Infectious Mononucleosis

Sir,—The "new look" at infectious mononucleosis by Dr. H. Pullen (12 May, p. 350), though certainly including much sound clinical information, hardly justifies the epithet "new."

The very brief, almost scatting, reference to the possible aetiological role of E.B. virus will, for example, have disappointed those seeking aetiological clues. Even a brief consideration of the past four years' work on this virus could have provided plausible reasons for some of the observations left unexplained under the headings of "Infectivity" and "Diagnosis.

Much information which was already available1 at least four years ago is omitted entirely from the article. The disease, for instance, is stated not to be notifiable; yet there are figures for notification in Northern Ireland and in Bristol covering several years. The often observed preponderance of the disease in females in the 15-20-year age group is not mentioned. Not all agree that the atypical mononuclear cells observed are indistinguishable from those of infectious mononucleosis (and I feel that malaria is over-emphasized as a cause for the appearance of the mononuclear cells). Many would stress that the persistence of the atypical cells in infectious mononucleosis, a feature not mentioned in the article, is at least as important a diagnostic point as their numbers. With regard to serology, Dr. Pullen does not mention the anti-i antibody, nor the now widely used rapid slide "spot tests" introduced by Davidson and his colleagues.2 The correct title of the older Pullen-Bunnell-Davidsohn test is "serology," not "diagnosis.

We have examined a large series of cases, and in no case found a "positive diagnosis"—no one has yet associated lower titres of appropriately absorbed sheep cell agglutinins with any other disease. The status of seronegative cases is not discussed, nor the importance of the timing of either serological or haematological tests in relation to the duration of illness.

Dr. Pullen is known to have a special interest in the rashes of infectious mononucleosis. I wonder if he has the impression, as I have, that rash and jaundice occur together or by chance in children? He leaves the incidence of obvious jaundice vague; would he agree with a figure of about 5%?

Marrow depression is given in the article as the cause of agranulocytosis in infectious mononucleosis. These cases are exceedingly rare and relevant information is correspondingly meagre. But milder granulocytopenia is relatively common, and in these cases the evidence favours peripheral granulocyte destruction with marrow hyperplasia. The absence of marrow depression has been clearly documented by Carter,3,4 whose views on the bone marrow you have recently misquoted,5 so misleading others such as Drs. J. F. Boyd and D. Reid (21 April, p. 176).—I am etc.,

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Varicose Veins in the Tropics

Sir,—Mr. M. A. Hassan and others (3 March, p. 515) refer to the general belief that the incidence of venous disorders among the people of the developing countries of Africa is low. If the data on which this belief is based are reviewed in respect of varicose veins it is seen that the information comes from "occasional" observations by general practitioners or in hospital notes. The observations in the first case are found in published articles by earlier authors.

In February 1972, owing to the kindness of the Ministry of Health and the Service des Grandes Endemies of the Mali Republic, we were able to visit 10 traditional villages about 100 km south of Bamako and quite untouched by occidental medicine. We examined all the women (a total of 469) for the presence of varicose veins, using well-standardized criteria and methods.6 All were examined in the standing position. Their weight, height, number of living children, and number of births were noted. Owing to the lack of recent official records ages had to be estimated, and the women were divided into four groups: (1) under 25 years, (2) middle-aged, and (3) old. There were 237 in group (1), 166 in group (2), and 66 in group (3). Only evident or "clinical" varicose veins were recorded (see table). "Reticular" varicose veins were dilatations of large subcutaneous veins which did not concern the main saphenous trunks.

The prevalence rate of 10.9% (all types of varicose veins) in these women was therefore much higher than we were led to expect from the reports of others. It rose significantly (P < 0.001 (χ² test)) through the age groups, and the proportion of cases in which the veins were compared with those examined in the European cases. Finally, though 4.5% of the cases in our series were severe, we never found signs of chronic venous insufficiency or any other complication usually associated with the most severe varicose veins in Europe.—I am, etc.,

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Cognitive Deficits in Parkinsonism

Sir,—In your leading article on mental symptoms and Parkinsonism (14 April, p. 67) the important question of dementia was mentioned apropos of discussion of evidence reported by Celseia and Wasanameruk.1 I should like to raise some further points on this topic,


Adrenal Failure in Bronchial Asthma

Sir,—The beclomethasone dipropionate aerosol has been introduced into the treatment of bronchial asthma with a recommendation that the majority of patients on systemic steroids can be converted to its use with reduction or discontinuation of the oral dose. We believe this to be a potentially dangerous policy and would like to support the views of Dr. J. C. Bats and his colleagues (3 February, p. 290) with the following case report of a patient recently under our care.

A man aged 37 with a 30-year history of asthmatic asthma had received 5-15 mg of prednisolone daily for four years. Adrenal function was known to be deficient, with a basal plasma cortisol level of 4.5 μg/100 ml which reached only 8.5 μg/100 ml 60 minutes after 250 μg of tetracosactrin intravenously. In addition he was obese, with a moon face, striae, muscle wasting, and oedema. For these reasons he was started on treatment with a beclomethasone inhaler and the dose of prednisolone reduced in one month from 15 mg to 7.5 mg daily, without deterioration in respiratory function. The dosage was maintained at this level so that he was not bereft of systemic steroids.

He developed, at home, "viral" gastroenteritis for which he received symptomatic treatment from his family doctor. This appeared to be a mild illness. There were no signs of adrenal insufficiency and no obvious deterioration in his respiratory condition. Six hours after consulting his doctor he died suddenly. At necropsy the cause of death was bronchial asthma with severe bronchial oedema.

Both adrenal glands were small and atrophic. There was no evidence of myocardial infarction or cerebrovascular accident. Adrenal failure, we feel, could have contributed to this patient's sudden death.

Patients with adrenal suppression due to steroid therapy may be at considerable risk during the stress of an intercurrent illness when oral steroids have been reduced or discontinue due to the presence of beclomethasone aerosol.—We are, etc.,

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