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 emergency scans as a priority when necessary.¹ As with all hospitals there is an on call radiology service available to interpret scans. In the case of "out of hours" computed tomography 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 resource 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 tomography. 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 tomography in the case of head injury. However, 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 tomography to increase in this type of patient. It is also important to point out that although there were fewer than 200 scans ordered by A&E staff, many 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 scanner, it seems illogical 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 neurosurgical transfer, irrespective of the results of the computed tomography, that the transfer should take priority. This is the case however in a small minority of head injured patients. As our data point out, even after scanning only one in six patients requires neurosurgical transfer. Thus five out of six patients avoid an unnecessary and potentially hazardous transfer.^{1,2} 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 system, which does not have significant 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 true 24 hour service it is vital that a culture change occurs and that all A&E departments have ready and rapid access to the tools of investigation they require on a 24 hour basis.

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- 2 Andrews PJD, Piper IR, Deardon NM, et al. Secondary insults during intrahospital transport of head injured patients. *Lancet* 1990;335:327-30.

"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 resuscitative 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 protocols, was started. In total she received 7 mg of adrenaline and 2 × 200J 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 full of altered blood. Intravenous heparin was continued. A full recovery ensued.

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

The accepted best treatment for "massive" pulmonary embolisms is angiography followed by surgical embolectomy. Several studies have demonstrated that thrombolysis can be followed by restoration of normal pulmonary circulation.²⁻⁶ 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 resuscitated employing rapid bolus thrombolysis. In our opinion, this patient was saved by initial mechanical disruption and peripheralisation 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 would welcome correspondence from other physicians who have used these agents in similar circumstances.

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- 1 Stein PD, Goldhaber SZ. Alveolar-arterial oxygen gradient in the assessment of acute pulmonary embolism. *Chest* 1995;107:139-43.
- 2 Dalla-Volta S, Palla A, Santolucando AM, et al. PAIMS 2: alteplase combined with heparin versus heparin in the treatment of acute pulmonary embolism. Plasminogen Activator Italian Multicenter Study 2. *J Am Coll Cardiol* 1992;20:520-6.
- 3 Goldhaber SZ, Markis JE, Kessler CM, et al. Perspectives on treatment of acute pulmonary embolism with tissue plasminogen activator. *Semin Thromb Hemost* 1987;13:221-7.
- 4 Goldhaber SZ, Haire WD, Feldstein ML, et al. Alteplase versus heparin in acute pulmonary embolism: randomised trial assessing right ventricular function and pulmonary perfusion. *Lancet* 1993;341:507-11.
- 5 Goldhaber SZ, Meyovitz MF, Markis JE, et al. Thrombolytic therapy of acute pulmonary embolism: current status and future potential. *J Am Coll Cardiol* 1987;10:96B-104B.
- 6 Levine MN, Hirsh J, Weitz J, et al. A randomised trial of a single dosage regimen of recombinant tissue plasminogen activator in patients with acute pulmonary embolism. *Chest* 1990;98:1473-9.

Spontaneous carotid artery dissection

EDITOR,—I read with interest the case report of Mirza *et al* on spontaneous carotid artery dissection.¹ We would like 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 dysarthria and was confused. Clinical examination was normal. Past medical history revealed occasional migraine of four to five attacks accompanied by tunnel vision. He also had a history of head injury in Christmas of 1996 when he was punched and butted with a brief loss of consciousness but with no obvious neurological deficit.

Computed tomography and electroencephalography 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 speech disorder. Clinical examination was again normal. A repeat MRI scan showed that the left internal carotid artery had canalised 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 Mirza *et al* that a diagnosis of carotid artery dissection should

be considered in those patients presenting to an A&E department with features suggestive of migraine.

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- 1 Mirza Z, Hayward P, Hulbert D. Spontaneous carotid artery dissection presenting as migraine—a diagnosis not to be missed. *J Accid Emerg Med* 1998;15:187–9.

The authors reply

The letter from Gupta and Moalypour further illustrates that carotid artery dissection is not as uncommon as had been thought. The range of symptoms recognised as being due to the condition and its diagnosis will no doubt increase as doctors become aware of it and imaging techniques and access to them improve. At present we are still learning the true incidence and the natural history of this challenging condition.

Minor injuries units

EDITOR,—Mabrook and Dale's paper on the minor injuries unit in Horsham will doubtless be cited as further evidence of the viability of such facilities.¹ However, closer inspection of their data suggests otherwise.

Firstly, we know that 50% of patients attending minor injuries units could have either self treated or seen their general practitioner (GP).² This implies that only 3472 of the Horsham patients had a significant injury. Of these, 1342 had to be seen by the accident and emergency (A&E) consultant, 234 were referred to the major A&E unit, and 93 were referred to the ear, nose, and throat/ophthalmology departments. This leaves just 1803 appropriate patients who were treated by emergency nurse practitioners (ENPs) during the 12 month period. This equates to 3.5 patients per nurse per working day. Hardly an efficient use of experienced nurses.

Secondly, the paper talks about quality but fails to say whether the ENPs accurately managed soft tissue injuries or whether the antibiotics they prescribed were appropriate. Nor are we told how many ENP patients later self referred to their GP or to the major A&E unit. Nor does the planned reattendance rate reported (23%) suggest a particularly efficient or confident department.

As the pressure to close small and medium sized A&E units continues, more and more communities will be offered minor injury units instead. The public should understand that such units are both untested and inefficient of resources.

