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]

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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 French[2] 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.

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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]

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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.

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