cerebral blood flow studies of Lassen,² Larsen et al,³ and Orgogozo and Larsen⁴ provide powerful evidence that the supplementary motor areas modulate the motor programming of repetitive and sequential movements, including articulation, rather than attention. Since the supplementary sensory areas seem to have sensory functions analogous to the motor functions of the supplementary motor areas,¹ I suspect the supplementary sensory areas have something to do with the actual processing of sensory information, including the comprehension of spoken language, that goes far beyond attentional mechanisms. What this processing entails, however, remains to be discovered.

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Pure motor hemiparesis and thalamic hemorrhage

To the Editor: We read with interest the article by Dr. Weisberg about pure motor hemiparesis.¹ In 3 of 33 cases, hemorrhagic lesions were detected in the capsular region, two extending from the thalamus. In another report² he noted that thalamic-ganglionic hematoma was found in 232 of 300 patients with intracranial hemorrhage and that 83% of the patients were 40 to 60 years old. These reports suggest that thalamic hemorrhage rarely causes pure motor hemiparesis, especially in childhood, and only two cases of thalamic hemorrhage have been reported in children.^{3,4} We now describe acute hemiplegia caused by thalamic hemorrhage in a 4-year-old-girl.

She was well until she began to vomit on February 6, 1978. That evening, she had a right hemiplegic gait and could not lift the right arm. The next day, she was admitted. She had no fever or headache. There was a right hemiparesis; tendon reflexes were slightly exaggerated on the right; there was no Babinski sign. The optic fundi were normal. Except for right facial weak-

ness, cranial nerve functions were intact, as was sensation. There was no nuchal rigidity, but the Kernig sign was present. CSF was xanthochromic. The next day CT revealed a hematoma in the anterior part of the left thalamus, surrounded by brain edema and extending to the left internal capsule (figure). She was treated with steroids. On the eighth hospital day, cerebral angiography showed dilation of the left thalamoperforating artery and intense visualization of the choroidal plexus; no apparent cause of the hemorrhage was demonstrated. Thereafter, she improved. On the seventeenth hospital day, CT no longer revealed hematoma, and a small area of edema was noted. After a month, she was almost normal.

In this case no cause of the hemorrhage was found in spite of various investigations, so it seems to be an example of spontaneous intracerebral hemorrhage;⁵ a small vascular malformation may have been the cause.

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Corrections

"Multiple sclerosis and viruses: An overview" by Stuart D. Cook and Peter C. Dowling, July 1980, Part 2, p. 81, left column, paragraph 3, line 9, should read "... and in the 10 ($p < 0.05$, ...)"

"Is L-DOPA drug holiday useful?" by Lorne K. Direnfeld, Robert G. Feldman, Michael P. Alexander, and Margaret Kelly-Hayes, July 1980, p. 786, left column, paragraph 3, line 7, should read "... One patient was discharged on the same dose as on admission and two patients were taking more L-DOPA."

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Corrections

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