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- 1 Mabrook AF, Dale B. Can nurse practitioners offer a quality service? An evaluation of a year's work of a nurse led minor injury unit. *J Accid Emerg Med* 1998;15:266–8.
- 2 Dolan B, Dale J. Characteristics of self-referred patients attending minor injury units. *J Accid Emerg Med* 1997;14:212–14.

The authors reply

The aim of the paper was to evaluate whether an experienced trained nurse can treat minor injuries and ailments in a minor injury unit and not to justify the existence of such units. However, if healthcare trusts decide to commission a unit the year's study has shown that ENPs can be used to provide a successful alternative service.

The points raised by Mr Leaman are arguments that can be used against the existence of minor injuries units, which might well be valid, however this is not what the authors intended to raise in this paper.

All patients who attended the unit were initially assessed and treated by the ENPs. Patients who were referred to the consultant were patients who required follow up and would have been referred even if they had been treated by a casualty officer. It is true that a high number of patients were reviewed in the unit. This is because the consultant has an interest in the management of the common fractures that do not require orthopaedic intervention.

In order to monitor the ENPs' work during the year of evaluation, all the patients' notes were reviewed by the consultant to ascertain whether patients had been diagnosed correctly and treatment had been carried out according to protocols set. Reviews of patients served to monitor the effectiveness of the treatment given by the ENPs.

As to not having kept a record of how many patients treated by the ENPs then self referred to a GP or an A&E department.... Is it possible to keep records of this without a national integrated monitoring system? Patients self refer for second opinion all the time regardless of where they have been initially treated.

Risk of fire outweighed by need for oxygen and defibrillation

EDITOR,—We read with interest that Cantello *et al* from St George's Hospital have repeated part of an experiment we conducted (at the same institution) examining ambient oxygen concentrations during simulated cardiopulmonary resuscitation.² Unfortunately, it is unclear exactly where their gas samples were taken. They state that "the oxygen level beside the manikin on the trolley surface did rise from 22% at the axilla to 28% directly below the reservoir valve" but do not define sampling points nor the time course of the experiment. This lack of detail may be responsible for Dr Ward's supposition (in his comments attached to the letter) that Cantello *et al* measured 28% oxygen concentrations at the axilla. This would be, indeed, a potential hazard as this is a standard paddle position during defibrillation.

We demonstrated a risk of raised oxygen concentrations in areas where oxygen (which is heavier than air) can pool, notably the axilla, when a disconnected ventilation device (Waters' bag, self inflating bag, and intensive care ventilator) is left resting on the pillow. Oxygen concentrations were not raised if the breathing systems were left connected to the manikin or were removed to a distance of greater than 1 m behind the head. The advice of the Resuscitation Council (UK) "that the breathing system be ... disconnected from and distanced from the patient" is sound if the distance be specified as greater than 1 m.

Although Dr Ward's comments are not supported by the findings of Cantello *et al* we feel that, in the case of a disconnected and not adequately distanced breathing system, they

do apply. It is a simple thing to move the source of oxygen away from the patient in the accident and emergency department. In the intensive care unit, where disconnection of breathing systems may present more complex problems, it may be safer to leave the patient attached to the ventilator.

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The authors reply

We were pleased to find that McAnulty and Robertshaw's work regarding oxygen concentration during simulated cardiopulmonary resuscitation² confirms our research, and are grateful for the opportunity to discuss our methodology in greater detail.

With the manikin and ventilation bag set up as explained in our previous letter,¹ the oxygen level recorded at the sternum and apex paddle positions and also at the mouth remained at 21%. Each position was observed for 10 minutes and the oxygen reading was noted to stabilise after two minutes. Therefore, with the analyser raised above the trolley around the manikin's anterior, there is no change in oxygen concentrations.

Referring to both our earlier letter on the subject and supporting letter by Dr Ward,¹ the real debate is not how far to remove the ventilation bag before defibrillation but whether one should remove the oxygen at all before defibrillation. Which is the greater risk? The risk of fire due to defibrillation over an oxygenated area, or the risk of dislodging the endotracheal tube, reducing oxygen flow to a patient in dire need, and delaying life saving defibrillation?

As a training issue, we concur with Dr Ward, "awareness of the problem is likely to reduce the incidence [of fire],¹ and Robertshaw and McAnulty, "it is most important to avoid arcing by ensuring correct placement of paddles and electrolyte pads before defibrillation..."²

In summary, the risk of fire is remote in properly performed defibrillation; the risks to the patient caused by taking the time to remove the oxygen, and the possibility of dislodging the endotracheal airway before defibrillation are too great. Whether in the accident and emergency department or the intensive care unit, we reaffirm our belief that oxygen should not be removed before defibrillation.

1 Cantello E, Davey TE, Koenig KL. The question of removing a ventilation bag before defibrillation. *J Accid Emerg Med* 1998;15:286–9.

2 Robertshaw H, McAnulty G. Ambient oxygen concentrations during simulated cardiopulmonary resuscitation: should ventilatory devices be disconnected before defibrillation? *Anaesthesia* 1998;53:634–7.

Child Protection Register—time for change

EDITOR,—The Child Protection Register neither protects children nor is it a good register. It is used in accident and emergency (A&E) departments across the country several thousand times a day as an investigation. What is the sensitivity and specificity of this test, what positive or negative predictive value has it got? Many, if not all, A&E department clinicians