have been those of LKA, although Lang et al did not clarify the type of apraxia. A neuropathologic study of this patient revealed conspicuous cortical atrophy in the right precentral and postcentral gyri. Asymmetric perirolandic cortical atrophy could account for the apraxic disorders, as a lesion in the sensorimotor cortex induces LKA on the contralateral side. In agreement with the neuropathologic findings, single-photon emission computed tomography (SPECT) studies of our four patients consistently showed an asymmetric decrease in cerebral blood flow in the perirolandic cortices.

The areal distributions of cortical involvement of our patients resembled those of the patient reported by Lang et al, with some differences. In addition to LKA and parkinsonism, three of our four patients presented with constructional disabilities and two with supranuclear vertical gaze palsy. SPECT studies of the three patients with constructional disabilities suggested cortical degeneration in the posterior parietal region, which was not detected in the report by Lang et al. Supranuclear gaze palsy occurring in our patients resembled that of progressive supranuclear palsy (PSP). On the basis of the above clinical manifestations, our patients were diagnosed as having CBGD. Neuropathologic studies suggested that CBGD might share the degenerative process of PD or PSP. The distribution of cortical damage of CBGD resembles that of parietal PD, and the distribution of subcortical damage of CBGD resembles that of PSP. Leiguarda et al reported that ideomotor apraxia is the most frequent type of apraxia in CBGD, but we disagree. The report by Lang et al supports our observations that LKA is the most frequent type of apraxia in CBGD, as the perirolandic cortices are frequently involved in CBGD as well.

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Reply from the Author: We would like to thank Drs. Okuda and Tachibana for their comments. There is a clear need for more clinicopathologic correlation in patients proven to have suffered from CBGD. As indicated in the table in our article, there are many sources of potential misdiagnosis. This disorder involves both the cerebral cortex and the basal ganglia, the latter resulting in the universal presence of an akinetic-rigid syndrome (at least in the cases that can be diagnosed clinically in life). The accompanying bradykinesia, rigidity, and dystonia often make adequate testing for various types of apraxia extremely difficult and at times impossible. These disturbances frequently result in "a breakdown of previously skillful movements, manifested by disturbances of fine finger movements." If this is taken as the definition of LKA, as suggested by Drs. Okuda and Tachibana, then certainly we would agree on its extremely common occurrence. However, as indicated in our table, it is the misinterpretation of additional neurologic features as apraxia that has commonly resulted in patients being sent to us with a misdiagnosis of CBGD when, in fact, they suffered from some other asymmetric extrapyramidal disorder. In hopes of avoiding the overdiagnosis of CBGD, in the absence of several other classic features such as stimulus-sensitive myoclonus and cortical sensory loss, we would insist on the presence of ideomotor apraxia rather than LKA in the clinical support of a diagnosis of CBGD. We believe that a similar diagnostic approach may account for the findings of Leiguarda et al, with which Drs. Okuda and Tachibana take issue.

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References

Correction
In "A reduction in serum glucocorticoids provokes experimental allergic encephalomyelitis: Implications for treatment of inflammatory brain disease" by Reder et al, which appeared in the December 1984 issue (Neurology 1994;44:2289-2294), figures 4A and 4B were transposed. With the figures in the text as they are now, figure 4A is DEX-treated and figure 4B is the saline control. The discussion in the text is correct.
Correction
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