letters to the editor

Please submit letters for the editor's consideration within three weeks of receipt of *Clinical Medicine*. Letters should ideally be limited to 350 words, and sent by email to: clinicalmedicine@rcplondon.ac.uk

Structured clinic letters

Editor – It is very necessary to study efficient and effective communication between hospital and general practice. Tom Parks and others (*Clin Med* April 2011 pp 205–6) found GPs to prefer structured letters but what about the person in the middle – the patient? For many years I dictated the outpatient letter to, and with, the patient in their presence in clinic and sent a copy to the GP. This ensured that the patient agreed with the facts, targets and treatment and that the GP knew what information the patient had been given. I surveyed 120 GPs in North Bristol and 119 were very happy with this and I also had a similar result in Gloucester. I know of other consultants who use this system. Apart from being basic good manners it offers a wonderful opportunity to discuss with, and inform patient, family and doctor.

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Cardiac involvement in systemic lupus erythematosus not only limited to pericarditis

Editor – We read with great interest the article by Perry and colleagues (*Clin Med* June 2011 pp 268–70) on acute systemic lupus erythematosus (SLE) presenting as pericarditis.

The incidence of cardiac involvement in SLE at post-mortem is approximately 40%, but only 6% of patients had echocardiographic evidence of impairment, and only one death in a cohort of over 500 patients was attributable to cardiac involvement.[1]

The key first step in investigation of SLE-related cardiac disease is the electrocardiogram for analysis of arrhythmias, ischaemic change, and left ventricular function. There are a number of possible cardiac manifestations of SLE, the most common forms being pericarditis, myocarditis, nonbacterial verrucous endocarditis, coronary artery disease, coronary arteritis, premature coronary atherosclerosis, congestive heart failure, cardiac arrhythmias, pulmonary hypertension and conduction disturbances.[2]

While there is no particular consensus on what imaging is required when cardiac involvement with SLE is suspected, the most reasonable second step is transthoracic echocardiography as with many cardiac diseases. Echocardiography can help diagnose SLE-related pericarditis, pericardial effusion, systolic dysfunction, valvular involvement, and cavity thrombus formation with a good sensitivity.[3] The amount of information which is gained from an echocardiogram is especially valuable in such patient cohort. We generally suggest doing an echocardiogram on patients presenting with pericarditis to rule out the above. It can also be organised as an outpatient test if early discharge is contemplated.

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Acute systemic lupus erythematosus on the acute medical take: are we missing anything?

Editor – I read with great interest the above case by Perry and colleagues (*Clin Med* June 2011 pp 268–70). To assess lupus anticoagulant the assay that is widely available is the Russell's viper venom test. Unfortunately, as pointed out by Perry *et al*, this test cannot be used when patients are on warfarin. Recently, I was educated about the presence of another assay called the Taipan viper venom test. This assay can be used to detect the presence of lupus anticoagulant even if patients have been commenced on warfarin. With respect to this particular patient maybe the doctors would like to explore this option? It would help them exclude or confirm the diagnosis of secondary aPL syndrome and hence decide the duration of anticoagulation accordingly. The pitfall is that this assay is available only in London and Manchester as far as I am aware.

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In response

We thank Garg *et al* for their feedback. Cardiac involvement in SLE is indeed important. In our case with a history consistent with pulmonary embolus, the absence of marked cardiovascular compromise on clinical examination, CTPA confirming pulmonary embolus and showing no evidence of pericardial effusion. Echocardiography was performed as a later

investigation rather than as an immediate investigation in the acute setting.

We would also like to thank Madan for the guidance, and we will look into the possibility of checking Taipan venom time in this patient.

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Hyper acute stroke unit services

Simon Liu and colleagues (*Clin Med* June 2011) should have clarified that the quoted number needed to treat (NNT) of 3.1 with thrombolysis did not refer to the more often used group outcome comparison of 'independent' versus 'dependent and dead', but to expert derived estimates for one additional patient to have a better outcome by one or more grades on the mRS (modified Rankin Score).[1,2] This would include patients moving from mRS 5 to mRS 4, for example, who would remain in the 'dependent and dead' category of outcome.

Jeffrey Saver's 2004 modelling paper concluded 'for every 100 patients with acute stroke treated with tissue plasminogen activator, approximately 32 will have a better final outcome and three have a worse final outcome as a result of treatment'. Thrombolysis has the potential to harm as well as cure! Saver also stated in the paper 'the NNT for tPA treatment to avert one case of dependence or death after stroke, defined as an mRS of 2 or more, is 8.4' based on the NINDS study.[2] A NNT of 8–10 is probably more recognised by physicians for the effectiveness of thrombolysis.

Work from Australia documenting the real-life three-month outcomes after thombolysis suggests that Saver's experts may have underestimated the benefits of thrombolysis in the group of patients presenting with more severe strokes.[3] Bray and colleagues in Melbourne found that of 24 patients presenting with stroke and a mRS of 4, the outcome at three months was that five of the group had an mRS of 0, 6 mRS of 1, 3 mRS of 2, 5 mRS of 3 and 1 an mRS of 4. Only four patients had a worse outcome with one dying (mRS 6) and three having a mRS of 5. For the 43 patients presenting with a mRS of 5 there were similar favourable improvements; 19 returned to independence (mRS 0–2) at three months post-stroke, with a further six dying and five remaining on a mRS of 5.

The access to hyper acute stroke care and thrombolysis in London has improved in recent years. Those people with severe strokes in particular need to get to hospital as soon as possible because early thrombolysis could make a major difference to their future care needs.

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The NHS: assessing new technologies, NICE and value for money

Editor – I read with interest Stevens' article (*Clin Med* June 2011 pp 247–50) giving an historical perspective of the inception of the National Institute for Health and Clinical Excellence (NICE) and development of its core activities. I would question, however, the assertion that NICE 'held its ground' over industry pressure to approve Relenza®, when less than a year after its first decision not to approve, it made a u-turn and approved (in admittedly a restricted way) the use of the drug for the following flu season, much to the horror of many GPs.[1] While the relevant paragraph is factually correct, as it refers to its 'first' decision, I would not want the casual reader to be unaware of the conclusion to that particular episode. NICE has had many question the legitimacy of its decisions made in a maelstrom of political and industry pressures.[2] One wonders whether an interpretation of the coalition government's plan to devolve rationing decisions to a more local level is an attempt to escape that perception.

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In response

Patel is right that NICE exists in a world of 'a maelstrom of political and industry pressures'. But it makes every attempt to remain fair and objective. Not least is the appointment of independent appraisal committees who are protected from most media exposure.

It is true that our Relenza® (zanamavir) for influenza decision changed between the very first (strictly pre-NICE) appraisal and technology appraisal No15 (TA15) a year later.[1] But then the evidence changed too. The first appraisal was informed by three randomised controlled trials, all three of which excluded 'at-risk' patients. The nub of the appraisal concerned precisely these patients – the immunocompromised, the elderly or those with other co-morbidities. In TA15 the evidence base included 800 at-risk individuals, including one trial of people with chronic respiratory disease. This was sufficient to reasonably model the effectiveness and cost-effectiveness of zanamavir not just on ameliorating an episode of flu, but in reducing the likelihood of exacerbating the co-morbidity.