west Pacific, are still malarious and constitute a reservoir of imported infection. A W.H.O. report¹ quoted some 1,500 cases of malaria imported into six countries during the past few years. There were 876 such cases in 1963-1965 in the U.S.S.R.²

The burden of imported malaria falls now heavily on the U.S.A., where, since 1966, the number of cases rose dramatically from fewer than 100 per annum to 2,855 cases in 1967 and to 2,610 cases in 1968, chiefly in the military personnel returning from Vietnam.³ Although malaria in the civilians returning to the U.S.A. from tropical areas was seen only in 123 cases, the fact that most of them occurred in students and teachers calls for special vigilance when it comes to these and similar occupational groups and particularly Voluntary Service Overseas volunteers returning to this country from service abroad. To this one might add the increasing number of far-ranging tourists, and the following case may serve as a good illustration of hazards of modern version of *Innocents Abroad*.

"A 60-year-old woman living in Hartford, Conn., U.S.A., developed on 7 March 1969 nausea, vomiting, and diarrhoea. On 9 March her temperature reached 103° F. (39.4° C.), but became normal on the next day, and she was able to do some shopping. Three days later she showed the same symptoms as before, with the temperature 102° F. (38.9° C.) and some disorientation. Her physician found no neurological abnormalities and prescribed tetracycline, although he was told on that day that the patient and her husband had just returned from a tourist trip to East Africa, where they spent three weeks without using any prophylactic antimalarials. On 13 March the patient continued vomiting and became dyspnoeic and very weak. She was transported to the hospital while comatose and cyanotic. The blood examination showed 10-20% of red blood cells infected with *P. falciparum*. She died before any treatment could be started."

The tidal wave of present mass travel is of growing public health significance. In 1967 there were not fewer than 138 million international tourists, and the average annual growth of this phenomenon was 9% during the past three years.⁴ Only repeated warnings addressed to the public and to the medical profession could prevent the repetition of tragic cases such as the one quoted above and others discussed by Maegraith and by Watson at the Cardiff Symposium of the Royal College of General Practitioners.⁵ Attention should be drawn to the excellent pamphlet⁷ offering general guidance on the prevention and recognition of imported infectious disease.—I am, etc.,

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Prospects in Cardiology

SIR,—The poor prospects of obtaining a consultant post in cardiology implied in the reports of the Ministry of Health on Medical Staffing in Hospitals^{1,2} are confirmed by the present difficulties experienced by many senior registrars trained in cardiology in obtaining consultant posts. This, together with the evident lack of sufficient consultant appointments in cardiology in the country, suggests the need to investigate and clarify the position with a view to its improvement.

Such an investigation has been carried out by the Cardiology Committee of the Royal College of Physicians of London and the British Cardiac Society, with the co-operation of representatives of the Department of Health and Social Security. It soon became apparent that the true number of consultant cardiologists in the country was not known. Many consultants appointed as general physicians, and appearing as such in the official returns to the Department of Health, are in fact engaged in intensive cardiological work. Before carrying the investigation further the committee agreed to define three categories of cardiological work:

Group 1.—Investigative cardiological work in association with a cardiac medical/surgical centre as outlined in the report of the Joint Cardiology Committee of the Royal College of Physicians and the Royal College of Surgeons.³

Group 2.—Specialist cardiological work, such as acute coronary care, pacemaking, hypertension, etc.

Group 3.—The work of a general physician with a special interest in cardiovascular disorders, including cardiopulmonary and occlusive vascular disorders.

A letter was then sent to the 187 consultant physicians who are members of the British Cardiac Society asking each to state his (or her) present category in the official return from his major hospital, and also in which of the three groups he considered his present work to lie. Of the 141 consultants who replied, 47 were officially categorized as cardiologists and 94 as general physicians. Of the 47 cardiologists 44 were doing Group 1 work and three Group 2 work. Of the 94 categorized as general physicians 24 were doing Group 1 work and 27 Group 2 work. Thus of 68 members of the British Cardiac Society who are actually doing full investigative cardiological work 24 were officially categorized as general physicians. Out of the 30 doing specialist cardiological work 27 were officially categorized as general physicians. This sample, from members of the British Cardiac Society, shows that the Department of Health's figures for the number of consultant cardiologists are considerably too low. A further questionnaire is now being sent to consultant cardiologists in order to detail the number of senior registrars and their responsibilities in their units. It appears probable that there should ultimately be a larger establishment of senior registrar posts in cardiology, as these posts provide most important training opportunities for posts in any of the categories.

