of prophylaxis is less well founded when given outside the close and prolonged contact group.

In a hospital there seems to be no indication for precautions above normal hygiene in the wards. In the context of meningococcal infection immunocompromised patients are mainly young children and occasionally adults who are not yet immune, and many clinicians will prefer to admit patients with meningococcal infection to a side room until the diagnosis is established and treatment has been started, though it is doubtful whether this is strictly necessary. There is evidence that penicillin treatment suppresses but may not eradicate the meningococcus in the throat so during treatment the chance of transmission will be further reduced; rifampicin is conveniently given before discharge. Taking swabs from staff is generally unhelpful because in the time taken to process them the circumstances relating to carriage may well have changed. For an expanded discussion on the principles of prophylaxis the Communicable Disease Surveillance Centre, Public Health Laboratory Service, Colindale, may be contacted for a copy of the recommendations sent to microbiologists and community physicians.

D M JONES

Public Health Laboratory,
Withington Hospital,
Manchester M20 8LR

1 Cooke RPD, Riordan T, Jones DM, Painter MJ. Secondary cases of meningococcal infection among close family and household contacts in England and Wales, 1984-7. *Br Med J* 1989;298:555-8. (4 March.)

Calcium supplementation of the diet

SIR,—The clinical dangers of the recurrent letters from Drs Peter L Selby and Christopher J Robinson¹ are that some practitioners may conclude that they can use calcium supplementation alone as a preventive therapy for postmenopausal osteoporosis and thus might ignore the available effective treatments.

It is 30 years since the idea that calcium deficiency was implicated in osteoporosis was first forcefully advanced in the United Kingdom,² yet there are still no convincing data to show that increased dietary calcium intake, above and beyond that currently prevalent in the United Kingdom, has a significant effect on the prevention of osteoporosis. The review of Drs J A Kanis and R Passmore,³ which was extremely extensive, supports this view, and Drs Selby and Robinson do not cite any new reports to refute it.

As we emphasised in the correspondence after the publication of our study on dietary calcium intake and postmenopausal bone loss,⁴ there was no positive contribution of dietary calcium intake to skeletal mass, and their flawed interpretation of our data does not alter this conclusion.

They also appear to have failed to grasp the point that Drs Kanis and Passmore³ were obviously making from the balance studies of Malm⁵; this was to do with the accuracy of these studies but the trends they showed. Thus, after a change in dietary calcium, Malm's prisoners had not reached a new steady state of calcium balance even after a year or more of follow up. Furthermore, the negative calcium balance induced by a decrease in dietary intake became progressively less negative with time, and it may thus take many months, if not years, for a new steady state to be achieved.

Of course, the inaccuracies of traditional calcium balance studies are now irrelevant since they have been superseded by techniques which can accurately assess the density of the skeleton directly. Using such methods, it has been shown that bone density clearly reflects the incidence of fracture.^{6,7} Drs Selby and Robinson therefore cannot continue to ignore the various studies

which show no influence of dietary calcium intake on bone density in those anatomical sites which are clinically relevant to osteoporotic fracture—namely, the distal forearm, the vertebrae, and the proximal femur.^{8,9} Nor can they ignore the epidemiological data for the United Kingdom,¹⁰ which do not support a role for dietary calcium intake in the pathogenesis of postmenopausal osteoporosis. Indeed, we now have extensive data to support this conclusion.¹¹

I would not disagree that calcium is one of the dietary constituents necessary to build and maintain the skeleton. There are, however, no accurate data to show exactly what calcium intake is needed to optimise or maintain bone density at any age. Certainly for the adult population of the United Kingdom the current average dietary calcium intakes seem adequate.

I fail to understand how the critical analysis of the literature from Kanis and Passmore negates either the directives of consensus conferences or advances in the prevention and treatment of osteoporosis. Surely it is only likely to negate hypotheses no longer tenable and will act as a major stimulus for those with hypotheses to test to actually do the appropriate studies rather than accept such hypotheses as facts.

