Dying for palliative care

EDITOR,—It seems appropriate that articles communicating poor recognition of the palliative care philosophy should have space “afforded” to them.¹ They emphasise the crisis in caring for dying people despite the creation of a specialty and reiterate results of other studies showing that patients are still dying unprepared and suffering.² The reason for this is twofold.

Firstly, there is still a reluctance to inform patients with cancer of their diagnosis and, through a process of informed consent, to negotiate the appropriateness of active treatment.³ Greater emphasis should be placed on distinguishing between cure and care. Furthermore, cure should not be perceived as the only successful outcome of treatment, with anything less as failure.

Secondly, a deficit in training exists, which is exacerbated by an inappropriate reliance on specialists in palliative medicine. Most patients do not die in hospices, and so the philosophy of care is most frequently practised by inadequately prepared generalists in the community and hospitals.

Dying remains a taboo subject, and the recent articles and correspondence convey a message: where is palliation, dignity, and death as a normal part of the life cycle? In addition, why do some doctors avoid admitting that a patient is dying and why are they reluctant to provide sufficient symptom control?

Training in palliative care, for both undergraduates and postgraduates, must be urgently addressed to prevent continuing suffering of terminally ill patients who are not in hospices. As the head of the Cancer and Palliative Care Unit at the World Health Organisation's headquarters, Dr J Stjernsward, stated, “Nothing would have more immediate effect on quality of life and relief from suffering, not only for cancer patients but also for their families, than implementing the knowledge accumulated in the field of palliative care.”⁴

RODGER CHARLTON
Senior lecturer in primary health care

School of Postgraduate Medicine,
University of Keele,
Department of Primary Health Care,
North Staffordshire Hospital,
Stoke on Trent ST4 7QB

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Computed tomography in first uncomplicated generalised seizure

Should be performed irrespective of any history of alcohol misuse

EDITOR,—Ronald A Schoenenberger and Sabine M Heim draw conclusions about the investigation of a first generalised seizure that may not be generally applicable to patients seen in an accident and emergency department.¹ The CAGE questionnaire may or may not be a fair means of assessing alcohol dependence in English speaking respondents and may be less reliable when translated into another language (what is the German for “eye opener” anyway?).

Thirteen patients were excluded because computed tomography was not performed, in six cases because the patients refused to consent to it. Did they refuse scanning because of local brain lesions causing altered behaviour? While no patient answering “yes” to two or more questions on the CAGE questionnaire had a focal brain lesion, two

had potential additional causes for their seizure other than alcohol withdrawal, which in one case was a disorder of the central nervous system. One of the dangers of this paper is that it will discourage the search for other potentially important causes of a seizure in patients who are perceived to be dependent on alcohol—for example, HIV encephalopathy, as found in this study.

The fourth point in the box containing clinical implications states that “patients without neurological symptoms and who did not misuse alcohol did not have focal lesions on computed tomography.” In fact, 20 patients in this group had focal lesions on computed tomography.

It is also stated that, by not offering computed tomography to patients on the basis of lack of focal lesions and probable alcohol withdrawal, it “would thus greatly reduce the number of scans that yield no positive diagnosis.” It would reduce the number of scans by about a third, which is a minority.

We think that this paper is misleading in suggesting that we can reliably determine patients who will not have focal lesions by computed tomography by neurological examination and use of the CAGE questionnaire. Computed tomography should probably be offered to all patients who present with a first seizure, whether they misused alcohol or not. Whether computed tomography needs to be performed within the first 24 hours or can safely be left longer in the absence of further seizures was not addressed by this study.

ALASTAIR HAY
Senior house officer
MICHAEL BALL
Research registrar

Department of Neurology,
Royal Hallamshire Hospital,
Sheffield S10 2JF

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Should always be performed

EDITOR,—In their prospective series of 119 patients admitted after a first seizure Ronald A Schoenenberger and Sabine M Heim found that no focal lesion would have been missed if computed tomography had been performed only in patients who either presented with a focal deficit or did not misuse alcohol and did not have a focal deficit.¹ They conclude that although computed tomography of the brain remains necessary in most patients after a first seizure, it may not be absolutely necessary in patients who have uncomplicated seizures related to alcohol and in whom neurological examination yields normal findings.

We retrospectively reviewed the records of 247 adults admitted to our emergency unit after a first generalised seizure.² Criteria for inclusion were nearly the same as those in Schoenenberger and Heim's study. In every case neurological examination had been performed by a senior physician and computed tomography of the brain had been performed. The characteristics of our population were similar to those of Schoenenberger and Heim's patients. Alcohol dependence was the only cause in 49 patients (alcohol withdrawal in 37 of them) and was at least a cofactor in 91 patients. Indeed, alcohol dependence was ascertained in 42 patients in whom the cause was cerebral haemorrhage (eight), infarction (10), tumour (six), metabolic disturbance (six), drug intoxication (six), and cranial trauma (six). Thus the rate of focal lesions among patients dependent on alcohol was 25% (24 patients). In patients in whom the result of neurological examination was normal the rate of focal cerebral lesions was significantly higher in those who were not dependent on alcohol than in those who were (24/109 v 6/70, $\chi^2=5.525$, $P=0.019$). Nevertheless, the rate of focal lesions in

the patients dependent on alcohol in whom the results of the examination were normal—9%—is not negligible. Indeed, if in patients dependent on alcohol the absence of a focal deficit had been considered to be a reliable criterion of absence of brain damage, early diagnosis of six focal lesions—two infarctions, one haemorrhage, one glioblastoma, and two subdural haematomas—would have been missed.

