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) differences¹ 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 cancer¹ and in a recent correspondence, Odeh² 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 PGE₂ compared with surrounding tissue.⁸ This PGE₂ may play a role in pathophysiological processes including tumour-related angiogenesis⁹ and depression of cellular immunity.¹⁰ Tumour-derived PGE₂ may therefore further tumour growth in the colon.¹¹ By contrast, inhibiting PGE₂ 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