Finally, as dopamine has been considered to take part in the seizure control system, our results suggest that recurrent seizures could induce an increase in the turnover in dopamine, resulting in a secondary depletion in striatum and SN.

Franck Semah, Viviane Bouilleret, Maria-Joao Ribeiro, Orsay, France

Disclosure: The authors report no disclosures.

1. Bouilleret V, Semah F, Chassoux F, et al. Basal ganglia involvement in temporal lobe epilepsy: a functional and morphologic study. Neurology 2008;70:177–184.
2. Goldstein DS, Nadi NS, Stull R, Wyler AR, Porter RJ. Levels of catechols in epileptogenic and non-epileptogenic regions of the human brain. J Neurochem 1988;50:225–229.
3. Ribeiro MJ, Vidailhet M, Loc'h C, et al. Dopaminergic function and dopamine transporter binding assessed with positron emission tomography in Parkinson disease. Arch Neurol 2002;59:580–586.
4. Deransart C, Le-Pham BT, Hirsch E, Marescaux C, Depaulis A. Inhibition of the substantia nigra suppresses absences and clonic seizures in audiogenic rats, but not tonic seizures: evidence for seizure specificity of the nigral control. Neuroscience 2001;105:203–211.
5. Deransart C, Riban V, Lê B, Marescaux C, Depaulis A. Dopamine in the striatum modulates seizures in a genetic model of absence epilepsy in the rat. Neuroscience 2000; 100:335–344.
6. Depaulis A, Moshé SL. The basal ganglia and the epilepsies: translating experimental concepts to new therapies. Epileptic Disord 2002;4(suppl 3):S7–S93.
7. Starr MS. The role of dopamine in epilepsy. Synapse 1996; 22:159–194.

CORRECTION

Are initial demyelinating event recovery and time to second event under differential control?

In the article "Are initial demyelinating event recovery and time to second event under differential control?" by T. West et al. (*Neurology*® 2006;67:809–813), the authors concluded from their statistical analysis that the disease-modifying therapy conferred an increased risk of a second event. On repeat analysis, they discovered that the numerator and denominator had been reversed and that the disease-modifying therapy actually conferred decreased risk of a second event. The univariate HR should have been reported as 0.38 with a 95% confidence interval from 0.23 to 0.62. This error does not change the other results of the study. The authors regret the error.

CORRECTION

Familiality in brain tumors

In the article "Familiality in brain tumors" by Deborah T. Blumenthal and Lisa A. Cannon-Albright (*Neurology*® 2008; 71:1015–1020), the titles of tables 1 and 2 are incorrect. They should read as follows:

Table 1 Relative risks for brain tumor among first-degree relatives of patients with brain tumor

Table 2 Relative risks for brain tumor among second-degree relatives of patients with brain tumor

The publisher apologizes for the errors.

CORRECTION

Neurology 2008;71;1841
DOI 10.1212/01.wnl.0000339383.07826.36