (haemoglobin 11.0 g./100 ml.), and moderate thrombocytopenia (platelets 65,000/c.mm.). Penicillin was stopped, after which the temperature became normal, symptoms disappeared, and the haematological values rapidly returned to normal.

A second attack of infective endocarditis occurred in December 1966, and  $\alpha$ -haemolytic streptococci were again cultured from the blood. Intramuscular benzylpenicillin was given for four days while waiting for sensitivity studies. His treatment was then changed to phenethicillin 1 g. six-hourly together with probenecid 1 g. six-hourly, because it has previously been shown by one of us<sup>1</sup> that oral therapy is effective and kinder to patients when the organism causing infective endocarditis is fully sensitive to penicillin. The temperature, which had quickly fallen to normal, rose again during the second week of treatment. Although serum levels of phenethicillin were high compared with the sensitivity of the infecting organism it was thought wise to return to parenteral treatment with benzylpenicillin. The leucocyte count fell slightly during the first three weeks of treatment. During the fourth week the patient became quite unwell, with generalized muscular pains, sweating, and sore throat. The pharynx was red, but there was no ulceration and no significant bacterial growth from a throat-swab. Haemoglobin was 11.1 g./100 ml.; leucocytes 800/c.mm. with 4% neutrophil polymorphs, platelets 275,000/c.mm. Bone marrow showed normoblastic erythropoiesis but absence of granulocytes beyond the myelocyte stage of development. Megakaryocytosis was normal. Penicillin was stopped, after which the temperature returned to normal within 24 hours. The leucocyte count rose to 5,500/c.mm. (62% neutrophil polymorphs) after three days and remained normal. After 30 days haemoglobin had risen to 12.5 g./100 ml., and apart from symptoms of the heart condition he has remained well.

Serological investigations: Erythrocytes—Group O rhesus positive, phenotype ccDE. Direct Coombs test positive. Eluate showed weak direct agglutination with pooled Group O red cells suspended in both AB serum and saline containing 40 mg./ml. benzylpenicillin at 0° C., 22° C., and 37° C. High titre agglutination was found using an indirect Coombs test with broad spectrum antihuman globulin, antiIgG, and antiIgM, again at all three temperatures. Similar results were obtained by testing the patient's serum with Group O cells treated with penicillin. Both serum and eluate reactions against penicillin-treated red cells were inhibited by previous exposure for 30 minutes to penicillin, IgM or IgG. There was no evidence of blood group specificity of the antibody present in the eluate or the serum. Thirty days after the penicillin was stopped the direct Coombs test was negative and no penicillin red cell antibodies could be demonstrated. Leucocytes: There was direct agglutination of two out of four leucocyte suspensions. All four suspensions showed agglutination by complement fixation techniques carried out by Dr. W. J. Jenkins at the N.E. Metropolitan Blood Transfusion Centre (unpublished). Addition of phenethicillin 10 mg./ml. or benzyl penicillin 7 mg./ml. caused no change in the strength of reactions using the com-

plement fixation test. When the investigations were repeated 30 days after stopping penicillin therapy no agglutination could be demonstrated by either technique.

As in the previously reported cases, comparatively high doses of penicillin had been administered. The most striking feature of our patient was the occurrence of agranulocytosis on two occasions following penicillin and the demonstration of leucocyte antibodies during the second attack. As far as we are aware thrombocytopenia has not been previously observed.—We are, etc.,

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#### REFERENCE

- <sup>1</sup> Gray, I. R., Tai, A. R., Wallace, J. G., and Calder, J. H., *Lancet*, 1964, 2, 110.

