improved recognition, understanding and management. Only if comprehensive data are collected, can worrying clinical prognosticators be identified.

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In response

Editor – Sandilands and Bateman raise an important point in regard to the safety of induced emesis and rightly remind us that it is no longer routinely recommended in cases of poisoning. It was not the intention of the previous report to advocate its use as a primary management step in cases of poisoning. Indeed, it can be seen from the case report that this was not part of the management strategy which was guided both by reference to the online TOXBASE and by discussion with the National Poisons Information Service. We concede that there is only anecdotal, dated and rather tenuous basis for the induction of emesis and perhaps the mention of induced emesis in the original report belongs in the same history books as those used by our patient to glean her knowledge of yew toxicity!

In cases of rare poisoning such as this there is little evidence on which to guide management. A review of 10 years' data from the American Association of Poison Control Centers Toxic Exposure Surveillance System revealed only four cases of life-threatening complications of yew ingestion.[1] TOXBASE lists only eight references on which it bases its guidance and information to emergency departments throughout the UK. All of the measures recommended on TOXBASE and highlighted by Sandilands and Bateman were attempted but none appeared to improve the clinical situation at the time.

Given this, it is worth reporting that the young lady described in the case report has presented once again with deliberate self-poisoning with yew foliage. She absconded from the supposedly secure psychiatric unit where she was an inpatient following her previous presentation. She made her way directly to where she knew the yew trees were growing and once again consumed a quantity of shoots and leaves which she washed down with a fizzy drink which she brought specifically for this purpose. Although she was apprehended quickly she was observed continuing to consume yew leaves even after apprehension having hidden some in the pockets of her trousers. She was brought immediately to the emergency department where, because she presented within 60 minutes of ingestion, she was given 50 g of oral-activated charcoal. Although she developed a marked sinus tachycardia, she remained clinically well with a blood pressure of 125/90 mmHg and peripheral oxygen saturations of 99% while breathing room air. She was discharged back to the psychiatric unit the following day. While induced emesis may not be recommended, she has, unintentionally, provided a single patient case-control 'study' into the effectiveness of the early use of oral-activated charcoal. The lack of effect seen from late administration of charcoal is in keeping with current guidelines and published studies showing a steady reduction in toxin absorption with time.[2]

Guha is right to mention the QT prolonging and proarrhythmic potential of amiodarone. While caution with the use of antiarrhythmic medications is certainly prudent, in this case amiodarone was given in the setting of a cardiac arrest with shock refractory ventricular tachycardia (VT) (and later repeated because of the apparent success of the initial administration) and is in keeping with current UK resuscitation guidelines to provide standard advanced life support if cardiac arrest occurs. TOXBASE already acts as a comprehensive database and we hope that publication of rare case reports such as this will help to inform future practice.

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Angina without 'strangling and anxiety of the breast'

Editor – Cooper and colleagues remind us that cardiac pain may be present only in the neck and arm, without there being any chest pain (*Clin Med* April 2011 pp 201–2). Very rarely, cardiac pain is felt in more unusual positions. Lanza and colleagues reported a case in which cardiac pain consisted purely of headache.[1] The rarity of this presentation may be judged by the fact that the article has never been cited.

I saw a man in his mid-60s who had cardiac pain confined to the vertex of his head. (His exact age was unknown because he was born in a remote village in a developing country and there was no official record of his birth.) He had woken from sleep with sudden onset of severe pain at the top of his head. It was the worse pain he had ever experienced. It lasted about eight hours. There was no meningism or abnormal neurological findings. Subarachnoid haemorrhage was suspected but a computed tompgraphy (CT) brain scan and lumbar puncture were normal. An electrocardiogram (ECG) showed anterior ST elevation consistent with an anterior myocardial infarction. The ECG appearance was initially attributed to a subarachnoid haemorrhage.[2] That view of the ECG was not revised even after subarachnoid haemorrhage was discounted. After discharge from hospital, he reported similar but less severe pain confined to the vertex of his head when walking uphill. It disappeared almost immediately once he rested. He had identical pain associated with anterior ST segment depression during a treadmill exercise test. Coronary angiography showed a single severe stenosis in the left anterior descending

artery. During percutaneous coronary intervention, when there was balloon inflation, he had identical head pain with ECG changes but with no chest discomfort. After coronary intervention he was free of the pain on exertion.

About half of patients with a subarachnoid haemorrhage have ECG abnormalities.[2] Failure to recognise that ECG abnormalities are common in patients with subarachnoid haemorrhage can lead to them receiving inappropriate cardiac treatment and delayed investigation for subarachnoid haemorrhage. In the case I describe, the localisation of the pain resulted in initial misdiagnosis of cardiac pain as subarachnoid haemorrhage.

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Emergency medical readmission: long-term trends and impact on mortality

Editor – We read with interest the study by Glynn *et al* (*Clin Med* April 2011 pp 114–8) describing long-term trends in emergency medical readmissions and the impact on mortality. There is much interest in emergency readmissions at present and a view that many readmissions are preventable.

In 2002–03, we undertook an audit of 28-day emergency readmissions from 14 general medical (including care of the elderly) wards in our 800-bedded acute trust serving a predominantly deprived population. As part of that audit, we solicited patients' views on their emergency readmission. There were 642 emergency readmissions in 4,801 medical discharges (13%) over a seven-month period, of 606 for whom notes were available, 202 (33%) had died by the time we undertook the survey and 15 had moved district. We wrote to the remaining 389, and 119 (31%) responded.

Interestingly, 85% of patients said that their readmission was for the same problem as the index admission (25% heart, 24% chest, 33% unsure of condition, other conditions all <5%). With hindsight, 40% of patients felt that they were not ready for discharge after their index admission, 45% felt that the readmission might have been prevented with better care or a longer index admission, 40% of patients felt an early follow-up outpatient appointment would have prevented readmission, 28% felt readmission could have been prevented by better post-discharge support from the primary care team and 20% felt social service input after discharge could have prevented readmission.

Listening to our patients may also help prevent emergency readmissions.

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How do I manage a patient with suspected acute pulmonary embolism?

Editor – I read with great interest Sheares' excellent review article on the management of patients with suspected acute pulmonary embolism (PE) (*Clin Med* April 2011 pp 156–9). I would, however, like to comment on the author's recommendations regarding the treatment of high-risk PE, previously known as massive PE.

Sheares, citing the study of Jerjes-Sanchez *et al*[1] which states that thrombolysis improves survival in patients with high-risk PE. However, the author neglects to report the observations from the International Cooperative Pulmonary Embolism Registry.[2] Although admittedly somewhat counterintuitive, the findings of this landmark study were that thrombolysis did not reduce mortality or recurrence of PE at 90 days in high-risk PE.

Sheares confines the role of surgical embolectomy in high risk PE to patients who have failed thrombolysis or in whom thrombolysis is contraindicated. However, there is an emerging body of evidence supporting the use of primary embolectomy. Successful surgical embolectomy, using temporary cardiopulmonary bypass, was first reported by Denton Cooley 50 years ago.[3] Thirty years later, Gulba *et al* compared the outcome of 13 patients with massive PE treated with surgical embolectomy and 24 such patients treated with thrombolysis.[4] The surgically treated patients had a lower death rate as well as lower rates of bleeding and recurrence of PE. More recently, Fukuda *et al* have reported an operative mortality of only 5% in patients with massive PE undergoing emergent pulmonary embolectomy.[5]

Accordingly, primary surgical embolectomy should be considered favourably in centres with on-site cardiothoracic surgery. Given that the author's institution is an internationally acclaimed cardiothoracic centre, I would welcome her comments on her experience in this area.

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