English girl visiting the country from Nigeria. Her illness was mild and her symptoms settled without treatment.

I should like to report the case of a 63-year-old New Zealand man who came to England via South Africa and Rhodesia. While in Rhodesia he developed a febrile illness, and approximately 14 days after the onset of his symptoms he came to Charing Cross Hospital. Although conscious, he was confused and unable to give an accurate history. The tongue was dry and furred, there was air hunger, and he had a macular rash all over the body. His temperature was 101°F. (38.3°C.) and the pulse was 120 per minute. The lungs were clear clinically and radiologically and there were no other abnormal physical signs.

On admission, a leucocyte count was 8,300/cm³, of which 92% were neutrophils. The Paul Bunnell test was negative, and no malarial parasites were seen. The urine was normal and culture was sterile. Two days later, the leucocyte count was 15,000/cm³, and the blood film showed a few atypical lymphoid cells. The cerebrospinal fluid was normal. By this time, the patient was delirious and he was so ill that hydrocortisone 100 mg six hourly, penicillin, 2 mega units six hourly, and chloramphenicol 500 mg six hourly were given empirically by intravenous infusion, two days before the tests indicated that the diagnosis was typhus. The response to therapy was immediate. Overnight, the cerebral clouding vanished and the patient became rational. The temperature was normal within 24 hours and remained so thereafter. Hydrocortisone was continued for two days and chloramphenicol for two weeks.

The Weil-Felix reaction showed an agglutination titre of 1/180, for *Proteus* OX-K, and the rickettsial complement fixation test was positive against the epidemic typhus antigen to a dilution of 1/250. Neither epidemic louse born typhus nor scrub typhus are recorded in South Africa or Rhodesia. The most likely causative organism was the African tick-borne typhus *Rickettsia conorii*. Unfortunately, the Weil-Felix reaction is not specific in this case, and there is considerable antigenic overlap between the types of typhus.

It is important to realize that tick-borne typhus is a risk in people coming to Britain from Africa and that the illness can run a very severe course.—I am, etc.,

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Ocular Bobbing

SIR,—Ocular bobbing is a distinctive movement disorder seen in a variety of related forms which, in a recent review, we classified as "typical," "monocular," and "atypical."¹

"Typical" ocular bobbing occurs in patients with paralysis of horizontal conjugate eye movements and consists of abrupt, spontaneous downward jerks of the eyes with a slow return to the mid-position. The "monocular" type reflects co-existing unilateral third nerve paresis in addition to the bobbing. The "atypical" type includes either a variation unexplained by associated ocular motor palsy, or bobbing with intact spontaneous or reflex horizontal eye movements. We presented nine cases and discussed the causes and prognostic significance of the disorder.

In our literature review, we neglected to include two Japanese language publications on the subject, which I mention here for completeness.

Okamoto *et al.*² described a 7-year-old boy with an intrinsic pontine tumour who had the "typical" form of ocular bobbing. Kawanami *et al.*³ misused the term "ocular bobbing" in describing a patient with ocular myoclonus. Regularity, pendular phases, and associated synchronous movements of mid-line structures should readily distinguish ocular myoclonus from bobbing.

Since publication of our report, Borit mentioned ocular bobbing occurring in Leigh's necrotizing encephalomyelopathy, a metabolic disorder of infancy and childhood.⁴—I am, etc.,

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¹ Susac, J. O., Hoyt, W. F., Daroff, R. B., and Lawrence, W., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1970, 33, 771.

² Okamoto, S., Hayashi, M., and Shintani, A., *Clinical Neurology (Tokyo)*, 1968, 8, 257.

³ Kawanami, S., Shida, K., and Kuroiwa, Y., *Clinical Neurology (Tokyo)*, 1969, 9, 447.

⁴ Borit, A., *Archives of Ophthalmology*, 1971, 85, 438.

* A leading article on this subject appeared on 8 May, p. 295.—Ed., *B.M.J.*

Wound Dehiscence after Caesarean Section

SIR,—Dr. James Mowat and Dr. John Bonnar (1 May, p. 256) are to be congratulated on their report showing the advantage of the transverse incision. I would suggest that the incision they describe should continue to be known as the Pfannenstiel, both to give honour to the great gynaecologist who described it, and also to distinguish it from the transverse rectus-cutting incision, which finds little favour, and the skin-only transverse incision, which combines the disadvantages of both vertical and transverse incision.

