all adults. It is likely that ischaemia, rather than haemorrhage, was the more frequent pathogenic mechanism.

The other disease which you did mention is diabetes mellitus. Diabetes, like hypertension, is a predisposing factor. In large series the frequency of previously recognized diabetes was 10-14%.^{1,2} However, when glucose tolerance tests are performed many new cases are discovered, the frequency of abnormal results increasing with age.¹ The frequency of overt and latent diabetes in patients with Bell's palsy is thus similar to that previously reported in oculomotor palsy.³ Patients with Bell's palsy frequently show abnormal blood vessels in their conjunctivae and nail-beds, and I have therefore postulated that diabetic microangiopathy may be a factor responsible for the nerve lesion.

Your leading article mistakenly attributes to Dalton the evidence that herpes zoster infection can cause Bell's palsy. Ramsay Hunt suspected this possibility when he described the syndrome which now bears his name, and it was Aitken and Brain⁴ who demonstrated high titres against the virus in some patients with typical Bell's palsy without vesicles. Though the possibility of an unrecognized viral infection causing Bell's palsy is interesting most patients lack any evidence of such a preceeding or concurrent infection. I have tried to isolate viruses from patients with Bell's palsy and also to detect antibodies to several viruses, but the results have been negative thus far.

The seasonal variation in the incidence of Bell's palsy which is frequently mentioned and which you quote is of course only circumstantial evidence that the disease is infective. As Leibowitz⁵ remarked, similar climatic influences have been shown on the occurrence of myocardial infarction (and also cerebral vascular disease). Leibowitz has subsequently demonstrated⁶ (and not in the paper you mistakenly quoted) clustering of cases of Bell's palsy. This interesting finding is open to similar alternative interpretations.

A final point concerns the theory that water retention is a predisposing factor in the development of Bell's palsy. I have found that pregnant women are more susceptible to Bell's palsy than expected, particularly just before delivery, and also in the puerperium.⁷ Water retention could be a factor there too.—I am, etc.,

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¹ Korczyn, A. D., *Lancet*, 1971, 1, 108.

² Hauser, W. A., Karnes, W. E., Annis, J., and Kurland, L. T., *Mayo Clinic Proceedings*, 1971, 46, 258.

³ Korczyn, A. D., *Lancet*, 1971, 1, 862.

⁴ Aitken, R. S., and Brain, R. T., *Lancet*, 1933, 1, 19.

⁵ Leibowitz, U., *Neurology (Minneapolis)*, 1966, 16, 1105.

⁶ Leibowitz, U., *Brain*, 1969, 92, 109.

⁷ Korczyn, A. D., *Acta Neurologica Scandinavica*, in press.

No Smoking and Life Expectancy

SIR,—In recent months the *B.M.J.* and numerous others have discussed the dangers of smoking. Everywhere, however, there appears one great omission. In its anxiety to persuade the public to abandon tobacco and the consequent illnesses and disability produced as a late penalty, it is important that the medical profession should also be far

sighted beyond a mere clinical field. It is not sufficient to abolish an undesirable disease if, as a consequence, more misery is produced later. A parallel situation has occurred in recent years where the prevention of malaria by removing an agent of population control has led to widespread starvation.

When the effects of reduced smoking bear fruit, we shall have an increased life expectancy and a larger number of elderly citizens. The people who have smoked are at present likely to die at an age when the younger generation is finding them a burden which they are unwilling to bear. We must therefore couple with our exhortations for the abolition of smoking a call for advanced creation of suitable homes and geriatric services to cope with the new population bulge that will develop.—I am, etc.,

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Diagnosis of Cystic Fibrosis

SIR,—I would like to comment on Dr. A. G. F. Davidson and Professor Charlotte M. Anderson's (6 November, p. 362) reference to the screening of meconium for the presence of albumin.

They state, quite correctly, that 10-15% of patients with cystic fibrosis have no clinical evidence of malabsorption and that such cases may not show a positive meconium test.

