The authors reply
Thank you for giving us the opportunity to clarify certain points raised by Mr Scholes.

The rapid access to computed tomography on a 24 hour basis in the Leicester Royal Infirmary is accomplished by ensuring that at least one of the on site radiographers is trained to use the scanner and they are available to perform as a priority when necessary. As with all hospitals there is an on call radiology service available to interpret scans. In the case of “out of hours” computed tomodiography the radiologist is informed as early as possible when a patient requires a scan of the head. This usually means that the radiologist arrives either before or during the scanning process. Thus, this system employs a radiographer who is already on site and there are no additional personnel implications. It is obviously imperative that at least one of the radiographers who is working in the hospital at any time is trained in the use of computed tomodiography. This is ensured by training as many radiographers as possible and especially those who cover A&E to use the scanner.

Mr Scholes has concentrated on the use of computed tomodiography in the case of head injury patients as we have demonstrated, 45% of the emergency scans which we carried out in our department were for medical indications. As A&E staff become more “proactive” in the investigation and management of critically ill patients we would expect our need for and use of computed tomodiography to increase in this type of patient. It is also important to point out that although there were fewer then 200 scans ordered by A&E staff any further scans were requested by in house teams particularly on patients admitted directly through the medical and paediatric admission units. Where a hospital has made such a large capital investment in installing a scan room it would not to make best use of it on a 24 hour basis.

We are in complete agreement that where it is apparent on clinical grounds, and after neurosurgical consultation, that a head injured patient will require as a neurosurgical transfer, irrespective of the results of the computed tomodiography, that the transfer should take priority. This is the case however in a small minority of head injured patients. As our data point to as potential problems with only a scan of six patients requires neurosurgical transfer. Thus five out of six patients avoid an unnecessary and potentially hazardous transfer. Transfers to the regional neurosurgical unit in Nottingham take approximately 30 minutes by road from the Leicester Royal Infirmary.

In conclusion, we agree with Mr Scholes that policies and protocols on indications for computed tomography and transfer are dependent on local resources and should be decided upon by consultation between the district general hospital and the neurosurgical centre to which they refer. We have described our experience on the basis of what we have demonstrated. We have not have signified resource implications as it makes best use of existing on site personnel. As the specialty of A&E moves into the 21st century and becomes a 24 hour service it is vital that it culture change occurs and that the A&E and radiology departments have ready and rapid access to the tools of investigation they require on a 24 hour basis.


“Empirical” thrombolysis in catastrophic pulmonary embolism

EDITOR,—We report a case of pulseless electrical activity thought to be caused by catastrophic pulmonary embolism. The early and “empirical” use of thrombolysis, accompanied by prolonged resuscitation efforts, appears to have been lifesaving. We seek to draw a distinction between “catastrophic” pulmonary embolism, which causes pulseless electrical activity and “massive” pulmonary embolism, which is a term used in the literature to describe cases of pulmonary embolism associated with hypotension.

A 69 year old woman attended the accident and emergency department having collapsed in her general practitioner’s surgery. She was extremely anxious, tachypnoeic (33 bpm), blood pressure 70/30 mm Hg, and heart rate 130 bpm. Electrocardiography (ECG) showed a classical right ventricular strain pattern. Anteroposterior radiography of the chest showed no sign of cardiac failure or pneumothorax. Arterial oxygen saturation was 96% on high flow oxygen.

Although the history was not typical and the ECG changes non-diagnostic, a diagnosis was made of massive pulmonary embolism with hypotension. She was given an intravenous bolus of 5000 units of unfractionated heparin. Arterial blood gases showed an oxygen tension of 15 kPa and a carbon dioxide tension of 4.1 kPa on 95% fractional inspiratory oxygen. The alveolar-arterial oxygen gradient was in keeping with pulmonary embolism.1

Shortly after the bolus of heparin, cardiorespiratory arrest occurred. Resuscitation, following standard life support protocol, was attempted. In total she received 7 mg of adrenaline and 2 x 2000 J DC shocks for pulseless ventricular tachycardia with a bolus of 10 mg of recombinant tissue plasminogen activator (rt-PA) followed by an infusion of 90 mg over two hours. In intensive care, a dobutamine infusion of 5 mg/kg/min was started. The rt-PA infusion was stopped after 90 mg when her mouth was found to be extremely bloodstained. Intravenous heparin was continued. A full recovery ensued.

Subsequent investigations including echocardiography showed only trivial mitral regurgitation. Cardiac enzymes and a low creatine kinase (muscle and brain) fraction of 2.8%, which is not diagnostic of acute myocardial infarction. Total creatine kinase was raised markedly in keeping with prolonged resuscitative efforts.

The accepted best treatment for “massive” pulmonary embolism is angiography followed by surgical embolectomy. Several studies have demonstrated that thrombolysis can be followed by restoration of normal pulmonary circulation.2 There is little written about the management of “catastrophic” pulmonary embolism and we believe that this is the only case reported in which a patient with a pulseless rhythm suspected to have sustained a massive pulmonary embolism on clinical grounds alone, was successfully treated employing rapid bolus thrombolysis. In our opinion, this patient was saved by initial mechanical disruption and perfusion of the clot by resuscitation followed by rapid clot lysis with rt-PA.

There may be a role for the use of bolus thrombolysis in cases of pulseless electrical activity due to suspected pulmonary embolism, and we would welcome correspondence from other physicians who have used these agents in similar circumstances.

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Spontaneous carotid artery dissection

EDITOR,—I read with interest the case report of Mizra et al on spontaneous carotid artery dissection.1 We wished to highlight a similar case, who presented to our accident and emergency (A&E) department recently.

A 36 year old man presented with a five day history of discomfort in the left side of his neck associated with a gradual onset of a left temporal headache and diminished left temporal vision. The headache was increasing in severity and he had also developed neck pain and was confused. Clinical examination was normal. Past medical history revealed occasional migraine of four to five attacks accompanied by tunnel vision and aura. He had a history of head injury in Christmas of 1996 when he was punched and bunted with a brief loss of consciousness but with no obvious neurological deficit.

Computed tomography and electronencephalography were normal. The headache and neck stiffness continued and he then had a two minute episode of numbness in his right arm. Magnetic resonance imaging (MRI) suggested a left internal carotid artery dissection and he was thus started on heparin and warfarin.

He made a complete recovery and remained well until he represented nine months later after his warfarin was stopped with an ache behind his left eye associated with neck stiffness but there was no visual impairment or dysphagia disorder. Clinical examination (MRI) suggested a left internal carotid artery dissection and there was no evidence of a new dissection. No specific treatment was given and he remains well.

This report further supports the view expressed in the article of Mizra et al that a diagnosis of carotid artery dissection should