USA) has no further supplies, nor does any other company sell this antiserum, and it is not clear when amyloid A antiserum will be available commercially.

In contrast, the measurement of serum C reactive protein concentration is easy and readily accessible. C reactive protein is the classical acute phase protein,[2] the circulating concentrations of which correlate extremely closely with those of serum amyloid A protein in all published studies[1] and which were shown as long ago as 1981 to provide the same information in renal allograft recipients as that now reported for serum amyloid A protein by Professor Maury and colleagues.[3] C reactive protein is easy to isolate and purify, potent antisera are readily available, assay by the whole range of immunochemical techniques is well established, and several excellent and relatively inexpensive methods are commercially available—for example, EMIT homogeneous enzyme immunoassay or fluoroimmunoassay from Syva Company, Palo Alto, California, USA; or rate immunonephelometry from Beckman Instruments Inc, Fullerton, California, USA. Both of these companies have subsidiaries in the United Kingdom and Europe. For routine monitoring of the acute phase response, which is extremely useful in the management of a wide range of different conditions[1 4] as well as in renal allografting, serial assays of serum C reactive protein are therefore the method of choice.

M B PEPYS

MRC Acute Phase Protein Research Group,
Department of Medicine,
Royal Postgraduate Medical School,
Hammersmith Hospital,
London W12 0HS

[1] Pepys MB, Baltz ML. Acute phase proteins with special reference to C-reactive protein and related proteins (pentaxins) and serum amyloid A protein. *Adv Immunol* 1983;**34**:141-212.
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Metabolism of narcotics

SIR,—We were intrigued by the suggestion from Dr Henry McQuay and Dr Andrew Moore (21 January, p 237) that the kidney may play an important part in morphine metabolism as well as in excretion. We have since extracted the case records of patients admitted to this unit during the years 1981-3 who had biochemical evidence of impaired hepatic or renal function.

Patients were divided into three categories: (*a*) those with evidence of impaired hepatic function (excluding those with co-existent renal failure or pulmonary metastases); (*b*) those with evidence of impaired renal function with or without impaired hepatic function; (*c*) those with impaired renal function but without coexistent impaired hepatic function (a subset of *b*). Criteria of impaired function were a plasma aspartate transaminase activity or plasma creatinine concentration of more than four standard deviations above the mean (>50 IU/l for aspartate transaminase; >180 μmol/l (2·0 mg/100 ml) for creatinine). The median dose of oral morphine sulphate solution received by each of the three groups every four hours was compared with the median dose for all patients receiving morphine in 1982 (n = 246). This larger group was analysed using a microcomputer database system.[1] The table shows the results.

As is the standard practice, the dose of morphine had been optimised by titrating the dose upwards until the patient's pain was controlled. The finding that patients with impaired hepatic function received normal doses but that patients with impaired renal function were controlled on below average doses lends support to the suggestion that the kidney may play a major part in the conjugation of morphine. We recognise, however, that our data are open to several alternative interpretations.

CLAUD F B REGNARD
ROBERT G TWYCROSS

Sir Michael Sobell House,
Churchill Hospital,
Oxford OX3 7LJ

[1] Hanks GW, Thomas PJ. A microcomputer-based system for therapeutic audit and retrospective research in palliative care. *Med Inf (Lond)* 1982; **7**:113-8.

Details of patients with biochemical evidence of impaired hepatic or renal function who had received morphine compared with details of all patients who had received morphine in 1982

Category	No	M:F ratio	Median age (range)	Median four hourly dose of morphine (mg) (range)	Median time on morphine (days)	Median plasma AST (IU/l) (range)	Median plasma creatinine (μ/l) (range)
All patients receiving morphine in 1982	246	0·9:1	62 (5-95)	20 (2·5->200)	9	—	—
Patients with impaired hepatic function	25	0·4:1	53 (43-73)	20 (5-260)	24	82 (50-350)	—
Patients with impaired renal function*	14	1:1	60 (49-75)	7·5 (5-100)	11	—	232 (197-1394)
Patients with impaired renal function†	9	0·8:1	59 (49-73)	5 (5-100)	5	—	294 (311-1394)

AST = Aspartate transaminase.
*Including five with impaired hepatic function.
†Excluding five patients with impaired hepatic function.
Conversion: SI to traditional units—Creatinine: 1 μmol/l ≈ 0·01 mg/100 ml.

