SIR,—Dr. P. Moxon (11 March, p. 693) draws his inferences from very tenuous reasoning. Neither I nor, I believe, any of the other doctors who signed the doctors and overpopulation letter (8 January, p. 108) would wish the emphasis to shift from prevention to termination of pregnancy. Before the Abortion Act is made the scapegoat for the increased number of handicapped children, Dr. Moxon should remember that in the 12 years before the Act was passed the numbers of educationally subnormal and mentally handicapped children almost doubled. (These figures are taken from "Family Planning and Population Control," a paper read by Dr. R. D. Haigh at the 4th Biennial Conference of Maternity Liaison Committees in the Sheffield Regional Hospital Board Area on 16 November 1971.)

To say that the United Kingdom would have a falling population by 1974 if present trends continue ignores the Registrar General's return for the quarter ending 31 March 1970. This points out that 70% of all births in the United Kingdom occur to women aged 20-29. The numbers of these potential mothers in 1981 and those at the most fertile ages in 1991 can be predicted with some confidence. Table 3 on p. 53 shows that there will be a 20% increase in 1991 and a 23% increase by 2001. "This increase in the population of women means that unless a very substantial and quite unforeseen fall in fertility rates should occur, the number of future births will show a rising trend, a little more sharply in the 1980s than in the 1970s."

As to his warning about an increased dependency ratio, of course we realize that a falling population means that for a period of 50 years there will be a relative increase in the over-65 age group. This is a small disadvantage compared with the eventual results of an ever-expanding population. For a nation that twice within a lifetime has sent its young men to die in their hundreds of thousands, it would seem to be a relatively trivial burden to face.—I am, etc.,

P. J. HORSEY

Compton,
Winchester, Hants

SIR,—Dr. P. Moxon (11 March, p. 693) raises a number of points. The birth rate has been dropping and stood at 16.2 in 1970. This indeed is below the rate for 1931, but there is no consideration of the death rate at that time. Our death rate now is about 12; in the 1930's it was about 16—that is, replacement rate. Consequently our present birth rate must approach 12 before the population will stabilize. With a birth/death rate of 16/12 our population will continue to grow at about 0.4-0.5% per year. If this, say, were reversed and our population allowed to decline at the same rate the proportion of active, working people would be exactly the same as now. (It is not only the old who need support—children are also very dependent members of the population.) In addition many over 60s, now required to retire, could continue active, useful lives. (Incidentally, as a post-script, the 1971 birth rate announced recently shows an increase over 1970.)

The original signatories (8 January, p. 108) of which I am one, did not propose political control of population. What I, and I'm sure the others, want to see are the

facts laid simply and understandably before the public. Given this type of education and the wherewithal to voluntarily limit family size then much, and possibly all of the problem in these Islands would be solved without such political control. Since a massive educational programme is the linch-pin of this type of action then Dr. Moxon's point that only the educated will respond can be seen in a somewhat different light.

With an increase in public knowledge, plus increased availability of contraceptive methods, it is likely that the abortion rate will drop and not "have to rise to about 400 per 1,000 births to eliminate population increase." Prevention is better than cure.—I am, etc.,

JAMES BRIGGS

Department of Pathology,
Frenchay Hospital,
Bristol

Donors for Organ Grafting

SIR,—Your leading article on donors for organ grafting (4 March, p. 582) concluded by saying: "There is, however, an urgent need for more organs, and these might be available if the medical profession and public were more aware of the lives that could be saved by donations from the dead."

I have been requested to carry out a survey to estimate the possible number of donors for organ grafting which could have been made available in one year in a typical peripheral hospital. The preliminary findings at this hospital for the year 1971 were a total of 190 deaths. Of these, 13 could have been suitable candidates. Their ages ranged between 15 and 59 years, the average age being 43. Six of these cases had to be ventilated and subsequently ventilation was discontinued. The cause of death: intracranial haemorrhage, 5; head injuries, 3; brain tumour, 1; overdoses, 2; brought in dead, 2 (died approximately 10 minutes before arrival and certified dead. Subsequent necropsy showed coronary occlusion but no evidence of widespread arterial disease).

This hospital is within easy reach of the London Hospital, where there is a renal transplant team on call 24 hours a day, who have a mobile unit fully equipped for the preservation of kidneys.—I am, etc.,

M. W. SALAH

St. Margaret's Hospital,
Epping

Syphilis as a Cause of Backache

SIR,—I should like to report an uncommon presenting symptom of secondary syphilis.

