frequent intervals. The results quoted above do suggest that it may be desirable to treat all those over the age of 70 with prophylactic antibiotics.

Summary

The family doctor is in an excellent position to study the natural patterns of an influenza epidemic. This paper reports the effects of influenza epidemic of January to March, 1959, on a south-east London general practice.

Over a period of nine weeks (849 (14%) patients in this practice were seen, and were diagnosed as suffering from influenza. It is estimated that of those who treated themselves are included the total incidence in the practice was around 20%.

The liability to infection seemed to vary with age. The highest rates were noted in school children and adolescents, and the lowest rates in the elderly.

The clinical features were those common in other epidemics. Investigations showed that the epidemic was a mixture of influenza viruses A and B.

Chest complications occurred in 80 patients (9.5% of all cases of influenza). Most of these complications were pneumonias (50), and many patients were severely ill and responded only slowly to powerful antibiotics. These complications were highest in those aged 70 and over (73% of cases) and in those aged 60-69 (36%), and lowest in those aged 10-39 (4%).

Compared with previous epidemics, the age patterns in the 1959 epidemic were very similar to the Asian influenza outbreak of 1957. The rates of complications were similar to other winter epidemics of influenza. The autumn Asian influenza had a much lower rate of complications.

If these figures are representative of the country as a whole then it is estimated that there were 7-10 million case of influenza almost 1 million chest complications, and some 5,000 deaths.

Future planning in this field should include a nationwide study of the effects of influenza in general practice, the possibility of an efficient vaccine, and the advisability of using prophylactic antibiotics in the elderly.

It is a pleasure to thank Dr. G. E. H. Callebaut and Miss E. B. Blake for co-operation in coping with the work of this epidemic.

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The new edition of the *Clean Air Year Book*, 1959, published by the National Society for Clean Air, Palace Chambers, Bridge Street, London, S.W.1 (68 pages, 2s. net, 2s. 4d. by post), is an informative and useful guide to the new Clean Air Act and the Orders and Regulations made under it. Many other aspects of the work being done for the prevention of air pollution are the subject of the articles, and a new feature is a list of local authorities with smokeless zones or smoke control areas. Concise information is given about the work and services of the Society, and of other bodies dealing with the problem, together with a guide to the literature on the subject, a selected list of recent papers and articles, and a catalogue of films and filmstrips on air pollution, fuel efficiency, etc. Many readers will find helpful a synopsis of the case for clean air, for the guidance of speakers and writers, which is supplemented by a brief history of air pollution in Great Britain—from 1273 to 1958.

SICKLE-CELL/HAEMOGLOBIN-J DISEASE

BY

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Haemoglobin J was first described in an American negro by Thorup *et al.* (1956). Since that time reports of the presence of this haemoglobin have come from various parts of the world—Indonesia (Huisman *et al.*, 1957; Lie-Injo Luan Eng, 1958), Algeria (Cabannes *et al.*, 1958), India (Raper, 1957; Sanghvi *et al.*, 1958), Singapore (Ager *et al.*, 1958), French Canada (McCabe *et al.*, 1957).

Most examples of this haemoglobin that have been reported hitherto have been found in association with haemoglobin A. However, in one of the cases reported by Sanghvi *et al.* (1958) the haemoglobin J trait was present together with thalassaemia minor, although there was no evidence of interaction between them. Cabannes *et al.* (1958) mention three instances of association between haemoglobin D and haemoglobin J. The purpose of this communication is to report the presence of haemoglobin J in association with haemoglobin S.

Case Report

A 42-year-old man of fair complexion had been perfectly well until 1950, when he attended hospital for indigestion. In 1953 a duodenal ulcer was diagnosed, which did not respond well to two years of medical treatment. In 1955 a partial gastrectomy was performed, after which he remained well for three years. In April, 1958, he was admitted to another hospital with jaundice, and Weil's disease was diagnosed. In view of the finding of a positive sickling test and a rather abnormal blood film his blood was sent to us for further investigation to exclude sickle-cell anaemia. We could confirm the positive sickling test; the type of sickling was intermediate between holly leaf and filamentous.

Other results were as follows: haemoglobin, 11.7 g. per 100 ml.; foetal Hb, 0.5%; R.B.C., 4,400,000 per c.mm.; P.C.V., 40%; M.C.V., 90 cubic microns; M.C.H.C., 29%; reticulocytes, 4%. The osmotic fragility of the red cells was slightly decreased. The blood film showed numerous target cells (Fig. 1). On electrophoresis of haemoglobin a

FIG. 1.—Stained blood film showing numerous target cells.

