may have a direct action on the adrenal cortex, reducing responsiveness to adrenocorticotrophic hormone, there may be an additional factor in certain cases involving failure of the adrenocorticotrophic hormone release expected in response to falling serum cortisol concentrations.

It is interesting that in the work cited[2] patient mortality was reduced appreciably after administration of cortisol to those individuals with subnormal serum cortisol concentrations. The beneficial effect of such cortisol administration in the 11 cases mentioned was related to the initial prevailing plasma adrenocorticotrophic hormone concentration. Of the six patients in whom the adrenocorticotrophic hormone concentration rose as expected only one died compared with four deaths among the five patients in whom there was no rise.

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Ethical dilemmas of brain failure in the elderly

SIR,—The letters from Professor Elaine Murphy and others (7 January, p 61) fail to reflect the substance of either my own proposals or the published evidence in several important respects.

Firstly, although the basis of my proposals has little to do with numbers but more to do with the principle of individual rights to consultation, it is necessary to question the assertion that "the numbers are containable." Apart from the references cited in my original paper (10 December, p 1775), several others are equally difficult to ignore. A recent study of dementia concluded that "events are moving at a frightening pace and ... the 'quiet epidemic'[1] is no misnomer."[2] In 1980 Kramer referred to a "pandemic" of disease among the elderly.[3] Failure to perceive the problem may stem from ignorance of the extent of dementia in the community.[4,5] The 1983 conference of the Association of Health and Residential Care Officers was told by its president that the increasing numbers of people with dementia in old folk's homes were rapidly tipping the balance to a point where the quality of life of other residents was seriously affected. Professor Murphy's opinion flies in the face of the evidence.

Secondly, "effective planning" has been a catch phrase of medical, social, and political bodies for many years, but there is little evidence of either the will or the means to provide adequate facilities.

Finally, the suggestion that I am advocating a "solution" or euthanasia by adopting a policy of non-intervention in large numbers of the elderly is erroneous and pernicious. A two year mortality of 50% in dementia must mean that sizable numbers are, at any one time, in a near terminal state. The practice of defensive medicine is an effective means of deferring the need to define the reasonable limits of medical effort. In intensive care units arbitrary judgments of when to stop have been replaced, for logistic and humanitarian reasons, by the definition of "brain death." The elderly with advanced brain failure present an analogous set of circumstances, albeit with a different timescale, but with prognostic criteria that may be quantifiable.

The medical response is in some danger of being seen to arise from a threatened posture which denies that a problem exists, denies the patient the right of representation, and denies access by "the old man's friend" to those for whom it offers the kindest prospect. It would be a pity if these denials were construed, by the elderly in particular, as another example of professional paternalism or even arrogance.

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[1] Anonymous. Dementia—the quiet epidemic [Editorial]. *Br Med J* 1978;i:1-2.
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SIR,—I read the article by Dr George S Robinson just after a traumatic discussion with relatives of an elderly man who had been admitted 10 days before because of falls. Now recovered but partially sighted this man wished to return home to be with his wife, who has early senile dementia, on Christmas Day—their 65th wedding anniversary. The five daughters and two sons in law present at the discussion were adamantly opposed to his wishes and insisted that I should put their elderly parents into a home—a course of action that the father does not want. Moving into an institution can cause premature death,[1] but in their distressed love they believed that social euthanasia was right. Likewise, 15% of the relatives of patients dying at home discussed with the general practitioner the question of terminating the patient's life compared with only 3% of the patients.[2]

Johnson said to his biographer Boswell: "No Sir, let it alone. It matters not how a man dies but how he lives for the act of dying lasts so short a time." A bill of rights for the aged patient is certainly needed, but for those of us who spend our lives practising with the aged—and for whom the management of death is a commonplace event—such a bill would not give us the right to let the patient die but would give the patients the right to live their lives as they want to. Then, perhaps, hospital based home care would become the reality.

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[1] Millard PH. Depression in old age. *Br Med J* 1983;**287**:375-6.
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SIR,—If Dr Robertson feels aggrieved by being attacked for advocating euthanasia, which he clearly did not, I feel equally affronted by the summary of my paper[1] as proposing "to substitute money for care" (7 January, p 62). In fact, quite the reverse is the case. My objective has been to restore dignity and autonomy to the carers and their demented charges, to enable them to avoid being at the whim of the local authority and their local hospital service. Although, as Professor Murphy pointed out, there are excellent units caring for patients with dementia, there are also areas of the country where care provided by the statutory authorities is far from adequate.