A 25-year-old labourer attended a casualty department in the small hours of the morning complaining of persistent low backache of recent onset. Subsequent examinations by an orthopaedic surgeon and rheumatologist failed to make a diagnosis, but by this time, two weeks after initial presentation, the backache had ceased, a generalized eruption having developed. The patient was seen by a dermatologist, who, suspecting secondary syphilis, referred the patient to this department.

At this stage, three weeks after first attendance, the patient manifested several of the more common symptoms and signs of secondary syphilis: sore throat and malaise; a widespread maculo-papular, squamous, erythematous eruption on the trunk

and flexor surfaces of the arms; palmar and plantar syphilides; a squamous eruption on the scrotum; syphilitic balanitis; anal condylomata lata; snail track ulcers on the tonsils; and a generalized lymphadenopathy.

Treponema pallidum was demonstrated from the anal condylomata lata. Reiter protein complement fixation test; cardiolipin Wasserman reaction, and fluorescent treponemal antibody test were positive. Venereal Disease Reference Laboratory slide test was positive with serum diluted 1 in 64. Erythrocyte sedimentation rate was 50 mm in the first hour.

The patient admitted to a previous episode of urethral gonorrhoea and recent frequent promiscuous homosexual liaisons, usually after meeting men while drinking in public houses in the locality. He completely recovered after treatment with 13.5 megaunits intramuscular procaine penicillin given over 15 days, a Jarisch-Herxheimer reaction being sustained after the first injection of penicillin.

Stokes *et al.*¹ gave the term ostealgia to the indefinitely localized bone pains of secondary syphilis for which no pathological background could be found. They stated that traditionally the leading symptomatic feature of the bone changes of early and recurrent syphilis was pain, nocturnal in character, exaggerated by heat, and relieved by movement of the affected part. Other authorities, such as Catterall,² state that osteoscopic pains, characteristically worse at night, may occur in secondary syphilis—however, usually in the long bones and due to a periostitis.

In recent years the incidence of early syphilis has declined to such an extent that its more unusual manifestations are not often seen. Nevertheless as Jefferis³ has stated, two or three cases of this potentially crippling and sometimes fatal disease are still seen each week in the larger metropolitan clinics. For the reason that syphilis may still be the great imitator this case is reported.

I should like to thank Dr. J. L. Fluker for his encouragement for me to report this case in his care.—I am, etc.,

MICHAEL WAUGH

West London Hospital,
Charing Cross Group of Hospitals

¹ Stokes, J. H., Beerman, H., and Ingraham, N. R., *Modern Clinical Syphilology*, 3rd edn. Philadelphia and London, Saunders, 1944.

² Catterall, R. D., *A Short Textbook of Venereology*. London, English Universities Press, 1965.

³ Jefferis, F. J. G., *British Medical Journal*, 1970, 3, 225.

Sickle-cell and Altitude

SIR,—We were gratified by the interest in our paper on "The Sickle-cell and Altitude" (4 December, p. 593) and would like to thank the four correspondents from Ghana (1 January, p. 53; 8 January, p. 113; and 15 January, p. 177).

We would agree that the sickle-cell trait is more widespread than in specifically Negro races and, in retrospect, we regret the omission from our paper of a statement on its geographical distribution. Our critics have interpreted this omission as ignorance on our part, but we would suggest that this fact is so well established that at the time we considered such a statement to be unnecessary. It is true that two small surveys in the Lake Copais and Petromagoula areas of Greece revealed sickle-cell

trait incidence of 26.9%¹ and 20.6%² respectively, and while accepting that such foci of the gene do exist, it is important not to lose sight of the major areas where problems associated with the sickle gene are more likely to occur. There is no doubt that should screening programmes for aircrew be adopted, these must be applied regardless of race. The present policy of some authorities in screening only people of Negro origin would miss examples of sickling in other ethnic groups and is to be deplored. Dr. R. A. Lewis (8 January, p. 113) feels that we have misquoted a statement from his book,³ and in fairness to this author we would like to produce the relevant passage in its entirety.

