with the history of left ventricular (LV) failure and angioplasty and would explain the negative enzymes. Although hypercalcaemia has, on rare occasions, been described as mimicking ST elevation due to its effect on the ST segment, there is no reason that it would cause the pathologic Q waves seen in leads V1 to V5. In contrast, pathologic Q waves with persistent ST elevation are typical of LV aneurysm.

The case was described as a 'lesson of the month'. I believe that the lesson here is not the one that was presented but just the opposite. From the presented data one can conclude that common things occur most commonly, incomplete data (lack of prior ECG and no echocardiogram) may lead to incorrect diagnosis, and that even reputable journals allow information to be published that is inaccurate and misleading.

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Response to both letters

Sado and Greaves have raised the possibility of left ventricular (LV) aneurysm, hypertrophic cardiomyopathy (HOCM) and pericarditis as a differential diagnosis. Falk suggests that LV aneurysm was the most likely diagnosis. We agree that the points raised are valid. The patient certainly had a significant history of ischaemic heart disease. Her past cardiac history included non-ST elevation myocardial infarctions (NSTEMIs) with left ventricular failure (LVF). Seven months before the published event, she was admitted with NSTEMI and LVF. The angiogram showed normal left main stem, significant stenosis of left anterior descending (LAD) artery, moderate stenosis of distal right coronary artery, and obtuse marginal. She subsequently had elective percutaneous coronary intervention (PCI) to her LAD, with three bare metal stents, as there would have been difficulties to insert drug-eluting stents due to calcification and tortuosity. She had a further admission with NSTEMI and LVF three months before the published event, due to instent restenosis, which was treated with balloon dilatation only.

On both occasions the LV systolic function was well preserved with mild hypokinesia of the anteroapical and inferoapical walls; no evidence of LV aneurysm on either occasion. An echocardiogram (ECHO) at the time of the first PCI did not show any evidence of LVH or HOCM. Therefore we felt LV aneurysm or cardiomyopathy were unlikely based on previous investigations. Moreover the patient did not have any significant cardiac event between the last PCI and the published event. We agree that a repeat ECHO during the published admission (unfortunately not undertaken) or post-mortem (declined by family) would have clinched or refuted the differential diagnosis of LV aneurysm.

Supporting our theory, the most recent ECG before this published admission showed Q waves in V3 and V4 with ST coving and T wave inversion. Therefore we felt that the profound ST segment elevation during published admission was secondary to severe hypercalcaemia.

The chest pain history was not suggestive of pericarditis. As saddle-shaped ST elevation is only seen in V5, the ST segment elevation in other leads does not suggest pericarditis. Moreover the PQ/PR segment appears normal. Therefore we felt pericarditis to be an unlikely diagnosis. To conclude we feel that the ECG changes are secondary to profound hypercalcaemia rather than LV aneurysm or hypertrophic cardiomyopathy.

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The story of Axel Munthe

Robert Allan comments that 'the true sequence of [Axel Munthe's] life is unravell[ing] and it becomes evident that many events are either imagined or at least embroidered', but surely this was already evident to the averagely intelligent reader from Munthe's own text even before the research by Alex Paton and Bengt Jangfeldt (*Clin Med* June 2009 pp 204–5). Munthe even asks the reader's forbearance for having perhaps described the man he would have liked to have been, rather than the man he actually was. The following examples drawn from the 1932 impression of *The story of San Michele* confirm that Munthe never intended us to take him too seriously[1]:

> *I am aware that some of the scenes in this book are laid on the dangerous borderland between the real and the unreal, the dangerous No Man's Land between fact and fancy…I do not ask for better than not to be believed.*
>
> Pre-preface, 'To those who have read this book', p xiii

> *But I could see quite distinctly a little man as big as the palm of my hand sitting cross-legged on the table carefully pulling at my watch chain and bending his grey old beard on one side to listen to the ticking of my repeater.*
>
> Chapter VII, 'The tallow candle and the goblin', p 139

> *'[Axel Munthe] was an unbeliever', St Ignatius went on. 'A blasphemous scoffer, a liar, an impostor, an enchanter full of black magic, a fornicator…'. 'He was fond of children', said St John. 'He was fond of their mothers too', growled a Patriarch in his beard.*
>
> Chapter XXXII(b), 'In the old tower', p 515

Unless they believed that Munthe really did spend an evening in the company of a goblin, it is hard to understand the sense of betrayal felt by some readers on subsequently discovering that Villa San Michele was designed by an architect or that the Sphinx was bought in an antique shop, rather than dug up from Nero's villa under supernatural guidance. Weaving fact and fiction beautifully together, Munthe told a story that still remains particularly inspirational to those of us in the medical profession.