I mentioned earlier (11 September, p. 639) the routine screening programme being carried out in Newcastle using this technique. One of the aims of this experiment is to find out how many cases of cystic fibrosis are missed for this, or any other reason.

We do not know as yet if absence of malabsorption automatically means that a meconium test is negative, but even if this is so we feel it is still more than worth while to diagnose the remaining 85-90% at birth.—I am, etc.,

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"Stroke Units"

SIR,—In your leading article (6 November, p. 313) you suggest that we seriously consider the development of stroke units in this country, similar to those which have grown up in the United States. The conditions in the two countries are not, however, comparable. The majority of strokes occur in older patients and in the United Kingdom we already have a national network of geriatric units, which have done their best, with limited resources, to develop rehabilitation services. For many years these units have attracted stroke patients and many important contributions to the British literature of stroke have originated from such departments.

When the geriatric assessment unit of the Glasgow Royal Infirmary group of hospitals was established at Lightburn Hospital in 1968 one ward of 30 beds (15 male and 15 female) was set aside for stroke rehabilitation. A few patients are admitted from their own homes direct to this unit, but most strokes occurring in the area served by the hospital group are admitted in the first instance to the general medical wards of Glasgow Royal

Infirmary, where they undergo intensive care, if appropriate, and have ready access to neurosurgical and investigative facilities. Many patients are treated to conclusion in the medical wards, but each year between 150 and 200 patients are referred by the physicians in charge to the geriatric service, and are transferred, after assessment, to the stroke ward in the geriatric unit. One-quarter of these patients are under the age of 65, but none has objected to being treated alongside older patients. The stroke rehabilitation ward is organized along the lines of a standard geriatric rehabilitation ward, with close collaboration between doctors, nurses, physiotherapists, occupational therapists, speech therapist, medical social worker, and after-care services. The additional activities include a daily recreation programme organized by a ward hostess, pre-discharge home assessment, post-discharge follow-up, a club for relatives, group speech therapy, and group therapy for relatives of aphasic patients. A day hospital is being planned.

This arrangement has worked well, and similar collaboration between general physicians, neurologists, neurosurgeons, and geriatricians might prove a suitable pattern for the management of stroke patients in Britain. The unit depends heavily on its small staff of physiotherapists, occupational therapists, and speech therapists, and the continuing shortage of these grades of staff is the major obstacle to the needed expansion of stroke rehabilitation throughout the country.—I am, etc.,

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Epitaph for the M.C.H.C.

SIR,—Dr. F. Nour-Eldin (30 October, p. 298) takes exception to the statement of Dr. M. S. Rose that neither the M.C.H.C. (mean corpuscular haemoglobin concentration) nor the M.C.H. (mean corpuscular haemoglobin) convey anything of particular clinical value any longer (16 October, p. 169) and then goes on to raise a number of points.

It must be made quite clear that Dr. Rose is referring specifically to the M.C.H.C. as calculated by automatic apparatus and is contrasting this with the M.C.H.C. derived from the haemoglobin level and the packed cell volume measured by manual methods. That these two figures do not always mean the same thing has now become obvious as is explained by Dr. J. M. England and others (23 October, p. 232).

(1) The M.C.H.C., as measured by the Coulter Model "S," does not fall in hypochromic anaemia except in the severest cases of iron deficiency. Therefore, when measured in such apparatus, it cannot "return to normal on successful treatment of hypochromic anaemias." The M.C.V. (mean corpuscular value), on the other hand, begins to fall relatively early in iron deficiency and is an excellent parameter both for assessing the degree of the deficiency and for following the response to therapy.

(2) The traditional teaching of textbooks of haematology is that the absolute values, the M.C.V. and M.C.H., are unacceptably inaccurate because they depend upon the red cell count, which has a large inherent error—of the order of $\pm 400,000$ cells out of a total of 5,000,000 cells if a visual count is done on 500-1,000 cells.¹ The introduction