practice, who generate a high income, over health authority patients.

Sophisticated bureaucratic props for the internal market such as those proposed by Clarke and McKee could spell yet another expensive diversion in the development of NHS information systems and bode ill for the health of the population. Moreover, since the NHS budget is cash limited, funds for improving purchaser and provider information systems would have to come from the resources allocated to patient care. In the United States, where spending on information systems and data analysis is high, administrative costs can be as high as 40% of provider budgets.[3] Nevertheless, this has not resulted in cost containment or improved health care.[4,5] The real questions that we have yet to address in our information systems are whether the internal market will preserve equity in health care and safeguard against implicit clinical rationing and queue jumping.

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EDITOR,—Aileen Clarke and Martin McKee do not emphasise the inadequacy of the consultant episode as a measure of efficiency although it is used for this purpose in corporate contracts.[1]

Consider a typical, but hypothetical, purchasing dilemma for a district health authority that has set aside £120 000 for treating infertility and is trying to choose between therapeutic options A and B with different costs and effectiveness (table). In terms of clinical outcome treatment A is the most efficient use of resources because it leads to 40 live births as opposed to 30 from treatment B. But the health authority, constrained by an obligation in its corporate contract to increase consultant episodes, will be tempted to opt for treatment B. Counting the clicks of a turnstile is not a good way of measuring efficiency in the NHS.

Variables that may influence district's choice of treatment for infertility

	Treatment A	Treatment B
Cost per case (£)	1000	800
Live birth rate (£)	33	20
Cost per live birth (£)	3000	4000
Live births per budget	40	30
Consultant episodes per budget	120	150

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EDITOR,—Aileen Clarke and Martin McKee's editorial on the consultant episode well exemplifies the Kafkaesque world that the NHS has entered since the invention of the internal market.[1] Concern over the possibility that opportunistic providers might discharge and then readmit patients simply to clock up another treatment episode has led to proposals of several solutions. These include greater analysis of multiple admissions, employing a panel of clinicians to advise on what should properly be regarded as a single episode, and devising more sophisticated (and more expensive) information systems.

The available resources for health care are finite, and the more money we spend on collecting and analysing data the less we will have to purchase health care. I suspect that the treatment being offered to control "opportunism" will cost more than the disease itself.

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Confounding in epidemiological studies

EDITOR,—Contrary to our view,[1] Alfredo Morabia asserts that "it is unlikely that many associations identified in epidemiological studies are due to confounding."[2] He supports this with the observation that "examples of suspected causal associations that have later been entirely attributed to a hidden confounding variable are rare." This is true, but Morabia should consider the size of the denominator as well as the numerator. How many diseases are there for which the aetiological mechanisms have been sufficiently elucidated to enable retrospective judgment of the ability of previous epidemiological studies to identify the true cause or causes? Very few. AIDS is an exception in fulfilling this criterion. A case-control study reported in 1982 that the "independent" relative risk for the use of amyl nitrite "poppers" was 12·3.[3] The corresponding value for the lifetime number of sexual partners was 2·0. Only after the identification of the probable hidden confounding variable—infection with HIV—in 1983 was the theory that poppers may cause AIDS generally abandoned.

In the absence of the ability further to estimate directly the proportion of epidemiological associations that have been identified that are truly causal, we should view the process in reverse. Instead of using empirical evidence to make inferences about whether an association may be causal we begin with the knowledge that one or more exposures are contributory causes of a disease and consider what empirical evidence might be generated.

Consider a disease for which there are a limited number of exposures that contribute to its occurrence. Virtually all such causal exposures will be correlated to some degree with many other exposures. For example, the amount of any given dietary factor consumed is correlated with the amount of most of the hundreds of other dietary components consumed. Given the small number of truly causal exposures relative to all possible exposures, when epidemiologists study the disease it is more likely that correlates of the causal exposures will be included than that the causal exposures themselves will be. This is what happened in the studies that, in the absence of knowledge about HIV, identified poppers, cytomegalovirus, and antibodies to rectally introduced semen (among others) as potential causes of AIDS. If the causal exposures are not included, or even if they are included but measured imprecisely, exposures that are most strongly correlated with the causal factors will emerge as independent risk factors for the disease.[4-7] This suggests that many epidemiological associations are likely to be due to confounding.[8]

Morabia asserts that confounding bias is unlikely to be sufficiently large to explain the observed association when the association between exposure and disease is strong. As the example of poppers and AIDS illustrates, this view is inappropriately complacent. We and others have shown—without any need to invoke extreme or unlikely circumstances—that strong independent associations can arise solely as a result of a lack of control over confounding.[4-7 9]

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Letting vegetative patients die

EDITOR,—The decision to stop feeding patients in a permanent vegetative state is scientifically correct and difficult to reject from an ethical point of view.[1] But the value of feeding and hydrating a patient, even one in a persistent vegetative state, is probably more emotional than medical for doctors and relatives as it is the only way they have of feeling that the patient is still alive and that they are helping him or her.

