is widely used in both acute and chronic cardiac care. Traditionally for decades, any patient presenting with chest pain is instantaneously administered high flow oxygen. This concept originally started as we realised oxygen could ease angina pain.[1] It was subsequently believed that this would ease myocardial ischaemia in patient's presenting with acute coronary syndrome (ACS). It quickly became norm to administer high-flow oxygen therapy to patients presenting with acute chest pain.

However, more recently, there have been many reports of harmful effects of high-flow oxygen in ACS patients where the patient might not be hypoxic. High flow oxygen has been shown previously to reduce cardiac output,[2] attribute to arterial vasoconstriction[3–5] and also to increase systemic vascular resistance.[6] More recently, two systematic reviews suggest that the routine use of high-flow oxygen in uncomplicated myocardial infarction may result in a greater infarct size and possibly increase the risk of mortality.[7,8]

The Resuscitation Council UK, the National Institute for Health and Clinical Excellence and the British Thoracic Society have recently appreciated this concern of oxygen therapy in ACS patients and have changed their guidance accordingly. They all now suggest that oxygen therapy should be reserved for ACS patients with hypoxia (O_2 saturation below 94%).

From my current clinical experience, oxygen is still widely administered to ACS patients without hypoxia. This practice needs to change across the NHS and it will only happen with constant multidisciplinary education and the introduction of local oxygen prescription guidance in ACS patients.

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Acute renal failure in diabetes: looking beyond diabetic retinopathy

Editor – We read with interest the report by Sen Gupta and colleagues on a challenging presentation of antineutrophil cytoplasm antibody (ANCA)-associated vasculitis (*Clin Med* August 2011, pp 368–71). However, we are concerned by their recommendation that thromboprophylaxis should be instigated in such cases.

Gastrointestinal (GI) bleeding is a well-recognised complication of acute kidney injury (AKI), occurring in approximately 15% of patients in one series, where it was associated with prolonged hospital admission and an increased risk of death.[1] Factors such as uraemic platelet dysfunction and stress (peptic) ulceration secondary to critical illness are thought to contribute to this risk. For this reason, gastro-protection with H_2 receptor antagonists or proton pump inhibitors is often advised in AKI (although there is no randomised control data to support this recommendation). The recent exposure to non-steroidal anti-inflammatory drugs was an additional risk factor for GI bleeding in this case.

This particular patient also demonstrated features suggestive of systemic vasculitis at first presentation (including AKI with an active urinary sediment, thrombocytosis, severe anaemia, reduced alveolar-arterial gradient with infiltrates on the chest radiograph) and the possibility of life-threatening pulmonary haemorrhage (present in 12–29% of patients with microscopic polyangiitis[2]) should be considered at the outset. No reference was made in the report to assessing gas transfer factor which, if increased, would be a useful indicator of pulmonary haemorrhage in this setting. The severe anaemia may also have alerted to the possibility of gut vasculitis.

Finally, this patient required a renal biopsy to secure a diagnosis and allow the initiation of definitive treatment; this procedure can often be delayed or complicated in patients who have received anticoagulants inappropriately.

For these reasons, we would caution against the routine use of thromboprophylaxis in patients with AKI, particularly in those with features suggestive of coincident pulmonary or gut haemorrhage. It should also be noted that anticoagulants, such as low-molecular weight heparins, may accumulate unpredictably in renal failure and thus require dose-modification in accordance with estimated glomerular filtration rate.

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In response

We thank McAdoo and Pusey for their interest in our case report and for their

comments on the bleeding risks in AKI. In response to the points raised:

1 Our standard policy in AKI is to use gastroprotective proton pump inhibitor therapy as they suggested. In this case we used lansoprazole 30 mg once daily considering the associated GI bleeding risk given the patient's recent use of non-steroidal anti-inflammatory drugs and the steroid therapy for treatment of his ANCA-positive vasculitis.
2 Pulmonary haemorrhage was considered and gas transfer factor was measured (64%). As it was not elevated, we did not mention it in the case report.
3 We agree that low-molecular weight heparins may accumulate in renal failure and that their use in AKI is controversial. However, this patient undoubtedly had a higher than usual risk of thrombosis and we therefore selected to use a low dose of daily enoxaparin (20 mg).

On balance, although there is a significant GI and pulmonary haemorrhage risk in AKI and in ANCA-positive vasculitis, this man also had a sizeable risk of thrombosis given his diabetes with hyperosmolality on presentation, AKI and sepsis. We therefore maintain that thromboprophylaxis should be considered in similar cases, given the trial evidence[1,2] but that this should be balanced against the calculated bleeding risk which should be assessed for each case.

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The inpatient neurology consultation service: value and cost

Editor – The paper by Douglas and colleagues emphasises the importance of neurological advice for the diagnosis and management of hospital inpatients with acute neurological disorders (*Clin Med* June 2011 pp 215–17). This is usually available in the district general hospital (DGH) attached to the regional neurology centre but not so readily elsewhere. This can create difficulties since the present default model of management of acute medical emergencies is admission to an acute medical unit (AMU) followed by triage to an appropriate specialist consultant physician. Without neurologists in the DGH, who rarely have inpatient beds, there is nobody for patients with acute neurological symptoms to be triaged to.

The recent report *Local adult neurology services for the next decade* published jointly by the Royal College of Physicians and Association of British Neurologists, draws attention to this inequitable standard of care for patients with acute neurological disorders in many DGHs.[1] It also refers to other evidence[2] that liaison neurology can halve the length of stay of patients with acute neurological emergencies and result in more accurate diagnosis, confirmed by Douglas *et al*'s paper. One of the major recommendations of the report is that acute neurology services run by neurologists should now be specifically commissioned and provided in the DGH so that patients with acute neurological disorders get earlier access to a neurology opinion and treatment.

Some simple calculations show that rapid access to neurology advice, as well as potentially improving outcomes, would easily pay for the appropriate neurological time by saving costs through reductions in the length of stay and number of admissions. If 120 patients have an average length of stay of four days each, this amounts to 480-bed days per three months at a cost of £72,000 (assuming £150 per bed day). Reducing the length of stay to two days saves £36,000 per three months or £144,000 per year. Halving these estimates to an average length of stay to two and one day respectively, still amounts to £72,000 per year, more than enough to pay for the necessary neurological sessions.

There are therefore sound financial reasons as well as potential improvements in care if patients with acute neurological disorders are seen by the people best able to look after them, ie neurologists. General physicians, specialist physicians themselves, quite rightly in my experience, see no reason why they should continue to manage patients with acute neurological disorders any longer. This is surely the responsibility of neurologists! The case for acute neurology run by neurologists is incontrovertible and DGH trusts should be encouraged to appoint acute neurologists to do this.

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In response

We are grateful for Dr Bateman's comments and agree that there are obvious financial, as well as clinical, advantages for patients with acute neurological disorders to be seen by neurologists. It is worth noting that the data from our paper[1] were generated between September and November 2005, at a time when the majority of our inpatient work was based on a ward referral review system, rather than a system of direct admission to neurological care. The service was subsequently reconfigured and patients with acute neurological problems are now first admitted to the medical assessment unit, followed by triage and transfer to the neurology ward. This practice broadly follows the suggestions of the report by the