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 scathing, reference to the possible aetiological role of E.B. virus will, for example, have disappointed those seeking recent information. 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 "Transmission."

Much information which was already available[1] 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 of rubella are indistinguishable from those of infectious mononucleosis (and I feel that malaria is over-emphasized as a cause for the appearance of atypical 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 Davidsohn and his colleagues.[2] The correct title of the older Paul-Bunnell-Davidsohn test was pleasing to see, but I have Davidsohn's support in rejecting the idea of a titre of 1/40 being necessary for a "positive diagnosis"[1]—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.[3] I wonder if he has the impression, as I have, that rash and jaundice occur together more often than by chance? 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[4] with marrow hyperplasia. The absence of marrow depression has been clearly documented by Carter,[4 5] whose views on the bone marrow you have recently misquoted,[6] so misleading others such as Drs. J. F. Boyd and D. Reid (21 April, p. 176).—I am, etc.,

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4 Carter, R. L., *Journal of Clinical Pathology*, 1966, **19**, 279.
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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 studied 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 referred to as "personal communications," not available for critical examination,[1] and in the second they are based on case histories taken for other reasons, the patients usually being bedridden.[2] It is therefore not surprising that in a special survey of this problem in an African country the prevalence of varicose veins was found to be quite different from the 1% or less reported by earlier authors.

In February 1972, owing to the kindness of the Ministry of Health and the *Service des Grandes Endémies* 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.[3 4] 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 three age groups: (1) young, (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 ($P < 0.001$) (χ^2 test) through the age groups, and the proportion of cases in which the large compared with the small saphenous vein was affected was greater than in 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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2 Dodd, H., *Lancet*, 1964, **2**, 809.
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Number and Percentage of Cases of Varicose Veins among 469 Women in Three Age Groups

Veins Affected	(1) Young (n = 237 (50·5%))	(2) Middle-aged (n = 166 (35·4%))	(3) Old (n = 66 (14·1%))	All (n = 469 (100%))
Large saphenous	2 (0·8%)	5 (3%)	1 (1·7%)	8 (1·7%)
Small saphenous	—	2 (1·2%)	3 (4·6%)	5 (1·1%)
Subcutaneous ("Reticular")	12 (5·1%)	17 (10·2%)	9 (13·6%)	38 (8·1%)
Total	14 (5·9%)	24 (14·4%)	13 (19·9%)	51 (10·9%)

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 fears of Dr. J. C. Batten and his colleagues (3 February, p. 296) with the following case report of a patient recently under our care.

A man aged 37 with a 30-year history of bronchial 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 osteoporosis. For these reasons he was started on treatment with a beclomethasone inhaler and the dosage 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 steroid.

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 recorded as right lower lobe pneumonia. 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 discontinued in favour of beclomethasone aerosol.—We are, 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 Celesia and Wanamaker.[1] I should like to raise some further points on this topic,

because it seems that to focus on the presence or absence of dementia in Parkinsonism may in fact distort the picture through oversimplification.

The assessment of dementia can be simply a subjective, clinical evaluation, which is, unfortunately, most often how it is made in studies on Parkinsonism. It can also be made by looking at the actual degree of cortical atrophy. An example of this is Selby's[2] study of 250 Parkinsonism patients where he found 57·2% had such degeneration. Finally, and probably most usefully, is to carry out appropriate psychometric assessment. I have had occasion to review the studies where full assessment of level of intelligence of patients with Parkinsonism has been made using such well standardized tests as the Wechsler Adult Intelligence Scale (W.A.I.S.), the Mill Hill Vocabulary Scale, and Raven's Progressive Matrices. There is no argument that individual cases may have dementia but what is outstanding is that in none of these studies (seven in number) was the mean I.Q. for the Parkinsonism patients below the average range—namely, I.Q. 90 to 109.

In two of the studies[3 4] the performance I.Q. on the W.A.I.S. was in the low 90s, but this could be attributed to direct impairment of motor ability affecting speed of performance on those subtests where motor activity is necessary. In some studies the verbal I.Q., though in the average range, was well above the population mean of 100. The overall deduction from these studies seems to be that a group of patients with Parkinsonism may be no more intellectually impaired than a random group of people of comparable age from the general population. This does not mean that they have no cognitive deficits. Recent carefully designed psychological investigations have produced evidence of cognitive deficits which operate much more selectively than a general intellectual deterioration.[5-7] These studies have all controlled the effects of motor impairment per se on level performance. The findings concur in implicating the basal ganglia as having a role in perceptual organization.

Apart from interesting, theoretical considerations about the role of subcortical brain centres in higher mental processes, there is pragmatic value in such studies. If areas of subtle deficit can be carefully mapped then therapeutic techniques can be evaluated by measuring changes in these areas. I have already some preliminary evidence from a small pilot study that treatment by levodopa not only improves actual motor performance but also reduces the extent of the "central processing" deficit, at least over a period of up to five months after the start of treatment.—I am, etc.,

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Hemifacial Spasm

SIR,—After reading your leading article (16 December 1972, p. 624) I have waited to see, from your correspondence columns, whether any of your readers had hit on a method other than surgical for the relief of hemifacial spasm. To my surprise I appear to be able to teach my grandsons how to suck eggs.

