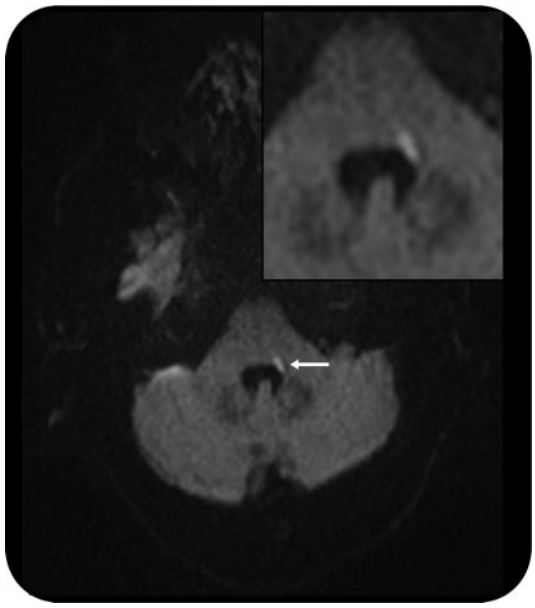
An unusual cause of isolated vomiting

Figure Diffusion-weighted image showing diffusion restriction in the left area postrema (arrow)



A 76-year-old woman with hypertension and diabetes presented with acute-onset vomiting. Her neurologic examination was unremarkable. When vomiting continued despite antiemetic therapy, brain MRI and magnetic resonance angiography were performed. These revealed an isolated region of restricted diffusion in the left area postrema (figure) together with basilar artery stenosis and filiform stenosis of the left vertebral artery.

The rich vascularization of the area postrema, which is supplied from branches of the anterior cerebellar and the vertebral arteries,[1] may account for the lack of reported cases of infarctions of this medullary structure. While one small case series found that surgical ablation of the area postrema relieved patients of intractable vomiting,[2] our patient showed severe vomiting following infarction of only part of the area postrema. These contrasting findings imply that ischemia induced irritation, but not suppression, of the area postrema in our patient.

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1. McKinley MJ, McAllen RM, Davern P, et al. The Sensory Circumventricular Organs of the Mammalian Brain. Berlin: Springer-Verlag; 2003:32.
2. Lindstrom PA, Brizzee KR. Relief of intractable vomiting from surgical lesions in the area postrema. J Neurosurg 1962;19:228–236.

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INTELLECTUAL ENRICHMENT LESSENS THE EFFECT OF BRAIN ATROPHY ON LEARNING AND MEMORY IN MULTIPLE SCLEROSIS

To the Editor: Sumowski et al.[1] showed that greater intellectual enrichment lessened the negative effect of brain atrophy on cognitive performance in multiple sclerosis (MS). Patients with MS with high intellectual enrichment performed similarly in memory tasks regardless of brain atrophy values on MRI while brain atrophy was associated with lower performances in patients with low intellectual enrichment.

We previously showed that educational background protects cognitive efficiency in patients with newly diagnosed relapsing-remitting MS (RRMS).[2] Low-educated (LE) patients with RRMS, but not high-educated (HE) patients, had lower performances than matched healthy controls on most neuropsychological tests including the memory test used by Sumowski et al. However, cognitive scores between LE and HE controls showed no difference. LE and HE patients with RRMS did not differ regarding age, gender, disability, or MRI measures, so the effect of education on cognition was not attributable to a different distribution by chance.

Sumowski et al. only observed a correlation between brain atrophy and cognitive performance in patients with low intellectual enrichment. In our study, almost none of the MRI measures correlated with cognitive scores in LE patients, which suggests that a limited amount of tissue damage could be sufficient to induce cognitive disturbances in these patients. In addition, lower compensatory capacities in these patients could not counteract the cognitive deficits related to brain involvement. By contrast, cognitive performances of HE patients were negatively correlated with MRI measures, indicating that education-dependent cognitive compensation, as we showed for cognitive compensation in RRMS,[3] could be limited by tissue damage accumulation and disease progression.

Both studies demonstrated 2 different intellectual enrichment variables and the protective effect of a strong intellectual background on brain damage. However, we obtained different results according to MRI correlations, which could be explained by differences in the 2 MS populations, including disease course (we studied only early RRMS); number of years of education (16.1 ± 2.3 years vs 13 ± 2.9 years); or disease duration (10.5 ± 7 years vs 2 ± 2.19 years).