Mother care for children in hospital

SIR,—Dr Michael Ryan's USSR letter (4 February, p 381) attracted our attention at once as "Mother Care for Children in Hospital" is the name of the organisation that was the precursor of the National Association for the Welfare of Children in Hospital. The original organisation was founded in 1961 to encourage the implementation of government policy on the welfare of children in hospital, and our present name was taken from the 1959 Platt report.

Our recent survey shows that in England we are halfway there in ensuring that children in hospital have access to their parents,[1] but there is a desperate lack of accommodation for the parents of young children who wish to stay in hospital. We have written to every region about access and accommodation and have the impression that our survey has surprised and shamed some hospitals into looking at their own practice at ward level.

Dr Ryan's reports of doctors in Russia who think "mothers in hospital are unauthorised persons who merely distract the staff with idle questions" mirror rather neatly comments reported to us from surgeons who exclude parents on operating day. The Nuffield Foundation has given us a grant to examine hospital policies concerning operating day, and we are holding conferences on the subject on 7 April at Devonshire Hall in Leeds and on 14 June at the King's Fund Centre. Young children need their parents' support and comfort on the day of operation.

In contrast, a survey of neonatal units conducted in December 1983 and January 1984 shows that there are very few restrictions on parents being with their babies in these units, but parents do not always share fully in the care and we shall be looking at the barriers to shared care in eight conferences during Special Care Baby Month (March 1984) and at the King's Fund Centre on 22 May 1984.

Once most hospital staff accept the principle of care being shared between parents and professionals we see our task to be to reduce the barriers to complete cooperation. The best British hospitals probably lead the Western world in this aspect of child care; in the Third World the separation of mother and child may not even have been contemplated.

JEAN LOVELL-DAVIS
Director

National Association for the Welfare of Children in Hospital,
London SE1 8UE

[1] Thornes R. Parental access and family facilities in children's wards in England. *Br Med J* 1983; **287**:190-2.

The WRVS and the elderly

SIR,—Dr Tony Smith's articles on the care of the elderly in Denmark and the Netherlands (8 October, p 1053; 14 January, p 127) underline the increasing problems of nursing the aged. In the United Kingdom poignant examples of the problems can be seen in nearly every adult ward—especially of the smaller hospitals. They are termed "social cases"—not fit to live alone and not ill enough to justify hospital treatment with its high attendant costs. Their growing numbers are the despair of the medical and administrative staff alike, and the problem is now really too big for the National Health Service and local authorities to cope with financially.

In recent correspondence with the chairman of the Women's Royal Voluntary Service I asked her what could be done about it. She replied that the WRVS would certainly like to extend its existing number of homes (24 residential and one full care nursing home), which provide places for 675 elderly people and are run as residential clubs, usually in properties given to them or purchased by the WRVS Trust. She also stated that its priority was to provide "extended care"—ideally care for those who are beyond what is provided in the clubs and which takes place in an extension to or in the grounds of the existing home.

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.

RODERICK S ROSS

Dunston Hill Hospital,
Gateshead,
Tyne and Wear NE11 9QT

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.

K T WOO
R P S EDMONDSON
E J C LEE
G S C CHIANG
C H LIM

Singapore General Hospital,
Singapore 0316

[1] Lim CH, Woo KT, Chiang GSC. Correlation of proteinuria and histopathology in asymptomatic glomerulonephritis. *Ann Acad Med Singapore* 1982;**11**:9-14.
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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.

J P WATSON

Department of Psychiatry,
Guy's Hospital Medical School,
London SE1 9RT

[1] Oswald I. Sleep disorders. In: Kendell RE, Zealley AK, eds. *Companion to psychiatric studies*. Edinburgh: Churchill Livingstone, 1983.

**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.

J MARKS

Girton College,
Cambridge CB3 0JG

A N NICHOLSON

Royal Air Force Institute of Aviation Medicine,
Farnborough,
Hants

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.

F C MARTIN
A G RUDD

Geriatric Unit,
Hammersmith Hospital,
London W12 0HS

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