### Gas Mixtures for Calibration

SIR,—The accuracy of blood  $PO_2$  and  $PCO_2$  measurements by the electrode techniques depends, among other factors, on the accuracy of the gas mixtures used to calibrate the electrodes. As the preparation and analysis of these calibrating gas mixtures requires more skill than the operation of the electrodes, most users now purchase their calibrating gas mixtures. The demand for numerous individual cylinders of "special" mixtures is now very great; their preparation, analysis, and delivery are expensive. In our experience three gas mixtures greatly facilitate blood  $PO_2$  and  $PCO_2$  measurement by spanning the most important range for both gases; they are 4%  $CO_2$ , 13%  $O_2$ ; 6%  $CO_2$ , 11%  $O_2$ ; and 10%  $CO_2$ , 7%  $O_2$ . We therefore approached the British Oxygen Company with the suggestion that they provide these gas mixtures on loan stock. They agreed and undertook to prepare the mixtures with a tolerance of  $\pm 0.5\%$  and, as an optional extra service, to supply a certificate of analysis stating the composition to  $\pm 0.05\%$ . This note is to draw attention to this service and report the agreement between the manufacturers and our own analyses.

Ten cylinders of each gas mixture were received between November 1966 and January 1968, and analysed with the Lloyd-Haldane apparatus. Each cylinder was analysed in duplicate. Room air was analysed at each session and the results were as follows:  $CO_2$ , 0.04%, S.D. 0.022;  $O_2$ , 20.92%, S.D. 0.028 ( $n=23$ ). B.O.C. analysed the cylinders either by gas chromatography or by the Bone and Wheeler absorption technique. All the cylinders supplied contained gas mixtures within the specified tolerance of  $\pm 0.5\%$ . The comparison between the certificates supplied by B.O.C. and our analyses were as follows:

	$CO_2$	$O_2$
Mean difference	+0.048 (B.O.C. and R.P.M.S.)	-0.003
S.D. of difference	0.063	0.078
S.E.M. of difference	0.0115	0.014
Maximum difference	0.22	-0.18
P	<0.001	<0.5

The reason for this small but significant difference is unknown.

There was no significant difference in the agreement for either gas at any of the three nominal ranges.

The general use of these gas mixtures in cylinders on loan should save time and

money. The user must decide whether to analyse the cylinders himself or to accept the manufacturer's certificate. If one assumes that our analyses gave the "right" values, acceptance of the values on the B.O.C. certificates would rarely have caused estimates of blood-gas tensions to be more than 1.0 mm. Hg "wrong." Whether the user analyses a cylinder or accepts the certificate it is wise to use the electrodes to compare a fresh cylinder with the old one before the old one is completely empty.

These gas mixtures are now available from British Oxygen (Special Gases Department, the British Oxygen Company, Deer Park Road, London S.W.19). We are grateful to Mr. R. C. Heape, Mr. J. Pennington, and Mr. J. H. Scawin for their enthusiastic collaboration.

—We are, etc.,

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#### REFERENCE

- <sup>1</sup> Lloyd, B. B., *J. Physiol. (Lond.)*, 1958, 143, 5P.

### Hereditary Quivering of the Chin

SIR,—I have been interested to read your leading article (20 July, p. 138) and the ensuing correspondence about hereditary quivering of the chin. I have this condition myself and so has my sister. My father, grandfather, and at least two of his sibs and his mother also suffered with this. We have all found it to be precipitated by rapid movements of the eyes such as playing table tennis or any ball game or watching birds flying, etc. It quite frequently occurs during sleep, and I have many times been woken up by an attack. It has been most troublesome in childhood and puberty, attacks tending to become infrequent and shorter in duration with age, although different members of the family have been affected more badly than others.

I am at present trying to compile our complete family incidence, and would appreciate being informed by any other affected persons or by their doctors if they have any patients with this complaint.—I am, etc.,

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### Cerebral Malaria

SIR,—It was with great interest that the preliminary communication entitled "Use of Dexamethasone in Cerebral Malaria," by Professor A. W. Woodruff and Dr. C. J. Dickinson (6 July, p. 31), was read at the 93rd Evacuation Hospital, Long Binh, Viet Nam.

