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, Wilts. BRIAN REEVES.

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, Chelmsford. DAVID BROWN.

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 subcontinent. 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 A_2 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.,

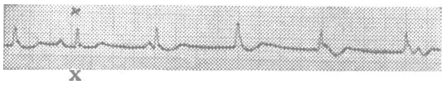
P. C. SRIVASTAVA.
S. VARADI.

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 $\frac{F.E.V._{.1}}{V.C.}$ 55%. Other respiratory function tests were not done because the patient with his "small mouth" could not tolerate the mouthpiece 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, Anniesland, Glasgow.

REFERENCE

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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 questionaries 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-

ment are present in the cytoplasm of the parasite,[3] whereas with the other three species, in all forms larger than the ring forms, 20–50 individual grains can be seen. It does happen occasionally, but not often, that in *P. vivax* only ring forms may be found, but if the species is mistaken for *P. falciparum* it is of no importance. There is something to be said for advising pathologists to send all malaria films to specialists for confirming species diagnosis, even after treatment has been started.

Quite recently we were sent films of a patient who had never left the shores of the United Kingdom. He had had a blood transfusion,[4] and died several weeks later from what was believed to be malignant malaria and black-water fever. It was in fact a human and fatal case of Babesia (*Piroplasma divergens*), and the blood transfusion had no part in the infection. The so-called black-water fever was in fact redwater fever. The parasites so very closely resemble morphologically the ring forms of *P. falciparum* that only an experienced worker could be expected to make the differentiation. Although this is the first case of human Babesia ever to be reported in the United Kingdom and only the third anywhere in the world, it does, I think, justify the above remarks. Incidentally this patient, as with the other two known cases, one in Yugoslavia[5] and the other in California,[6] had had a splenectomy. I would add that the diagnosis of piroplasmosis was confirmed by Professor P. C. C. Garnham.[7]

All the piroplasmata of cattle, horses, and other animals were at first placed in the genus *Piroplasma* or *Babesia*. Piroplasmosis is endemic among cattle, sheep, and rodents in many parts of the United Kingdom, and, although the number of people who have had a splenectomy is small, it nevertheless may be of some importance, especially among farmworkers and veterinary surgeons.—I am, etc.,

Malaria Reference Laboratory, Horton Hospital, Surrey. P. G. SHUTE.

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7 Garnham, P. C. C., and Bray, R. S., *J. Protozool.*, 1959, 6, 352.

Diverticulosis and Diverticulitis

SIR,—May we be allowed to comment on Dr. E. Goulston's letter (11 November 1967, p. 359)? A statement in inverted commas is attributed to us (2 July 1966, p. 50) which we never made. Our letter stressed that diverticulitis does not occur in African negroes living tribally on unrefined grains, and was also rare in United States negroes when they lived on the same class of foodstuff. Dr. Goulston, writing from Addis Ababa (6 May 1967, p. 378), stated that no case of diverticulitis had ever been reported from Ethiopia either, but that diverticulosis had been seen. One of us replied (22 July 1967, p. 243), with supporting barium-meal studies, that this is exactly what would be expected with a changing diet, diverticulosis being regarded as the precursor condition, and Mr. N. S. Painter supported us (12 August 1967, p. 434). We see no new argument in Dr. Goulston's latest letter.

With regard to Dr. S. N. Salem's letter (23 September 1967, p. 800) pointing out the absence of diverticulitis in Kuwaitis, though the diet is frequently refined, this appears to present no difficulty. It has been demonstrated that even in Westernized countries diverticulitis is rare under the age of 40 (23 September 1967, p. 762), showing that a very long period of time is needed to produce the condition, and it is therefore too early yet to assess the colonic consequences of the change in the Kuwaitis' diet, which are relatively recent. We are confident that cases of diverticulitis will start up both in Kuwait and in Addis Ababa during the next decade or so, probably earlier.

With regard to the paper by Dr. Manousos and others (23 September 1967, p. 762) showing intestinal transit times in normal and diverticulosis subjects, we see no difficulty here either. These subjects were all from our own population, which even 25 years ago was shown by J. N. Morris[1] to be taking regular aperients in 60% of its numbers, and we note that over 90% of the "normals" in the present paper retained food in the gut three, four, five, or even more days after its ingestion. We can well believe that the transit times in diverticulosis subjects may be shorter than these times, since we believe that the condition arises from the efforts of the colon in certain people to overcome just this type of delay, but we equally believe that all these transit times are very different from the natural transit time. We plan to do a series of comparable studies in Africans living on unrefined carbohydrates. The distinctive soft stools of these Africans which are passed twice daily have been described by H. Dodd,[2] who visited them in 1964, and we shall be surprised indeed if the transit times are not correspondingly illuminating.

