Although echocardiography was unremarkable and neither bronchial nor cardiac biopsies were performed, the presumed explanation for the symptomatology was allergic alveolitis and myocarditis. The decision not to biopsy, but to treat on empirical grounds was based on clinical judgement. The response to treatment justified the conservative management.

Numerous organ systems have been involved in clozapine-induced allergy; myocarditis (Bandelow *et al*, 1995), colitis (Friedberg *et al*, 1995), pancreatitis (Chengappa *et al*, 1995), hepatitis (Thatcher *et al*, 1995) and cutaneous reactions (Stoppe *et al*, 1992) have been reported. Those reactions are invariably associated with eosinophilia.

To date, only one case of clozapine-induced allergic asthma has been reported (Stoppe *et al*, 1992). This case suggests that a separate entity be recognised: that of clozapine-induced allergic alveolitis. In this case the patient's symptoms showed rapid progression.

Pyrexia, although frequently a benign side-effect of clozapine, in fact, indicates the need for thorough investigation and clinical vigilance. The possibility of potentially lethal cardio-respiratory complications should be considered early, especially where there is eosinophilia, and a chest X-ray, electrocardiogram, erythrocyte sedimentation rate and echocardiograph may be regarded as necessary investigations for pyrexia.

The diligent reporting of such cases may assist in the identification of predictors of such potentially lethal allergic compilations, for example age, history of smoking, length of clozapine treatment and previous cardiorespiratory disease.

In terms of progress, the pyrexia abated two days after the cessation of clozapine. A chest X-ray performed five days after discontinuation of the clozapine showed complete resolution of the interstitial shadowing. The erythrocyte sedimentation rate dropped to 42. The patient showed dramatic clinical improvement.

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Prolonged bradycardia complicates antidepressive treatment with venlafaxine and ECT

Sir: We describe the case of a patient given venlafaxine and electroconvulsive therapy (ECT), where cardiovascular complications led to interruption of the ECT.

A 42-year-old man with a severe depressive disorder and suicidal ideation failed to improve despite venlafaxine (300 g/day) combined with flurazepam (15 g/day) over 44 days. Thoracic X-radiography, electroencephalogram (EEG), computed tomography, laboratory testing and serial electrocardiogram (ECG) recordings were all normal during venlafaxine treatment and before ECT. On the 45th day venlafaxine was reduced to 150 g/day (flurazepam unchanged). On the 46th day, 0.75 g atropine, 100 g propofol, 0.5 g norcurone and 50 g succinylcholine were given immediately before ECT (ThymatronDG, bilateral brief pulse, 100.8 mC). A generalised cramp was induced lasting 40 seconds. A rapid reduction in heart rate was followed by an asystole. After another atropine dose (0.5 g), a bradyarrhythmia (22–40 bpm) developed (for about 90 seconds) which spontaneously led to a bradycardial sinus rhythm. During three hours a normal sinus rhythm returned. Depressive symptoms completely receded with hypomania lasting about 16 hours; afterwards the depressive profile returned to its full extent. The cardiovascular events observed prompted further tests (echocardiography, 24-hour recording of ECG and blood pressure, stress-ECG) which produced normal results. Therapy administered during this phase (lithium, moclobemide, lorazepam) was unsuccessful. Six weeks later a second ECT course (seven rightside unilateral, one bilateral; 100.8–201.6 mC) did not produce cardiovascular complications. All psychopharmaceuticals except lorazepam had been discontinued 14 days before and the same pre-medication as given for the first ECT session. Starting with the third ECT, depressive symptoms gradually resolved.

Temporary arrhythmias and repolarisation abnormalities can occur in cardiologically-healthy subjects and may be physiological side-effects of ECT (Abrams, 1992). However, in our patient such effects were observed only after the combined use of ECT and venlafaxine and not at all after eight ECTs without antidepressants. ECT may enhance central serotonergic responsivity (Shapira *et al*, 1992). Venlafaxine inhibits both serotonin and noradrenaline uptake. We hypothesise that our patient experienced long-lasting bradycardia because increased serotonin in the central nervous system directly affected brainstem cardiovascular regulation (Shvaloff & Laguzzi, 1986). Alternatively, unknown interactions between venlafaxine and the anaesthetics used may have caused the adverse effect. Nevertheless, clinicians should note that prolonged bradycardia can complicate the combined use of venlafaxine and ECT.

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Doxazosin for reboxetine-induced urinary hesitancy

Sir: Reboxetine is a selective potent noradrenaline uptake inhibitor with clinically documented antidepressant properties. The drug is usually well tolerated; however, especially in elderly male patients, urinary hesitancy and/or retention can be a troublesome side-effect (Berzewski *et al*, 1997). Recently, we have found that this side-effect can be mitigated by the co-administration of doxazosin, an α_1-adrenoceptor antagonist indicated for the treatment of urinary retention associated with prostatism (Dollery, 1991).