finally lodged in the sphenoidal sinus, narrowly missing the hypophysis.

A new 16-mm. film, entitled "Dead Safe" and running for 27 minutes, illustrates the safe handling of guns. It is available for loan, without charge, from I.C.I. Film Library, Millbank, London S.W.1.

I am grateful to my colleague Mr. John Ogg for allowing me to see the two cases illustrated.

—I am, etc.,

Salisbury General Hospital, BRIAN REEVES.
Wilts.

Oxytocic Drugs in Labour

SIR,—Might I suggest to Dr. R. H. G. Lyne-Pirkis (17 February, p. 447) that the compromise that he is seeking is to inject the oxytocic *immediately after* the baby has been completely delivered, and *not* with either the crowning of the head or the appearance of the shoulder.

It is my teaching that as soon as the child arrives the accoucheur—midwife or doctor—should first palpate the relaxed uterus to ensure that there is no "undiagnosed twin" present, then without any delay 1 ml. of Syntometrine (ergometrine maleate and oxytocin)—already in the syringe—is injected intramuscularly. This procedure has obviated the trapping of such a twin, and it has kept the incidence of postpartum haemorrhage in the unit in the region of 3%.

—I am, etc.,

St. John's Hospital, DAVID BROWN.
Chelmsford.

Lead-poisoning from Unusual Source

SIR,—Dr. M. A. Warley and others (13 January, p. 117) point out the very real danger of lead-poisoning from a mascara-like substance ("surma") commonly used by the gentle sex originating from the Indian sub-continent. The following case shows that natives of this island are not exempt from similar hazards.

We found an apparently inexplicable case of lead-poisoning in a 15-year-old English girl admitted in October 1967 for the investigation of amenorrhoea and anaemia. There was a four-month history of fatigue, pallor, and a sickly feeling in the stomach making her disinterested in her breakfast. Two months previously slight jaundice of the eyeballs was noticed by her family. Moderate pallor and very slight subicterus of the sclerae were the only abnormal clinical findings. The blood count revealed a slightly hypochromic or normochromic anaemia with aniso-poikilocytosis and polychromasia (Hb 8.5–9.1 g./100 ml.; P.C.V. 29–31%; reticulocytes 15–21%). Many red cells showed coarse basophilic stippling and siderotic granules. The bone marrow exhibited a very active normoblastic erythropoiesis, numerous "ringed" and ordinary sideroblasts, siderocytes, and fairly rich reticulum iron. Haptoglobins were absent; the direct Coombs test was negative (on both reticulocytes and non-reticulocytes); the serum bilirubin was raised (3 mg./100 ml.); and haemoglobin A₂ was normal. The diagnosis of haemolytic anaemia due almost certainly to lead-poisoning was made.

When almost all hope of tracking down the source of lead intake was given up it was noticed by one of us (S.V.) that the patient had on a bright salmon-pink lipstick. This

turned out to be ointment belonging to the patient's grandmother—used by her for various skin blemishes and bunions—a proprietary preparation sold over the counter, which the patient, being a lip-biter, had been using for the previous 18 months as a lip-salve and/or a lipstick for its pretty colour. It could be calculated that she had ingested about 3 oz. (100 g.) of this ointment containing 67% basic lead carbonate B.P.C. (A. H. Allen and Partners, Public Analysts Laboratory, Sheffield), the total intake being in the neighbourhood of 45 g. of elemental lead. Subsequent chemical investigations, as expected, were confirmatory of lead-poisoning (lead in urine up to 1,000 µg./l.). The patient is now well, her haemoglobin is normal, and she is menstruating.

The common denominator between the two forms of lead-poisoning, that reported by Drs. Warley, Blackledge, and O'Gorman, and ourselves, is the existence of potentially dangerous sources of lead accessible to unsuspecting individuals. Although in our case the jar carried on its label the warning "For outward use only," one wonders whether this is sufficient.

What will surely interest Dr. Warley and his colleagues is that when in November 1967 the diagnosis of lead-poisoning was established in our patient one of us (P. C. S.) suggested to a countrywoman of his, a lady doctor, that her complaints of irritability, weakness, and diminished appetite might be due to the use of "surma." Subsequently her urine was found to contain 118 µg. of lead per litre and the blood 28.6 µg./100 ml. Three weeks after discontinuation of this cosmetic she felt completely fit.

