Patient	UPDRS		MMS		BPRS		Hallucinosis score	
	B	A	B	A	B	A	B	A
1	68	64	17	18	49	24	4	1
2	63	72	19	18	42	25	3	0
3	62	74	16	17	45	30	2	0
4	79	79	16	18	40	23	4	0
5	43	38	18	19	38	21	4	1
6	34	25	17	17	39	29	4	1
Mean (SD)	58·17 (16·63)	58·67 (21·98)	17·17 (1·17)	17·83 (0·75)	42·17 (4·17)	25·33* (3·50)	3·50 (0·84)	0·5* (0·55)

*p<0·05 *vs* B values (Wilcoxon's test).

Table: **Results before (B) and after (A) risperidone**

beginning of the study and during treatment with the unified Parkinson's disease rating scale (UPDRS), mini-mental state (MMS), and brief psychiatric rating scale (BPRS); moreover, the patients filled out, with family assistance, a hallucinosis questionnaire (scoring 0–5).

The doses of risperidone were increased up to a maximum of 0·67 (0·47) mg per day (range 0·25–1·25); the mean duration of the treatment was 14·67 (7·34) weeks (range 8–24). Hallucinations improved significantly in all patients and disappeared in 3 (50%); the BPRS scores significantly improved, whereas the MMS remained unchanged (table). The basal motor symptoms measured by UPDRS did not worsen and the total daily levodopa doses did not change (before 556 [209] mg; after 622 [204]). The number of hours spent in an "on" state before and after risperidone was unchanged. Risperidone was well tolerated; only 2 patients reported mild increases in salivation and 1 other had temporary hypotension.

These results suggest that risperidone may effectively and safely control hallucinations in levodopa-treated patients with Parkinson's disease without causing a worsening of extrapyramidal symptoms.

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Symptomatic junctional bradycardia due to lithium intoxication in patient with previously normal electrocardiogram

SIR—Symptomatic tachyarrhythmia and bradyarrhythmia induced by lithium have been reported in patients with pre-existing conductive disease and abnormal electrocardiograms (ECG). We report a patient who had a previously normal ECG who presented with symptomatic junctional bradycardia due to lithium intoxication.

A 75-year-old woman collapsed in an orthopaedic hospital a week after an operation. She had no chest pain, but her ECG showed bradycardia for which she was given two doses of 600 mg of atropine intravenously and urgently transferred to our hospital. She had no history of cardiovascular disease or diabetes mellitus but had been taking phenytoin for epilepsy and lithium for depression for more than 20 years. Warfarin had been administered after surgery. On arrival, she was conscious but withdrawn, her heart rate was 42 per min, blood pressure 90/60 mm Hg, general examination was unremarkable, and a 12-lead ECG showed junctional bradycardia with narrow complexes and no evidence of ischaemic or intraventricular conductive disorder. All drugs were discontinued. She had evidence of renal dysfunction (urea 10 mmol/L, sodium 135 mmol/L, potassium 4·2 mmol/L, and creatinine 211 μmol/L). Magnesium, calcium, and cardiac enzymes were normal. Her phenytoin and warfarin serum concentrations came back within the therapeutic range but lithium was 2·13 mmol/L (normal 0·8–1·2). After rehydration her condition improved and by the fourth day her ECG had returned to sinus rhythm with no evidence of ischaemic or conductive abnormalities. She has remained well and symptom-free until now (5 months after discharge).

Sinus arrest and asystole have been reported in a patient with severe lithium intoxication but his recovery and follow-up ECG showed right bundle branch block and left axis deviation.[1] Rosenquist and colleagues concluded that lithium treatment at therapeutic concentrations is unlikely to cause conductive disorders in patients with normal hearts and ECGs.[2] In contrast, our case shows reversible symptomatic junctional bradycardia associated with lithium intoxication in a patient whose ECG suggests otherwise normal conduction. Electrophysiological study was not felt to be justified in view of her complete recovery. There has been no mention of phenytoin in therapeutic doses causing such an effect or potentiating lithium toxicity to the heart.

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Iliac artery dissection in α_1-antitrypsin deficiency

SIR—The role of α_1-antitrypsin (α_1-AT) deficiency in the pathogenesis of arterial aneurysm or dissection is controversial. Schievink and colleagues (Feb 19, p 452) report 3 cases of ruptured intracranial aneurysm and 1 of spontaneous dissection of the cervical internal artery among 362 patients with α_1-AT deficiency. Likewise, the heterozygous PiMZ phenotype is more common than predicted in patients with abdominal aortic aneurysms.[1] On the other hand, Elzouki and Erikkson (April 23, p 1037), recorded no arterial abnormality in a retrospective study of 30 consecutive homozygous PiZZ patients who underwent necropsy. A shortened lifespan of homozygous PiZZ patients might explain these negative results. We report a patient in whom iliac artery dissection revealed α_1-AT deficiency.

A 34-year-old man presented with a regressive acute arterial occlusion of the left leg which arose 48 h before