Dr Bennett offers little evidence to suggest that nursing homes are the "best buy" for our dependent elderly. Perhaps some of the public money being used to fund the expansion in private nursing home places might be better spent on geriatric wards or in helping people to remain in their own homes.

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1 Capewell AE, Primrose WR, MacIntyre C. Nursing dependency in registered nursing homes and long term care geriatric wards in Edinburgh. *Br Med J* 1986;**292**:1719-21.
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SIR,—We compliment Dr J Bennett (4 October, p 867) on bringing an important topic to general notice. However, there are points with which we would disagree.

The Purkiss rating for dependency is not known to us, and the reference suggests that it is not generally available for critical analysis. As such it is unsatisfactory to use such a scale, and a more generally accepted scale might have been that of Akhtar.[1] We would also like to know which mental test questionnaire was used to assess confusion, as subjective analysis can be notoriously misleading.

We would also draw different conclusions from the facts presented. Compared with nursing homes the long stay wards had a greater proportion of dependent patients (grades 3 and 4)—60% *v* 80%. The long stay wards were, however, deficient in qualified nursing staff and physical environment. Thus, before NHS facilities are further extended into the community or alternative care explored, the staffing and environment of NHS long stay wards should be made at least as good as those of private nursing homes, which are grossly subsidised by public money. The figure of 7·8 beds per 1000 population aged 65 years and over is not, to our knowlege, in general use. The DHSS has suggested to regional health authority chairmen that the recommended norm should be reduced from 10 beds to 8·5 beds per 1000 population aged 65 years and over. This is a topic which is hotly contested.[2]

It is indeed a laudable aim to promote meaningful dialogue between the NHS and private sector in terms of further education and continuing good relations. However, it would be dangerous to include private beds in overall NHS planning for the elderly in a health district. Private beds are not planned but driven, either up or down, by market forces. It is interesting that 18% of the nursing home patients were admitted from outwith the Brighton health district and thus they would be difficult to include in any planning. In addition, there appears to be no control over the suitability of admissions to private homes, other than the ability to pay. It is possible that some of the 36% of patients admitted directly to nursing homes from their own homes may have had rehabilitation potential.

Clearly the Brighton geriatric service is underbedded, as are many around the country. Equally clearly, the DHSS is paying considerable amounts of money for a service in the private sector over which it has no direct control.

Finally, although the situation in Brighton is interesting, it is not necessarily typical. This is exemplified by the numbers of nursing home residents who pay their own fees. This being the case, heavy reliance on private nursing homes is not a pattern of care which can, or indeed should, be pursued elsewhere.

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1 Akhtar AJ, Broe GA, Crombie A, McLean WMR, Andrews GR, Caird FI. Disability and dependence in the elderly at home. *Age Ageing* 1973;**2**:102-11.
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Platelet function defects in chronic alcoholism

SIR,—Some points deserve comment in the study by Dr D P Mikhailidis and colleagues (20 September, p 715) of reduced platelet aggregation and thromboxane A_2 production in chronic alcoholism.

Firstly, the control subjects differed from the study patients in more respects than just their alcohol intake: they were younger, they did not have liver disease, and they were not given drugs such as chlormethiazole or chlordiazepoxide during the study. All of these factors have effects on platelets and platelet activity[1-3] and might have influenced the results of the tests of platelet behaviour in this study. Attributing the difference in platelet behaviour between the patients and controls to the effects of chronic alcoholism alone is therefore not justified.

Secondly, inclusion in the study only of patients who had taken no medication in the two weeks before admission was presumably based on the patients' reporting of medication taken. In a group of chronic alcoholics admitted for alcohol withdrawal this must be suspect. Undeclared ingestion of aspirin in the week before admission cannot be discounted and might have accounted for the inhibition of platelet thromboxane production and aggregation seen in some of the patients on day 1 and the recovery of these platelet activities over the subsequent two weeks.

Thirdly, no mention is made of any changes in blood coagulation activity which may have resulted from the liver damage in the alcoholic patients. Platelet behaviour can be altered by changes in the concentrations of the vitamin K dependent coagulation factors,[4] and changes in platelet behaviour and in bleeding time in the alcoholic patients may have been secondary to such coagulation abnormalities.

These criticisms cast some doubts on the validity of the authors' conclusion that chronic alcohol ingestion in itself exerts an inhibitory effect on platelet function.

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AUTHORS' REPLY—Dr Jones states that our control subjects were younger than the patients; this difference was not significant ($p>0·09$; Mann-Whitney test). To eliminate any doubts we also matched (within three years) control subjects with patients; our conclusions remain the same.

