Fisher, for their invaluable help in running the clinic; and Mr. E. S. McLeod, of the Department of clinical photography, Nottingham General Hospital, for providing the photograph of the apparatus. The bulk of our equipment was obtained from the Orphan Homes of Scotland, Bridge of Weir, Renfrewshire, but it may also be obtained from N. H. Eastwood, 5, Farmleigh, Southgate, London, N.14; and from Down Bros. May and Phelps, of New Cavendish Street, London, W.1. The pads and alarm units of these makers differ considerably.

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VISUAL DISTURBANCES ASSOCIATED WITH TRIVALENT ANTIMONY SALTS

A REPORT OF TWO CASES

BY

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The causes of optic neuropathy are almost legion. Duke-Elder (1940) has classified them as follows.

1. Local inflammatory conditions.
2. General inflammatory conditions: (i) central nervous (? virus) infections; (ii) syphilis; (iii) tuberculosis.
3. Leber's disease.
4. Endogenous toxins: (i) acute infective disorders; (ii) septic foci (teeth); (iii) metabolic (deficiency diseases).
5. Intoxications by exogenous toxins: tobacco, methyl and ethyl alcohols, lead, arsenic, thallium, apiol, quinine, ergot, optochin, felix mas, aniline.

The condition is fortunately uncommon in Kuwait. If one excludes cases associated with proved intracranial neoplasm, hypertension, or nephritis, only two examples have been seen in the native population during the 10 years or so in which the company has been developing its medical services. During 1957 about 500 patients were seen daily at the out-patient clinics.

Case 1

This patient was a young man about 25 years old. He was well nourished and of fair physique. He had had repeated attacks of left-sided renal colic, and urinary schistosomiasis was first diagnosed at the beginning of September, 1956. His weight was 120 lb. (54.4 kg.). Routine physical examination revealed nothing abnormal, and he was given five intravenous injections of 12 mg. of sodium antimony tartrate at 12-hourly intervals, with lucanthone hydrochloride ("miracil D"), 0.55 g. b.d., for three days over the same period. An E.C.G. taken at the end of the course showed only mild toxic changes. Viable ova of *S. haematobium* were again present in the urine at the beginning of October, and he received a standard course of intramuscular lithium antimony-thiomalate ("anthiomaline") to a total of 40 ml.

He continued to void viable ova, and on October 29 received intramuscular stibophen in the usual dosage to a total of 50 ml. He was still voiding viable schistosoma ova on November 21, and it was decided to embark upon an extended course of treatment with sodium antimony tartrate. He was given three intravenous injections weekly— $\frac{1}{2}$ gr. (32 mg.), 1 gr. (65 mg.), $1\frac{1}{2}$ gr. (100 mg.)—and then 2 gr. (130 mg.) thrice weekly.

On December 22 he complained of dimness of vision of the right eye. By this time 21 gr. (1,365 mg.) of sodium antimony tartrate had been given, and from this date no schistosoma ovum has been found in the urine. Visual acuity for the right eye on December 31 was 5/60, the left eye 6/6. Nothing abnormal was seen in the anterior segments, but haemorrhages and exudates were present at the right macula. Both disks appeared normal, as did the left macula. The right fundus was unchanged when examined in the middle of January, 1957, at the beginning of February, and on February 14. A week later, however, some improvement was noted, and on February 25 visual acuity in the right eye was 6/24. The haemorrhages steadily cleared and none was present on March 20, when visual acuity was 6/12 in the right eye. The exudate was lessening in extent and in area, though vision was still 6/12 in the right eye on April 11. On April 29 he said that he was better. It was noted that there were very few retinal exudates and that visual acuity was 6/6 each eye. He continued to have no complaints as regards his eyes, and further examinations at the beginning of June and in September showed no abnormality of the fundi and both eyes to be 6/6.

The patient continues to suffer from attacks of renal colic, but has no other morbid symptoms. Repeated skiagrams of the chest and repeated stool examinations have showed no abnormality, but he has a persistent eosinophilia of 20-23%, with a white cell count of 5,800-8,000.