On the basis of the recommendations for the staffing of cardiac units suggested by the joint committee of the two Colleges there will be a need for more consultant cardiologists doing Group 1 work, and from this it follows that more Group 2 cardiologists

will also be needed. Since most of those senior registrars who now hold a post in cardiology will ultimately hold Group 1 posts, or in some cases Group 2 or sometimes Group 3 posts, it would appear that the prospects in cardiology implied in the forecasts by the Department of Health may present an unduly gloomy view, and that registrars should not be discouraged from seeking further training as senior registrars in cardiology.—We are, etc.,

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JOHN MCMICHAEL,
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J. F. GOODWIN,
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Decompression Sickness and Contact Lenses

SIR,—A 23-year-old woman wearing micro-corneal contact lenses was one of a party of six undergoing a dry dive in a recompression chamber. The initial air pressure was equivalent to 200 ft. (66 m.) water gauge (89 lb./sq. in.; 20.3 kg./sq. cm.), later dropping slightly to 190 ft. (60 m.) as the chamber cooled. After ten minutes under pressure a decompression according to the Royal Navy tables was carried out, with stops of five minutes at 20 ft. (6 m.) and ten minutes at 10 ft. (3 m.). On leaving the chamber she complained of blurring of the vision of her right eye, which appeared to have a white film over it, the left eye being also slightly affected.

On examination with an ophthalmoscope, a haze of fine bubbles could be seen under her contact lens, which could not be removed either by rinsing and refitting the lens or by changing over lenses. The bubbles could be seen with the lens removed, the surface of the cornea appearing slightly granular. On replacing the contact lens it could be seen that the bubbles remained stationary on the surface of the eye, while the lens itself moved up and down as the subject blinked. The bubbles slowly diminished in size and after some 30 minutes had completely disappeared.

There would seem little doubt that the bubbles formed in the superficial layers of the cornea, since blinking and all attempts at mechanical removal were ineffective, and are thus of some interest as a visible example of decompression sickness and as a possible hazard to contact-lens wearers. Presumably, as the cornea is vascular, the condition was due to surface absorption of nitrogen in an analogous manner to the production of skin itching following compressed-air work.

Since the cornea is not normally the site of decompression sickness and the Royal Navy tables are capable of decompressing safely a wide range of tissue half times, it may reasonably be inferred that the contact lenses were responsible. A possible explanation is that there was a certain amount of

hilarity and movement (presumably including eye movement) during the compression phase due to the combined effects of warmth and nitrogen narcosis, thus assisting the flow of nitrogen-laden tears between the lens and cornea, while during decompression the chamber was, as is usual, rather cold and foggy, with the subjects relatively immobile, thus minimizing any flow of tears which might have helped to wash out the nitrogen. As the other eye was only slightly affected, it must be presumed that there was a difference in the fit, and hence in the flow of tears under the two lenses, causing either less absorption or better elimination of gas from the other eye.—I am, etc.,

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Bowel Motility and Colonic Cancer

SIR,—Recent studies¹ and comments² on cancer of the colon indicate that valuable information may be forthcoming from intensive studies of bowel motility and faeces composition in populations with contrasting prevalences of this type of cancer.

In South African Bantu age specific mortality is only a tenth of that in whites.^{3,4} In preliminary studies on this subject we have noted two points of interest. In the Bantu (children and students studied thus far) a third have three motions daily compared with only 1–2% in whites. Moreover, in terms of exposure of bowel mucosa to digesta, carmine studies have revealed that in the Bantu the mean transit time of the longest third is less than the mean time of the quickest third among whites. The finding to be stressed is that even in Bantu and whites with the same frequency of defaecation (say, one to two motions *per diem*) traces of carmine continue to be seen far longer in stools of whites than in Bantu; in whites the period is sometimes five to seven days. In addition, therefore, to the presumably vulnerable constipated moiety of population,^{1,4} there is this further moiety with regular motions, not constipated, who, unknown to themselves and undetectable clinically, have unduly prolonged times of traversal of digesta. This is the condition which conceivably provides the favourable *milieu intérieur* required for carcinogenesis, whether of exogenous or endogenous origin.

The second finding to be emphasized concerns the differing ability to produce a stool quickly on request. Eighty to ninety per cent. of rural Bantu children have this capacity, 70–80% of partially sophisticated urban Bantu, but fewer than 10% of white children. Previously^{5,6} we noted that when young white adults consumed a very high-residue Bantu-type of diet for several days they still could not produce a stool on request in the same facile manner as Bantu. Evidently, with “civilization,” voluntary control of the defaecation reflex is largely lost. May not differences in physiological tone of the colon be associated with different susceptibilities to carcinogenic agents?

The critical question, of course, is whether the moiety with protracted exposure to possible carcinogens is in fact that moiety in which sufferers from bowel cancer are to be found. Elucidation of this problem will be difficult. For example, verbal questioning regarding bowel habits, etc., is often worthless; we find a poor correlation between frequency from recall and frequency by keeping a weekly chart. Further,

not recollectable to patients, their bowel habits, etc., at 50–60 years may differ markedly from those at 30–50 years.

