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 nutrition and hydration (whether orally administered or medically assisted) are sometimes withdrawn not because a patient is dying but precisely because the patient is not dying (or not dying quickly enough) and someone believes the patient is perceived as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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- 3 US Bishops Pro Life Committee. Nutrition and hydration; moral and pastoral reflections. *Origins* 1992 Jan 30;705-12.
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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 reported² 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.³ 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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- 1 Goddard C, Paton C. Hyponatraemia associated with paroxetine. *BMJ* 1992;305:1332. (28 November.)
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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

screening is the worried well,⁶ those who think "My doctor thinks I can be seriously ill when I feel perfectly fit. How can I accept her reassurance that a complaint does not need high technology work up and specialist referral?"

We must not abandon stringent criteria¹⁰ for implementing screening programmes again.

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Lung cancer and breast cancer in women

EDITOR,—It isn't just in Glasgow that the incidence of lung cancer is overtaking that of breast cancer in women.¹ Looking at a measure that is cruder than, but perhaps as important as, incidence—that is, numbers of deaths—shows that lung cancer now kills more women than breast cancer does in many parts of England and Wales.²

Mortality figures for 1990 show that lung cancer is a bigger killer of women than breast cancer in the Northern region and in major cities such as Liverpool (198 deaths from lung cancer, 130 deaths from breast cancer); Leeds (169, 160); Manchester (129, 109); Newcastle (112, 54); and Nottingham (70, 61). The pattern is not, however, confined to the industrial cities of the north: it is also seen in such places as Westminster (53, 49); Kensington and Chelsea (40, 32); Southwark (63, 38); and Watford (15, 13).

Any death from cancer is a tragedy, but deaths from lung cancer are doubly so as the disease is almost entirely preventable. Those concerned for women's health, in national and local government and the health service, must increase their efforts to reduce cigarette smoking and its unnecessary death toll. It would be a start if the health minister dropped her objections to the proposed European ban on tobacco advertising.

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Botulinum toxin for hemifacial spasm

EDITOR,—A J Lees describes the useful role of injection of botulinum toxin in treating various types of dystonia.¹ The editorial mentions hemifacial spasm but does not make clear that this is not a dystonia. There is increasing evidence that

hemifacial spasm is a hyperactive cranial nerve syndrome, along with trigeminal neuralgia, glossopharyngeal neuralgia, and, possibly, some cases of vertigo.² The aetiology of these conditions is generally agreed to be compression of a cranial nerve within the posterior fossa by an aberrant or ectatic cerebral blood vessel. The site of this vascular contact is generally the exit zones of the fifth or seventh nerve and can now be imaged with magnetic resonance imaging.³ This chronic pulsatile compression is thought to create local demyelination with consequent ephaptic transmission, which results in paroxysms of hyperactivity causing pain or spasms.^{4,5}

Microvascular decompression entails displacing and translocating the offending blood vessel via a small retromastoid exposure. The blood vessel is maintained in a new position with small Teflon slings. Intraoperative electromyography has shown that hemifacial spasm stops as the offending vessel is retracted and resumes if the vessel is allowed to spring back into position.⁶ Microvascular decompression results in a long term cure rate of 92-98% in hemifacial spasm (over 10 year follow up) and of over 80% in trigeminal neuralgia.^{7,8} The operative approach has now been miniaturised so as to be minimally invasive, with consequent rapid postoperative recovery and discharge from hospital on the fourth day on average. In the past surgical morbidity has been a concern, but this is negligible when the procedure is done by an experienced surgeon.

Botulinum toxin produces relative paralysis of facial muscles, with recovery over months and consequent recurrence of spasms. Increasing numbers of patients are disenchanted with the lifelong prospect of repeated treatment, and for them microsurgery now offers a safe and permanent cure.

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Doctors' retainer scheme

EDITOR,—The doctors' retainer scheme¹ currently favours only women working very part time and could usefully be expanded to include all doctors who have done their training in general practice but have not gained a permanent post. All general practitioners spend some time in this situation, and some (male and female, for various personal reasons) choose to work as locums or assistants for several years before settling in their final practice. All suffer from a lack of financial and educational support during this time. Without continuing education the benefits of vocational training can be lost, so the cost of administering an expanded doctors' retainer scheme would be amply repaid.

Nicola Toynton is wrong to state that only assistants and doctors on the retainer scheme must

pay their own defence fees.¹ Locum general practitioners are a much larger group who must do this, and a locum's gross pay is severely reduced by other expenses too, including education.

Toynton's idea of a free pass for educational activities could usefully apply to locums. Like assistants and doctors on the retainer scheme they get no postgraduate education allowance. They must lose a week's pay if they go on a course, as well as having to pay in full the expensive course fees. It is scandalous that the new reforms have created financial penalties that discourage the education of enthusiastic general practitioners who are keen to acquire new ideas. Instead, the allowance is restricted to principals, including some who are old, uninterested in change, and about to retire. This wastes a lot of teaching effort and NHS money. A better scheme would be to make the postgraduate education allowance available to all doctors who work regularly in general practice in the NHS. The benefits would be noticed by everyone.

In the current system those at the start of their careers (who have the most to benefit and the most to contribute) get the worst education. This could so easily be changed with an expanded doctors' retainer scheme.

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- 1 Toynton N. Doctors' retainer scheme. *BMJ* 1992;305:1378-9. (5 December.)

EDITOR,—Doctors on the retainer scheme are the menials of general practice, and Nicola Toynton rightly associates this with the poor uptake of places on the scheme.¹ Doctors with family commitments who wish to keep in touch with work gain little support from the scheme, which offers inadequate finance and no appropriate educational input for their special needs. They are often not kept in touch with practice affairs and lack the benefit of following up patients—both things taken for granted by their colleagues. All they can be sure of is a constant parade of other people's patients to be squeezed in before the next family deadline.

From an educational perspective, these doctors are clearly in a training rather than a service post. A quota of postgraduate sessions is useless when the need is for personalised training in the practice. My practice is one of an apparent minority in which the retained doctor has an informal educational supervisor (the trainer in the partnership), who tries to meet at least some of her training needs. A short weekly session, during school hours or with the retained doctor's children present in the surgery, can help considerably by allowing feedback, discussion of cases, and the provision of career information.

General practitioners traditionally give scant attention to their own career development, yet some simple education measures in the practice can greatly improve the lot of the disadvantaged doctors on the retainer scheme.

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EDITOR,—Nicola Toynton draws attention to the doctors' retainer scheme and makes suggestions for its improvement.¹ In the North West Thames region these suggestions have already been implemented. The regional postgraduate dean has delegated the overall administration of the scheme to the regional adviser in general practice, and at local level the scheme is supervised by general practice tutors rather than clinical tutors. It is the