"With respect to occupational ability, individuals with G-6-PD deficiency do not suffer from any occupational handicap, and people with sickle-cell trait are only at risk when the type of work entails frequent exposure to reduced oxygen pressure. Although there are military and civilian pilots who have sickle-cell trait and who have not suffered from any disability during their career, there is still some risk involved, especially that of splenic infarct. It is therefore advisable to discourage individuals with sickle-cell trait from entering a career which involves frequent airplane flights. On the other hand, it is not necessary to dismiss or ground individuals who have already been trained or employed before the sickling trait was noted. These individuals deserve the benefit of medical supervision."

We apologize to Dr. Lewis for our inadvertent substitution of the word "patient" for an individual who deserves the benefit of medical supervision. The use of the word individual or patient for those rare cases of the sickle-cell trait in which symptomatic episodes are or might be related to the presence of Hb S is a semantic problem. In his book Dr. Lewis has summarized the situation:

"Sickle-cell trait is not a disease. For this and other reasons it is almost impossible to make the clinical distinction between a person with normal haemoglobin and one with sickle-cell trait. The only clues to the existence of sickle-cell trait are idiopathic haematuria and infarction following airplane flight." To these should be added the hazards of anaesthesia, and Dr. F. I. D. Konotey-Ahulu⁴ stated "While the difference in haemoglobinopathy between normal homozygotes and persons with the sickle-cell trait is qualitative, that between persons with the sickle-cell trait and those with sickle-cell anaemia is quantitative; so that the same conditions which would cause crises in patients with sickle-cell anaemia could, if pushed to a further unphysiological degree, also cause in-vivo sickling in persons with the sickle-cell trait." We submit that exposure to altitude is such a condition.

It has been inferred that cases of infarction in flight previously reported as the sickle-cell trait were perhaps examples of other sickle-cell syndromes. It was suggested that ignorance of Hb C prior to 1951 may have confused the diagnosis but only one case report preceded that time. Case reports since 1951 (which include all but Sullivan's (1950) case) have been queried by your correspondents on the grounds that there was insufficient evidence for the trait. It has been pointed out that there is a close similarity between the haemoglobin electrophoretic patterns in the sickle-cell trait and in some cases of sickle-cell β -thalassaemia. However, the concentration of Hb A in sickle-cell β -thalassaemia uncommonly exceeds 30%, whereas in the sickle-cell trait it is usually above 60%. In short, the Hb S band is more prominent in sickle-cell β -thalassaemia and the Hb A band is

more prominent in the sickle-cell trait. The strict diagnostic criteria adopted by Dr. Konotey-Ahulu (15 January, p. 177) for the sickle-cell trait are admirable, and with these in mind we would like to consider the following case reports. Smith and Conley⁵ reviewed the haemoglobin electrophoretic pattern in 15 cases of splenic infarction occurring in flight. In 11 of these they noted two bands, and particularly commented that "the amount of normal hemoglobin exceeded the amount of sickle hemoglobin. This is the pattern in the sickle-cell trait." Rotter *et al.*⁶ described three cases of splenic infarction in which investigation indicated Hb A levels of 56%, 56.2%, and 57.5% respectively. Thompson⁷ reported a case of splenic infarction in which the AS pattern was present with 40% Hb S.

It appears to us that these 15 cases are acceptable as examples of the sickle-cell trait and we feel that there is no reason to entertain any other diagnosis. We consider that the association between the sickle-cell trait and splenic infarction at high altitude is established, and it is against this background that we were surprised at the incredulity surrounding Case 1 of our article (the Ghanaian sickle-cell trait case who developed an infarctive crisis during flight in an unpressurized aircraft). However, in this case we must agree that the available haematological investigations have not excluded a diagnosis of sickle-cell disease. The surgeon in charge of this case (Wing Commander Keiller) has forwarded all available details to Lieutenant-Colonel R. O. Addae as requested (1 January, p. 53), and we share his interests in further investigations to establish the genotype in this patient.

It was also suggested that the four fatal cases reported by Jones *et al.*⁸ at 4060 feet (1234 m) were possibly not sickle-cell trait carriers. Elsewhere Binder and Jones⁹ stated that three of the four cases had haemoglobins A and S present on electrophoresis with "an obvious predominance of Hb A in each case." As stated earlier, we consider this is good evidence for the sickle-cell trait. At this time, however, it is impossible to relate the significance of these fatalities to members of the general population with sickle-cell trait.