Dr Jones's reference 3 deals with chlorpromazine and not chlordiazepoxide and chlormethiazole, which we used. This reference is even less relevant since it reports that chlorpromazine affected only serotonin induced aggregation and we did not use serotonin. Furthermore, (*a*) platelet function was abnormal on day 1, well before drugs were administered, (*b*) "several" patients received these drugs, implicit in this statement is that some did not. Obviously we carefully assessed the results of this latter group and they were similar to those of the other patients; (*c*) chlordiazepoxide does not influence platelet counts or bleeding time[1]; (*d*) our findings were comparable with those of others who managed patients differently.

Dr Jones thinks our findings could be ascribed to the undisclosed ingestion of aspirin or other drug. However, it is unlikely that most of the 27 patients were both taking drugs and hiding this fact from us. Moreover, the effect of aspirin on platelet function lasts a week or so whereas some of our patients required double that time for platelet function to return to normal; other non-steroidal anti-inflammatory drugs may even require less time for normalisation.[2] Surely Dr Jones does not believe that aspirin caused the thrombocytopenia and rebound thrombocytosis, which were also reported by others. Finally, does the significant correlation between alcohol consumption and bleeding time in 17 patients with two different types of liver histology reflect a random event related to secretive aspirin ingestion?

Clearly the only factor that correlated with the changes in platelet function was abstinence, since: (*a*) liver histology (ranging from relatively minor changes to cirrhosis) was largely irrelevant and was unlikely to have improved dramatically during two weeks' abstinence, (*b*) alcohol consumption correlated with bleeding time, and (*c*) we cited evidence that alcohol affects bone marrow function.

It is well known that liver disease affects coagulation factors and we are aware that these in turn can influence platelet function.[3] Our study, however, assessed platelet function and did not extend to coagulation factors. We felt that, within the constrains of space in the *BMJ*, we should comment only on work that we actually carried out ourselves.[4]

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1 Lackner H, Hunt VE. The effect of Librium on haemostasis. *Am J Med Sci* 1968;**256**:368-72.
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Boys with late descending testes

SIR,—In our short report (27 September, p 789) we noted that we "found a hydrocele either at birth or at 3 months in seven (39%) of the 18 late descenders who had a cryptorchid testis at 1 year, compared with five (19%) of the 27 who did not." This suggested to us that another source of "retractile" testes may be boys whose testes were descended at birth but who were noted by us

to have hydroceles at that time. We have now examined a group of such boys at 1 year of age.

Of the 17 boys re-examined, four have non-scrotal cryptorchid testes (as defined in our report) at 1 year, three bilateral, and one unilateral. The remaining 13 boys still have descended testes. There was no evidence of hydrocele in any of the 17 boys at 1 year of age.

Though a much smaller proportion of these boys than of our late descenders have "become cryptorchid," this group is clearly a population at risk needing careful follow up with a view to orchidopexy. These findings would appear to add further weight to the suggestion that cryptorchidism may be acquired after birth by the resorption of the processus vaginalis.

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Exposure of midwives to nitrous oxide

SIR,—It is unfortunate that the article by Dr A J Munley and colleagues (25 October, p 1063) is marred by obviously missing copy [see Correction p 1280]. What is clear, nevertheless, from their report is that levels of exposure of midwives to nitrous oxide in four hospitals without "appreciable active ventilation" in the delivery rooms resulted in concentrations which were not significantly less than 100 ppm in three of them, even with the use of "scavenging" in the worst.

Scavenging presents considerable technical problems with the on demand equipment which is used for maternal analgesia. In association with a senior chemical inspector of the Health and Safety Executive we have carried out a similar study in the first stage rooms of the University Hospital of Wales, the results of which also emphasise the importance of proper ventilation.[1] In this unit (built in 1972) similarly unacceptable levels were found but investigation showed that the room ventilation had been improperly maintained and adjusted. With six changes per hour, time weighted averages of less than 100 ppm could be achieved in all areas. We would recommend regular checks of performance to ensure that the ventilation system is achieving this. Theoretical calculations show that the National Institute for Occupational Safety and Health (US) standard of 30 ppm for nitrous oxide could be achieved only by room ventilation with 32 changes per hour, which is neither reasonably practicable nor comfortable.

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1 Atherton NJ, Slowey HF, Rosen M, Vickers MD. Nitrous oxide pollution in the labour ward. *Br J Anaesth* (in press).

Munchausen's syndrome by proxy

SIR,—Dr Laweh Amegavie and colleagues (4 October, p 855) report factitious bleeding in an infant whose mother appeared to have no formal psychiatric disorder. Women known to our department have used an ingenious variety of tactics to make their children ill.