In conclusion, in accordance with previous reports^{3,4} and our experience, we believe that computed tomography should always be performed in patients presenting after a first seizure, even those presenting with chronic alcohol dependence in whom the result of neurological examination is normal.

P LAFOND

B TARDY

F ZENI

A VIALLO

Y PAGE

Consultants in emergency medicine

J C BERTRAND

Professor of medicine

Emergency and Intensive Care Department,
Hôpital Bellevue,
CHU Saint Etienne,
42055 Saint Etienne Cedex 2,
France

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Is grossly oversold

EDITOR,—Ronald A Schoenenberger and Sabine M Heim conclude that “both the diagnostic yield and the resulting interventions justify computed tomography of the brain in most adult patients in casualty departments who have had a first generalised seizure,” but their evidence does not support this statement.¹ They found a particularly high proportion of cerebral tumours, possibly because only patients over the age of 16 were included in the study. In the national general practice study of epilepsy a definite cause for seizures could be ascertained in only 61% of 564 patients; in only 6% of cases were the seizures due to cerebral tumour.² We believe that this is more representative of the true incidence of symptomatic epilepsy in a population. The authors also included patients with intracranial haemorrhage and physical signs in their first study, when other patients were excluded because “clinical circumstances allowed the definite cause of the seizure to be diagnosed.” A further anomalous point is that two patients who were said to have idiopathic epilepsy had focal neurological deficits.

The authors present their data in a way that grossly oversells the value of computed tomography after a first seizure. Subtraction of the fifth from the second columns of the authors' table I shows that only 13 of the 119 patients had a focal lesion on scanning without physical signs. In one of these, the patient with a viral encephalitis, a neurological deficit was apparent on examination but the scan was normal. It may fairly be said, therefore, that in this series computed tomography found a focal lesion unaccompanied by physical signs in 12 of the 119 patients. The number of patients in whom interventions are likely to have been both effective and stimulated by the scan alone rather than by the results of clinical examination is therefore just three—two with meningioma and one with arteriovenous malformation.

We are particularly concerned that the box giving clinical implications states that "computed tomography is recommended as part of the diagnostic investigation for adults with a first generalised seizure." Approximately 4.7% of men and 3.7% of women in the whole population will have at least one epileptic seizure during their life,³ and we need cost effective policies. The decision whether scanning should be performed depends, as always, on a careful history (which will include the possible role of alcohol, as discussed by Schoenberger and Heim) and physical examination. We believe that previously suggested practice remains valid: people who have had a first seizure should be scanned if the clinical onset of the seizure was clearly partial or if there are focal neurological signs that are not clearly accounted for by known pre-existing neurological damage such as cerebral palsy or embolus.⁴

ANTHONY HOPKINS
Director

Research Unit,
Royal College of Physicians,
London NW1 4LE

SIMON SHORVON
Consultant physician

National Hospital for Nervous Diseases,
London WC1N 3BG

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Authors' reply

EDITOR,—The CAGE questionnaire as a screening tool for alcohol dependence has shown clinically useful test characteristics in various English speaking populations in accident and emergency departments.¹ We agree that a formal study of the reliability and validity of a German translation have not been published, but the CAGE questions have been validated in French² and used in research in Spanish.³ We cannot think of any major cultural boundaries that would prohibit the capture of a similar construct of alcohol dependence in a German translation of only four quite simple questions.

We cannot be sure why six patients refused to consent to computed tomography. It was our clinical impression, however, that almost all of these patients had suffered a seizure due to alcohol withdrawal rather than seizures due to focal brain lesions.

The fourth point in the box containing clinical implications is indeed wrong. As the figure in our paper shows, patients with no neurological deficit who misused alcohol showed no focal lesions on computed tomography. Not offering computed tomography to these patients would have saved one third of all scans in this series, which we think is a substantial number.

We believe, however, that we have drawn our conclusions cautiously enough. In the discussion we state that the high yield may justify routine computed tomography, and the third clinical implication in the box iterates this. Confidence intervals are given in every case, allowing an evaluation of the significance of the findings, and the limitations of the study are extensively covered. Under these circumstances, research seeking to reduce unnecessary use of ancillary tests in times of cost constraints should be allowed and encouraged. Further research increasing the total sample size may confirm our results with narrower confidence intervals. Such a study would help to enhance the certainty with which clinicians can

replace the use of non-discretionary tests by indications based on evidence.

