
Hemiplegia in posterior cerebral artery occlusion

M. Hommel, MD; G. Besson, MD; P. Pollak, MD; P. Kahane, MD; J.F. Le Bas, MD; and J. Perret, MD

Article abstract—We report 4 patients with hemiplegia due to a posterior cerebral artery occlusion. Associated clinical signs were aphasia, alexia or a neglect syndrome, hemianopia, and hemisensory loss. Hemiplegia was due to infarction in the lateral midbrain. The level of the occlusion in the posterior cerebral artery may be located distal to the junction with the posterior communicating artery.

NEUROLOGY 1990;40:1496-1499

The occlusion of a posterior cerebral artery (PCA) causes a spectrum of manifestations, including homonymous visual field defects, abnormal visual perception, and neuropsychological signs and symptoms. Usually, there is little or no weakness.¹ Two reported cases of hemiplegia with PCA territory infarction were not verified with imaging studies or autopsy.² Only the case of Caplan et al³ had anatomic verification; it was a lateral midbrain infarct. We report 4 patients with hemiplegia associated with infarction in the territory of the PCA.

Case reports. Patient 1. A 66-year-old man with a history of cigarette smoking was admitted for sudden right hemiplegia. On admission, there was complete right hemiplegia affecting face, arm, and leg, with a Babinski sign, right hypoesthesia, and right homonymous hemianopia. The head and eyes were turned toward the left. He was aphasic with impaired comprehension, loss of repetition, and he was able to perseverate only in rare, stereotyped, poorly articulated phonemes. CT without contrast injection 3 days after the onset showed a hypodensity in the left posterior thalamus and left occipital and temporal lobes (figure 1A). MRI 24 days after the stroke (CGR magniscan with a 0.5 Tesla superconducting magnet in joined axial T₂-weighted images [TE = 60, 120 msec; TR = 2,000 msec] of 9-mm thickness, from the medulla to the vertex) confirmed CT and also showed an infarction in the anterolateral part of the left cerebral peduncle (figure 1B). The patient did not improve, remained bedridden, and died 7 months later.

Patient 2. A 58-year-old hypertensive man with a history of myocardial infarction and valvular aortic stenosis complained of left temporal cephalalgia for 3 days. He then developed a right ataxia. He was admitted on the 4th day with a right hemiplegia affecting the face, arm, and leg, and a Babinski sign. He had a right anesthesia and right homonymous hemianopia. Upward gaze was limited. The eyes and head were turned toward the left. He was dysarthric, fluency was de-

creased, and semantic paraphasia, neologisms, and perseverations were present in spontaneous speech. Comprehension was severely impaired. He was alexic. CT, performed without contrast injection on the 4th day after the stroke, showed a hypodensity affecting the thalamus and the occipital and the inferior part of the temporal lobes. An MRI study on the 12th day, in axial T₂-weighted images of 10-mm thickness with contiguous slices, confirmed the CT findings and also showed an infarct in the left ventral peduncle (figure 2). One year later, sensory perception had improved, and he was able to walk slowly, but was dependent in all activities of daily living. He remained alexic and aphasic with decreased fluency and voice volume. Comprehension, however, had recovered, repetition was normal, and the paraphasia, neologisms, and dysarthria had disappeared.

Patient 3. A 76-year-old right-handed woman with a history of hypertension and myocardial infarction was admitted for heart failure. On the next day, she was found with a complete right hemiplegia affecting the face, arm, and leg, and a Babinski sign. The head and eyes were turned toward the left, and she had a right homonymous hemianopia. She was alert but mute and did not respond to verbal orders. Painful stimuli elicited normal reactions on the left side but stereotyped extensor responses on the right. Pupil size and reactivity, as well as ocular movements, were normal. CT without contrast injection on the same day showed a hypodensity affecting the left midbrain, posterior thalamus, occipital lobe, and the inferior and medial part of the temporal lobe (figure 3). She died from cardiogenic collapse 4 days later. No autopsy was obtained.