At this stage we would like to restate the purpose of our original communication which was to draw attention to the fact that even in pressurized aircraft, cases of sickle-cell haemoglobin C disease and sickle-cell β -thalassaemia are at some risk. As Dr. Konotey-Ahulu has pointed out, these cases may have normal haemoglobin levels and it follows that they may escape detection unless adequate screening is applied. An association between the sickle-cell trait and splenic infarction in unpressurized aircraft at high altitude appears to us to be established, but no cases have been reported in pressurized aircraft.

Finally, with regard to aircrew we wish to reiterate our comment that "It would seem most important that a firm, lasting, and internationally acceptable policy be defined on this subject by the authorities responsible for aircrew licensing."

The relationship of the sickle-cell trait to altitude is but one aspect of the much wider issue relating to the possible pathogenetic significance of the sickle-cell trait. There is much ignorance in this field at present, and we are sure that all workers

would join in the hope that future studies will serve to clarify the situation, not only in respect to altitude, but also to other areas.—We are, etc.,

R. L. GREEN

Air Corporations Joint Medical Services,
Speedbird House,
London Airport

R. G. HUNTSMAN

St. Thomas's Hospital Medical School,
London S.E.1

G. R. SERJEANT

Abnormal Haemoglobin Research Unit,
University of Cambridge

1 Choremis, C., Zannos, L., Ikin, E. W., Mourant, A. E., and Lehmann, H., *Lancet*, 1957, 2, 1333.

2 Choremis, C., Zannos-Marioleas, L., and Kattamis, M. D. C., *Lancet*, 1962, 1, 17.

3 Lewis, R. A., *Sickle States: Clinical Features in West Africans*, Accra, Ghana Universities Press, 1970.

4 Konotey-Ahulu, F. I. D., *Lancet*, 1969, 1, 267.

5 Smith, E. W., and Conley C. L., *Bulletin Johns Hopkins Hospital*, 1955, 96, 35.

6 Rotter, R., Luttrell, W. F., Peterson, W. L., Stock, A. E., and Motulsky, A. G., *Annals of Internal Medicine*, 1956, 44, 257.

7 Thompson, G. R., *British Medical Journal*, 1963, 2, 976.

8 Jones, S. R., Binder, R. A., and Donowho, E. M., *New England Journal of Medicine*, 1970, 282, 323.

9 Binder, R. A., and Jones, S. R., *New England Journal of Medicine*, 1970, 282, 1158.

Mercury Poisoning from Wheat

SIR.—We would like to draw attention to an outbreak of poisoning from the mercurial compound Granosan M (ethyl mercury p-toluene sulphonanilide), which has ravaged Iraq in the last two months. The poisoning occurred among farmers whose wheat grain had been dressed with the fungicide. The number of hospital-admitted cases exceeded 5,500, and the deaths reached 280.

A similar outbreak on a smaller scale occurred in this country in 1961 and has been reported.^{1,2} The present outbreak is being thoroughly studied and will be reported in due course.

It will be greatly appreciated if doctors from other countries with experience in this field, particularly as regards treatment, would correspond with the undersigned.—We are, etc.,

SALEM F. DAMLUJI

SADOON TIKRITI

Department of Medicine,
Medical College,
Baghdad University,
Baghdad, Iraq

1 Jalili, M. A., and Abbasi, A. H., *British Journal of Industrial Medicine*, 1961, 18, 303.

2 Damluji, S., *Journal of the Faculty of Medicine, Baghdad*, 1962, 4, 83.

The Haemoglobinopathies

SIR.—My old friend Professor H. Lehmann (26 February, p. 571) is quite correct. True sickling is no doubt a property of Hbs S and C Harlem/Georgetown, in which valine replaces glutamic acid in position 6 of the β -chains. However, according to Harris and Kellermeyer,¹ Hb Memphis, an α -chain variant in which glycine replaces glutamic acid in position 23, may produce sickling by the same mechanism. These authors also state that Hbs F Alexandria (not F as stated in Professor Lehmann's letter), Barts, I, and Porto Alegre can induce a sickle shape-change.—I am, etc.,

G. W. G. BIRD

Regional Blood Transfusion Service,
Birmingham

1 Harris, J. W., and Kellermeyer, R. W., *The Red Cell*, Cambridge, Massachusetts, Harvard University Press, 1970, p. 179.