splenic infarction during flight, in a black man, Dr. Green and his colleagues produced the statement: "A positive sickle-cell test with a normal haemoglobin level suggested he was a sickle-cell trait carrier, though electrophoresis was not performed." The possibility of SC disease in this case appears to have been ignored because of the "normal haemoglobin level." Evidence of 377 consecutive Hb SC disease patients on whom haemoglobin estimations have been done in the steady state (see Table) shows levels of at least 12.0 g/100 ml in 49, 12.8 g/100 ml in 37, 13.5 g/100 ml in 36, 14.3 g/100 ml in 14, and 15.0 g/100 ml or more in 18 (the normal Ghanaian male-female mean³ being 12.7-14.8 g/100 ml).

TABLE.—Haemoglobin Levels in Sickle Cell Disease Patients in the Steady State.†

Haemoglobin (S/100 ml)	SS	SC	SFh	STh
16.5		**	*	
15.75				
15.00		16		
14.25		14	**	
13.50	*	36	*	**
12.75	***	37	*	***
12.00	***	49	*	****
11.25	***	25	*	***
10.50	***	57	*	****
9.75	***	41	*	***
9.00	***	37	***	***
8.25	***	23	***	***
7.50	***	28	*	***
6.75	***			**
6.00	***			
5.25	***			
4.50	***			
3.75	***			
Mean	7.998	11.202	11.137	10.317
SD	1.645	2.210	2.767	2.113
Total	463	377	16	30

†BMD Biomedical Computer Program 07D output. Each star represents a patient. Where the total number of patients for the same range of haemoglobin level exceeds 10 the exact number is shown after the row of stars. Note that 154 (41%) of 377 Hb SC patients had haemoglobin levels of 12.0 g/100 ml or more. Inter-genotype differences in Hb levels are highly significant ($P < 0.001$).

By far the most striking thing about the haemoglobin electrophoresis of sickle cell β -thalassaemia in West Africa is an "AS" pattern with no more fetal haemoglobin than is found in sickle-cell anaemia (Fig.). The

inexperienced confronted with such an electrophoretic strip without the control, and without values for A_2 and S, will almost certainly call it sickle-cell trait. The authors have failed to satisfy themselves and your readers that the Ghanaian nurse, information on whom they obtained second hand, was in fact sickle-cell trait and not sickle-cell β -thalassaemia. While they admitted that "no sickling crises were reported among African competitors with the sickle-cell trait competing in the Mexican Olympic Games at 7,000 ft (2,135 m)" they nevertheless accepted without question the recent controversial report⁴ of four cases that "violent exercise and residence at a moderately high altitude (4,000 ft; 1,220 m)" was capable of precipitating a fatal sickling crisis in sickle-cell trait carriers. Actually, in their paper entitled "sudden death in sickle-cell trait"⁴ the authors relied on the "SA" electrophoretic pattern without quantification of A_2 , A, or S in three of their cases, and in the fourth who did not even have haemoglobin electrophoresis they were content to call it sickle-cell trait because "a sickle-cell preparation at autopsy was positive."

There is no controversy regarding sickle-cell disease and altitude. The controversy is about the uncritical way the authors have used the term sickle-cell trait throughout their article in relation to aviation. The sickle-cell gene is found in much of the non-negro world, for example in the Mediterranean countries and in South India. Indeed, in the Lake Kopais region of Greece 1 in 5 of the population has the sickle-cell trait⁵—a proportion which is exactly the same as that in Ghana. Any international pronouncement on the sickle-cell trait will, of course, also affect Greeks and people of Greek descent.

I am grateful to Professor Bela Ringelmann, Department of Chemical Pathology, University of Ghana Medical School, for his advice and help; to Professor Herman Lehmann, M.R.C. Abnormal Haemoglobin Unit, University Department of Biochemistry, Cambridge, for confirming the haemoglobin electrophoresis of the S β -thalassaemia strip shown in the Figure; and to Mr. R. G. Carpenter and Miss J. Nixon of the Department of Human Ecology, Cambridge, for their help in computer analysis of data.

—I am, etc.,

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IgE Levels in Asthmatics

SIR,—The observation of "two distinct forms" of IgE by Mr. D. Jacobs (11 September, p. 642) may simply represent an artefact resulting from the processing of serum to concentrate the IgE.

Starting with PS myeloma IgE serum, purification usually results in an IgE

preparation giving a single band on Ouchterlony plates.¹ However, in one preparation in which the purified IgE was concentrated to 10 mg/ml two distinct precipitin bands were observed when this preparation reacted with commercial antihuman IgE (Miles Laboratories) (see Fig.). We suspect that the second band probably represents aggregated or otherwise modified IgE expressing new antigenic determinants which become visible only with very concentrated preparations.

PHOTOGRAPH OF OUCHTERLONY A & B

Ito *et al.*² have reported new antigenic determinants resulting from the processing (heating) of IgE preparations which may be an additional example of the possible artefactual nature of the observation reported by Mr. Jacobs.—We are, etc.,

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Gamma Benzene Hexachloride as a Scabicide

SIR,—I recently returned from four months' relief work among East Bengali refugees in India, about half of whom were suffering from scabies. All patients infested by scabies, whether as a presenting complaint or an incidental finding, were taken by a nurse to a quiet corner of the hospital compound, stood upon a piece of plastic sheeting, stripped naked, and painted swiftly, exclud-

Figure.—Haemoglobin electrophoresis tris-barbiturate pH 8.9 of blood from sickle-cell β -thalassaemia person (left) and normal control (right). Note from top to bottom increased A_2 band, 6.1%; large S band 70%; and smaller A band 21% in S β -thalassaemia, while in the normal control a smaller A_2 , 2.1% and an A band are found (from Konotey-Ahulu and Ringelmann⁷). Haemoglobin F in this sickle cell β -thalassaemia patient was only 2.3%. Mean Hb F levels in 184 HbSS 34 HbSThal, and 27 HbS-persistent F patients in Accra were 7.7%, 7.4%, and 20.3% respectively.