Patient 4. A 69-year-old man had a history of cigarette smoking, hypertension, and, 13 years before the stroke, a left trigeminal neuralgia attributed to a fusiform aneurysmal vertebral artery kink demonstrated with angiography. He was admitted for the progressive 3-day evolution of a left hemiplegia. On examination, the hemiplegia affected the face, arm, and leg, with a head and eyes deviation toward the right. There was no Babinski sign. Hypoesthesia for fine discrimination, light touch, joint position, pain, and temperature was ob-

From the Department of Clinical and Biological Neurosciences, Stroke Unit and Magnetic Resonance Unit, Centre Hospitalier Universitaire de Grenoble, Grenoble, France.

Received January 23, 1990. Accepted for publication in final form April 3, 1990.

Address correspondence and reprint requests to Dr. Marc Hommel, Clinique Neurologique, Centre Hospitalier Universitaire de Grenoble, BP 217 X 38043, Grenoble, Cedex, France.

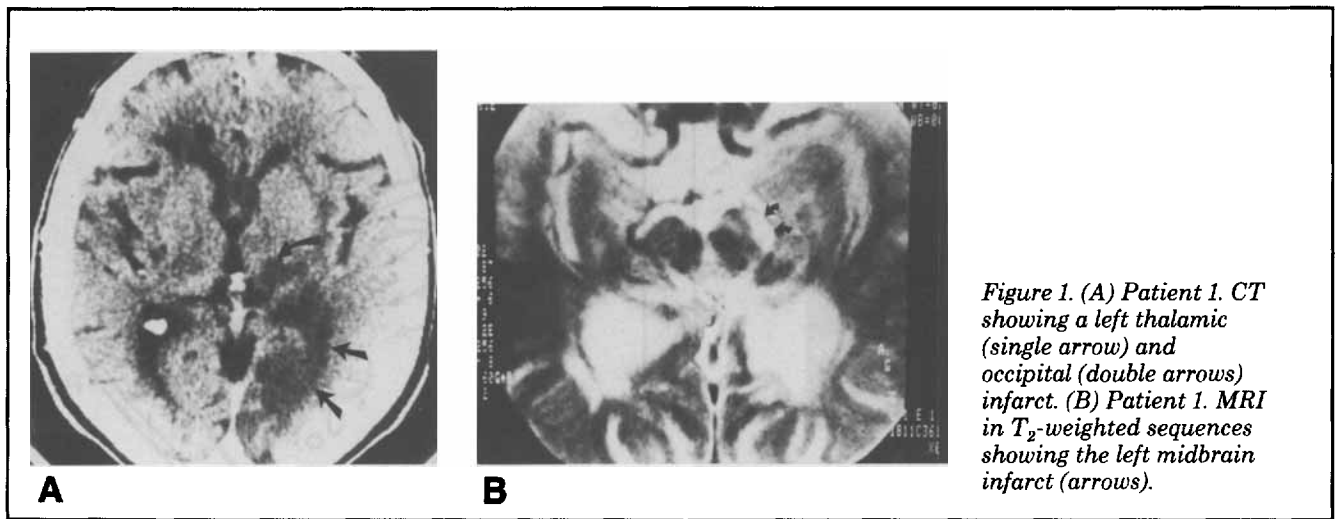


Figure 1. (A) Patient 1. CT showing a left thalamic (single arrow) and occipital (double arrows) infarct. (B) Patient 1. MRI in T₂-weighted sequences showing the left midbrain infarct (arrows).

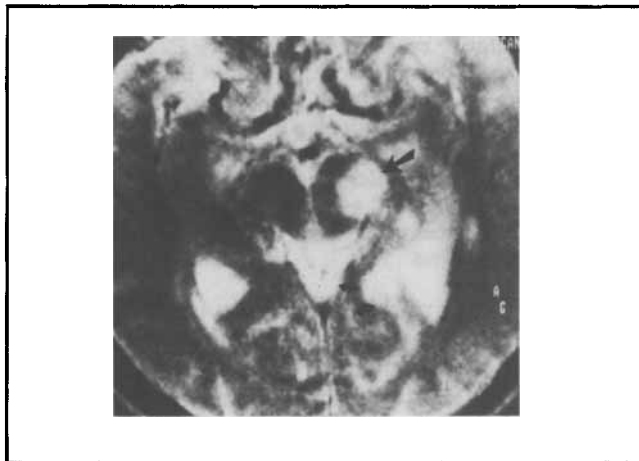


Figure 2. Patient 2. MRI showing the left midbrain (single arrow) and occipital (double arrows) infarct.