The more flexible attitude now taken by officials of the Department of Health and Social Security means that those without savings are able to make good use of private care. However, the failure to maintain adequate inspection of these facilities, the failure to make objective measurements of incapacity, and the continued means testing of benefits has resulted in dementia remaining the illness that becomes a focus for family feud in those with some capital and an excessive drain on national resources when institutional care is unnecessary.

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[1] Kellet JM. A new approach to the elderly. *Br Med J* 1982;**285**:1588-9.

Standardisation of oral anticoagulant treatment

SIR,—Dr N K Shinton (8 October, p 1000) correctly points out that present recommendations for optimum prothrombin time ratios should be related to the type of thromboplastin and that the international normalised ratio should be given.

Recently, a batch of British comparative thromboplastin was established by the WHO's Expert Committee on Biological Standardisation as the second international reference preparation against which all future reference preparations have to be calibrated. This preparation (BCT/253, human plain) is to replace the first international reference preparation (67/40, human combined), which has an international sensitivity index of 1·0 by definition. The international sensitivity index for BCT/253 has been determined by an international collaborative study as 1·085 (SEM 0·013).[1] Another batch of British comparative thromboplastin (BCT/099) has been certified by the European Community Bureau of Reference under the auspices of the International Committee for Standardisation in Haematology and assigned an international sensitivity index of 1·048 (SEM 0·015).[2]

The Manchester comparative reagent, the routine counterpart of British comparative thromboplastin, used in over 90% of British hospitals, has been calibrated against the international reference preparation at the National (UK) Reference Laboratory for Anticoagulant Reagents and Control and assigned an international sensitivity index of 0·99. The Manchester comparative reagent is a liquid, phenolised extract whereas the British comparative thromboplastin reference thromboplastins are lyophilised preparations. The international sensitivity index of each successive batch of Manchester comparative reagent is always kept within 2½% of a target value of 0·99, and the interbatch variation of the international sensitivity indexes of 10

successive batches was found to be 1·36% (CV). British comparative thromboplastin is monitored externally using a different procedure, which allows for a slightly greater variation between successive batches.[3] The international sensitivity indexes of successive batches are, however, extremely unlikely to differ by more than 5% and the differences between the two British comparative thromboplastin reference preparations (BCT/099 and BCT/253) are well within the published acceptance limits for external monitoring.[3]

International normalised ratios are provided with all batches of British comparative thromboplastin and Manchester comparative reagent and should be used in reporting results of prothrombin time whenever clinical trials are performed, and if a patient is to travel abroad. In Britain the situation is simplified by the fact that the international normalised ratio and British ratios obtained with the routine reagent Manchester comparative reagent are interchangeable within the established therapeutic range of 2·0 to 4·0.

Some confusion has arisen in published reports concerning the additional term British corrected ratio. The British corrected ratio is obtained by calibrating other thromboplastins —that is, national reference preparations, commercial reagents, and home made human brain extracts—against British comparative thromboplastin using the original calibration method.[4] This procedure is no longer necessary in hospitals in Britain as the use of Quick test reagents other than Manchester comparative reagent is now insignificant. For manufacturers' products and national reference preparations the newly established international normalised ratio terminology should be introduced to provide a uniform method of reporting. In view of the cumulative, clinical experience in anticoagulant dosage with British ratios it may be helpful, however, to use the terms British ratio and international normalised ratio concomitantly until the new system is fully implemented and becomes familiar to clinicians world wide.

Finally, Dr Shinton's example of a prothrombin time ratio of 3·1 with rabbit thromboplastin being equivalent to a ratio of 5·0 with British comparative thromboplastin pertains to a particular rabbit thromboplastin (RBT/79), certified by and available at the European Community Bureau of Reference[2] and also established by WHO as one of the international reference thromboplastins.[5] The table compares RBT/79 with another rabbit thromboplastin and shows that accurate calibration of the preparations is required when relating ratios with international normalised ratios. Other commercial rabbit thromboplastins give different international sensitivity index values.

