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In response

We thank Quantrill and Webbe for their response, and agree completely both that an important role of pharmacists is to educate, and that better systems are needed for providing feedback to prescribers about any errors made. We believe that feedback is complementary to pharmacist attendance on consultant ward rounds, and that both approaches are required. Pharmacists attending ward rounds are likely to be more aware of patients' current priority medical problems, and are able to discuss drug therapy with senior members of the medical team, resulting in the higher intervention rate demonstrated in our paper. Separately, better feedback on prescribing errors, particularly to junior doctors, is also needed, to facilitate learning. Several studies have shown that junior doctors get little or no feedback on their prescribing errors at present. We recently completed some exploratory work with junior doctors and pharmacists to explore these issues, and found a key barrier to be pharmacists unable to ascertain the identity of the prescriber. We are therefore considering piloting the use of name stamps, and are designing a controlled study to explore the impact of providing feedback to our junior doctors. We would encourage Quantrill and Webbe to publish their findings in more detail so that others can build on them further.

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'The tubercular diabetic'

Editor – We read with great interest the article by Bailey and colleagues (*Clin Med* August 2011 pp 344–7). Treatment of people with tuberculosis (TB) and diabetes is indeed complicated. Not only does rifampicin potentially adversely alter the pharmacokinetics of gliclazide,[1] glipizide,[2] pioglitazone,[3] nateglinide[4] and repaglinide,[5] but like isoniazid, it may increase insulin requirements.[6] Liver and nerve toxicity from anti-TB drugs may be difficult to distinguish from diabetes-associated non-alcoholic fatty liver disease and peripheral neuropathy respectively and for those with co-morbid HIV infection with access to treatment, there is the added complication of antiretroviral-associated insulin resistance.[7] TB itself may precipitate hyperglycaemia by a stress hormone response and there is some evidence of glucose intolerance in TB patients reverting to normal in up to 75% of patients after three months of TB treatment.[8]

We wholeheartedly endorse Bailey and Grant's conclusion that TB and diabetes demand increased attention from clinicians and academics if we are to ensure that future patients receive optimal management of both conditions.

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In response

We read with appreciation the comments of Chandrasekara and Hardy. The management of concomitant tuberculosis and diabetes mellitus remains challenging and highlights two important factors. Firstly, that our level of clinical suspicion of dual pathology here in the UK needs to be raised so that management can be optimised, including appropriate adjustment and monitoring of medication. Secondly, that as diabetes progresses in low-income countries we need to consider collectively how best to manage this chronic disease in resource-limited settings and indeed this is a focus of our ongoing research.

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Oxygen therapy in acute coronary syndrome: current NICE recommendations

Editor – I read with great interest the concise guidance by O'Driscoll and colleague (*Clin Med* August 2011, pp 372–5) on emergency oxygen use in adult patients. Oxygen therapy

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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

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