the centres; many are seen in peripheral clinics. Similarly, there are disabilities other than neurological ones that require rehabilitation skills and other professionals—not all of them medical—with such skills.

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Why women are not receiving anti-Rh prophylaxis

SIR,—I noted with concern Dr P A Gover's claim that women are refusing to receive anti-D immunoglobulin because of the fear of the acquired immune deficiency syndrome (9 May, p 1232).

I have had a number of queries from obstetricians from various parts of Britain about this question, and I have pointed out to them that millions of ampoules of anti-D immunoglobulin have been administered throughout the world and, without doubt, in the past some of it has probably been obtained from virus positive material, but no case of a recipient developing antibodies to human immunodeficiency virus (HIV) has been reported to my knowledge. This is particularly so with regard to intramuscular immunoglobulin, the preparation used in Britain. Not only is all source material tested for antibodies to HIV but the processing of the plasma destroys any viral particles evading this net.

There is no reason, therefore, for mothers to refuse to have anti-D immunoglobulin injections prophylactically. If this fear does become widespread then Dr Gover is correct in stating that the number of unprotected mothers will increase and the encouraging fall in mortality and morbidity due to Rh haemolytic disease will cease, or even be reversed, which would be not only tragic but unnecessary.

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Mortality from myocardial infarction in different types of hospitals

SIR,—The observations of Dr Robert Reznik and colleagues in Australia (2 May, p 1121) are of great interest to clinicians and health care planners in Britain. Dr Reznik and coworkers, having stratified the coronary care facilities offered by 18 hospitals into four grades of refinement or complexity, found no difference in overall hospital mortality among patients with myocardial infarction in the four groups. The referral of high risk patients to the more specialised units did not account for the findings. This message is unlikely to surprise many practising doctors who realise that until recently the principal benefit of any coronary care organisation was the provision of immediate resuscitation from ventricular fibrillation.

The practice of coronary care has undoubtedly become over elaborate in some units, with little obvious benefit to the patient. Perhaps the most important practical advance over the past 20 years has been the development of the role of the nurse as a resuscitator who will provide defibrillation with the minimum of delay. It is therefore hardly surprising that the absence of a staff cardiologist or in house cardiac surgical services, coupled with only basic monitoring facilities, did not result in a higher inpatient mortality. What we really need to know is a great deal more about the nursing care that was provided.

Evidently, resuscitation was successful in the "small country district hospital." It seems unlikely

that the visiting doctor or general practitioner was well placed to be effective in this respect, and therefore the nurse was clearly the key factor. If patients were resuscitated by nurses the description of nurses as "comparatively inexperienced" requires elaboration. Indeed, without more data with regard to nursing numbers and experience it is impossible to relate this study to our own domestic practice. Nursing statistics are doubly important when attempting to interpret the study's comparison of mortality in general wards with that in units. Unfortunately, however, it would be extremely difficult to interpret this section, even with the extra data, as so much clearly depends on individual hospital admission policies.

Any manager in Britain who is tempted to look covetously at resources now "wasted" on the coronary care unit should reflect that with general medical wards often staffed by only one or two nurses the chances of observing, let alone correcting, cardiac arrest would be remote. Finally, although defibrillation, and to a lesser extent pacing, have been the mainstay of coronary care practice in recent times, the promise of thrombolysis is such that a policy of early admission to hospital and high quality nursing care is likely to be of increasing importance.

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Housing conditions and ill health

SIR,—Dr Claudia J Martin and colleagues (2 May, p 1125) do not seem to have considered the obvious confounding factor of unemployment in their analysis. This seems particularly surprising as they state that there were no adults (excluding pensioners) in paid employment in 45% of the households that they studied. Though the evidence that unemployment causes ill health is still sparse, it is clear that unemployment and illness are closely associated in those of working age and their close relatives.¹⁻⁵

If the authors did test the factor of unemployment but omitted to tell us then we can assume only that they fail to acknowledge its importance. By not testing this factor, however, they have jeopardised the value of their results. No doubt the irony is unintended, but they begin their abstract with the comment: "Lack of empirical evidence that living in damp houses has detrimental effects on health may partly be due to inadequate research." People who live in glass houses (damp or dry) should not throw stones.

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AUTHORS' REPLY.—As we indicated in our paper, it is obviously impossible to allow for all confounding factors in assessing the relation between damp housing and health. The role of unemployment, however, should perhaps have been considered more fully as it is quite clear that it is a major cause of ill health.

Certainly, we found that income was inversely related to Nottingham health profile scores—that is, the lowest income group had the worst perceived

health. There were, however, no significant differences in physical health between adults living in damp and non-damp houses. Moreover, there were no differences in the unemployment rates between these two groups. We did not, therefore, pursue the importance of unemployment in relation to health differences between adults living in different housing conditions; the effects of housing conditions on the health of adults in this sample would not have been influenced by unemployment, even though unemployment may have had a more global effect on their health.