However, his greatest legacy may be in the area of wildlife conservation, eg Chapter VII 'The bird sanctuary. The wings of the angels', p 448:

> *The mountain of Barbarossa is now a bird sanctuary. Thousands of tired birds of passage are resting on its slopes every spring and autumns, safe from man and beast.*

Several years ago, I chanced upon a copy of an Italian weekly magazine from around

1948 containing a short article describing how, despite sharing his Royal Palace, Munthe stopped speaking with King Gustav V for a period each year corresponding to the Swedish hunting season (of which the King was an enthusiastic participant). As someone who has visited Italy almost every year since I was born, I have been struck by the year-on-year increase in birdsong (and the correspondingly reduced biting insect life) in both countryside and city, as the Italians gradually wean themselves off blasting everything that flies out of the sky.

RICHARD QUINTON
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Reference

1 Munthe A. *The story of San Michele.* London: John Murray, 1932.

A history of the gardens of the Royal College of Physicians of London

Editor – I was delighted to read Arthur Hollman's article on a neglected area of the College's history (*Clin Med* June 2009 pp 242–6). May I make some minor additions to the story?

At Knightrider Street, the Royal College of Physicians (RCP) almost certainly did not have a garden on the premises. Contrary to the usual perception, the RCP did not own the whole house. After Linacre's death, the majority of the building, including the garden, was clearly the property of Merton College, the RCP having only two rooms at the front, the will stating:

> *And ferther I will and bequeth the chappell and the chamber over the chappell wythin my howse where I now dwell wyth in the citie off London to the College of Phicicons of London and to ther successors for ever…*

The College therefore would have had to look elsewhere for a garden.

At Amen Corner, there was undoubtedly a garden. In Stow's *Survey of London*, under 'Farringdon Ward, infra or within' he states that:

> *Now to turn up again to the north end of Ave Mary Lane, there is a short lane which runneth west some small distance, and there is closed up with a gate into a great house; and this is called Amen Lane.*

This is presumably the house which the College bought, and is shown on the copperplate map. However the copperplate map is somewhat misleading on the issue of the garden. As noted in the article, it probably dates from 1558, and in 1611 the Stationers' Company moved their hall from St Paul's Churchyard to 'Abergavenny House', or 'Bergavenny House' (rebuilt in 1654). This is referred to in all RCP legal manuscripts as forming the southern boundary of the College's site, and extended up to the City Wall, taking up the southern part of the garden shown on the copperplate map. The statement by Munk, referred to in the article, about a 'College garden…reached to the church of St Martin Ludgate' is actually referenced to McMichael's statement to this effect in *The gold-headed cane*,[1] and I can only assume that McMichael had deduced this from the later woodcut Agas map (which is based on the copperplate).

ANDREW HILSON
Harveian Librarian
Royal College of Physicians

Reference

1 MacMichael W. *The gold-headed cane.* London: John Murray, 1828.

Sepsis and septic shock: inching forwards

Editor – It was great to see the article by Jonathan Cohen and colleagues highlighting the large challenge that recognition and basic treatment of sepsis still brings (*Clin Med* June 2009 pp 256–7). I share their concerns that it is not part of the core competencies or syllabus for Modernising Medical Careers. Our sepsis audit, in keeping with a lot of national data, demonstrated the time to antimicrobials for patients with severe sepsis (any two of systemic inflammatory response syndrome (SIRS) criteria + one feature of end-organ dysfunction) was over seven hours in some cases. This prompted the production of a one-sided A4 document to be used as a pro forma and audit tool; a sepsis guidelines/antibiotic prescribing guidelines card that is attachable to the trust photo ID badge.

In view of this we set up two sepsis symposia to promote early recognition and delivery of a sepsis care bundle. The symposium each comprised of three training sessions of a total of 24 doctors over a two-hour period.

Introduction

The introduction covered the audit data and the evidence for a need to change.

Session A: sepsis simulation with 'Sim man'

This session was based on a real scenario that had occurred a few weeks previously, when a patient with severe community-acquired pneumonia had not been treated with antibiotics or fluids for six hours and subsequently died. This was a 10-minute scenario for each team, culminating in improving observations/survival or a pulseless electrical activity (PEA) cardiac arrest depending on the group's ability to recognise and effectively treat the underlying sepsis. This was followed by structured feedback for each group.

Session B: the 'box'

This was a 20-minute brainstorming session where the junior doctors were asked to plan, construct and populate a box with the components needed to investigate and promptly treat sepsis. This resulted in the production of a 'suspicion of sepsis' box containing intravenous (iv) Tazocin®, 500 ml normal saline, a giving set, two cannulae, a blood culture phlebotomy pack, an arterial blood gas syringe, a tourniquet, water for injections and flushes. The juniors were also asked to formulate what should be written on the box with instructions, warnings and guidelines.

Session C: iv antimicrobial administration/blood culture phlebotomy/priming iv giving sets

This was a practical session involving a senior nurse teaching the juniors how to