Equivalent ratios for various rabbit thromboplastins in oral anticoagulant control

International normalised ratio	Rabbit thromboplastins	
	RBT/79*	Simplastin (lot No 4H612)†
2·0	1·6	1·3
3·0	2·2	1·6
4·0	2·7	1·8
5·0	3·1	2·0

*Certified reference material available from Community Bureau of Reference (Brussels), with an international sensitivity index of 1·413 (SEM 0·036), established by WHO in 1982 as the international reference thromboplastin, rabbit, plain, with an international sensitivity index of 1·4 (rounded off figure of 1·413).
†Commercial thromboplastin (rabbit lung, brain) calibrated by Dutch Reference Laboratory for Anticoagulant Control, with an international sensitivity index of 2·4.

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5 WHO Expert Committee on Biological Standardization. Thirty third report. *WHO Tech Rep Ser* 1983:687.

Infection with netilmicin resistant Serratia marcescens

SIR,—We welcome the distinction made in the report by Dr D A Lewis and others (3 December, p 1701) and in your leading article (p 1651) between outbreaks of *Serratia marcescens* due to a common source and cross infection via hands.

The role of contaminated hands in the transmission of *Klebsiella* sp is well established[1 2] and it seems increasingly clear that the epidemiology of *Serratia* is similar. We have recently investigated Dr Lewis's strain of netilmicin resistant *S marcescens* and outbreak strains of *Kl aerogenes* with a view to finding an explanation for the epidemiological behaviour of these organisms, which may well differ from that of other opportunistic Gram negative bacilli.

Using a technique that mimics the contamination of staff hands and is described fully elsewhere,[3] we found that Dr Lewis's strain of *S marcescens* and multiply resistant outbreak strains of *K aerogenes* survive considerably longer on artificially contaminated fingertips than *Pseudomonas aeruginosa*, *Escherichia coli*, or *Enterobacter cloacae* (table). This ability to survive on skin may well be a major determinant for the epidemic spread of *Serratia* and *Klebsiella* spp.

In serratia outbreaks it is clearly important to exclude a common source or failures in standard hygienic practice. In their absence, Dr Lewis's finding of the outbreak strain on staff hands and our finding of its enhanced survival on fingers provide a rational explanation for the importance of hand disinfection in the successful control of this and similar serratia outbreaks.

Survival of netilmicin resistant Serratia marcescens compared with other Gram negative bacilli after inoculating fingers with approximately 10^4 bacteria

Time after inoculation (min)	Log $_{10}$ reduction recoverable colony forming units/finger of:				
	S marcescens	*Klebsiella* K21	*Ps aeruginosa* NCTC 10701	*E coli* NCTC 10418	*Enterobacter cloacae*
10	2·10	1·89	2·96	2·70	3·82
60	2·85	2·40	4·00	4·00	4·00

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Impact of audit on preventive measures

SIR,—Dr D M Fleming and Dr M S T A Lawrence (17 December, p 1852) have produced an interesting report of the impact that audit of medical records has on preventive measures in general practice. They mention especially improvements in the recording of blood pressure and rubella immunity. Both their original paper[1] and this later report, however, contain a statement to the effect that rubella immunity was based on a record of the presence of rubella antibodies or of rubella immunisation.

I would suggest that this is an inaccurate assumption: a record of a rubella immunisation procedure is not the same thing as a record of the detection of rubella antibodies. Although this difference does not matter as far as the research into the effect of audit is concerned, it does matter clinically.

Seroconversion rates after rubella immunisation are high for both RA 27/3 and Cendehill vaccines, though the RA 27/3 vaccine is shown to be superior in many studies. Freestone analysed the pooled results of 21 clinical trials of both vaccines and found a seroconversion rate of 98·6% for RA 27/3 and 96·2% for Cendehill.[2] Recently a study compared the vaccines when given to women post partum and found seroconversion figures of 97·6% for RA 27/3 and 82·2% for Cendehill.[3]

A general practice study that tested 883 women using the single radial haemolysis technique, found two seronegative patients whose records carried clear evidence of Cendehill vaccination five years previously.[4] They represented 6·1% of the seronegative patients in their 15-24 years age range and later became positive after receiving RA 27/3.

Even if some seronegative but vaccinated women do in fact have undetectably small though nevertheless protective amounts of antibodies, there would still be some who, for technical reasons—for example, bad storage of vaccine—would remain susceptible.

The danger of equating rubella immunisation with an immune state lies in the fact that a