—We are, etc.,

P. C. SRIVASTAVA.
S. VARADI.

Department of Haematology,
Northern General Hospital,
Sheffield, Yorks.

Scleroderma Heart Disease

SIR,—The report on the cases of scleroderma heart disease by Drs. E. Fletcher and P. Morton (16 December, p. 657) prompts us to write about a patient at present in hospital.

This is a 51-year-old male who was admitted in November 1967 with progressive shortness of breath of two months' duration. Other complaints were numbness of hands and loss of 2 stones (12 kg.) in weight over the same period. Bowels open twice a day on average, the stools often being pale. He had a slight cough but no chest pain. On examination he was dyspnoeic at rest, slightly cyanosed, but had no clubbing. Skin over hands and face was moderately "tight." Heart was clinically normal and blood pressure was 115/70 mm. Hg. Jugular venous pressure was not raised and there was no oedema. There were coarse crepitations over the lower chest on both sides. Investigations: Chest x-ray showed slight cardiac enlargement and dense mottling of the lower lung fields, Hb 10.2 g./100 ml., W.B.C. 7,000/cu. mm., E.S.R. 80 to 110 mm. (Westergren); urea 50 mg./100 ml., electrolytes normal; albumin 3.5 g./100 ml., globulin 2.6 g./100 ml. with an increase in the γ fraction; latex and Rose-Waaler negative; L.E. cells demonstrated three times; complement fixation tests to *M. pneumoniae*, *R. burneti*, and psittacosis-L.G.V. all negative. E.C.G. on the day of admission revealed nodal rhythm with interference-dissociation. The QRS marked "X" appears

to be a captured ventricular beat. (He was not on digitalis.) Rhythm reverted to normal with atropine, and subsequent E.C.G.s did not show any evidence of myocardial infarction.

He has now recovered from the acute episode, though he is dyspnoeic on moderate exertion. Basal crepitations and x-ray appearances of diffuse pulmonary fibrosis persist. Vital capacity is 1.5 litres (predicted 3.3 litres) and F.E.V.₁ 55%. Other respiratory function tests were not done because the patient with his "small mouth" could not tolerate the mouth-piece of the instruments. He probably has systemic sclerosis with primary cardiac involvement.

Though a variety of E.C.G. abnormalities have been described in such cases we found no record of nodal rhythm with A–V dissociation. This case exemplifies the non-specific nature of E.C.G. changes found in scleroderma.¹

We thank Dr. Ian Short for permission to publish this case.

—We are, etc.,

FRANCES WREN.
M. GOVINDARAJ.
Knightswood Hospital,
Annisland, Glasgow.

REFERENCE

- ¹ Oram, S., and Stokes, W., *Brit. Heart J.*, 1961, 23, 243.

Malaria in Britain

SIR,—The correspondence on malaria in Britain has pinpointed the importance of early diagnosis of the species *P. falciparum*. For several years past we have been following up imported malarias as they have been officially reported. We have made it a practice of asking for the loan of the blood film on which the diagnosis of malaria and the species of parasite was made. The questionnaires sent out by us have shown that with quite a number of patients they did not report sick for several days after fever began. Many patients said they did not think it could be malaria because they had taken prophylactic drugs regularly until the day they left the malarious area. In over 90% of all cases of *P. falciparum* malaria in this country prophylactic drugs were discontinued either on the day of leaving the area or a few days later. In not a single case investigated by us did the patient take his prophylactic drug regularly for one month after leaving, as advocated by Professor B. G. Maegraith.¹

As Dr. P. Rees and Dr. D. H. Smith (20 January, p. 179) have pointed out, species diagnosis is of very great importance.² It is all too frequently assumed that if the films show parasites larger than the signet-ring forms then it must be other than *P. falciparum*. This is far from correct and of the very greatest importance. Primary infections of *P. falciparum* invariably show growing trophozoites if the attack remains untreated for more than seven or eight days. It cannot be overemphasized that developing forms of *P. falciparum* in the peripheral circulation are a danger signal and call for urgent and drastic treatment. The detection of these forms is very easy if it is remembered that never more than one or two lumps of pig-