Further studies are needed to understand the compensatory effect of intellectual background on cognitive efficiency preservation in patients with MS. The ceiling effect due to the progression of the disease should also be elucidated.

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Reply from the Authors: We reported negative correlations between brain atrophy and cognition in patients with MS with lower reserve, but not patients with higher reserve.[1,4] In contrast, Bonnet et al.[2] found only one correlation between MRI measures and cognition in patients with lower reserve, but several correlations among patients with higher reserve.

The cognitive reserve hypothesis states that patients with higher reserve can withstand more severe neurologic disease before suffering cognitive decline.[5] The negative impact of neuropathology on cognition is weaker among higher reserve patients, as we have shown in MS[1,4] and others have shown in aging and Alzheimer disease.[6,7] In this context, the findings of Bonnet et al.[2] conflict with the cognitive reserve literature, and therefore prompted this review of their study.

Bonnet et al. defined low reserve as educational attainment less than 12 years. However, the LE MS group had much lower verbal intelligence than the LE control group. Considering that verbal intellectual decline is very rare in MS,[8] the MS group probably had lower intelligence before disease onset. The observed cognitive differences between the LE MS and control groups likely represent premorbid differences rather than disease-induced cognitive decline, especially given the short disease duration of the MS group.

Because lower cognition among patients with MS was likely developmental rather than disease-induced, a correlation between MRI measures and cognition cannot be expected. Among HE patients with MS, Bonnet et al. reported correlations between MRI parameters and performance on 8 cognitive tasks. However, patients with MS only differed from controls on one task, suggesting that most of the correlations were due to an uncontrolled nondisease variable, such as age, which likely correlates with both cognition and MRI measures. If so, correlations should also be evident among controls, but these analyses were not performed. Finally, differential statistical power is another explanation for fewer correlations among LE patients (n = 19) relative to HE patients (n = 24), especially given the high risk of type I error (5 MRI parameters × 15 cognitive scores = 75 correlations performed for each group, with an uncorrected α of 0.05).

The cognitive reserve literature supports the notion that neuropathology is more likely related to cognition among patients with lesser reserve,[1,4–7] and the exception posed by Bonnet and colleagues[2] appears confounded.

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Disclosure: See original article for full disclosure list.

1. Sumowski JF, Wylie GR, Chiaravalloti N, et al. Intellectual enrichment lessens the effect of brain atrophy on learning and memory in multiple sclerosis. Neurology 2010;74:1942–1945.
2. Bonnet M, Deloire M, Salort E, Dousset V, Petry KG, Brochet B. Evidence of cognitive compensation associated with educational level in early relapsing-remitting multiple sclerosis. J Neurol Sci 2006;251:23–28.
3. Bonnet M, Allard M, Dilharreguy B, Deloire M, Petry KG, Brochet B. Cognitive compensation failure in multiple sclerosis. Neurology 2010;75:1241–1248.
4. Sumowski JF, Chiaravalloti N, Wylie GR, DeLuca J. Cognitive reserve moderates the negative effect of brain atrophy on cognitive efficiency in multiple sclerosis. J Int Neuropsychol Soc 2009;15:606–612.
5. Stern Y. Cognitive reserve. Neuropsychologia 2009;47:2015–2028.
6. Bennett DA, Wilson RS, Schneider JA, et al. Education modifies the relation of AD pathology to level of cognitive function in older persons. Neurology 2003;60:1909–1915.
7. Rentz DM, Locascio JL, Becker JA, et al. Cognition, reserve, and amyloid deposition in normal aging. Ann Neurol 2010;67:353–364.
8. Chiaravalloti ND, DeLuca J. Cognitive impairment in multiple sclerosis. Lancet Neurol 2008;7:1139–1151.

CORRECTION

An unusual cause of isolated vomiting

In the Neuro*Image* "An unusual cause of isolated vomiting" by R. Schlaeger and M. Sollberger (*Neurology*® 2010;75:1303), the small ischemic lesion shown in the figure is not in the left area postrema, but likely a few millimeters above. The authors are still convinced that the depicted lesion is related to the therapy-refractory vomiting, either by affecting parts of a network of interacting neuronal structures related to vomiting or by irritation of the area postrema, for instance by ischemia-induced, transitory bleeding on the lower surface of the fourth ventricle. The authors regret this error.