IN 1965, Fisher and Cole described a cerebrovascular syndrome, they called homolateral ataxia and crural paresis, characterized by cerebellar-like ataxia, weakness and pyramidal signs involving the limbs of the same side, the leg more than the arm. In 1978, based on the clinicopathological results in three patients with this syndrome, Fisher identified an old infarct cavity in the basis pontis at the level of the junction of the upper one-third and lower two-thirds on the side opposite the neurological deficit as the causative lesion. He also proposed the designation "ataxic hemiparesis" for this syndrome. Although this syndrome has been recognized as one of the lacunar syndromes of Fisher et al., lesions other than lacunes, such as tumors or demyelinating processes, could conceivably produce the syndrome.

Recently we examined 2 patients with primary pontine hemorrhage, that had a syndrome compatible with the ataxic hemiparesis of Fisher.

Case Report
Case 1
A 55-year-old hypertensive man, noted the sudden onset of dysarthria and weakness of the left leg and the left arm following occipital heaviness and nausea on November 3, 1981. He was unable to walk because of unsteadiness. Three hours later he was admitted to Tokai University Hospital. On admission his blood pressure was 160/110 mm Hg. He was alert and well oriented. The neck was supple.

Oculomotor functions and pupils were normal. The left nasolabial fold was shallower and the tongue deviated to the right. The left eye was artificial due to trauma in his youth; visual fields and extraocular movement of his right eye were normal. No pupillary abnormalities were detected. There was fine horizontal nystagmus on left lateral gaze. Speech was dysarthric. The nasolabial fold on the left was flattened; mild left hemiparesis was present particularly in the distal portion of the left leg. Deep tendon reflexes were normal, but Babinski sign was positive on the left. Sensation was normal. There was left dysdiadochokinesis, and the finger-nose-finger test, shin-tapping test and heel-shin test showed impairment out of proportion to his weakness.

CT scan on the day of admission showed a small recent hematoma in the right dorsal part of the rostral basis pontis (fig. 1). ECG and EEG examination were normal. Retrograde vertebral angiography was normal.

After admission, nystagmus rapidly disappeared, and the dysarthria and the left hemiparesis decreased in severity. CT scan on November 26 was now normal. Though the ataxia on the left side gradually improved, slight cerebellar signs still persisted with positive Babinski sign at discharge (December 7, 1981).

Case 2
This 39-year-old woman had been well until the evening of March 20, 1982, when she noticed dysarthria following a floating sensation for several minutes. She could not pick up a coin using her left hand because of clumsiness. Shortly afterwards, she noticed unsteadiness in walking. On admission, about four hours after onset, her blood pressure was 180/110 mm Hg. She was alert and well oriented. Neck was supple. Oculomotor functions and pupils were normal. The left nasolabial fold was shallower and the tongue devi-
Figure 1. Unenhanced CT scan of case 1 on the day of admission shows a small fresh hematoma located in the right dorsal part of the rostral basis pontis.

Figure 2. Unenhanced CT scan of case 2 on the day of admission shows a small hematoma located in the right dorsolateral aspect of the pons.

The mechanism of the development of homolateral pyramidal and cerebellar signs resulting from one small lesion is the mechanism of the development of homolateral pyramidal and cerebellar signs resulting from one small lesion. How can a unilateral lesion of the basis pontis produce only contralateral cerebellar signs? Regarding the mechanism underlying the contralateral
cerebellar signs, Fisher speculated that either the pontine nuclei sending fibers to the opposite cerebellum were damaged or crossing fibers from the opposite pontine nuclei were interrupted, but he did not conclude why the cerebellar signs were not bilateral in either case. In each of Fisher's cases the lesion was very small, and in each of our cases too, the hematoma located in the upper basis pontis was rather small. The reasons for the development of contralateral cerebellar signs from these small lesions in the basis pontis might be as follows; 1) the pontine nuclei may be more vulnerable or they are more localized than the crossing fibers, and/or 2) ipsilateral cerebellar signs resulting from the interruption of the crossing fibers are apt to be promptly compensated for by the majority of the intact fibers. The former possibility seems more likely, and thus we consider that a lesion of the ipsilateral pontine nuclei is probably responsible for homolateral ataxia. Further clinico-pathological studies are necessary to confirm this hypothesis.

The third interesting problem is the side of lesion and dysarthria. The lesions in all 3 cases of Fisher and in the present 2 cases were in the right brainstem. Among those 5 patients, 4 showed dysarthria, while ataxic hemiparesis due to left brainstem lesions did not reveal speech disturbance. This is in agreement with the results of Lechtenberg and Gilman, who suggested that speech function was most commonly affected by damage to the left cerebellar hemisphere.

References
Ataxic hemiparesis in patients with primary pontine hemorrhage.
K Kobatake and Y Shinohara

Stroke. 1983;14:762-764
doi: 10.1161/01.STR.14.5.762
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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