Nevertheless, it is unclear why Asians have a higher prevalence of ApoEε3/3 but also higher prevalence and earlier onset of WD compared to Caucasians. It may be that ApoEε3/3 provides only mild neuroprotection. Definition of WD onset is also sometimes difficult: the presymptomatic ‘cause-unknown’ purpura2 is usually identified only after explicit gut haemorrhage; the earlier-onset cases may be induced by some unusual event such as head trauma, or neural inflammation.

Finally, although ApoE4 has been often considered a risk factor in Alzheimer’s disease, we found no negative effect of ApoEε4 in WD.

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Bladder rupture: a rare complication of tonic-clonic seizures

Sir,

A 52-year-old man taking flufenazine, resperidone and trihexyphenidyl for schizophrenia had two generalized tonic-clonic seizures within an hour. During the second, he was incontinent of faeces but not urine. In a nearby hospital, he was treated with iv phenytoin and a urinary catheter was inserted which drained 10 ml of bloodstained urine. The patient was subsequently transferred to our care because of continuing coma and anuria.

At the time of transfer, he was apyrexial and normotensive. His abdomen was distended and the sign of shifting dullness could be elicited. Abdominal ultrasonography showed an empty bladder and confirmed the presence of ascites. The ascitic fluid contained 700 mg/dl protein, 135 mg/dl sugar, and 850 cells/mm3 (90% neutrophils); smear was negative for acid-fast bacilli and malignant cells. He was treated with phenytoin, ceftriaxone, metronidazole and iv fluids. Haemodialysis was carried out on days 3 and 5 after admission because of a rising serum creatinine and continuing anuria. On day 6, following a hypotensive episode, a cystogram was performed, which showed extravasation of dye into the peritoneal cavity (Figure 1). Rupture of the urinary bladder was diagnosed and surgical repair considered but not implemented because of the patient’s improving general condition. Seven days later, both repeat cystogram and serum creatinine were normal, and he was discharged.

In our patient, presence of haematuria, absence of urinary output and increasing abdominal distension were highly suggestive of a ruptured urinary bladder. Following grand mal seizure, urinary incontinence is common, whereas our patient had faecal incontinence only, suggesting that bladder rupture might have occurred during one of the

Correspondence
convulsions. Our patient had a seizure in the morning after overnight retention of urine. The anticholinergic effects of his drug treatment for schizophrenia may have been a contributing factor. Moreover, schizophrenic patients have been reported to have detrusor hypereflexia.1 Full bladder with possible outflow obstruction in presence of raised intraabdominal pressure during convulsions could have resulted in a ruptured bladder in our patient. The cystogram in our patient revealed the rent in the dome of the diaphragm. The bladder is protected anteriorly by pubic bone, inferolaterally by the urogenital diaphragm and obturator internus, leaving the dome unprotected, covered only by peritoneum. Our patient may have some similarity with two patients who developed bladder rupture following electroconvulsive therapy.1,2 In these patients, bladder rupture was attributed to powerful abdominal muscle contraction during an unmodified seizure on a distended bladder on a background of anticholinergic medication.

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Increase of heat shock protein with exercise

Sir,

Dr Press and colleagues1 make a forceful case in their editorial for the beneficial effect of exercise in the prevention of coronary artery disease. We would however like to point out that there is a large body of literature on the role of heat shock protein (HSP) in providing cellular protection in a variety of stresses.2 Exercise induces HSP in skeletal muscle, and exercise training increases HSP in the skeletal muscle of old rats.2 A role for HSP in cardioprotection has been postulated, and several studies addressing the cytoprotective effect of HSP in experimental models of cardiac ischaemia have been summarized in a review by Benjamin and McMillan.2

An approach to preventive measures is often difficult, as one is often trying to correct a risk that may not be perceived as imminent. Efforts to reduce cardiac risks by voluntary measures such as exercise often require a great deal of motivation, and the desired outcome (i.e. avoidance of a coronary event several years later), may not be completely appreciated by individuals. Additionally, physicians vary in their ability to emphasize the beneficial effects of exercise to their patients. This pattern of behavior is known as ‘clinical inertia’, and has been linked to poor control of hypertension and diabetes mellitus, despite awareness of evidence-based guidelines.4

The editorial1 is highly significant in its efforts to compile and summarize a large body of evidence on the beneficial effects of physical exercise in the prevention of coronary artery disease, which could be applied to a majority of our patients.

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Figure 1. Cystogram showing Foley balloon in the peritoneal cavity with extravasation of contrast medium.