Case 2

A well-nourished young man of good physique, aged about 23, and weighing 145 lb. (65.8 kg.). He had several attacks of right-sided renal colic, and *S. haematobium* ova were first found in the urine at the beginning of October, 1956. He had no other complaint and routine physical examination revealed no abnormality. He was given lucanthone hydrochloride, 0.6 g. b.d. for three days, receiving five intravenous injections of 14 mg. of sodium antimony tartrate at 12-hourly intervals during the same period. An E.C.G. taken on October 8 at the end of this course showed toxic effects of antimony upon the myocardium.

Viable schistosoma ova were demonstrated in the urine on November 26. A standard course of 40 ml. of lithium antimony-thiomalate intramuscularly was completed by December 23, but again viable ova were seen in the urine.

On January 12, 1957, he complained of dimness of vision of the right eye for the past five days. It was noted that he had "white eyes," and that nothing abnormal could be seen in the anterior segment or in the fundi. He continued to complain of poor vision in the right eye. On February 2 he was found to have amoebiasis and was admitted for treatment of this, receiving emetine and bismuth iodide, $1\frac{1}{2}$ gr. (100 mg.) daily for 10 days.

When the eyes were examined on February 9, intra-ocular tension was normal. Again nothing pathological was seen in the anterior segments or in the fundi. Vision for the right eye was 6/12, for the left 6/6. He was given a course of lucanthone hydrochloride 1.3 g. daily for three days, since when no viable ovum of *S. haematobium* has been found in the urine. Visual acuity for the right eye was now reduced to 1/60, the left remaining at 6/6. The pupils were equal in size; the fundus on the right side appeared granular, but the macula was otherwise normal. He said that he was better on March 12, when the visual acuity of the right eye was 2/60. By March 20 it was 6/60, by the 28th 6/24, by April 7 6/18, and by May 29 it was once again 6/6. Frequent examinations subsequently have always resulted in 6/6 for each eye, both fundi being normal.

The patient was admitted to hospital on July 7, 1957, when he said that he had vomited blood. No abnormality was found even after a barium meal, fractional test meal, haemoglobin estimation, and repeated stool examinations, and it was thought that he had been mistaken. At various

times he has had skiagrams taken of the chest, and many stool and blood examinations. The only noteworthy feature of all these is that he has a persistent eosinophilia of 15-23% with a white cell count of 7,000-10,000.

Discussion

In neither case is there any obvious indication that the cause of the condition is to be found in the first four of Duke-Elder's categories; nor had either patient been given isoniazid (Sutton and Beattie, 1955), digitalis (Wagener *et al.*, 1946), barbiturates (Carrillo *et al.*, 1938), streptomycin (Sykowski, 1951), gold (Foster, 1943), chloramphenicol (Wallenstein and Snyder, 1952), or sulphonamides (Mansour, 1945). They had not been exposed to carbon monoxide (Casanovas, 1944), bitten by snakes (Guttmann-Friedmann, 1956), or had a cold permanent wave (Robson and Cameron, 1949); they were not addicted to *Catha edulis* (Baird, 1952), or infested with ascaris. They both smoked cigarettes, but not heavily—fewer than 20 a day. There are two factors common to each history: each patient had received trivalent antimony injections, and both developed neurological symptoms at about the same time, the interval being only a few days. The latter suggests that the cause of the illness may have been infective and not related to the antimony.

Summary

Two cases of optic neuropathy associated with visual disturbances and indefinite fundus changes are described as occurring during or following a course of injections with trivalent antimony compounds. The association has not been previously reported.

The patients developed symptoms within a few days of each other, and an infective basis cannot be excluded.

I am grateful to the Kuwait Oil Company for permission to publish; to Dr. K. P. S. Menon, formerly medical officer, Kuwait Oil Company—whose ophthalmic notes I have freely used; and to Sir Stewart Duke-Elder and Dr. J. Guthrie, the chief medical officer of the Kuwait Oil Company, for help and advice.