FIG. 2—Electrophoretic patterns (on starch gel), reading from left to right: CC, CS, SS, SJ (patient), AH, AA. Application at arrow.

fast component in addition to haemoglobin S was found. The mobility of the component at pH 8.6 was about half way between A and H (Fig. 2). Blood samples were sent for reference purposes to Dr. Lehmann, of London, and to Mr. Robinson, of Detroit. They both reported that the fast-moving component was identical with haemoglobin J. Scanning showed that the composition of the patient's haemoglobin was about 40% S and 60% J.

Discussion

The episodes of jaundices was not thought to be a sickle-cell crisis clinically, and this view would seem to be justified. In fact, the patient suffers no disability attributable to his blood condition. He has a mild but well-compensated haemolytic anaemia, which is similar to some of our cases of sickle-cell/haemoglobin-C disease. In a series of 45 cases (MacIver and Went, 1958) of this latter condition a number were without any symptoms.

Investigation of the patient's family was interesting but disappointing. Both parents were stated to be dead, and the patient has no children and no full brothers or sisters. However, his mother, who was of negro stock, had six children by two other fathers, one of whom was Chinese. Both children by the latter and two of the other three children had the sickle-cell trait. The remaining child showed normal haemoglobin A only. It is justifiable to deduce from these findings that the mother carried an S gene. The father must therefore have contributed the J gene.

The patient stated that his mother "got him in Panama," and that his father was an American resident there. The predominantly Caucasian appearance of the patient, taking into account the negro mother, tends to support this possibility. To trace the father or his relatives would seem to be hopeless.

Summary

A case of sickle-cell/haemoglobin-J disease is reported. The interaction between the genes for haemoglobin S and J is not marked, resulting in a mild well-compensated haemolytic anaemia. Family studies suggest that the haemoglobin J was contributed by a Caucasian from North America.

We are grateful to Dr. E. S. Shoucair for referring the case to us, and to Drs. H. Lehmann, J. V. Neel, and A. R. Robinson for their kindness in comparing the characteristics of the fast haemoglobin in this case with other examples in their possession.

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ACETAZOLAMIDE IN SICKLE-CELL ANAEMIA

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In 1957 Hilkevitz recommended acetazolamide ("diamox") for the treatment of patients with sickle-cell anaemia. Since then little has been reported on experiences with this drug in sickle-cell anaemia, although unpublished observations were said to be disappointing. However, there is at least one communication, by Charmot and Reynaud (1958), who found that sickle-cell crises were relieved by acetazolamide. The drug is a carbonhydrase inhibitor and, quite apart from any effect on the sickle cells, it would be expected to cause a metabolic acidosis. It is also known to produce a fall in serum potassium. Greenberg and Kass (1958) have laid emphasis on the importance of the blood pH in the causation or prevention of sickle-cell crises, and they produced crises by treating patients with ammonium chloride and acetazolamide, but not with acetazolamide alone. Tenney and Tschetter (1959) found that the metabolic acidosis caused by acetazolamide in rats was accompanied by a fall in oxygen consumption. It occurred to us that sickle-cell anaemia patients might have failed to respond to treatment with acetazolamide because of the associated acidosis.

Present Investigation

We decided to treat (at the General Hospital, Lagos, Nigeria) patients for two weeks with acetazolamide and, at the same time, to give them large amounts of sodium bicarbonate and potassium chloride to counteract the metabolic acidosis and hypokalaemia associated with prolonged acetazolamide therapy.

Table I summarizes the ages of the patients, their

TABLE I.—Patients Treated with Acetazolamide

Case No.	Age in Years	Weight (kg.)	Acetazolamide (mg./kg. Day)	Daily Supplement (g.)			
				NaHCO ₃		KCl	
				1st Week	2nd Week	1st Week	2nd Week
1	7/12	7.7	24	3	6	0.8	1.5
2	2	9.1	21	6	12	1.5	3
3	24	11.3	17	6	12	1.5	3
4	34	10.9	17	3	6	0.8	1.5
5	5	15.9	12	6	12	1.5	3
6	5	13.6	14	6	12	1.5	3
7	7	18.6	10	6	12	1.5	3
8	9	30.0	12	9	18	2.8	4.5
9	8	19.6	9	6	12	1.5	3
10	15	57.3	7	9	18	2.8	4.5
11	20	36.4	10	9	18	2.8	4.5