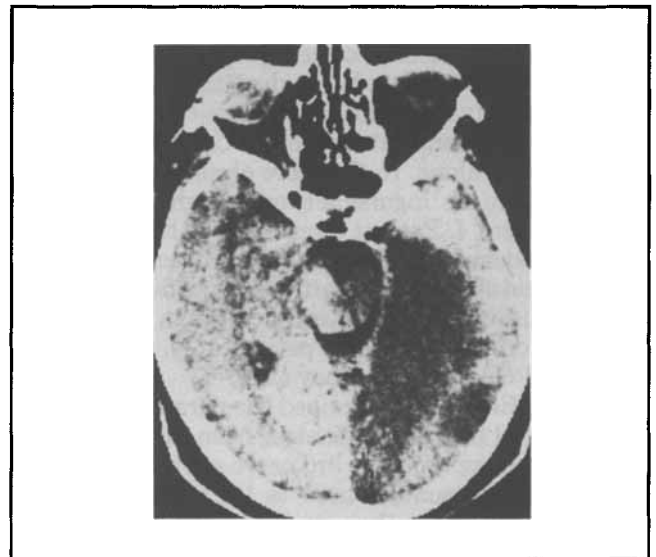


Figure 3. Patient 3. CT showing the left midbrain, temporal, and occipital infarct.

served on the left side. Visual fields were normal. There was a disorientation for time but not for place. After 7 days, the disorientation had disappeared, and a left spatial neglect syndrome was observed. CT without contrast on day 4 after the stroke showed a hypodensity in the posterior part of the right thalamus. An MRI study, performed on day 35 after the stroke in T₂-weighted axial images, also showed an infarct in the right midbrain (figure 4). During the follow-up, he had a deep venous thrombosis, pneumonia, and an acute cholecystitis. Sensory perception fully recovered, but he had a residual hemiparesis. Six months later, he needed minimal help in daily activities, and some left motor neglect remained.

Discussion. A 3rd nerve palsy with crossed hemiplegia, Weber's syndrome, is a well-known midbrain syndrome. When due to infarction, the arterial occlusion is located at the origin of the PCA, where the paramedian branches supplying the 3rd nerve nucleus and fascicles, and the short circumferential branches supplying the lateral part of the midbrain, have their origin.⁴ None of our cases had a 3rd nerve palsy or tegmental signs or symptoms indicating a midbrain lesion, with the exception of an upgaze paresis in patient 2.

Sometimes, in PCA occlusion, paresis and sensory loss occur. There are 2 proposed hypotheses: Johansson⁵ suggested, in a CT study, possible involvement of the pyramidal tract in the internal capsule, or associated lacunes in the corona radiata. Benson and Tomlinson² suggested midbrain infarction in 2 cases with neuropsychological signs and symptoms of PCA territory involvement and radioisotope brain-scanning evidence. Caplan et al,³ in an anatomic case, showed infarction in the lateral midbrain. In each of our cases, the hemiplegia was associated with an infarct in the midbrain. In 3 patients, the sensation could be assessed and was decreased or abolished. This loss could have been due to either thalamic involvement or midbrain lemniscal system infarction.⁶

Conjugate deviation of the head and eyes toward the side of the lesion is not common in PCA occlusion. Case 1 described by Caplan et al³ had the same anatomic lesions as ours, and as observed in our 4 cases, a contralateral head and eye deviation.