In a limited poll[2] of Italian neurologists 39% of the doctors who declared themselves to be in favour of euthanasia for patients in a persistent vegetative state (78% of the participants) were nevertheless against withdrawal of feeding, and 10% were uncertain. Considering that most of these neurologists came from universities or research centres, these results are unlikely to have been due to poor knowledge of this issue or of the positions taken by the American Academy of Neurology[3] and the Institute of Medical Ethics.[4]

Should we consider this attitude scientifically incorrect, professionally wrong, or simply irrational? All of these, of course. But doctors are human, and what is scientifically or ethically correct is not necessarily acceptable emotionally.

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EDITOR,—Bryan Jennett quotes a single Catholic theologian as saying "that to persist with tube feeding can convey stupidity and cruelty, not

compassion and love."[1] The same also appears in the BMA's discussion paper on the withdrawing of tube feeding and is incorrectly stated as representing the Roman Catholic view.

To elicit an authentic Catholic teaching, however, it is best to go to a proper Catholic source such as the Vatican Statement of 1980: "Euthanasia includes not only active mercy killing but also the omission of treatments when the purpose of the omission is to kill the patient"[2] or that of 50 American bishops early this year: "The harsh reality is that some who propose withdrawal of nutrition and hydration from certain patients do directly intend to bring about the patient's death ... nutrition and hydration (whether orally administered or medically assisted) are sometimes withdrawn not because a patient is dying but precisely because the patient is not dying (or not dying quickly enough) and someone believes the patient is perceived as having an unacceptably low 'quality of life,' or is imposing burden on others."[3]

Bishop Christopher Budd on behalf of the English and Welsh bishops, in the statement *Euthanasia and Hard Cases*, stated "A PVS patient is not dead in any normal or technical sense of the word. In spite of the expression 'permanent vegetative state' Tony Bland is still a live human being, he is not a vegetable, his soul has not left him.—Nor in fact is he dying. Accordingly he ought not to be killed by being starved to death any more than he should be killed by lethal injection. The quest for 'death with dignity' does not justify the deliberate taking of a human life, no matter how good the intention or how great the strain of waiting."[4]

I hope the above clarifies the Roman Catholic position. The Linacre Centre established by the English and Welsh metropolitan bishops for the study of health care ethics in the Hospital of St John and St Elizabeth, London, is always available for consultation.

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EDITOR,—One of the outcomes highlighted by the recent ruling on Tony Bland (Hillsborough Stadium disaster) is the importance of providing good quality rehabilitation and the need for continuing care for patients in the persistent vegetative state.[1,2] This is also emphasised in the recommended guidelines published by the ethics committee of the British Medical Association.[3]

Although the Royal Hospital and Home, Putney, provides an excellent service for patients in the persistent vegetative state,[4] rehabilitation, especially in the long term and including placements, should be nearer the family home and hence available at district level. If, as recommended by the Royal College of Physicians,[5] all districts have a comprehensive medical rehabilitation service it should include rehabilitation and support for people in the persistent vegetative state. It is important for district (or supradistrict) rehabilitation units to have access to, or admitting rights to, health biased continuing care or residential units.

Most disabled people should be placed in the community, but it must be recognised that for many the demand on the NHS is such that they may need to continue staying in a health biased continuing care unit. Generally for these clients it would also be more cost effective. Patients in the persistent vegetative state generally fall into this category.

Many medical rehabilitation units have selection criteria that discriminate against people with a poor prognosis—again, a group that includes patients in the persistent vegetative state. Support from and access to continuing care units as we have in Hillingdon guards against fears of long term bed blockage in the acute rehabilitation unit and thus helps to provide a complete rehabilitation service to support the acute unit of the district general hospital.

The incidence of one or two patients in the persistent vegetative state per 100 000 of population is probably an underestimate. In Hillingdon (population 250 000) we have six patients. The figure is thought to be relatively high (and possibly more accurate) because the availability of the service is known locally. Besides preventing blockage of acute beds by patients in the persistent vegetative state lying in medical and surgical units with inadequate and inappropriate attention, the continuing care unit also helps concentrate appropriate expertise in the care of these unfortunate people.

