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 Yugoslavia⁵ 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.,

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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 food-stuff. 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. Morris¹ 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.,

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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.,

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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

pustular eruption follows, which tempts the patient once more to resume treatment.

Three patients with this syndrome have been referred to me within the last few months. The most severely affected had suffered from rosacea for five years and for the last four had applied betamethasone-17-valerate ointment daily. This had controlled the papulo-pustular eruption but her face had become more and more red. On examination she showed severe telangiectasia not only over the cheeks and central part of the face but strikingly over the whole face up to the hair line, where she had applied the ointment. The skin was soft, velvety smooth, and atrophic and showed subcutaneous ecchymoses similar to senile purpura. Another less severely affected patient had used betamethasone-17-valerate ointment daily for six months for rosacea. Within two days of stopping the ointment an angry eruption of pustules appeared over her whole face, which became much swollen. Having been taken by surprise with this rebound pustulation in the first of the three patients, the second patient had been warned what to expect and her eruption slowly improved with oral tetracycline and a bland lotion. A month later the telangiectasia was less prominent.

Telangiectasia may also become visible in skin conditions other than rosacea. A fourth patient used betamethasone-17-valerate ointment daily for a year for the treatment of a mild dermatitis of her eyelids. The skin of the upper eyelids became plum-coloured with numerous telangiectases, but the condition had almost recovered six months after ungu. aquosum had been substituted for the betamethasone-17-valerate ointment.

While these patients have used potent corticosteroid creams for an excessive time, it is probably unwise to apply fluorine-containing corticosteroid applications with great penetrating properties to rosacea.—I am, etc.,

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Screening for Anaemia

SIR,—The results of the screening survey for anaemia reported by Dr. P. C. Elwood and others (23 December, p. 714) are most interesting and show many features in common with other surveys.

During the course of a study of anaemia in a working-class industrial general practice¹ a 10% random sample was examined, with the aim of getting as complete a sample as possible. It was felt that a general practitioner is in a unique position and may be able to get a more complete sample than other less personal approaches. This was done, 99.6% of the random sample of 347 patients being examined. The results were examined to see if those who do not co-operate in a survey were more or less anaemic than those who do co-operate.

The patients in the sample, considering adult non-pregnant women only, were placed in three groups:

(1) Those patients who attended surgery for any reason. Most of these were presumably ill to some extent.

(2) Those who came up with a relative or were sent for through a relative who had attended at surgery. With this group were included those who came in response to a written invitation.

(3) The last group consists of those who failed to respond to a written request and required visiting in order to obtain samples.

Owing to varying hours of shift work some members of this group required several visits in order to find them at home. This group would probably not be included in any survey conducted by an outside person or unit.

Age	Group 1		Group 2		Group 3	
	Anaemic	Normal	Anaemic	Normal	Anaemic	Normal
14	0	6	1	15	0	2
24	1	7	0	9	1	2
34	1	1	0	10	1	0
39	3	3	1	6	2	2
44	4	2	1	10	0	4
54	2	12	0	6	0	4
64	1	5	0	8	2	3
Total	12	36	3	64	6	17
Mean Hb in g./100 ml.	10.4				11.0	
S.D. ±	0.9				0.6	

In this particular survey the greatest number of anaemic patients and the more severely affected were found among those already attending the surgery. Further searching did uncover a few more cases of anaemia, but this was hardly worth while in view of the

effort involved. Moreover, of those patients in group 3 who were anaemic two failed to attend for treatment and one defaulted before her haemoglobin level had returned to normal.

Although the discovery of anaemia in two patients was an important factor in the early diagnosis of carcinoma of the colon, there were no cases of serious organic disease discovered as a result of screening the random sample. This was also the experience of the authors of the M.R.C. Survey² of 1945: "No obvious cause for the anaemia was found in any instance as a result of routine physical examination."

Every encouragement should be given to doctors to obtain accurate haemoglobin estimations on those patients who come to them. Anaemia is a common cause of poor health, and is often an important clue in the diagnosis of more serious disease, but there appears to be no need to go to great lengths to discover new cases.—I am, etc.,

Cambridge.

R. H. MORGAN.

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Mycoplasma in Leukaemia

SIR,—In 1964 one of us (Negroni¹) reported the isolation of infectious agents from patients with leukaemia; on the basis of some biological characteristics the infectious agents were classified as viruses. This conclusion was refuted by Grist and Fallon,² and by Girardi and others,³ who showed that the infectious agents were mycoplasmas. Negroni^{1,4,5} had also found that sera from leukaemic patients neutralized, at a low titre, the infectious agents more frequently than sera from non-leukaemic individuals. This was not confirmed by Grist⁶ or by Fallon.⁷ Because of these conflicting results a series of sera previously tested by Negroni, and stored since then at -20°C . were retested at Glaxo Laboratories. The results of these tests are reported here.

Twelve samples of sera from leukaemic and non-leukaemic individuals were coded so that their origin was unknown to the workers at Glaxo Laboratories. For each serum two different serological techniques, the neutralization test in tissue culture (Negroni²) and the metabolic

inhibition test in broth (Taylor-Robinson *et al.*⁸), were used. Only when the results of these tests were known was the identity of the serum revealed and the Glaxo results compared with those obtained previously by Negroni. The tissue culture neutralization tests were carried out in both laboratories in the same way. Ten-fold serial dilutions of Negroni's mycoplasma were titrated in the presence and absence of 1/10 dilution of serum using two tubes per dilution. At Glaxo, in addition, serial twofold dilutions of serum were tested against a fixed dose of mycoplasma in the metabolic inhibition (colour) test.

The results are shown in the Table. There was general agreement between the results obtained in the two laboratories; thus all three samples from healthy individuals were negative in both laboratories. There was also agreement between laboratories for five of the remaining positive sera, but it is clear that low levels of inhibition of mycoplasma growth in the tissue culture may be detected in one laboratory and not in another (sera 5, 8, 10, and 11). These results show that

Serological Tests with Human Sera

No.	Diagnosis	Glaxo		I.C.R.F. Mill Hill T.C. Infectious Doses Neutralized by 1/10 Diln. Serum	Agreement
		Metabolic Inhib.	TC Infectious Doses Neutralized by 1/10 Diln. of Serum		
		Serum Diln. Endpt.	100 TCD ₅₀		
1	Healthy	<0	<0	0	+
2	"	<0	<0	0	+
3	"	<0	<0	<10	+
4	Acute monocytic	1/40	‡ at a 100*	100	+
5	Unspecified leukaemia	<0	<0	10	-
6	Chronic lymphocytic	1/20	10	(in 2 tests) 10	+
7	"	1/10	10	(in 3 tests) >100	+
8	Acute	<0	<0	10 100	-
9	Chronic myeloid	<0	<0	(in 2 tests) 0	+
10	" lymphocytic	<0	10	0	-
11	Unspecified	<0	<0	10 ‡ at 100* (2 tests)	-
12	Acute myelo-monocytic	1/40	10	10	+
13	Immune rabbit antiserum	1/80	100	1,000	+

* ‡—1 tube out of 2.