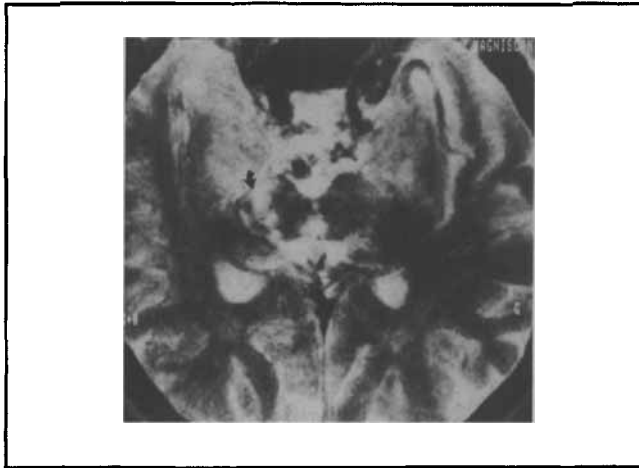


Figure 4. Patient 4. MRI showing the infarct in the right midbrain (arrow).

Within the 1st few days after the stroke, the neuropsychological features in our patients (aphasia, alexia, and neglect), the presence of hemiplegia, hemisensory loss, hemianopia, head and eyes deviation, and verbal language difficulty make it difficult to distinguish main trunk middle cerebral artery from PCA occlusion on clinical grounds alone. In the more chronic period, we were not able to distinguish a specific right PCA syndrome in patient 4. Patient 2 remained aphasic with a pattern of reduced fluency and voice volume, normal repetition, and absence of dysarthria, often described in thalamic aphasia.⁷ But we have difficulties in ascertaining, on this criterion and in 1 patient only, whether a specific left PCA syndrome may exist.

Foix and Hillemand⁴ described the paramedian and circumferential arteries, originating from the top of the basilar artery, supplying the 3rd nerve nucleus and fascicles, and the pyramidal tract in the ventral-lateral midbrain. According to their study, a hemiplegia without a 3rd nerve palsy did not seem possible. Zeal and Rhoton,⁸ in an anatomic study of 50 brains, divided the PCA into 3 parts: P1 from the origin in the basilar artery to the posterior communicating artery; P2 (mean length, 50 mm) was divided into an anterior (P2A) and posterior half (P2P), with P2A beginning at the posterior communicating artery and coursing around the peduncle, and P2P beginning at the posterior margin of the posterolateral midbrain and terminating at the posterior aspect of the midbrain within the ambient cistern; P3 proceeds posteriorly from the level of the pulvinar in the lateral aspect of the quadrigeminal cistern and ends at the anterior limit of the calcarine fissure. The average length of the P3 segment is 20 mm (figure 5). The thalamoperforating arteries arise from P1 and supply the anterior part of the posterior thalamus, hypothalamus, subthalamus, substantia nigra, red nucleus, oculomotor nucleus and nerve. The short circumflex arteries, present in 66% of brains, originate in 86% of the cases from P1, pass only a short distance around the brainstem before entering the brain, and send branches to the cerebral peduncle, contributing to the blood supply of the corticospinal tract. The corticobulbar and corticospinal pathways are also

