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 postcen-
tral 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 com-
puted tomography (SPECT) studies of our four patients consis-
tently 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 disabili-
ties and two with supranuclear gaze palsy. SPECT stud-
ies of the three patients with constructional disabilities suggest-
ed 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 distrib-
ution of subcortical damage of CBGD resembles that of PSP.
Leiguarda et al reported that ideomotor apraxia is the most fre-
quent type of apraxia in CBGD, but we disagree. The report by
Lang et al supports our observations that LKA is the most fre-
quent 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 lat-
ter resulting in the universal presence of an akinetic-rigid syn-
drome (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 move-
ments, 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 fea-
tures as apraxia that has commonly resulted in patients being
sent to us with a misdiagnosis of CBGD when, in fact, they suf-
ered 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 diag-
nostic approach may account for the findings of Leiguarda et al,
with which Drs. Okuda and Tachibana take issue.

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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 1994 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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