A baby subject to recurrent drowsiness and faecal vomiting (but with a soft abdomen and normal stools) was being spoonfed a suspension of chlordiazepoxide and faeces in hospital from a bottle found in the locker by the child's bed. Another infant was admitted with a fluctuating level of consciousness, loss of coordination, marked irritability, and frequent jerks resembling infantile spasms but with an electroencephalogram showing moderate slow wave activity. Her mother, a woman with a personal history of Munchausen's syndrome, had been poisoning the child with phenytoin. A third child, unconscious and with pinpoint pupils, had emergency burr holes drilled before Distalgesic poisoning was thought of and the child woke up with naloxone. Less fortunate was the infant in a children's ward for investigation of blackouts induced, as it turned out, by the mother pinching her nose. One day this led to prolonged cardiac arrest and gross brain damage. Recurrent admission of a child with mysterious episodes of unconsciousness turned out to be the result of phenobarbitone poisoning (the mother had had epilepsy as a child).

The only boy in this group was admitted unconscious after being given sodium amytal by a cantankerous neighbour who found the noise of children at play irritating. Mostly mothers poison their daughters.

A close relative to this problem is the parent who provokes sickness behaviour in the child, refuses to accept psychological mechanisms, seeks multiple opinions, insists on repeated investigations, and ruins the child's life. However, whereas the former group are model parents in their attitudes to medical and nursing staff till they are found out, the latter operate in combative and contemptuous style. Maybe they do not respect doctors because they fool them.

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Origin of AIDS

SIR,—Dr I Wendler and colleagues (27 September, p 782) examined sera collected between 1976 and 1984 from 6015 Africans without symptoms of the acquired immune deficiency syndrome (AIDS). They found only four with confirmed antibody against human immunodeficiency virus (HIV) and concluded that the data do not support an African origin of AIDS. Unfortunately, they included only few samples from central Africa, which has the highest seropositivity rates at present and from where most cases of serologically proved AIDS in Africans have come to attention in Europe, from cases treated as early as 1977[1,2] until now.[3]

That most rural areas of sub-Saharan Africa have been free of HIV until recently is not an argument against an African origin of the spread of HIV. Biologically it would make sense if spread had started in a small rural area, which may never be identified. Epidemiological features of HIV are such that urban life lends itself best to further spread of HIV, most rural areas being infected only afterwards. The sampling available to Dr Wendler and colleagues is not representative enough to answer the question where HIV started to spread, and quite a different picture might have emerged if they had centred their investigation on central African cities.

Other arguments, mainly from Africa, against an African origin of the spread of HIV seem equally invalid. If Africa was the source why was the syndrome first identified in American homosexuals, and not in Africa? An emerging syndrome as polymorphous as HIV disease needs diagnostic facilities, which were present in the US and hardly at all in Africa, and the early recognition of the US cases was greatly facilitated because they were concentrated in limited groups at risk and not spread over the general population, as is now the case in Africa. The argument that cases of AIDS reported from Africa to the World Health Organisation are recent and few is even more deceptive. It is clear from all published material that there is tremendous underdiagnosis and underreporting in Africa. Until 1985 Belgium reported more AIDS cases in Africans, frequently fresh arrivals, than the whole of Africa (WHO Collaborating Centre on AIDS, AIDS surveillance in Europe, 1983-6).

We do agree with the authors that the issue has been obscured by early reports of high positivity rates on stored African sera which would be considered false positives by present means. As the authors illustrate themselves, it is now possible to distinguish between true and false positive stored African sera. The earliest human serum in the world which has been acceptably identified as being truly anti-HIV positive is a sample taken in 1959 in Kinshasa.[3] It remains puzzling why false positivity with some but not all anti-HIV assays is a greater problem with stored sera from Africa than with sera from Europe or America.

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Opportunistic screening for cervical cancer

SIR,—The BMA's document *Cervical Cancer and Screening in Great Britain* (11 October, p 970) reinforces much of what we all believe to be necessary to provide an effective screening programme. However, I do not agree that laboratories should be expected to communicate the results of a test direct to the woman herself, as this would encroach on the rights and responsibilities of the collector of the smear and the general practitioners of the women concerned. The proper channel for such communications is through the collector of the smear, who must accept that responsibility or not be in the business of performing such tests.

There is another aspect of a national screening programme—that of opportunistic screening—and here I write both as a pathologist and as chairman of the medical advisory committee of the Women's National Cancer Control Campaign. The campaign has also been screening through mobile clinics at the request of health authorities, businesses, and other organisations for over 20 years, in order to reach women who through lack of motivation or convenient facilities are not being screened through statutory clinics or by their own general practitioners. Such opportunistic screening on two accounts is being increasingly restricted.

Firstly, the laboratories are overstretched, and secondly, district health authorities, who are only just now achieving a call and recall facility, through family practitioner committees, do not wish to have their computerised programmes interfered with.

Both are reasonable points but these restrictions result in a loss of tests that are often performed at a cost far below that of health authority provision. Opportunistic screening does exist, through the campaign, through self developed industrial programmes, or through private practice. It would be folly to ignore such an additional gratuitous facility, which the family practitioner committees' computer lists could easily assimilate.

My proposal to overcome this problem is to suggest to district managers and consultant pathol-