She went on to say that if I knew of any interested persons or suitable properties the WRVS would be glad to respond. Inquiries about endowments or gifting of properties should be addressed to the Secretary, WRVS Trustees Ltd, 17 Old Park Lane, London W1Y 4AJ.

Large scale development of this priceless voluntary effort is surely the light at the end of the tunnel; and the devotion and dedication of the WRVS must be encouraged by every conceivable method.

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Significance of microhaematuria in young adults

SIR,—We agree with Dr P Froom and others (7 January, p 20) that asymptomatic microhaematuria without proteinuria in young adults is generally benign, but, unlike them, we believe that it is worth while to screen young adults for microhaematuria.

In Singapore all national service registrants who are found to have urinary abnormalities on routine screening are referred to our unit for further evaluation. From 1970 to 1977 we investigated 176 such patients of whom 24 (14%) had microhaematuria alone.[1 2] All had normal blood pressures and renal function. Appearances on intravenous pyelograms were normal in all, and percutaneous renal biopsies showed 21 with diffuse mesangial proliferative glomerulonephritis (two with associated glomerulosclerosis) and three with minimal lesions: four underwent immunofluorescence studies (three with mesangial IgA deposits).

At the end of the follow up period (mean 86 (SD 30) months, range 45-142 months) seven (29%) had developed significant proteinuria (>0·5 g/24 h), a similar proportion to that found by Dr Froom and colleagues, of whom one also developed hypertension. None had renal impairment and in 10 (42%) urine analysis gave normal results.

Asymptomatic microhaematuria thus seemed to herald significant renal disease in a substantial proportion of our apparently healthy young subjects. Several reasons could account for the differences between our findings and those of Dr Froom and colleagues. Firstly, our patients are less highly selected. Their patients were presumably screened and found normal before entering the air force and therefore a much lower incidence of renal abnormalities would be expected.

Secondly, our patients, on the whole, had more persistent bleeding and higher red cell counts (38 (SD 39) per high power field) than those of Dr Froom and others, as they were investigated only if more than one consecutive urine analysis gave abnormal results (>3 red blood cells per high power field) because, as Dr Froom and others pointed out, this method of analysis is of low sensitivity and accuracy and one isolated abnormal result may not indicate any renal abnormality. This, together with the fact that Dr Froom's patients tended to be underinvestigated could explain why such a small proportion of their subjects appeared to have any pathological lesion.

Phase contrast microscopy of the urine, as described by Birch and Fairley,[3 4] now offers not only a more rational approach to deciding whether to investigate cases of asymptomatic isolated microhaematuria but also a more accurate and sensitive way of assessing its extent. Any isomorphic red cells in the urine warrant urological investigation.

Our current practice is to use this inexpensive and non-invasive method to screen patients with microhaematuria for evidence of non-glomerular bleeding. If this is found then full urological investigation is undertaken. Glomerular haematuria should be followed up regularly, as a substantial number of these patients develop significant renal disease.

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Drugs and insomnia

SIR,—The leading article by Dr John Marks and Group Captain Anthony N Nicholson (28 January, p 261) begins as a report of a conference concerned with drugs and insomnia but proceeds to general statements about the treatment of insomnia.

I am concerned that readers may think that this article summarises good therapeutic practice in treatment of insomnia. It does not, because it fails to emphasise the importance of adequate psychological and behavioural analysis of sleep complaints before consideration is given to prescribing hypnotics. Further, it does not mention psychological treatments that may be applicable to sleep problems. Our teaching is that there are almost certainly no indications for prescribing hypnotics for chronic insomnia, for which either there is a specific cause—for example, depression—or a psychological approach is required. In either case adequate behavioural assessment is essential.

This concept is clearly implied in the best current psychiatric textbooks[1] and is basic to developments in what is sometimes called behavioural medicine, which has much to offer in a wide range of medical disorders, including insomnia (Bootzin, behavioural treatment of insomnia, BMA Audio Cassette Programs, 200 Park Avenue South, New York NY 10003), but which is not yet familiar to many doctors.

Perhaps a succeeding leading article could draw attention to psychosocial procedures that may well make obsolete the use of hypnotics in all but a few cases.

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**The authors reply below.—ED, *BMJ*.

SIR,—The whole of our leading article was concerned with the consensus development conference on "Drugs and insomnia: the use of medication to promote sleep," held at the National Institutes of Health, Bethesda, 15-17 November 1983.

Dr Peter Hauri, Dartmouth Medical School, New Hampshire, who is the acknowledged authority on the behavioural aspects of insomnia, concluded in his presentation that hypnotics have a part to play in the behavioural treatment of insomnia. His opinion was well received by the other participants and was reflected in their report. Our article summarised the meeting, and careful reading will show that the behavioural aspects, which we also feel are important, were covered.

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Hypoxia in patients with acute hemiplegia

SIR,—We would be more cautious than Dr M J Walshaw and Dr M G Pearson (7 January, p 15) in interpreting the pathogenic implication of the observed hypoxia in patients with acute hemiplegia and the benefit of oxygen treatment.

There is no good evidence that moderately reduced arterial oxygen tension (Pao_2 above 7 kPa (52·5 mm Hg)), which limits availability of oxygen to functioning neurones, plays an important part in the development of cerebral infarction. The fraction of total arterial oxygen extracted by cerebral tissues can be increased at least twofold, for instance in response to reduced cerebral blood flow in acute stroke[1] and in patients with carotid artery occlusion without cerebral infarction.[2] Reduced cerebral blood flow due to major vessel occlusion results in an increased blood volume[3] redirected by autoregulatory responses, and under such conditions patients may have little additional response to increased $Paco_2$.[4] Thus the claim that relatively minor changes in Pao_2 or $Paco_2$ are likely to affect the size of cerebral infarct, and thus the prognosis of an acute stroke, must be viewed with caution.

On the other hand, although effective measures to reduce infarct size are needed urgently, none are widely acknowledged at present and priorities of management must be to prevent complications and maximise functional recovery. To this end attendants, particularly nursing staff, must concentrate on the care of pressure areas, hydration, avoidance of fixed postures, and the emotional needs of the patient. Such measures seem unlikely to be facilitated by the assiduous application of oxygen treatment.

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