It would seem to us that Dr. O. N. Manousos and others, in contending that the basic abnormality in diverticulosis lies in incoordinated colonic activity, puts the blame on the body, instead of—as set out in Mr. Painter's letter—on a change in the food or on some other abnormality in the environment. And it is in this connexion that we believe that the differential incidences in the negro are of such great importance.—We are, etc.,

Fareham, Hampshire. T. L. CLEAVE. G. D. CAMPBELL.

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1 Morris, J. N., *Lancet*, 1941, 1, 51.
2 Dodd, H., *Lancet*, 1964, 2, 910.

Incidence of Diverticulosis

SIR,—In my last 3,000 tabulated post-mortem results at the (Royal) Adelaide Hospital between 1940 and 1948 (when I retired) there were 78 examples of diverticulosis of the colon—57 in men and 21 in women (there were about twice as many men as women). This shows a decidedly greater incidence in men. There was one woman of 31 and one man of 43; in males there were 6 out of 434 in their 50s; 21 out of 555 in the 60s; 23 out of 343 in the 70s; and 6 out of 90 over 80. In women the respective figures were 2 in 217, 11 in 249, 4 in 145, and 3 in 40.

These results, about which there can be no question even if some instances were not recorded, were in a (then) almost pure British community. They differ markedly from those recorded by Dr. O. N. Manousos and others (23 September, p. 762) for the Oxford area, who found by radiological studies that 40% of people over 70 had diverticulosis. My figures are 36 examples in 589 persons over 70, or about 1 in 16. Those of us over 70 will now feel happier.

May I take this opportunity of calling attention to my tabulated results (in thousands) of 7,000 necropsies at the (Royal) Adelaide Hospital between 1920 and 1948. These were printed in the annual reports of that hospital under the title of "The Medical and Scientific Archives of the Adelaide Hospital." All pathological conditions, except trivialities, were recorded in the summary of each case, and this summary accompanied the tabulation. Thus it is easy to ascertain how many individuals had hypertrophied hearts or gallstones or melanosis of the colon or carcinoma of the lung, and what other lesions were also present in each case. These annual reports and medical archives contain a mine of information for the medical world to use if it knows of their existence and has access to them.—I am, etc.,

JOHN B. CLELAND.
University of Adelaide, Australia.

Preventing Dental Caries

SIR,—Your leading article (3 February, p. 267) "Phosphates to Prevent Dental Caries" recalled to me the custom of the people of Barotseland in the Upper Zambesi, to whom I was medical officer long, long ago.

For them salt was scarcely available and they had no sugar. For flavouring, it was their custom to sprinkle their food, chiefly mealie meal or cassava and fish, very freely with wood ash, preferably from burned river weeds. Their physique was good and dental caries extremely rare. They cleaned their teeth occasionally, rubbing them with a piece of wood, and I think they sometimes used ash as tooth-powder. Dr. E. C. Wood, our Public Analyst, tells me that the ash would contain a high proportion of potassium and perhaps calcium phosphate. Calcium sucrose phosphate might be more potent for good.

My friends who have a strong objection to "chemicals" being added to their food or drinking-water would probably eat wood-ash with gusto and feed their children on it. Some of us might augment our incomes by selling the product of our garden bonfires if that is not unethical. The preliminary results of the long-term study being undertaken in Australia make one very hopeful that the scourge of dental caries may be reduced by inexpensive methods which can cause no qualms of conscience.—I am, etc.,

Paignton, Devon. IAN D. DICKSON.

Varicose Ulcers and Use of Topical Corticosteroids

SIR,—Some weeks ago Dr. Clifford D. Evans and others (25 November, p. 482) drew attention to the adverse effect of topical corticosteroid applications on gravitational ulcers. I have recently recognized another undesirable effect of these preparations, when used to suppress the eruption of rosacea. They do suppress the pustule formation, but the telangiectasia may be made much more prominent, presumably because the dermal collagen is thinned. When the corticosteroid applications are stopped a severe recurrence of the