JOHN C STEVENSON

Cavendish Clinic,
London NW8 9SQ

- Selby PL, Robinson CJ. Calcium supplementation of the diet. *Br Med J* 1989;298:891-2. (1 April.)
- Nordin BEC. Osteomalacia, osteoporosis and calcium deficiency. *Clin Orthop* 1960;17:235-57.
- Kanis JA, Passmore R. Calcium supplementation of the diet. *Br Med J* 1989;298:137-40, 205-8.
- Stevenson JC, Whitehead MI, Padwick M, et al. Dietary intake of calcium and postmenopausal bone loss. *Br Med J* 1988;297:15-7.
- Malm OJ. Calcium requirement and adaptation in adult man. *Scand J Clin Lab Invest* 1958;10:36.
- Melton LJ, Wahner HW, Richelson LS, O'Fallon WM, Dunn WL, Riggs BL. Bone density specific fracture risk: a population based study of the relationship between osteoporosis and vertebral fractures. *J Nucl Med* 1985;26:24.
- Melton LJ, Wahner HW, Richelson LS, O'Fallon WM, Riggs BL. Osteoporosis and the risk of hip fracture. *Am J Epidemiol* 1986;124:254-61.
- Riis B, Thomsen K, Christiansen C. Does calcium supplementation prevent postmenopausal bone loss? *N Engl J Med* 1987;316:173-7.
- Riggs BL, Wahner HW, Melton LJ, Richelson LS, Judd HL, O'Fallon WM. Dietary calcium intake and rate of bone loss in women. *J Clin Invest* 1987;80:979-82.
- Cooper C, Barker DJP, Wickham C. Physical activity, muscle strength, and calcium intake in fracture of the proximal femur in Britain. *Br Med J* 1988;297:1443-6.
- Stevenson JC, Lees B, Devenport M, Cust MP, Ganger KF. Determinants of bone density in normal women: risk factors for future osteoporosis? *Br Med J* 1989;298:924-8. (8 April.)

NHS review

SIR,—We would like to point out that the government's policies as stated in *Working for Patients*¹ could have potentially disastrous implications for NHS hospitals in London. Furthermore, if a hospital were to become self governing this might worsen its position.

The financial problems of districts in London have arisen primarily because residents of these districts use acute inpatient services at a rate 50-100% above what their capitation based allocations would permit.^{2,3} The extra 3% for the higher costs in London proposed in the white paper will have a negligible impact on the scale of reductions required in acute inpatient care for London residents as London districts will receive capitation based allocations. The hospitals in London mostly treat patients from London, and thus implementing these allocations will mean fierce competition among these hospitals both for the reduced market within London and in their attempts to attract patients from outside.

The NHS hospitals will have to recoup high capital charges⁴ (approximate estimates suggest that these may equal their current running costs)

and meet the high costs of employing staff in London.⁵ Thus they are unlikely to be chosen by districts and general practitioners with practice budgets outside London as the best buy for services for their residents or patients. Similar reasoning may well be applied to the regional services currently based in London teaching hospitals: these could be far more cheaply provided elsewhere. The NHS hospitals would also be poorly placed to compete with private hospitals in London if the private hospitals can meet their capital charges from their private patients and price the services that they offer in competition with the NHS at marginal costs.

The effect of the government's proposed policies on London would be to put its districts in a position analogous to that of countries trading in a world recession, where protectionism is a common response. A London district might therefore seek to maximise care provided for its residents by its own hospital: by, for example, making contracts based on reductions in the core services to be provided for its residents by other hospitals. General practitioners and patients may, however, continue to use services as before, which will result in dispute over the standing of contractual obligations.

If a London hospital opted to be self governing it would then have greater freedom to pay its staff more and borrow capital.⁶ But it is sensible to introduce such competition for staff? Given the dramatic decline in school leavers, all NHS hospitals will face problems in recruitment. Allowing a few hospitals to pay more than standard rates may help them solve their problem, but only at the expense of others. In London, particularly, an inflationary pay spiral could easily follow, which could not be financed and would be likely, therefore, to result ultimately in hospital closures.

Although there are possible advantages for a London hospital in becoming self governing, these have to be set against the lack of a guaranteed market from a district's population. In addition, if it were a teaching hospital what would happen to its service increment for teaching (SIFT)? If this is allocated to the district where the medical school is located the district could conceivably negotiate teaching arrangements elsewhere. The only way for such a teaching hospital to survive through self government might be to realise its capital assets and move away from London. Would this long term solution gain the support of doctors with private practice in London? What impact would this upheaval have on training of medical students and research?

R G BEVAN

W W HOLLAND

N B MAYES

Department of Community Medicine,
United Medical and Dental Schools of
Guy's and St Thomas's Hospitals,
St Thomas's Campus,
London SE1 7EH

- Secretaries of State for Health, Wales, Northern Ireland, and Scotland. *Working for patients*. London: HMSO, 1989. (Cmnd 555.)
- Craig M. Estimating the resources required to train medical students and provide services: a survey of English teaching authorities. *Financial Accountability and Management* 1987;3:135-45.
- Bevan G, Brazier J. Financial incentives of subregional RAWP. *Br Med J* 1987;295:836-8.
- Department of Health. *Capital charges*. London: HMSO, 1989.
- Brazier J. Reviewing RAWP: accounting for cross-boundary flows. *Br Med J* 1987;295:898-900.
- Department of Health. *Self-governing hospitals*. London: HMSO, 1989.