When I started private eye practice over 30 years ago I saw in my first two years three patients with this distressing and socially embarrassing condition. As the twitching orbicularis was the chief offender, often causing sleeplessness, I wondered if one could perhaps damp down the impulses in the most distal branches of the facial nerve without causing actual paresis or paralysis of the particular muscles. I explained to the patient that my idea was to try injections of weak alcohol after local anaesthesia. Their reaction was willingness to "try anything." I started first with 10% and gradually worked up to 90% alcohol, as I found that fewer injections were needed with the latter and that the danger was minimal. In all, I have treated 15 patients, all of them with complete success.

For the orbicularis three prominent bony points are chosen: one just outside the margin of the bony orbit at the level of the outer canthus, the second just above, and the third just below the prominence of the malar bone. All are in a vertical line and the point of the needle should strike bone at all three points. The points are infiltrated with procaine and after five minutes a total of 0·5-0·75 ml of 90% alcohol is injected in divided doses. The patient should be warned that a hot, burning sensation may be felt in spite of the anaesthetic, but this soon passes off. A further warning should be given that at the first injection there may be a moderately severe reaction of redness and swelling, which, however, passes off in 2-3 days. Further injections are given at intervals of 2-3 weeks, until there is no further evidence of spasm. The orbicularis should be tested by asking the patient to "screw up" his eyes and then trying to force them open with one's fingers. Injections should not be carried to the point of paresis, and any relative weakness on the injected side should be a warning to stop treatment. The rest of the face can be treated similarly by injections along an extension of the same line into the cheek below the malar bone, opposite the corner of the mouth, and on the lower bony margin of the mandible.

One of my patients had violent contractions of the platysma, which was similarly successfully treated. In two patients there was a slight localized recurrence after 2-3 years. A single injection at the necessary points cured this. There is some temporary loss of sensation over the areas injected, and in one case there appeared to be a slight thickening of the tissues below the skin.—I am, etc.,

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Anaesthetic Contamination

SIR,—In reply to the letter from Drs. A. A. Spence, R. P. Knill-Jones, and D. D. Moir (21 April, p. 174), we agree that direct proof of any effect of long-term exposure of man to low concentrations of inhalational anaesthetic agents is at present lacking and that incrimination solely of halothane is particularly speculative. However, Allison and Nunn[1] have demonstrated gross effects, from brief exposure to inhalational anaesthetic agents, on microtubules, which are known to be related to the motile system of sperm tails.[2] Reproductive disturbances, including an increased proportion of female live births, have been demonstrated in laboratory animals.[3] A larger version of our pilot study might provide a simple, statistically acceptable method of demonstrating an effect upon anaesthetists in an area less sensitive to questioning than the incidence of stillbirth and spontaneous abortion and assist in deciding whether large-scale expenditure on "anti-pollution" devices is desirable.

Only children conceived while the father was engaged in full-time anaesthetic practice were included in our results. In their paper[4] Dr. Knill-Jones and his colleagues do not state that this criterion was applied to their finding of 50% female children (compared with 48·5% in controls) born to female anaesthetists nor do they mention it with regard to the subgroup under 30 years, in which they now report 53·6% of male children. The proportion of female live births found in our survey was compared with the birth pattern in the Sheffield Hospital Region, in which region the anaesthetists we surveyed were employed, and not only with the pattern in England and Wales as a whole, as they have inferred.—We are, etc.,

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2 Satir, R. P., in *The Contractile Process*, ed. A. Stracher, p. 241. Boston, Little, Brown, 1967.
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School Eye Clinics

SIR,—Mr. J. A. E. Primrose's letter (14 April, p. 117) is most important because he describes the time-honoured view of school eye clinics and the division between them and the hospital clinics. Some rationalization really is necessary.

Doubts have been raised on the effectiveness of treating squint and amblyopia if they are not discovered before school age. Attention has concentrated on hospital cases (usually of preschool age) of squint and amblyopia and there is a conspicuous lack of knowledge and method in treating the school child with these conditions—largely, I believe, because of this division into hospital and school clinics. I tried to foresee developments in the ophthalmic care of children in order to arrange the back-up organization to meet any changes, principally regarding the problems of squint and amblyopia. Two separate services treating variations of the same condition often occurring in the same families is illogical. Furthermore, the public health service, as we know it, will shortly disappear.

The next "development" may be a completely different screening programme either aimed at truly preventive ophthalmology, if that is found possible, or at least better directed to revealing treatable conditions at an age when they can be treated. The obvious defect in the present school screening programme is that by the time it detects squint and amblyopia they have probably been estab-