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The new Biochemical Laboratory which is being built in the Royal Infirmary, Edinburgh, at a cost of £56,000 is designed to do more than accommodate the routine analytical work of the hospital, important though that is. (At present this involves some 80,000 quantitative analyses a year and the work is growing both in volume and complexity.) Facilities will also be provided for work as the reference laboratory of the South East (Scotland) Region and for research. This last, important in any teaching hospital, is doubly important here, since the laboratory also acts as the University Department of Clinical Chemistry. The new building will release the present laboratories, which are now far too small for their purpose, to give urgently needed space to, for example, the Departments of Therapeutics and Cardiology. The plan is the work of Mr. A. A. Dixon, A.R.I.B.A., the Royal Infirmary architect. It gives routine laboratory space for 20 analysts, a range of special-purpose rooms for chromatography, electrophoresis, fluorometry, etc., eight or nine research rooms, and the accompanying offices, store-rooms, workshop, library, and reading rooms. It is hoped that the building will be completed before the next International Congress of Clinical Chemistry, which is to be held in Edinburgh in August, 1960.

SERUM CONTAINING THREE Rh AUTOHAEMAGGLUTININS

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The occurrence of auto-antibodies belonging to the ABO system has been known since they were first demonstrated by Dockeray and Sachs (1941), followed by a number of other workers whose papers are summarized by Race and Sanger (1958).

The occurrence of specifically identifiable auto-antibodies belonging to other systems in the sera from cases of haemolytic anaemia was first clearly demonstrated by Weiner, Battey, Cleghorn, Marson, and Meynell (1953), who showed the presence of anti-e both in the serum and adsorbed on the red cells of a patient who was e-positive. Numerous similar cases, likewise summarized by Race and Sanger (1958), have since been found. Each serum has, however, been shown to contain one or at most two auto-antibodies. In the present case three distinct Rh antibodies, each specific for one of the patient's antigens, were found in one serum.

Case Report

The patient, Mr. C, of Bucharest, suffered from chronic lymphatic leukaemia with secondary haemolytic anaemia controlled by cortisone and repeated transfusions. The results described refer to his blood before any transfusions were given.

The patient's cells showed agglutination in the direct anti-human-globulin test, and on treatment with papain or with compatible serum and dextran. It was thus possible to group his cells only by means of saline-agglutinating antibodies or after removal of auto-antibodies by elution. The results were as follows: A₁ CcDee (probably CDe/cde), M, S+, P+, Lu(a-), Le(a-b+), Fy(a-).

The serum was found to contain albumin-agglutinating anti-C+anti-D and incomplete anti-e demonstrable only by means of the anti-human-globulin test or the use of papainized cells. In albumin tests at 37° C. the titres were found to be 16 with CDe/CDe and with Cde/cde cells, and 8 with cDe/cde and cDE/cDE. Cells of genotype cde/cde were not agglutinated in albumin. A strong non-specific cold agglutinin was detectable at 4° C.

The anti-e was first detected by adsorption on to cde/cde cells and elution by the method of Weiner (1957), the eluate being tested against papainized cells. It reacted strongly with cde/cde cells, not so strongly with cDE/cde and not at all with cDE/CDE. When the original serum was tested by means of the indirect anti-human-globulin test, the only Rh genotype giving negative results was the rare cDE/cDE, of which we were fortunately able to obtain an example. Cells of genotype cDE/cDE showed zoning when an anti-globulin titration was done, being negative with the patient's undiluted serum and weakly positive with dilutions from 1/4 to 1/16.

The analysis of the serum, however, depends mainly on direct tests of the whole serum against cells of a variety of genotypes: there was sufficient serum only for a few absorption tests and for the one elution test already mentioned. A pure anti-C was prepared by absorption with cDE/cde cells, but absorption with Cde/cde removed most of the anti-D as well as the anti-C. Absorption with CDe/cDE removed all antibodies, showing anti-f to be absent, or