the same fault (Dr R D Turner, 25 February, p 643). In reply the authors defend their questions in the same way that I supported mine.[2] They admit they do not know of a perfect questionnaire. Perhaps bias, like beauty, lies in the eye of the beholder. Even the apparently neutral question "Would you like a cup of tea?" could be construed as being biased against coffee.

Two disturbing matters arise. Firstly, Dr Pringle and others found even more patients than I did who have worries about computerisation of medical records—31% compared with 19% on the issue of medical confidentiality—although their sample is admittedly much smaller. It is astonishing therefore that they conclude in their discussion that "patients have few worries concerning computerisation of their medical records."

Secondly, they say that "measures taken to avoid this threat [to confidentiality] may reduce the value of computers in medicine." What is paramount: the value of computers or the patients themselves? Discussion should now be directed away from argument over "biased" questionnaires and towards those patients' fears that have been clearly expressed. Just how are general practitioners using, or intending to use, computers going to deal with them?

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## Multiple spontaneous ruptures of tendons in renal transplant recipient

SIR,—Dr A Bradlow reports (4 February, p 364) on the spontaneous rupture of tendons in a patient who had had a renal transplant, and speculates whether it is related to immunosuppressive treatment and why this complication has not yet been reported.

Among 1000 patients who have had renal transplants, we observed two similar cases, and we feel that this is probably due to steroid treatment.

*Case 1*—A woman with chronic glomerulonephritis underwent haemodialysis for 14 months. At 26 years she had a renal transplant with en bloc child kidneys and we treated her with azathioprine 1·5 mg/kg and methylprednisolone 8 mg/day. After one mild rejection episode tolerance was excellent. Six years after she had had her transplant, she complained of pain and tightness in both heels. Two months later while rising from her bath, she felt a sudden pop in the right heel, where she had ruptured her Achilles tendon. The foot was immobilised for six weeks. Steroid dosage was changed to 16 mg on alternate days and after three years rupture has not recurred.

*Case 2*—A man with chronic glomerulonephritis underwent haemodialysis for two months before receiving a cadaver graft at the age of 39. No rejection episode occurred. Seventeen years later after a long walk he complained of pain in both heels, and a few days later, while driving, he braked suddenly and felt a pop around the right Achilles tendon. Pain and inability to extend the foot were still present after three weeks, when he attended the transplant clinic. A ruptured Achilles tendon with a palpable gap was found and successfully repaired by surgery. Biopsy of the tendon showed only non-specific lesions: areas of necrosis with some granulation tissue; no amyloid substance was found.

In both patients spontaneous rupture of the Achilles tendon occurred more than five years after a well tolerated transplantation. Like the patient of Dr Bradlow and others, our patients had normal renal function, no biological or radiological signs of hyperparathyroidism, and no evidence of amyloidosis or any other systemic disease. The fact that tendon rupture has been reported after local corticosteroid injection[1] and after oral steroid treatment in systemic lupus erythematosus, asthma, and renal transplantation suggests a causal role for corticosteroids.[2-4]

Tenderness and pain in Achilles tendons were experienced a few days or weeks before rupture,[4-6] probably because of minute tears in the tendon.[4] The suggestion that preventive measures applied at an early stage may reduce the risk of rupture is supported by a third case. A 50 year old man developed progressive leg weakness associated with bilateral heel tenderness three months after transplantation. Achilles tendons were exquisitely painful on palpation and a small haematoma appeared at the insertion site of the right tendon. This was interpreted as an impending rupture. Rest and elevation of the heel were advised, and steroid therapy was decreased—from 25 to 10 mg/day. Clinical and electrophysiological signs of myopathy disappeared, as did pain in the heel. Up to four years later the patient has not experienced rupture of the tendon.

Tendon rupture is thus a rare complication of renal transplantation. Corticosteroid treatment appears to be an important causal factor. Early diagnosis—at the stage of initial tear—and rest of the affected tendon might prevent subsequent rupture.

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## Ultrasonic measurement of spinal canal

SIR,—In their survey of back pain in general practice Dr J N Drinkall and his colleagues (14 January, p 121) report that ultrasonic measurement shows that the spinal cord is significantly narrower in those who suffer from back pain than in controls but consider measurement of the canal to be of little value for the management of back pain within general practice.

We believe that the measurement could be of value in occupational groups with a high prevalence of back pain. A study of the relation between the diameter of the spinal canal and back pain in miners found that those men with the longest histories and longest times off work and those who had to leave the coal face or the industry had significantly narrower canals than their colleagues.[1] In a six year period over 32% of lost time because of back pain was accounted for by men whose canal measurements lay within the narrowest decile.

We therefore suggest that the pre-employment measurement of the spinal canal by ultrasound, in combination with other known risk factors such as strength,[2] might identify those workers most susceptible to back pain. Thus there may be potential for effective screening, which could significantly reduce morbidity for the individual, with consequent benefits to industry and society.

The difficulty with the ultrasonic technique is that it is highly dependent on the skill of the operator and liable to error. Only a few centres have been able to confirm Porter's early results.[3-5] We are currently undertaking further evaluation of the method and a careful prospective study of the importance of spinal canal size and strength.

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## Paranoid psychosis induced by tocainide

SIR,—Further to the patients described by Dr P Currie and Dr D R Ramsdale (25 February, p 606) we report a case of acute paranoid psychosis and leucopenia related to the use of tocainide.

A 79 year old woman had a 12 year history of recurrent ventricular tachycardia that had failed to respond to many antiarrhythmic drugs, and she had required DC cardioversion on several occasions. After an episode of ventricular tachycardia tocainide, 400 mg three times daily, was added to her regimen of amiodarone and disopyramide. Several days later she developed progressive paranoid symptoms with hallucinations. Biochemical and bacteriological investigations showed no abnormality, but her peripheral leucocyte count had fallen from 5·4 to $2{\cdot}1 \times 10^9$/l. An acute confusional and aggressive paranoid state, probably of organic origin, was diagnosed.

In the absence of any other cause for her psychosis and leucopenia and in view of the temporal relationship to tocainide, this drug was withdrawn. Her psychosis and leucopenia both resolved quickly. All other drug treatment was unchanged throughout this period.

The dose given might have been high for a patient of this age, and unfortunately the plasma concentration of tocainide was not measured so that a toxic reaction could not be distinguished from an idiosyncratic one. We