specificity of 95% as compared to a bone marrow aspiration. This higher than expected value might result from an increase in ferritin with age and from the role of ferritin as an acute phase reactant.

Therefore, using 12 μg/l as a discriminant value as proposed by Jolobe and Rakicka in their study will exclude many elderly patients with iron deficiency anaemia.[5] As a consequence, elderly patients with a ferritin level >12 μg/l and iron deficiency anaemia will not be investigated adequately. We, like Guyatt *et al.*[1] think that the serum ferritin levels in the elderly should be interpreted differently from those in younger patients. A serum ferritin level less than or equal to 50 μg/l may be a useful indicator to take into account the possibility of iron deficiency in elderly patients. In equivocal cases and for higher ferritin levels, a bone marrow aspirate would be required. Further studies investigating the clinical usefulness of the different cut-off levels of serum ferritin in the diagnosis of iron deficiency in elderly patients are warranted.

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Age-related differences in simultaneous interarm blood pressure measurements

Sir,

Interarm blood pressure (BP) differences[1] have posed a new problem over recent years since the advent of thrombolysis as the treatment of choice in acute myocardial infarction, namely can a dissecting thoracic aortic aneurysm be excluded in a patient with a definite myocardial infarction and yet a systolic BP difference greater than 10 mmHg?

We report four patients who were admitted with an acute onset of constant, crushing, central chest pain radiating to the left arm who had such a systolic BP difference. The BP was initially read with a ward sphygmomanometer, and then repeated by a second observer with a different sphygmomanometer. All the patients were aged over 50 years, and two also complained of slight back pain. Pulses were palpable and equal in both arms, and all patients were in sinus rhythm, with no carotid or subclavian bruits. Myocardial infarction was diagnosed by a classical history, with electrocardiographic evidence of greater than 2 mm S-T elevation in at least two limb or chest leads. Chest X-ray showed a normal mediastinum in all cases, and thrombolysis was administered with no complications. All patients were discharged within one week, with the systolic BP difference unchanged.

The concern about dissecting thoracic aortic aneurysm at the time of their presentation proved unfounded, and yet must be considered, particularly if the chest pain radiates through to the back or back pain is predominant. Prior documentation of interarm BP differences may help reduce this concern, as well as reduce misclassification of BP status.

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Aspirin and risk of fatal colon cancer

Sir,

It has been proposed that regular aspirin use may decrease the risk of fatal colon cancer[1] and in a recent correspondence, Odeh[2] proposed two possible mechanisms. We write to suggest a third possible mechanism.

Non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin, indomethacin, piroxicam and sulindac inhibit the growth of colon tumours induced by chemical carcinogens in rodents.[3–6] It is proposed this beneficial effect of NSAIDs is related, at least in part, to their ability to inhibit prostaglandin (PG) formation.[7] Previous studies have shown that colon tumours produce increased amounts of PGE_2 compared with surrounding tissue.[8] This PGE_2 may play a role in pathophysiological processes including tumour-related angiogenesis[9] and depression of cellular immunity.[10] Tumour-derived PGE_2 may therefore further tumour growth in the colon.[11] By contrast, inhibiting PGE_2 synthesis could be tumouricidal by reducing blood flow to the tumour coupled with enhanced immunorejection.[2,12]

Thus, NSAIDs may possess tumouricidal properties in the colon. This would account for the beneficial effects observed in animal models and the low risk associated with regular NSAID ingestion. Of considerable interest would be the effect of NSAIDs on established colon cancers, where an initial report is encouraging.[13]

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Fulminating streptococcal septicaemia

Sir,

Fatal streptococcal septicaemia in previously healthy young people is rare.[1–3] We report two recent cases at our hospital that serve to remind of the lethal properties of some Lancefield Group A streptococci.

Case 1

A 35 year old woman, known to suffer from bulimia nervosa, and to abuse both laxatives and diuretics, was admitted with a 6 day history of headaches and fever, treated with simple analgesia by her general practitioner. Clinically, she appeared alert and orientated, but somewhat dehydrated. There was generalized muscle weakness but no focal neurological deficit. Blood pressure was 80/40 mmHg. Blood investigations revealed potassium 3.3 mmol/l, sodium 131 mmol/l, urea 19.2 mmol/l, glucose 5.4 mmol/l, haemoglobin 10.6 g/dl, white cells 3.9×10^9/l and platelets 104×10^9/l. The initial impression was of deranged biochemistry and symptoms secondary to her bulimia and a recent viral illness.

Ten hours later, she collapsed with peripheral circulatory failure, unrecordable blood pressure, erratic respirations and a mottled skin rash. Further tests revealed disseminated intravascular coagulation and metabolic acidosis (arterial pH 7.2). Electrocardiogram showed sinus tachycardia and chest X-ray showed a small opacity in the right mid-zone. Septic shock with multi-organ involvement was now suspected. Despite intravenous broad-spectrum antibiotics, fluids, dobutamine and dopamine infusions cardio-respiratory arrest ensued from which resuscitation was unsuccessful. Autopsy revealed bilateral bronchopneumonia. Blood cultures grew *Streptococcus pyogenes*, Lancefield Group A, type T12 M12.

Case 2

A previously healthy 35 year old man presented as an emergency. The history obtained from his wife was of a 4 day flu-like illness with myalgia and sore throat, again treated with simple analgesia. Clinically he was now semi-conscious, hypotensive, cyanosed and had a striking mottled purpuric rash on his trunk and lower limbs. He was apyrexial, the abdomen was soft and he had no meningism. Blood tests revealed metabolic acidosis (arterial pH 6.9), urea 18.1 mmol/l, white cells 15×10^9/l (90% neutrophils), platelets 80×10^9/l. There was evidence of disseminated intravascular coagulation and amylase was moderately elevated at 416 IU/l (normal laboratory range up to 110 IU/l). The differential diagnosis was of septic shock or acute pancreatitis. He was ventilated and given intravenous piperacillin, metronidazole and gentamicin. Plasma volume expanders and inotropic support with dopamine and dobutamine infusions were also initiated. However, his cardiovascular status deteriorated culminating in a cardio-respiratory arrest with electromechanical dissociation. Resuscitation was unsuccessful. Blood cultures grew *Streptococcus pyogenes*, Lancefield Group A, type T1 M1 and autopsy revealed no source of infection.

Group A streptococcal infections account for only 2% of bacteraemias.[1] Fulminant streptococcal septicaemia is rare and may occur in previously healthy adults without any recognized primary focus of infection.[2,3] The clinical scenario of hypotension and multi-organ failure is reminiscent of the staphylococcal toxic shock syndrome and the term 'toxic strep syndrome' has been suggested.[4] The pathogenesis of this toxic shock remains inconclusive. Group A streptococci do not possess endotoxin