SIR,—Many of your correspondents, like Dr Paul Hewish,¹ have an unfortunate tendency to confuse the government's proposals in the white paper *Working for Patients* with those in *General practice in the National Health Service: A New Contract*. This is presumably why you publish letters on both subjects under the general heading "NHS review."

Although there is some overlap between the two sets of proposals, they have very different implications. It is therefore essential that we respond to them separately. It is perfectly logical—and probably politically more effective—to oppose one but not the other.

The white paper is about fundamental changes to the NHS as a whole. It has no clear commitment to more money for NHS services—the only commitment on financing the NHS is that it will continue to be “financed *mainly* out of general taxation” (my italics). Although its stated objectives, such as more audit, are welcome, the core of its proposals relate to the imposition of an untested market oriented system on the NHS. Anyone who believes that such proposals are misconceived and likely to damage the interests of patients has every reason—a duty, even—to oppose the white paper. There is nothing blind about such opposition.

Most of the contract proposals are about the way general practitioners are paid and will affect patients much less directly. Some of them certainly need to be withdrawn or modified, including the unrealistic nationwide targets for immunisation and cervical screening and the obligation to carry out health promotion activities for which there is no clear evidence of effectiveness. However, let us demonstrate how reasonable we are by negotiating on the contract while opposing the white paper.

JOHN TEMPLE

Nottingham NG7 1QG

1 Hewish P. NHS review: no hope in blind opposition. *Br Med J* 1989;298:889-90. (1 April.)

SIR,—In view of the enormous increase in the information that we expect to receive from the information revolution proposed in the white paper, I thought I ought to let you know of the current state of affairs as regards information for plastic surgery.

A patient recently asked me where he could obtain the quickest plastic surgical appointment. My secretary rang the Department of Health, which told us that it did not have the information. My patient wrote to the department, which suggested that he should write to the various regional health authorities in England to give him a list.

I asked him to do so; the results are presented in the table. The information took my secretary, me, and my patient about three months to accumulate. I am now fearful that in future we will have to write to each individual region and hospital

Waiting lists for plastic surgery in regional and district health authorities in England

	Waiting time (weeks*)
South West Thames:	
Queen Mary's Hospital } Roehampton	2-5
St George's Hospital }	4
East Anglian†	
Mersey:	
Outpatient	21
Inpatient	69
West Midlands†:	
Dudley	6-24
North Warwickshire	9-10
Coventry and Warwickshire Hospital	5-20
North East Thames	> 16 months
South East Thames:	
King's College Hospital	0-33
Surgery	20
Guy's Hospital	0-5
Surgery	107
Salisbury	12
Surgery	2 years
North West Thames	27-5 months

*Unless stated otherwise.

†Doctor asked to write for information.

to find out not only their waiting lists but also the particular costs of each treatment that we require for our patients.

DAVID STEPHENS

London SE24 8SX

SIR,—Like Dr A Morgan and others¹ we are general practitioners in London who are enthusiasts for health promotion. We regard their enthusiasm for the new contract for general practitioners, however, as both naive and ill thought out.

The central drive of the contract—that is, a sharp increase in the element deriving from capitation—is thoroughly retrograde. It will force list size up and consultation time down. Single-handed colleagues will be driven to extensive non-NHS work to maintain their income, and incoming vacancies will become still rarer. The target figures for vaccination and cervical cytology are simply unobtainable in inner cities given the problems of list inflation, patient turnover, and ethnic minorities. Indeed, it seems to us quite extraordinary that anyone with a knowledge of modern urban practice would ever draft such proposals. The contract itself is wedded to the white paper's philosophy of competitive marketplace care, which is an inappropriate and inefficient method of health care provision. Inner city doctors like ourselves are likely to be especially penalised by the white paper's plans as hospitals opting out will probably leap frog over us and our patients to do business with megapractices holding budgets in the affluent south east. The community health services, which we find so valuable in health promotion, would be marginalised by the white paper's proposals.

The contract for general practitioners needs to be developed, and a few of the features in the proposed draft, such as an allowance for social deprivation, are appropriate.² But the philosophies of the present draft, its effect on general practitioners' income and terms of service, and the arrogant manner in which it is being imposed are unacceptable.

