Importantly, postoperative imaging in these three patients confirmed that the lesions were confined to the medial pallidum (without extension to the internal capsule or lateral pallidum as observed in four others).[1]

Although we did not perform magnetic resonance imaging or electromyography studies of the paraspinal muscles, we believe that this truncal lateroflexion results from dystonia or asymmetric rigidity and not from a unilateral paraspinal myopathy.

The main question is whether this leaning towards one side is merely a phenomenon of an advanced stage Parkinson's disease or a hitherto unrecognised delayed-onset consequence of unilateral pallidotomy in Parkinson's disease.

Unilateral pallidal lesions in rats result in curling and head turning towards the side contralateral to the lesion.[2] The rarely reported acquired unilateral pallidal lesions in humans seem to particularly give rise to contralateral limb dystonia, hemidystonia or hemiparkinsonism rather than to axial abnormalities.[3] If the leaning in our patients is directly related to the pallidum lesion, the delay of 4–9 years after pallidotomy is rather difficult to explain, although delayed-onset progressive dystonia has been reported in bilateral anoxic pallidal lesions.

Previous observations noted the common presence of scoliosis in Parkinson's disease and postencephalitic parkinsonism, which was usually concave to the clinically less affected side—that is, directed towards the side with more severe nigrostriatal pathology.[4 5] This is corroborated by animal studies, as rodents with unilateral lesions of the substantia nigra display a deviated spinal curvature and/or abnormal turning behaviour directed towards the lesioned side; however, when these animals are given dopaminergic agents, their body asymmetry reverses from ipsiversive to contraversive.[6] In a 6-hydroxydopamine rat model of Parkinson's disease, with a unilateral substantia nigra lesion causing ipsiversive body axis deviation without and contraversive turning with dopamine agonists, a unilateral pallidotomy (ipsilateral to the substantia nigra lesion) alleviated both body axis asymmetry and abnormal turning.[2] The human correlate seems to be the notion that the scoliosis to the right in patients with postencephalitic parkinsonism with clinically more left-side than right-side involvement was corrected after a right pallidotomy.[5]

Further extrapolation to our patients is impossible because animal models or human data that predict the net effect of a unilateral pallidal lesion in a system of bilateral but asymmetrical nigrostriatal dopamine deficiency and chronic exposure to dopaminergic agents on truncal posture are not available. Consequently, we do not know whether the Pisa syndrome in our patients parallels advanced Parkinson's disease or actually represents an unrecognised delayed effect of unilateral pallidotomies in patients with Parkinson's disease.

We would like this letter to serve as an invitation to continue reporting on the follow-up of pallidotomy in patients, including less obvious clinical and easily overlooked features such as a lean to one side.

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Informed patient consent was obtained for the publication of details of the patients.

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References

1 **Schrag A**, Samuel M, Caputo E, *et al*. Unilateral pallidotomy for Parkinson's disease: results after more than 1 year. *J Neurol Neurosurg Psychiatry* 1999;**67**:511–17.
2 **Henderson J**, Doherty K, Allbutt H, *et al*. Effects of pallidotomy on motor symptoms in an animal model of Parkinson's disease. *Behav Brain Res* 2006;**169**:29–38.
3 **Bhatia KP**, Marsden CD. The behavioural and motor consequences of focal lesions of the basal ganglia in man. *Brain* 1994;**117**(Pt 4):859–76.
4 **Duvoisin RC**, Marsden CD. Note on the scoliosis of parkinsonism. *J Neurol Neurosurg Psychiatry* 1975;**38**:787–93.
5 **Martin JP**. Curvature of the spine in post-encephalitic parkinsonism. *J Neurol Neurosurg Psychiatry* 1965;**28**:395–400.
6 **Schwarting RK**, Huston JP. The unilateral 6-hydroxydopamine lesion model in behavioral brain research. Analysis of functional deficits, recovery and treatments. *Prog Neurobiol* 1996;**50**:275–331.

CORRECTIONS

K Talbot. Amyotrophic lateral sclerosis, 2nd edn (*J Neurol Neurosurg Psychiatry* 2007;**78**:109). In this book review the acronym TMS was incorrectly expanded to ''traumatic masturbatory syndrome''; it should actually be ''transcranial magnetic stimulation''. In addition, the first sentence should read:

Amyotrophic lateral sclerosis, through its first edition, has become the standard text for clinicians and researchers in the field of ALS/MND.

The online version has been corrected. We sincerely apologise for these errors introduced on copyediting.

A Larner. How to examine the nervous system, 4th edn (*J Neurol Neurosurg Psychiatry* 2007;**78**:110). In this book review the book details were incorrectly published. The correct book review details are:

Edited by R T Ross. Published by Humana Press, New Jersey, 2006, £36.00 (hardback), pp 242. ISBN 1-58829-811-6.

The online version has been corrected. We sincerely apologise for this error introduced on copyediting.