gas into the subarachnoid space to the list of causes of headaches following dural puncture. Of course, all who have had experience with the pneumoencephalogram in the past will agree that non-accidental gas injection should be in the list.

We thank Dr Zeidan and Nahleh for their comments on our case report. In searching the literature on intracerebral bleeds after dural puncture, we left out the subdural haematomas as these occur rather more frequently. We agree that the types of headache play a role in the differentiation between different headaches. This was the reason behind the table in our article.1 In our patient the headache was postural: increasing on sitting up and decreasing and bearable when lying down. Neurological directed physical examination showed no abnormalities. Because of this characteristic feature we proposed the blood patch. We would not advise a CT-scan for all patients with a postural headache following dural puncture.

Concerning the use of low molecular weight heparin in patients with PDPH, the dose used in our patient was a normal dose not a high dose regimen. Patients with PDPH are, by the nature of the problem, confined to bed and in the early postpartum period are still in a hypercoagulable state. Until proven otherwise, we would think the risk of thrombosis and its complications far outweigh the risk of an intracranial bleed in patients with PDPH.

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Reduction in mortality from severe head injury following introduction of a protocol for intensive care management

Editor—Clayton and colleagues report a relative risk reduction in intensive care mortality of nearly 30% from severe head injury with the introduction of protocol-driven management to their hospital.1 Adequate cerebral perfusion pressure is the primary goal of this protocol.

I note with interest, that the Frenchay protocols target a blood sugar level of 4–7 mmol litre−1. Van den Berghe and colleagues2 described a relative risk reduction in intensive care mortality of 43% with introduction of tight glycaemic control (blood glucose 4.4–6.1 mmol litre−1) in a population of predominantly post-cardiac surgery patients in Belgium. Interestingly, the patient groups in the two centres are similar in terms of predominance of single organ failure and lower APACHE II scores. This type of patient may benefit significantly more from tight glycaemic control than general intensive care patients.

I would be interested to know the blood glucose target in the period before protocol introduction at Frenchay, and how well targets were actually achieved. It may be that the improved mortality was at least in part a result of the low-tech, low-cost adherence to tight glycaemic control.

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Editor—The importance of maintaining normoglycaemia and the potential for hyperglycaemia to further damage an ischaemic brain has been appreciated for some time.3 As a result, maintenance of normoglycaemia had become standard neurointensive care practice. The blood glucose target in our protocol was no different to that we aimed to achieve before the protocol was introduced. Indeed, many of the targets and interventions in the protocol were not new or redefined. The main function of the protocol was to ensure an adequate cerebral perfusion pressure by treating the mean arterial pressure and the intracranial pressure in a standardized and logical stepwise fashion. Dr Young is right to highlight the importance of Van den Berghe and colleagues’ findings3 of a reduction in mortality in patients receiving intensive care using a low-tech, low-cost treatment. However, this information was neither available to us at the time our protocol was introduced in1997 nor indeed by the end of our study period in 2000. There is therefore no reason why we would have changed our blood glucose target in 1997 and no reason to suspect that we pursued this target any more vigorously after the implementation of our head injury protocol.

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