Figure 2. Typical mild extremity asymmetry in a patient with hemiparkinsonism and hemiatrophy with left thumb and foot smaller.

on the hemiatrophic side; (4) neuroradiologic evidence of cerebral pathology or asymmetry. In spite of these similarities, Dr. Jankovic's patient did not have a slowly progressive course and displayed hemidystonia rather than an action-induced dystonia. We would therefore rather classify his patient as one with prominent dystonia and parkin-

sonism rather than the hemiparkinsonism/hemiatrophy syndrome that we described in our article.^{1,5}

The recognition and separation of patients who presumably have secondary parkinsonism from those with IPD is important for further studies of IPD. In addition, the identification of these patients may have prognostic significance, since the natural history of patients with secondary parkinsonism may differ from that of IPD.

Aron S. Buchman, MD
Christopher G. Goetz, MD
Harold L. Klawans, MD
Chicago, IL

References

1. Buchman AS, Goetz CG, Klawans HL. Hemiparkinsonism with hemiatrophy. *Neurology* 1988;38:527-530.
2. Jankovic J, Orman J. Botulinum A toxin for cranial-cervical dystonia: a double-blind, placebo-controlled study. *Neurology* 1987;37:616-623.
3. Burke RE, Fahn S, Gold AP. Delayed onset dystonia in patients with "static" encephalopathy. *J Neurol Neurosurg Psychiatry* 1980;43:789-797.
4. Pettigrew LC, Jankovic J. Hemidystonia: a report of 22 patients and a review of the literature. *J Neurol Neurosurg Psychiatry* 1985;48:650-657.
5. Klawans HL, Paleologos N. Dystonia-Parkinson syndrome: differential effects of levodopa and dopamine agonists. *Clin Neuropharmacol* 1986;9:298-302.

Correction

For the article "Absence of contrast enhancement on CT brain scans of patients with supratentorial malignant gliomas," published in the September issue (Chamberlain et al, *Neurology* 1988;38:1371-1374), the authors have submitted a revised table 1, with corrected figures:

Table 1. Sex and tumor location in 229 patients with supratentorial malignant gliomas

Type of tumor	Sex (M:F)	Location				
		Fron	Par	Temp	Occip	Other*
GM (n = 93)	2.7:1					
Enhancing (n = 89)		29	31	20	9	0
Nonenhancing (n = 4)		4	0	0	0	0
GA (n = 10)	2.3:1					
Enhancing (n = 7)		2	3	1	0	1
Nonenhancing (n = 3)		2	1	0	0	0
HAA (n = 74)	2.5:1					
Enhancing (n = 51)		17	18	8	3	5
Nonenhancing (n = 23)		10	8	4	1	0
MoAA (n = 52)	1.5:1					
Enhancing (n = 24)		9	10	1	0	4
Nonenhancing (n = 28)		3	8	14	3	0

Fron Frontal.
Par Parietal.
Temp Temporal.
Occip Occipital.
GM Glioblastoma multiforme.
GA Gemistocytic astrocytoma.
HAA Highly anaplastic astrocytoma.
MoAA Moderately anaplastic astrocytoma.
* Thalamus, hypothalamus, pineal, basal ganglia, corpus callosum.

Neurology[®]

Correction

Neurology 1988;38;1816-1816-a
DOI 10.1212/WNL.38.11.1816-a

This information is current as of November 1, 1988

Updated Information & Services

including high resolution figures, can be found at:
<http://n.neurology.org/content/38/11/1816.2.citation.full>

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
http://www.neurology.org/about/about_the_journal#permissions

Reprints

Information about ordering reprints can be found online:
<http://n.neurology.org/subscribers/advertise>

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright © 1988 by Edgell Communications, Inc.. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.