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Hyponatraemia associated with paroxetine

EDITOR,—Cait Goddard and Carol Paton reported on a patient with hyponatraemia associated with paroxetine therapy.[1] We describe two patients with a similar complication after treatment with paroxetine.

A 78 year old woman was given paroxetine for symptoms of anxiety and depression. Five days later she was referred as an emergency with confusion. She was taking lisinopril 5 mg daily, which had been started three weeks previously for hypertension. Examination did not reveal any abnormal physical signs. Blood pressure was 160/90 mm Hg with no postural change. Investigations showed plasma concentrations of sodium of 115 mmol/l, potassium 4·5 mmol/l, urea 5·2 mmol/l, and creatinine 70 μmol/l. Random blood glucose and serum calcium concentrations and chest radiographs were normal. Plasma and urinary osmolality were 256 (normal range 285-295) and 369 (inappropriately concentrated) mmol/kg respectively. Her fluid intake was restricted and paroxetine was discontinued. Lisinopril was stopped on day 2 in case of possible interaction. From day 4 onwards her plasma sodium concentration began to normalise (115, 109, 113, 120, 128, 130, 136 mmol/l on consecutive days).

The second patient was a 76 year old depressed man admitted to a psychiatric ward with nitrazepam overdose of 50 mg. Physical examination was normal. Plasma sodium concentration was 130 mmol/l and a chest radiograph was normal. He was given paroxetine the following day. A week later he became progressively confused. Repeat plasma sodium concentration was 112 mmol/l. Plasma and urine osmolality were 246 and 292 mmol/kg respectively. Chest radiography also confirmed concomitant bronchopneumonia. He was given 200 ml 2M saline slowly and paroxetine was stopped. His plasma sodium concentration improved gradually (115, 127, 133, and 141 mmol/l on consecutive days). Despite intravenous antibiotics his bronchopneumonia persisted and he died on the sixth day on the medical ward.

The mechanism of hyponatraemia could possibly be due to the syndrome of inappropriate secretion of antidiuretic hormone as suggested by the osmolality results. Cases of hyponatraemia due to fluoxetine, another selective serotonin reuptake inhibitor, have been reported[2,3] and are thought to be due to inappropriate secretion of antidiuretic hormone. Our first patient was also taking lisinopril but hyponatraemia associated with this is uncommon.[4] Bronchopneumonia cannot be categorically ruled out as the cause of hyponatraemia in the second patient, but his plasma sodium concentration normalised after paroxetine was stopped despite persisting bronchopneumonia, suggesting the former as the cause.

The cases lend support to Goddard and Paton's views that care should be taken when paroxetine and other newer antidepressants are prescribed.

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Screening for neuroblastoma

EDITOR,—Parker and colleagues cannot recommend "general implementation" of screening for childhood neuroblastoma.[1] But does the question "require further study"? Their screening test is of superb sensitivity and specificity compared to those of other programmes. Even this, meticulously applied to a huge cohort of children, gives no hard evidence that a single life was saved. Cases were missed, and as with mammography more benign subtypes seem preferentially detected.[2]

The paper omits any estimate of cost-benefit of a nationwide screening programme. The authors are not alone in this oversight. The latest of the *BMJ* editorials on rationing again fails to include population based screening on the agenda.[3] Governments are eager to promote these high profile programmes, but the cash comes from the same budget as prosthetic hips, care in the community, and erythropoietin.

We "ration" treatment known to restore quality life. Yet we accept the cost for a year of life gained from cholesterol screening of at least £110 000,[4] and the cost of a life saved from cervical smear screening of up to £300 000.[5] We ignore the social, psychological,[6] and administrative costs of investigating thousands of people who gain no personal benefit.

Why mount screening trials on huge numbers of subjects which may just scrape into statistical significance? Outside of the clinical trial quality control falls. False positives, false negatives, and recall rates rise. Registers do not include high risk subjects.[7] Bored practitioners search for needles in the haystack of pathology forms. Clients and doctors alike see aggressive "interval" tumours unabated. Morale and counselling atrophy. The client understands only that "It's a test for cancer" and stays away.

Slender $p<0·05$ values disappear in the real world. The price of the quixotic gesture is less money for existing treatment or for research towards better treatment.[8]

A recent letter in these columns points out the ethical gulf between treating someone who presents feeling unwell and someone who feels healthy and whose custom you solicit.[9] The final hidden cost of