Their report discusses only the "burst abdomen," but, as they suggest, the incidence of later incisional hernia should also be reduced by the Pfannenstiel incision. The times have surely gone when the gynaecologist's trademark was a ventral hernia in the lower abdomen, and the time has come when this incision should be the routine one in our speciality. It is not an easy one, and attention to technique is needed,¹ also good assistance, but most of all the co-operation of our anaesthetic colleagues in providing safe relaxation, which can best be provided by one of the methods of regional block.—I am, etc.,

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¹ Williams, B., *Journal of Obstetrics and Gynaecology of the British Empire*, 1960, 67, 853.

Aspirin and Renal Papillary Necrosis

SIR,—Professor H. E. de Wardner and Dr. K. G. Koutsaimanis (3 April, p. 45) are incorrect when they state that consumption of aspirin has yielded only two victims with renal papillary necrosis. They seem to have overlooked a number of clinical reports in which papillary necrosis has been associated with consumption of aspirin and not phenacetin or paracetamol.¹⁻⁷ Aspirin given alone or in combination with other analgesics

regularly causes papillary necrosis in experimental animals, and this lesion can occur in individuals taking large amounts of aspirin. In contrast, phenacetin does not produce typical papillary necrosis in animals, even when given in massive doses, and analgesic nephritis has not been described in man with phenacetin alone.

Analgesic abusers rarely take plain aspirin. They prefer mixed analgesics, which until recently usually contained phenacetin. The figures for the total national consumption of aspirin and phenacetin quoted by Professor H. E. de Wardner and Dr. K. G. Koutsaimanis give no indication of the relative incidence of abuse of these two drugs, and it is therefore quite unrealistic to assume that aspirin is "safer" than phenacetin on this basis. I am in complete agreement with Dr. R. S. Nanra and Dr. P. Kincaid-Smith (3 April, p. 45), and the nephrotoxicity of aspirin should not be underestimated. The evidence strongly suggests that analgesic nephritis in Britain is caused largely by aspirin consumed in the form of popular analgesic mixtures. Phenacetin probably plays a very minor role in respect of nephrotoxicity, but its effect on the central nervous system⁸ may account for its popularity with analgesic abusers.—I am, etc.,

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¹ Prescott, L. F., *Scottish Medical Journal*, 1969, 14, 82.

² Gault, M. H., Rudwal, T. C., Engles, W. D., and Dossetor, J. B., *Annals of Internal Medicine*, 1968, 68, 906.

³ Harvald, B., *American Journal of Medicine*, 1963, 35, 481.

⁴ Olafsson, O., Gudmundsson, K. R., and Brekkan, A., *Acta Medica Scandinavica*, 1966, 179, 121.

⁵ Bulger, R. J., Healey, L. A., and Polinsky, P., *Annals of the Rheumatic Diseases*, 1968, 27, 339.

⁶ Lawson, A. A. H., and Maclean, N., *Annals of the Rheumatic Diseases*, 1966, 25, 441.

⁷ Murray, R. M., Lawson, D. H., and Linton, A. L., *British Medical Journal*, 1971, 1, 479.

⁸ Prescott, L. F., Steel, R. F., and Ferrier, W. R., *Clinical Pharmacology and Therapeutics*, 1970, 11, 496.

Encouraging Recruits to Medicine

SIR,—From the start of my clinical training I have been taught that two of the marks of a good doctor are a sense of responsibility to his patients and the ability to see them as human beings. It now seems strange to be urged by Dr. D. J. MacMyn (1 May, p. 281) to regard my parents as a continuing source of financial support regardless of the effect it may have on them.

I have no doubt that my father would, if necessary, have postponed his retirement and lowered his standard of living to see me through medical school. I am sure that I "would have been none the worse for that." But would he have been?

The present shortage of doctors seems to indicate that the medical profession is in need of recruits whose parents' income forces them into a sober consideration of money.—I am, etc.,

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Superannuation and Substandard Lives

SIR,—The letters by Drs. K. D. Allardyce (17 April, p. 165), and P. H. Sutton (8 May, p. 342) show that the woolly thinking about medical "fitness" for superannuation schemes