

tion. In most cases, transient ischemic attacks, including transient monocular blindness, or cerebrovascular accidents result.<sup>8,9</sup> Occasionally, an oculomotor palsy associated with an ipsilateral headache is the only clinical manifestation.<sup>10</sup>

In the cavernous sinus, the sixth nerve lies adherent to the sheath of the carotid artery on its medial side and to the sinus wall on its lateral side.<sup>11</sup> The dorsal meningeal artery arising from the meningohypophyseal trunk sends a branch to the sixth nerve. An expansion of the wall of the intracavernous carotid could therefore produce sixth-nerve dysfunction, either by direct compression or by interruption of its blood supply. Abad et al<sup>7</sup> demonstrated a traumatic intracavernous saccular aneurysm in this location in a patient with a sixth-nerve palsy and Horner's syndrome.

Other cranial-nerve palsies are rarely associated with carotid artery dissections. Paralysis of the tongue secondary to compression of the hypoglossal nerve at the base of the skull has been noted on occasion.<sup>8</sup> Our patient complained of an unpleasant taste that persisted for days. Dysgeu-

sia has been noted previously and presumed to result from involvement of the chorda tympani.<sup>8</sup> We suggest that a more likely cause is compression of the glossopharyngeal nerve in the neck as it passes forward between the internal jugular vein and internal carotid artery. Lane et al<sup>12</sup> documented by angiogram an aneurysm of the internal carotid artery at the base of the skull that produced palsies of both hypoglossal and glossopharyngeal nerves without perversion of taste.

An isolated cranial-nerve palsy may be the only neurologic sign of an internal carotid artery dissection. Dysfunction of the cranial nerves traveling through the neck at the skull base is most common. The sixth cranial nerve can also be affected, because of its anatomic relationship to the intracavernous carotid artery. Associated hemicranial headache and signs of Horner's syndrome may provide additional diagnostic clues.

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## Ataxic Hemiparesis From Lesions of the Corona Radiata

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● Ipsilateral cerebellar and pyramidal signs suddenly developed in three patients. Computed tomography revealed small infarcts of the corona radiata in two patients and a small infarct in the posterior limb of the internal capsule in the third. Ataxic hemiparesis can result from lesions of the corona radiata as well as many other points along the corticospinal pathways.

(*Arch Neurol* 1983;40:449-450)

Ataxic hemiparesis is a syndrome in which cerebellar and pyramidal signs occur ipsilaterally.<sup>1</sup> Cerebellar

signs in the limbs are more prominent than pyramidal signs, and dysarthria is described in many of the published reports.<sup>2-6</sup>

In 1965, Fisher and Cole<sup>2</sup> argued that the responsible lesion was located in the region of the internal capsule or corona radiata. They could not confirm this hypothesis, as the one autopsied patient in their series had multiple lesions. In 1978, Fisher<sup>4</sup> described three more patients with ataxic hemiparesis, each of whom had an infarct in the contralateral pons. Since then, more patients with ataxic hemiparesis have been described, with tumors, infarcts, and hemorrhages located in the pons, midbrain, and internal capsule.<sup>7-10</sup>

We report three cases of ataxic hemiparesis, two with lesions in the corona radiata, supporting Fisher and Cole's original hypothesis, and one

with a lesion in the posterior limb of the internal capsule. All three patients showed the characteristic changes of infarction on computed tomographic (CT) scans.

#### REPORT OF CASES

**CASE 1.**—A 78-year-old, right-handed, hypertensive woman was visiting the hospital when she experienced the sudden onset of unsteadiness and slurred speech. She was able to sit down without falling. On her arrival in the emergency room ten minutes later, she was alert, oriented, and not aphasic. Her BP was 204/90 mm Hg. She was dysarthric and had mild weakness of the right lower part of the face, a pronator drift of the right arm, and slight weakness of the right leg. The muscle stretch reflexes were symmetrical, but a right Babinski sign was present. The finger-nose and heel-knee-shin tests revealed a marked dysmetria. The patient was unable to stand without aid. A CT scan

Accepted for publication Jan 6, 1983.  
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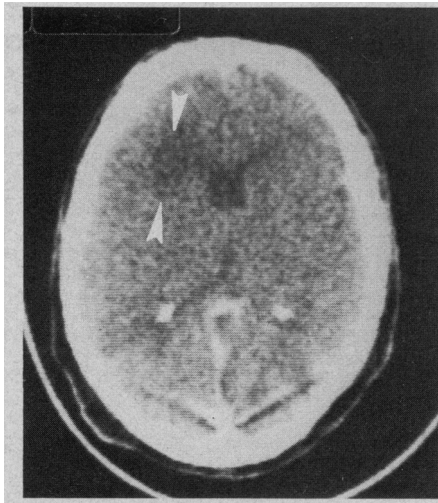


Fig 1.—Contrast-enhanced computed tomogram shows infarct (arrows) in left corona radiata near anterior horn of lateral ventricle.