DAVID WIDGERY
FELICITY CHALLONER
ANNA LIVINGSTONE

London E14 8HQ

1 Morgan A, Bower P, Barnard S, Bird C. NHS review. *Br Med J* 1989;298:747. (18 March.)

2 Widgery DJ. *The national health*. London: Hogarth, 1988:78-81.

Enhanced transmission of HIV to women in South Africa

SIR,—The editorial by Professor D C G Skegg¹ focused on the transmission dynamics of HIV infection and rightly emphasised the importance of sexually transmitted diseases and genital ulceration in enhancing virus transmission and their role in accounting for the varying rates of transmission found in different heterosexual risk groups.

In the sexually transmitted diseases clinic attached to this hospital about 6000 black patients with genital ulceration are seen each year. A recent survey of 100 men and 100 women with genital ulceration showed the men to have had more sexual partners over the three month period before their attendance at the clinic, the men admitting to a total of 190 partners (range 0-7) and the women to 114 (range 0-3). Recurrent attenders at the clinic and patients with genital ulceration were offered HIV antibody testing with consent from July 1988 to January 1989. The overall antibody prevalence was 2.5% (62/2461), 2.1% of men (32/1524) and 3.2% of women (30/937) being seropositive, giving a ratio of men to women of 1:1.5.

Sexually active men and women are generally affected by HIV infection in about equal propor-

tions in sub-Saharan Africa,² although an excess of female patients with AIDS was found in Ghana in 1986 after repatriation of prostitutes from neighbouring countries. Our study, however, shows a higher prevalence of HIV antibody in women even though no prostitutes were identified. This may be accounted for by the higher transmission rate of the virus from men to women.

Prostitutes and patients with genital ulceration are recognised risk groups for both infection with and transmission of HIV, and our studies suggest that, just as female prostitutes represent a focal group for spread of HIV infection into the male community, so may HIV positive men with genital ulceration and many sexual partners represent a key group for spread into the female population in our area.

Health education, counselling, and providing condoms for patients with genital ulceration should now be regarded as a priority.

NIGEL O'FARRELL
ISOBEL WINDSOR

City Health Department Sexually
Transmitted Diseases Clinic and
Department of Virology,
King Edward VIII Hospital,
Congella, Durban 4013,
South Africa

1 Skegg DCG. Heterosexually acquired HIV infection. *Br Med J* 1989;298:401-2.

2 Anonymous. AIDS in sub-Saharan Africa. [Editorial.] *Lancet* 1988;i:1260-1.

3 Neequaye AR, Neequaye J, Mingle JA, Adjei DO. Preponderance of females with AIDS in Ghana. *Lancet* 1986;ii:978.

Iron deficiency in adolescents

SIR,—Iron deficiency in children has been the subject of much recent interest following reports of impaired intellect and behaviour in children with iron deficiency reversed by treatment with iron,¹ and Dr Paul Armstrong has now found a high prevalence of iron depletion among Irish adolescents.² Recent British studies have shown high levels of iron deficiency in young children in hospital or areas with a large proportion of Asian families.³⁻⁵ In a study aimed at defining the prevalence of iron deficiency in white children and exploring the effects of economic deprivation we found unexpected results.

We studied children aged 9-15 months attending baby clinics in two areas of Newcastle, one an affluent suburb, the other a deprived inner city area. Capillary blood (0.25 ml) was collected from the great toe into a bottle containing edetic acid, and haematological variables were measured by Coulter counter. A blood film was examined and haemoglobin electrophoresis performed. Lower limits of normal for haemoglobin, mean cell volume, and mean cell haemoglobin were defined as the third centile,⁶ and iron deficiency was defined as subnormal mean cell volume or mean cell haemoglobin with an otherwise normal blood film and electrophoresis.

Children from the two areas showed the same age distribution, but the “deprived” children were significantly more likely to have had low birth weight, a hospital admission, or an unemployed parent. There was no significant difference in the haematological findings between the two areas (table). The overall prevalence of anaemia (13.5%) was similar to that found in other studies,⁷⁻⁹ but cell variables suggestive of iron deficiency were seen in many fewer cases (3.5% overall). The correlation between iron deficiency and anaemia was poor; of the five children with subnormal mean cell volume and mean cell haemoglobin, three had a normal haemoglobin, and of the 19 with low haemoglobin only two had mean cell volumes or haemoglobin values suggestive of iron deficiency. Direct measures of iron stores were not available, but seven of the 19 children with apparent anaemia had mean cell values above the median, making iron