As far as the health of children is concerned, we have now extended the analysis controlling for the presence of unemployment in the household. The original results (that is, of significant effects of dampness in relation to respiratory and gastrointestinal symptoms) remain unaltered. Dr Beale might, however, be interested to know that one symptom (diarrhoea) showed a significant main effect for unemployment, while two further symptoms (nerves and fever or high temperature) showed an interaction between dampness and unemployment, so that the highest rates were found in damp houses where there was also no one in employment.

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Internal market in the NHS

SIR,—Scrutator (16 May, p 1298) draws our attention to the vexed question of cross boundary flows and whether this aspect of funding health authorities should be liberalised to provide an internal market system within the National Health Service. Such a system would strengthen the funding of a good department balanced by the decline in a department in a neighbouring district with a lower workload. An internal market would bring real choice and consumerism into the NHS and is arguably the only thing that would make clinical budgeting a worthwhile exercise. I suspect that most consultants would be happy with clinical budgeting if the budget of a department were related to its workload and could thereby be increased by working harder.

An internal market would also take care of regional specialities and regional centres of excellence, which have no special recognition or regional funding. Top slicing would no longer be necessary as patients referred from one health authority to another for specialised treatment would "take with them" an amount of money appropriate to the treatment given.

This sounds like Utopia compared with our present bureaucratic tangle, and we might pause to ask why the NHS Management Board has found it unacceptable when we all thought that the board was introduced to revitalise the NHS and bring into it lessons from private industry. The reason is simple: the management objective is not really to do with efficiency or effectiveness but is fundamentally about control. Clearly, one objective is to obtain as high a level of service as possible for the money concerned, but the overriding objective of a health authority is to keep within its cash limit. Control is therefore the order of the day. Once doctors refer patients to neighbouring authorities and the bill has to be met by the health authority in which the patient lives control is lost. Similarly,

cross boundary flow funding for regional specialties would remove from the regional health authority the control over the volume of work that it has at present. Therefore, a proposal that is managerially sound and operates in the private sector and in health services overseas is politically unacceptable in Britain.

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Treating Paget's disease

SIR,—Dr D A Heath's article on the treatment of Paget's disease shows a worrying degree of complacency over this chronic condition (25 April, p 1048). The argument that most patients with this disease do not need to be treated is an old and cherished one, but we believe that Dr Heath has overstated it.

As we have gained experience with the use of intravenous aminohydroxypropylidene diphosphonate in the form of CGP 23339 (not yet commercially available),¹ which is clearly much safer and more effective than oral etidronate, our threshold for treatment has fallen progressively. We have now treated over 100 patients, and a striking feature is that many with rather vague bone pain have general ill health and malaise, which also shows sustained improvement after effective treatment. It seems possible that improved and fairly short treatment regimens with aminohydroxypropylidene diphosphonate will cure the disease without running the major risk of osteomalacia and bone fracture, which are seen with etidronate; the evidence so far is that the drug is extremely safe in both the short and the long term. If this is borne out by further experience the threshold for treatment will fall even lower. Treatment will become routine on diagnosis of Paget's disease, especially in younger patients, who obviously have a long time ahead for the disease to progress. With regard to many of the complications, it is obvious that cure will be the best means of prevention. Leaving it to the drug companies to set the pace for developing and applying new treatments for Paget's disease is in our opinion to shirk our responsibilities.

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1 Cantrill JA, Buckler HM, Anderson DC. Low dose intravenous 3-amino-1-hydroxypropylidene -1,1- bisphosphonate (APD) for the treatment of Paget's disease. *Ann Rheum Dis* 1986;45: 1012-8.

AUTHOR'S REPLY.—Professor Anderson's work on his first 20 cases treated with intravenous aminohydroxypropylidene diphosphonate was known to me when I wrote my review because he was good enough to let me have a preprint of his paper. The essence of his argument is that treatment with aminohydroxypropylidene diphosphonate is safer than oral etidronate and more effective and improves general ill health and malaise. He also seems to be suggesting that treatment may cure the disease.

In my review I discussed the safety of etidronate, and, when used at low doses, it does not seem to carry a risk of clinical osteomalacia or increased fracture rate. Aminohydroxypropylidene diphosphonate is undoubtedly more effective than etidronate in controlling the biochemical markers of the disease, but it remains to be shown that this carries any advantage over mere relief of pain. Informed judgment on the claim to improve ill health is impossible in the absence of proper randomised

controlled comparisons. Placebo responses are recorded in as many as 40% of patients with Paget's disease.

