reticulocyte count 49%, and a film showed gross spherocytosis and polychromasia. The white cell count was 10,600/cu. cm. and the differential included 15% eosinophils. The spleen was not palpable. The direct antiglobulin (Coomb's) test was negative on three separate specimens; tests for Heinz bodies and abnormal blood pigments were negative, as also were Ham's acid-serum test, L.E. cells, G-6-P.D. and pyruvate kinase in red cells, the Donath-Landsteiner test, and haemoglobin electrophoresis. At the height of haemolysis, serum bilirubin was 1.9 mg./100 ml. and urinary urobilinogen was increased; the mean corpuscular fragility was increased, 0.48%. Both gold and mefanamic acid were ceased on admission, he was transfused and the haemoglobin concentration has since been normal, the spherocytes and reticulocytosis and eosinophilia having disappeared eight weeks later.

This case is of interest firstly as a report of a severe iatrogenic haemolytic disorder, and secondly in raising the possibility that mefanamic acid may cause haemolysis in the absence of an immune mechanism. It is not possible to be sure that the haemolysis was attributable solely to mefanamic acid. Gold compounds are well-known to have myelotoxic effects and also to cause eosinophilia, but we can find no record of their causing haemolysis. The possibility that gold had a combined or synergic effect with mefanamic acid cannot be excluded, but seems unlikely. Gold therapy has since been reintroduced in this case and so far no haematological abnormalities have developed.

Normal ABO-compatible red cells were incubated for varying periods of time in plasma and serum specimens taken during the haemolytic episode, but neither spherocytosis nor a positive antiglobulin (Coomb's) test was obtained. These experiments were performed with and without added mefanamic acid and complement.—We are, etc.,

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REFERENCES

- 1 Scott, G. L., Myles, A. B., and Bacon, P. A., *British Medical Journal*, 1968, 3, 534.
- 2 Worledge, S. M., *Seminars in Hematology*, 1969, 6, 181.

Erythromycin and Clindamycin

SIR,—Dr. I. Phillips and his colleagues (11 April, p. 89) suggest that clindamycin might be useful in treating patients with chronic purulent bronchitis. In vitro studies have shown that erythromycin is somewhat more active against *Haemophilus influenzae* and clindamycin against *Streptococcus pneumoniae*.

My colleagues and I have already shown in a controlled trial that erythromycin was significantly less effective than a tetracycline (lymecycline) or a novobiocin-tetracycline mixture (Albamycin-T) in treating patients with chronic purulent bronchitis.¹ It was especially poor in preventing clinical deterioration during and immediately after antibiotic treatment. From Dr. Phillips and colleagues' evidence it seems very unlikely that clindamycin will prove any better.

though both it and erythromycin might be useful in occasional patients.—I am, etc.,

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REFERENCE

- 1 Pines, A., Raafat, H., Plucinski, K., Greenfield, J. S. B., and Solari, M., *British Journal of Diseases of the Chest*, 1969, 63, 206.

Entonox in the Ambulance Service

SIR,—A small error in technical nomenclature has crept into our article on the use of Entonox in the ambulance service (4 April, p. 41) where, towards the end of the Discussion section, we refer to Entonox as being "inflammable."

Entonox is not an inflammable gas, and the sentence should read: "Though Entonox supports combustion, the danger of ignition is also more theoretical than real, since the gas mixture will flow from the cylinder only when a negative pressure is applied."

We should like to take this opportunity of recording that, since the above article was prepared, several other ambulance authorities are being equipped with Entonox apparatus.—We are, etc.,

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Protection from Sunlight

SIR,—Drs. R. M. Fusaro and W. J. Runge (21 March, p. 731) are to be congratulated on the efficacy of their dihydroxyacetone and lawsone cream in erythropoietic protoporphyria. We hope that their excellent results will be obtained by others.

Our paper,¹ to which they refer, was concerned with clinical and chemical study of a family and only incidentally with observations on two patients of the effect of dihydroxyacetone/lawsone lotion. The patients started treatment in 1964 according to the method described by Fusaro and Runge,² and which did not mention the instability of the ingredients when dispensed as a single mixture. The lotion comprised: dihydroxyacetone 3%; lawsone 0.035%; and isopropyl alcohol and water *ad* 100%; prepared freshly. Instructions were to apply to sun-exposed areas six times in the first day, then once daily to once weekly depending on the amount of sun exposure expected.

The patient observed most closely was the propositus—an indifferent witness who claimed to have less discomfort when using the lotion. This did not prevent development of new lesions or of progressive ageing changes. Using the monochromator, Dr. Ian Magnus was unable to demonstrate significant protection from 400 mμ wavelength in his skin pretreated with the lotion. The patient died of hepatic cirrhosis in 1969.

The second patient was aged 19 years and developed new lesions while using the lotion. He also claimed discomfort was reduced, and was sufficiently impressed to

ask for further supplies of lotion. It is probably true, as suggested by Drs. Fusaro and Runge, that these indifferent results were due to instability of the lotion used.

We are grateful to the authors for introducing an apparently effective cream for this most inconvenient and occasionally fatal disease.³—We are, etc.,

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REFERENCES

- 1 Donaldson, E. M., Donaldson, A. D., and Rimington, C., *British Medical Journal*, 1967, 1, 659.
- 2 Fusaro, R. M., and Runge, W. J., *Journal of American Medical Association*, 1962, 182, 1120.
- 3 Donaldson, E. M., *British Journal of Dermatology*, 1969, 81, 945.

"Adult Pink Disease"

SIR,—I have recently seen a number of patients with what can only be described as "adult pink disease" in south east Wales. It is characterized by erythroedema and polyneuropathy. The erythema, due to capillary dilatation, is noticeable in those parts of the skin exposed to the light, and, if marked, must give rise to a significant degree of arteriovenous shunting. The associated water retention gives rise to protean symptoms and signs, such as headache, vertigo, joint stiffness, and dyspnoea on exertion. The appearance of a neuropathy can lead to confusion with compression (entrapment) "neuritis," and could result in an unnecessary and unsuccessful operation for the relief of the supposed compression. Acroparaesthesiae, which are often present, seem to be related to the water retention.

Symptoms of respiratory tract irritation and even bronchitis, accompanied by gastrointestinal upset, suggest that the causal agent is airborne. There are good grounds for thinking that contamination of the atmosphere is the factor responsible, since patients recover in about two weeks without specific therapy if removed from this area. Attempts to have the problem investigated by expert agencies in air pollution have so far produced no response. Until this is done, it seems we may expect to see more and more cases of "adult pink disease."—I am, etc.,

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Mon.

The Ophthalmologist

SIR,—I understand that some distress has been caused among junior residents after your recent publication in the "Unheard Voices" series (28 March, p. 813). I would like to make it plain that my remarks were general and I was not thinking of any particular person. My present registrar is very competent and well able to take over clinics if I am on leave. Previously clinics have had to be cancelled partly due to the difficulty many overseas graduates, and indeed Englishmen, have in understanding the local dialect and expressions; also the junior staff are not always free to cover my clinics, nor