Obviously, far more explicit data are needed on populations with contrasting prevalences of colonic cancer. In the present circumstances it would seem that only by impeding carcinogenic action may prophylaxis be envisaged. Screening centres have been advocated to detect the segment of population most prone to coronary heart disease, to detect the aglycosuric population who have abnormal glucose tolerance, etc. Would it be too far fetched to suggest that the younger adult population be screened, initially with carmine, to detect the moiety with very prolonged transit times, and that such be given dietary or therapeutic advice to accelerate excretion?—We are, etc.,

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Pigmentation in Megaloblastic Anaemia

SIR,—We have read with interest the description by Dr. N. Baumslag and Dr. J. Metz of spotty pigmentation of palms, soles, and tongue in lactating Africans with folate deficiency (21 June, p. 737). Clinicians in Ibadan are aware of the possibility of hyperpigmentation in patients with megaloblastic anaemia, as it has been seen in one Nigerian with vitamin B₁₂ deficiency, but it has not been noticed in folate deficiency, though about 200 women with severe megaloblastic anaemia associated with pregnancy are seen each year. No areas of hyperpigmentation were revealed by a detailed dermatological examination of six women with profound anaemia in pregnancy.

Failure to observe pigmentation in Ibadan may be because it has not been looked for closely enough, or it may be related to the different presentation of folate deficiency—anaemia is seen most commonly near the end of the second trimester, while the South African patients were seen during lactation. The South Africans may have been deficient for longer than the Nigerians, and shown an early stage of a skin lesion which is much more remarkable in vitamin B₁₂ deficiency,^{1,2} a condition which develops insidiously and is almost always long-standing at the time of presentation.

Biopterin is the coenzyme required for hydroxylation of phenylalanine to tyrosine, and it has been found in abnormally high concentration in the serum of folate-deficient pregnant Nigerians.³ Widespread disturbance of amino-acid metabolism has been found in pregnant Nigerians with megaloblastic anaemia, but no evidence of a failure to convert phenylalanine to tyrosine (Jacobs, S., and Fleming, A. F., unpublished observations). There is a lower than normal histidine α -deaminase activity in the skin from vitamin

B₁₂ or folate-deficient Indians with hyperpigmentation.⁴ None of these abnormalities of amino-acid metabolism help us to understand the accumulation of melanin in megaloblastic anaemia. A possible explanation is that it is the result of abnormal cell division.—We are, etc.,

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Hypokalaemia after Treatment with Duogastrone (Carbenoxolone)

SIR,—Dr. J. Forshaw's Memorandum on hypokalaemic muscle weakness complicating the use of Duogastrone (position-release carbenoxolone sodium) (14 June, p. 674) prompts me to record a similar case.

A woman of 55 developed pins and needles in the fingers of both hands while cooking one evening. This lasted only 30 minutes, but an hour later she noticed weakness of both hands and the next day her right arm was weak. Over the next 24 hours the weakness spread to both arms, the shoulders, and then to the thighs and back muscles and she had difficulty in holding up her head. She had no muscle pain, no difficulty in swallowing, and no visual symptoms. Her general health was good and there had been no diarrhoea or vomiting. She had never suffered from muscle weakness before, nor was there any family history of a similar condition. A duodenal ulcer had been demonstrated 23 years previously, but had always responded to conservative treatment. She had been taking Duogastrone, 200 mg. four times a day, for six weeks; the course of tablets had finished one day before the onset of her present symptoms.

On examination she was a fit-looking woman. General examination was negative except for a blood pressure which varied between 200/120 and 140/80, and a small nodule in the thyroid gland. There was slight weakness of jaw opening and weakness of the sternomastoids so that she had difficulty in lifting the head from the pillow. She had severe flaccid weakness in the limbs affecting proximal muscles more than distal and the arms more than the legs. There was no fasciculation or muscle tenderness, and the tendon reflexes were absent in the arms and at the ankles. The plantar responses were flexor. There was no impairment of sensation and the peripheral nerves were not thickened.

The relevant investigations were as follows: Serum electrolytes (mEq/l.): Na 149, K 1.7, Cl 101, CO₂-combining power 32. Serum enzymes (units): Aldolase 160, S.G.O.T. 244, S.G.P.T. 200. Glucose tolerance test (30 minute intervals following the ingestion of 50 g. glucose): 117, 162, 175, 206, 209 mg./100 ml.

Electromyogram (right deltoid): mean muscle action potential duration 10 msec. (normal 14.3 $\pm$ 20%) and incidence of polyphasic potentials 16% (normal 3–12%). Maximum effort showed a normal interference pattern of reduced voltage. Conduction velocities in the ulnar and lateral popliteal nerves were within normal limits. These results strongly suggested a myopathic lesion (Dr. J. S. Chopra). Electrocardiogram: depression of ST segment in leads 1, 2, AVL, V5, and V6; QT interval 0.4 sec: prominent U waves in chest leads.