

drochloride (Prolixin) and the thioxanthene thiothixene (Navane) follow haloperidol in potency.

The low-potency neuroleptic agents, including the aliphatic phenothiazine chlorpromazine (Thorazine) and the piperidine phenothiazine thioridazine (Mellaril), while associated with oculogyric crises, are much less likely to cause an oculogyric crisis. This can most likely be attributed to the inherent anticholinergic effect of these agents.<sup>2,3</sup>

ABRAHAM DOREVITCH, PIARMD  
Talbia Psychiatric Hospital  
PO Box 39  
Jerusalem, Israel

1. Clough CG, Plaitakis A, Yahr MD: Oculogyric crises and parkinsonism: A case of recent onset. *Arch Neurol* 1983;40:36-37.

2. Kessler KA, Waletzky JP: Clinical use of the antipsychotics. *Am J Psychiatry* 1981;138:202-208.

3. Johnston JA, Coleman JH, Callaway JT, et al: Protocols for the use of psychoactive drugs: Part I. Protocol for the treatment of psychosis with antipsychotics. *J Clin Psychiatry* 1980; 41:375-377.

### Pontine Ataxic Hemiparesis

*To the Editor.*—In a recent article, Ichikawa et al (ARCHIVES 1982;39:585-586) discussed the possible causes of ataxic hemiparesis. We report a case of ataxic hemiparesis with an ischemic lesion in the basis pontis, clearly shown by computed tomography (CT).

*Report of a Case.*—A 72-year-old man experienced weakness of the right arm and right leg on awakening and noticed some numbness of the right hand. On admission to the hospital two days later, his BP was slightly elevated (160/100 mm Hg). Physi-

cal and cardiac examination results were not remarkable. There was right-sided hemiparesis; strength was reduced to four fifths in the extensor muscles of the upper extremity and to three fifths in those of the lower extremity. There was a right plantar reflex to the extensor muscle, as well as mild ataxia in the right arm and severe ataxia in the right leg. The patient could not stand up without assistance. Sensation was normal. No visual field defects could be observed, and there was no speech disturbance. The patient had binaural perception deafness. The other cranial nerves, including those for ocular and facial motility function, were normal. There were no carotid bruits. The ECG and results of blood chemistry tests and CSF analysis were normal. An EEG showed a background rhythm of 8 to 9 Hz, sometimes slowing to 7 to 8 Hz; infrequent theta waves were recorded in the temporal leads. Brain-stem auditory evoked responses were not obtainable bilaterally due to hearing loss. A contrast-enhanced CT scan two weeks after onset showed a hypodense area (density, 17 hyperemia units; 10 mm in diameter) in the basis pontis on the left (Figure). Four weeks later, muscle strength was normal, but ataxia was still present.

*Comment.*—Ataxic hemiparesis is caused by different types of lesions, localized in the basis pontis, midbrain, or internal capsule and verified by CT scan or autopsy. Ischemic,<sup>1</sup> hemorrhagic,<sup>2</sup> or metaplastic<sup>3</sup> lesions in the basis pontis and a midbrain mass<sup>4</sup> have been described. Reports of lacunar infarcts in the internal capsule, shown by CT scan, are now abundant.<sup>5-12</sup> To our knowledge, this is the first case of CT-visualized pontine lacunar infarction in a patient with "typical" ataxic hemiparesis, as described by Fisher.<sup>1</sup> Sakai et al<sup>13</sup>

reported an atypical case of ataxic hemiparesis with trigeminal weakness, caused by a lacunar lesion in the basis pontis and confirmed by a CT scan.

E. VAN BUGGENHOUT, MD  
I. DEHAENE, MD  
M. VAN ZANDIJCKE, MD  
Department of Neurology  
AZ St Jan  
B-8000 Brugge  
Belgium

1. Fisher CM: Ataxic hemiparesis: A pathologic study. *Arch Neurol* 1978;35:126-128.

2. Schnapper RA: Pontine hemorrhage presenting as ataxic hemiparesis. *Stroke* 1982;13:518-519.

3. Radhakrishnan K, Malhotra AK, Shridharan R, et al: Ataxic hemiparesis: Clinical, electrophysiologic, radiologic and pathologic observations. *Clin Neurol Neurosurg* 1982;84:91-100.

4. Bendheim PE, Berg BO: Ataxic hemiparesis from a midbrain mass. *Ann Neurol* 1981;9:405-407.

5. De Renzi E, Nichelli P, Crisi G: Hemiataxia and crural hemiparesis following capsular infarct. *J Neurol Neurosurg Psychiatry* 1983; 46:561-563.

6. Donnan GA, Tress BM, Bladin PF: A prospective study of lacunar infarction using computerized tomography. *Neurology* 1982;32:49-56.

7. Iragui VJ, McCutchen CB: Capsular ataxic hemiparesis. *Arch Neurol* 1982;39:528-529.

8. Jokelainen M, Pilke A: Ataxic hemiparesis. *Arch Neurol* 1983;40:326.

9. Perman GP, Racy A: Homolateral ataxia and crural paresis: Case report. *Neurology* 1980; 30:1013-1015.

10. Rougemont D, Baron JC, Lebrun-Grandie P, et al: Débit sanguin cérébral et extraction d'oxygène dans les hémiparésies lacunaires: Etude sémi-quantitative par l'oxygène-15 et la tomographie d'émission. *Pathol Biol* 1982;30:295-302.

11. Tredici G, Bogliun G, Sanguineti I: Capsular ataxic hemiparesis. *Arch Neurol* 1983;40:326.

12. Weisberg LA: Lacunar infarcts: Clinical and computed tomographic correlations. *Arch Neurol* 1982;39:37-40.

13. Sakai T, Murakami S, Ito K: Ataxic hemiparesis with trigeminal weakness. *Neurology* 1981;31:635-636.

Left, Contrast-enhanced computed tomographic scan two weeks after onset of right-sided ataxic hemiparesis. Right, Hypodense area in left pons.