R SCHOENENBERGER
Research fellow

Division of Clinical Epidemiology,
Brigham and Women's Hospital,
75 Francis Street,
Boston, MA 02115,
USA

S HEIM
Registrar in neurology

Department of Neurology,
University of Basle,
Basle,
Switzerland

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Thrombolysis in patients with diabetes

More evidence that the treatment should not necessarily be withheld

EDITOR,—Helen Ward and John S Yudkin rightly draw attention to the lack of evidence that thrombolytic treatment for acute myocardial infarction may precipitate vitreous haemorrhage in patients with diabetic retinopathy.¹ Two additional points strengthen their argument that the treatment should not necessarily be withheld in such patients.

Firstly, consideration of the pathophysiology of diabetic vitreous haemorrhage suggests that it is an unlikely complication of thrombolytic treatment. Vitreous haemorrhage occurs in eyes with proliferative diabetic retinopathy almost invariably as the result of posterior vitreous detachment.² Separation of the posterior vitreous surface from the retina results in traction being exerted on adherent new vessels, which may then bleed. It is difficult to envisage how thrombolytic treatment could precipitate vitreous haemorrhage by this mechanism. Diabetic new vessels may occasionally bleed independently of a posterior vitreous detachment. For example, a sudden rise in ocular venous pressure associated with the Valsalva manoeuvre can produce a vitreous haemorrhage.³ While diabetic new vessels may be more fragile than normal retinal vessels, however, their structural integrity does not depend on the presence of thrombus. One would not therefore expect thrombolysis to precipitate bleeding except perhaps in the unusual situation of thrombus sealing a recent breach in a vessel wall.

Secondly, in the unlikely event that thrombolytic treatment did precipitate a vitreous haemorrhage the patient could generally expect a good visual outcome. Many vitreous haemorrhages clear spontaneously over several weeks or months. Dense vitreous haemorrhages that fail to resolve can be removed surgically by pars plana vitrectomy. This can be performed under local anaesthesia if the patient is unfit for general anaesthesia. The results of vitrectomy for diabetic vitreous haemorrhage are good, with vision improving appreciably in about four fifths of patients.⁴

D K NEWMAN
Registrar in ophthalmology
M P SNEAD
Senior registrar in ophthalmology
D W FLANAGAN
Consultant ophthalmologist
J D SCOTT
Consultant ophthalmologist

Department of Ophthalmology,
Addenbrooke's Hospital,
Cambridge CB2 2QQ

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Prophylaxis with aspirin should be considered

EDITOR,—Helen Ward and John S Yudkin have found no evidence that thrombolysis increases the risk of haemorrhage from retinopathy.¹ This will increase doctors' confidence in choosing whether to use thrombolytic drugs for myocardial infarction in patients with diabetes. But the argument that prevented use of these drugs in people at risk of vitreous haemorrhage has always been flawed. Vitreous haemorrhage usually causes transient blindness, indicating that there is proliferative retinopathy that needs treatment. Laser treatment and, if necessary, vitrectomy are extremely effective in preventing further vitreous haemorrhage, which, if it is not dealt with, may result in permanent blindness many years later because vitreous and preretinal scarring cause retinal traction and detachment. Even if thrombolytic drugs do cause vitreous haemorrhage the chance of it affecting vision in the long term is extremely small, which is what Ward and Yudkin confirm.

The question of withholding thrombolytic treatment might never have arisen if patients had more say in their treatment. The choice of having thrombolytic treatment or not affects the risk of dying and bleeding from proliferative retinopathy (or anywhere else, for that matter). Most patients would ignore the risk of bleeding because of the beneficial effect on the risk of dying. This does not mean that patients dictate treatment, but making medical decisions means sharing the same high quality information and understanding to arrive at a joint decision.

More importantly, Ward and Yudkin have an opportunity to create even greater benefit by endorsing wider use of aspirin. Cardiovascular disease is the commonest cause of death in people with diabetes, and there is an argument for routine prophylactic treatment, especially in patients with type 2 diabetes. These patients often have one or more complications at the time of diagnosis, including eye disease, and practitioners may be uneasy about wider use of aspirin in their patients for the same unfounded reasons that have limited use of thrombolytic treatment in hospital in patients with diabetes.

For many years it has been my practice to ask patients with proliferative retinopathy to ask their ophthalmologist whether aspirin treatment is acceptable. Even in patients with documented vitreous haemorrhage the response has invariably been affirmative. The early treatment of diabetic retinopathy study found no evidence of harmful effects of aspirin and recommended the use of aspirin in people with diabetes at risk of cardiovascular disease.² From the evidence I have, withholding prophylaxis with aspirin seems unfair.

It would be useful if Ward and Yudkin could augment their statement about thrombolytic treatment and give similar definitive information about the risks, if any, of the use of aspirin, both as prevention and as curative treatment.

K T MORIARTY
Honorary clinical assistant

Diabetes Unit,
Queen's Medical Centre,
Nottingham NG7 2UH

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