The results in the first 20 patients treated were submitted for publication in June 1986. Presumably the additional 80 patients have therefore been treated in the past 12 months. It seems unwise to talk in terms of cure with such limited follow up.

Whatever Professor Anderson and Mr Cantrill may say, despite the fact that for over eight years favourable reports of aminohydroxypropylidene diphosphonate have appeared, the drug is not available for general prescribing. Possible reasons for this were given in my review. The drug may be obtained on a named patient basis, but the widespread use of the drug in this way would be wrong as it would be bypassing the mechanism for regulating the use of new drugs. Named patient use should be reserved for patients who cannot be managed with existing treatment.

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Bone mineral density in Addison's disease

SIR,—Dr J P Devogelaer and colleagues claim to have produced evidence for an effect of adrenal androgens on bone mass (28 March, p 798). This is a subject that exercised the Leeds group for some time. Our description of reduced adrenal sex hormone concentrations in subjects with early or accelerated postmenopausal osteoporosis and the decline in these hormones in women at about age 60 led us to resurrect the idea of the adrenopause and propose that the postmenopausal woman with accelerated osteoporosis had undergone an early adrenopause.^{1,2} The idea of the adrenopause and that adrenal androgens are important in bone metabolism was, I believe, originally suggested by Albright and Reifenstein in 1948.³ As androgens serve as precursors for oestrogens, however, it has never been clear whether their effect, if any, is direct or indirect through oestrogen production.

The report by Dr Devogelaer and coworkers does not seem to resolve this issue. The rate of calcium loss and the postmenopausal oestrone concentration are known to be related,^{4,6} and the reduced oestrone concentration may therefore account for the lower bone mass in the postmenopausal patients with Addison's disease. The oestrone concentrations reported in their study seem, if anything, rather high.⁷

The preservation of bone in the men does not necessarily mean that the androgens are responsible. Men have an oestrogen concentration above that of postmenopausal women, and it has been suggested that this may contribute to the preservation of bone in aging men.⁴ Dr Devogelaer and colleagues do not report the hormone concentrations in the men with Addison's disease, but it is unlikely that the oestradiol concentrations would be much affected.

If we are looking for an effect of androgens, independent of any action through conversion to oestrogens, then we should look at androgen deficient, oestrogen replete subjects. The ideal model would be postmenopausal patients with Addison's disease receiving oestrogen replacement. The group in the paper by Dr Devogelaer and coworkers who come closest to this are the premenopausal women, who are oestrogen replete but have lost between a half and two thirds of their androstenedione and testosterone and practically all of their dehydroepiandrosterone. These patients, however, have not lost bone.

The relative balance between sex steroids and glucocorticosteroids may be important. Premeno-

pausal women seem to be fairly well protected against the effects of glucocorticoid treatment.⁸ It may well be that high glucocorticosteroid concentrations may be counteracted by high oestrogen concentrations. On the other hand, physiological replacement doses of glucocorticoids might do harm in the presence of extreme sex hormone deprivation—as in postmenopausal patients with Addison's disease.

The precise physiological role of the adrenal sex hormones in older life seems unclear. Why they should be secreted in response to adrenocorticotrophic hormone is also unclear, but it would make an attractive hypothesis to propose that androgen secretion, occurring hand in hand with the physiological secretion of glucocorticoids, protects some parts of the body, such as bone, from the catabolic effects of glucocorticoids. If so, however, whether the effect is direct or through the secondary oestrogen production remains unresolved.

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HIV testing and differential diagnosis

SIR,—Among the excellent points made in the ABC of AIDS, one recommendation sets my teeth on edge. This is the unqualified insistence that the diagnosis should be discussed with the patient before testing for human immunodeficiency virus (HIV).

In some contexts this is clearly excellent advice: in voluntary screening programmes, in genitourinary medicine clinics, when the patient is known to be in a high risk group, and when the patient expresses concern about the possibility. However, we can expect increasingly to see the condition presenting in general practice and in clinics of general and respiratory medicine, neurology, gastroenterology, and dermatology. The proportion of heterosexuals affected is likely to increase and patients may not be aware of exposure to the virus. Previously unsuspected groups may prove to be at risk. In any such individual patient HIV infection may be only one possibility, perhaps a remote one, in a long list of differential diagnoses, but it needs to be excluded or confirmed in the patient's interest and in the wider interest of following the progress of the epidemic.

Many of us would be deterred from ordering HIV antibody testing if we felt that standard practice entailed facing the patient with the possibility of AIDS in such circumstances. After all, we do not discuss the implications of leukaemia before ordering a white cell count or of bronchial carcinoma when ordering a chest radiograph. Even after receiving a positive or suspicious result we are likely to seek confirmation, either by repeating the