During the past 10 months we have been accumulating 50–80 falciparum malaria cases monthly, with 1–2 cerebral malaria cases per month. Dexamethasone has been part of the standard treatment, along with intravenous quinine. There have been no fatalities re-

corded. Post-cerebral malaria (? oedema) residua have been non-existent (by psychological and intelligence testing). We have used the drug as a life-saving measure in spite of its reported ability to exacerbate dormant amoebiasis and tuberculosis. Its effect on melioidosis is unknown.

We are taking our study one step further. It is a clinical impression that if an absolute contraindication to a short course of steroids (three days) does not exist, dexamethasone in doses of 4-6 mg. intravenously every 4-6 hours decreases the morbidity of the disease. Severe haemolysis (except in G.6-P.D. deficiency), cerebral malaria, renal and pulmonary complications may be prevented or rapidly reversed. We have been disseminating this information verbally. A protocol is being followed, and when significant case numbers are accumulated a report with positive results undoubtedly will be recorded. This brief letter may initially make physicians aware of what may be a significant advance in a disease that is still a world-wide problem.—I am, etc.,

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SIR,—Recently a Spanish sailor of 50 was admitted to a Liverpool hospital. He was transferred from his ship, which had just returned from the west coast of Africa via the Canaries, having been found unconscious in his cabin. Six days previously he had been seen on arrival in Bristol and given a course of tetracycline for a pyrexia which had started six days earlier, accompanied by headache and chest signs.

On admission he was deeply unconscious with no focal neurological abnormalities and was apyrexial. As his cabin locker had contained large quantities of proprietary medicines he was initially considered to have taken an overdose of drugs. Examination of the blood and urine, however, failed to confirm this, and later the same day he developed a fever of 100° F. (38° C.). A lumbar puncture was entirely normal, but blood films revealed infection with *P. falciparum* (malignant tertian malaria). The parasitaemia was 9%. Mature forms including schizonts were found in the peripheral blood. He was treated with parenteral chloroquine, and within 72 hours the blood was clear of parasites. Corticosteroids in the form of hydrocortisone hemisuccinate were given in high dosage, initially 200 mg. four-hourly, without any observable effect on the course of the illness. He remained comatose for five days after admission, and during this time showed evidence of a left hemiparesis and suffered a generalized epileptic attack. However, he then rapidly regained consciousness and made a full recovery with no residual neurological sequelae.

In this patient diagnosis was delayed several hours because the temperature was normal on admission. The importance of blood-film examination in any unconscious patient who has recently returned from a malaria endemic area cannot be too strongly emphasized. In fact the diagnosis might well have been made when the patient first presented six days earlier with fever and headache. Blood-film examination at that time would have averted a severe illness and what could have been a fatal outcome. With successful treatment of the infection prolonged coma is unusual in cerebral malaria, but this case not only confirms that it does occur but also demonstrates the possibility of

complete recovery even after five days' deep coma, and fully justifies persisting with strenuous efforts to support the patient until consciousness returns.

*Falciparum* malaria in the non-immune justly deserves the prefix "malignant," and in view of its increasing occurrence in Britain must be recognized promptly and treated efficiently if we are to avoid fatal or near fatal results.—We are, etc.,

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### Intraperitoneal Blood Transfusion

SIR,—The paper by Dr. J. M. Fowler and others (27 July, p. 220) certainly demonstrated the effectiveness of intraperitoneal blood transfusion in the adult, but as an aid to the treatment of severe anaemia there is a much simpler method.

If a short-acting diuretic is given during intravenous transfusion there follows a brisk diuresis which prevents circulatory overload. Fisher *et al.*,<sup>1</sup> using frusemide, have shown this method to be successful, while Ledingham<sup>2</sup> has transfused patients initially in congestive heart failure by adding ethacrynic acid to the transfused blood. Blood transfusions undertaken in this hospital on children with thalassaemia are always aided by a diuresis from frusemide, and over the past two years there have been no complications.