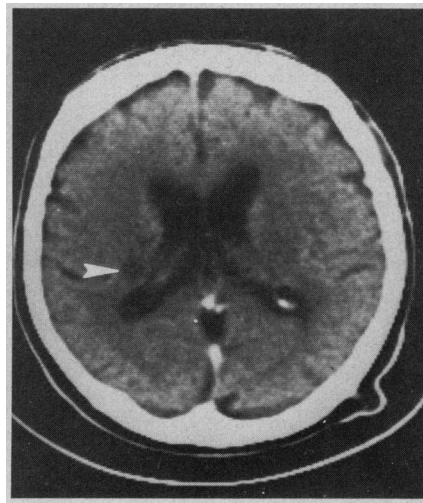


Fig 2.—Contrast-enhanced computed tomogram shows infarct (arrow) in right corona radiata near posterior horn of lateral ventricle.

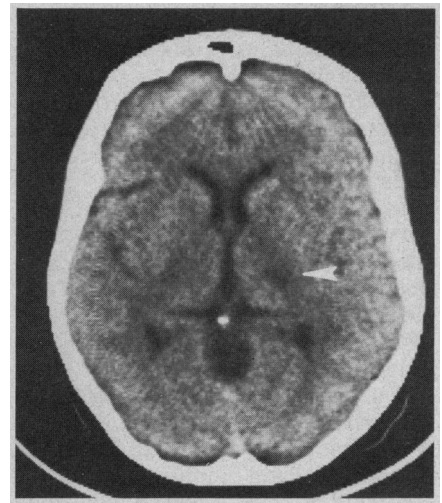


Fig 3.—Unenhanced computed tomogram reveals infarct (arrow) in posterior limb of left internal capsule.

done within one hour was normal. During the next four days, she became lethargic, and her hemiparesis worsened. By the seventh hospital day, she had recovered to the point that her neurologic status was the same as it had been on admission. The CT scan at that time revealed an infarct in the left corona radiata near the anterior horn of the lateral ventricle (Fig 1).

**CASE 2.**—A 67-year-old, right-handed, hypertensive man was at work when he suddenly began to fall toward the left. He managed to get home and remained there for the next three days. When improvement did not occur, he came to the emergency room. He was alert and oriented on arrival. His BP was 170/104 mm Hg. He had no dysarthria, but a mild weakness of the left lower part of the face was noted, along with a mild pronator drift of the left arm and weakness of the left leg. The muscle stretch reflexes were symmetrical, but a left Babinski sign was present. The finger-nose and heel-knee-shin tests revealed a marked dysmetria. The patient was able to walk with assistance. A CT scan performed on the sixth day revealed an infarct in the right corona radiata near the posterior horn of the lateral ventricle (Fig 2).

**CASE 3.**—A 61-year-old, right-handed, hypertensive woman found that she kept falling to the right when she tried to get out of bed one morning. She had stopped taking her antihypertensive medications three weeks earlier. When seen in the emergency room that day, her BP was 200/120 mm Hg. She was alert, oriented, and not aphasic. A mild weakness of the right lower part of the face was noted. In the opinion of her family, her speech was slightly slurred. There was a mild pronator drift of the right arm but no detectable leg weakness. Muscle stretch reflexes were symmetrical, but a right Babinski sign was present. The finger-nose and heel-knee-shin tests showed a marked dysmetria. The patient was ataxic but able to walk with assistance. A CT scan performed on the

fourth day revealed an infarct in the posterior limb of the left internal capsule (Fig 3).

#### COMMENT

The clinical features in each of our three patients were similar to those in previously described cases of ataxic hemiparesis. Pyramidal and cerebellar signs were present on the same side. Evidence of corticospinal dysfunction consisted of a mild arm drift, minimal weakness of the lower part of the face and the leg, and a Babinski sign on the involved side. In no case could the weakness explain the severe degree of limb incoordination and gait ataxia, which was indistinguishable from that found in cerebellar disease. Gait ataxia was severe enough to prevent any of the patients from walking without assistance. Two patients had some dysarthria, and one had normal speech.

These cases support the view that ataxia from supratentorial lesions is the result of interrupting corticopontocerebellar fibers. Patient 1 had an infarct of the anterior corona radiata. Such a lesion presumably interrupts fibers arising from the prefrontal and precentral areas, which go on to form the frontopontine tract (Arnold's bundle).<sup>11</sup> Patient 2 had an infarct in the posterior part of the corona radiata near the posterior horn of the lateral ventricle. A lesion so located interrupts fibers from the occipital and parietal lobes, whose fibers, together with those of the temporal lobe, join to form Türck's bundle in the retrolenticular and sublenticular capsule.<sup>11</sup> Patient 3 had an infarction of the posterior limb of the internal capsule,

which, again, interrupts fibers from the precentral cortex and has been described by others.<sup>5,6,8</sup> These three cases confirm the idea that ataxia with ipsilateral pyramidal signs, even in the presence of dysarthria and severe gait disturbance, does not always mean a pontine lesion as was suggested recently.<sup>1</sup> The cases reported herein, together with previously well-documented cases of midbrain and pontine lesions, provide convincing evidence that ataxic hemiparesis can result from lesions at many points along the corticopontine pathways, from the corona radiata to the midpons.

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