The technique is simplicity itself. The intravenous transfusion is started and 20 mg. of frusemide given intramuscularly. This dose is repeated if the urine output falls below the level of the fluid intake. This method has wide application for all severe anaemias, unless associated with renal disease; does not require the apparatus for exchange techniques, and retains an intact peritoneum.—I am, etc.,

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### REFERENCES

- <sup>1</sup> Fisher, J. A., Gibbon, J. E., Netscher, M., and Bradford, D. E., *Lancet*, 1966, 2, 545.  
<sup>2</sup> Ledingham, J. G. G., *Lancet*, 1964, 1, 952.

### Doctor or Social Worker ?

SIR,—I found your leading article (3 August, p. 265) on the Seebohm Committee's report<sup>1</sup> very pertinent and the remarks of Dr. J. D. Kershaw (24 August, p. 497) equally apt. I believe a few other points are also worth consideration.

Firstly, the expansion of community care programmes under medically qualified personnel capable of evaluating the service and the needs of the individual within the service would inevitably be stopped. Indeed, it seems certain that existing provisions would be curtailed. Many hospital consultants now recognize the value of comprehensive analysis of intertwining social and medical factors both within and without the hospital setting, and appreciate that, for the individual, the service should be fully co-ordinated. This is surely better done by administrators trained in medicine and sociology (i.e., social medicine consultants) and field workers with a

base in medicine and social work (i.e., health visitors).

Secondly, if workers dealing with the social factors of illness are entirely separated from the medical services a profoundly disturbing situation could arise. Patients referred for social difficulties with a medical causation—e.g., physical and mental long-term handicap—could be "treated" by social workers with little or no medical insight. This, I feel, is extremely likely to occur if those workers (e.g., psychiatric social workers) connected with the disabled are incorporated in the social work department. To some extent this already happens. Anyone in the profession must surely regard the development of a second-rate medical consultative service for the poor and socially vulnerable as highly undesirable.

Thirdly, the social factors concerned with the epidemiology would be much more difficult to elicit. This is because very few existing departments keep centralized records and are unlikely to develop them in the near future.

However, it is absolutely certain that social work should be organized in a single social work department. Indeed, the possibility of social workers being stationed in the field in the health centre working alongside general practitioners is an exciting one. Moreover, some may argue that the Seebohm Report's recommendations are based on the jealousy of a very new profession for the skill, power, and position of a very old profession. This, I feel, is not true. However, initially, before responsibilities are delineated, it is important to ascertain the true sociological role of the new social work department and to realize that a wrong analysis of the situation resulting in ill-trained staff in charge of the large resources governing medical services could cause inefficiency, lack of co-ordination, and the prevention of progressive developments.—I am, etc.,

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### REFERENCE

- <sup>1</sup> Report of the Committee on the Local Authority and Allied Personal Social Services, 1968, Cmnd. 3703. H.M.S.O., London.

### Purgatives and the Colon

SIR,—As the manufacturers of standardised senna (Senokot) we have been greatly interested in the studies of Dr. F. Avery Jones<sup>1</sup> and Dr. Barbara Smith<sup>2</sup> on the "cathartic colon" and especially in your assessment of their work (13 July, p. 74) "Purgatives and the Colon."

The histopathology described by Dr. Smith clearly provides valuable objective evidence of the cellular damage caused by prolonged purging, but whether the effects described are specific to a particular drug or drugs, or whether they are the non-specific effects of excessive purging, is open to question. Dr. Smith favours the idea of a specific causation, because, as she puts it, "it is almost impossible to wear out a physiological reflex in the presence of normal neurones." An alternative and, we think, a more likely possibility is to be found in the atrophy which results from disuse of all muscle tissue. In the case of the colon, motility mechanisms are easily disrupted, and we suggest that atony leading to atrophy of its