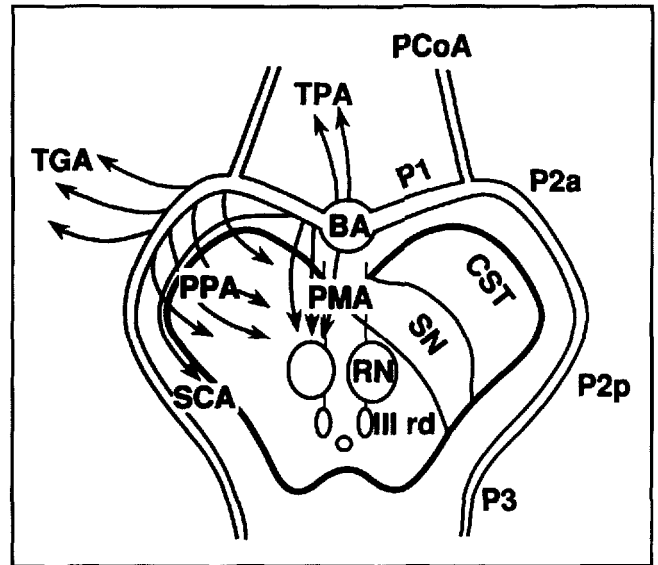


Figure 5. Axial diagram of the midbrain. BA = basilar artery; PCoA = posterior communicating artery; P1 = P1 part of the posterior cerebral artery (PCA); P2a = anterior segment of the P2 part of the PCA; P2p = posterior segment of the P2 part of the PCA; P3 = P3 part of the PCA; TPA = thalamoperforating arteries; TGA = thalamogeniculate arteries; SCA = short circumflex arteries; PPA = peduncular perforating arteries; PMA = paramedian arteries; CST = corticospinal tract in cerebral peduncle; SN = substantia nigra; RN = red nucleus; IIIrd = oculomotor nucleus.

supplied by the perforating branches originating from P2A in 94% of the cases and from P2P in the remaining 6%. The thalamogeniculate arteries have their origin at the P2A level. An arterial occlusion of the end of P1 or beginning of P2A could induce a midbrain infarction with hemiplegia and anesthesia without a 3rd nerve palsy. As the thalamogeniculate arteries have the same origin, an occlusion may also cause a posterior thalamic infarction.^{9,10} Our 4 cases had an infarction in the lateral midbrain involving the pyramidal tract and the posterior thalamus supplied by the thalamogeniculate arteries. Three patients also had an occipital and temporal infarction in the more distal territories of P3. These findings suggest that the occlusion was located at the P2A portion of the PCA in our patients.

References

1. Caplan LR. Posterior cerebral artery syndromes. In: Vinken PJ, Bruyn GW, Klawans HL, Toole JF, eds. Handbook of clinical neurology, vol 53. Vascular diseases Part I. Amsterdam: Elsevier, 1988:409-415.
2. Benson DF, Tomlinson EB. Hemiplegic syndrome of the posterior cerebral artery. *Stroke* 1971;2:559-564.
3. Caplan LR, De Witt LD, Pessin MS, Gorelick PB, Adelman LS. Lateral thalamic infarcts. *Arch Neurol* 1988;45:959-964.
4. Foix C, Hillemand P. Les artères de l'axe encéphalique jusqu'au diencéphale inclusivement. *Rev Neurol (Paris)* 1925;6:705-739.
5. Johansson T. Occipital infarctions associated with hemiparesis. *Eur Neurol* 1985;24:276-280.
6. Duvernoy HM. Human brainstem vessels. Berlin: Springer, 1978.
7. Cambier J, Graveleau Ph. Thalamic syndromes. In: Vinken PJ, Bruyn GW, Klawans HL, Frederiks JAM, eds. Handbook of clinical neurology, vol 45. Clinical neuropsychology. Amsterdam:

Elsevier, 1985:87-98.

8. Zeal AZ, Rhoton AL. Microsurgical anatomy of the posterior cerebral artery. *J Neurosurg* 1978;48:534-559.

9. Hayman LA, Berman SA, Hinck VC. Correlation of CT cerebral vascular territories with function: II. Posterior cerebral artery.

AJR 1981;137:13-19.

10. Takahashi S, Goto K, Fukasawa H, Kawata Y, Uemura K, Yaguchi K. Computed tomography of cerebral infarction along the distribution of the basal perforating arteries. *Radiology* 1985;155:119-130.



Capsular genu syndrome

Julien Bogousslavsky, MD, and Franco Regli, MD

Article abstract—We report 5 patients with unilateral infarct and 1 with hemorrhage limited to the genu of the internal capsule. The most prominent finding was contralateral facial and lingual hemiparesis with dysarthria. Three patients also showed unilateral mastication-palatal-pharyngeal weakness, and 1 had unilateral vocal cord paresis. Mild limb involvement was limited to hand weakness in 4 patients. Our findings suggest that the majority of motor corticopontine and corticobulbar fibers are located in the genu of the internal capsule. The faciolingual syndrome and its variants are highly suggestive of capsular genu stroke.

NEUROLOGY 1990;40:1499-1502

In his 1902 thesis, Ferrand¹ mentioned that partial forms of hemiparesis could occur in patients with lacunes in the internal capsule. The occurrence of partial forms of motor hemiparesis from capsular stroke depends upon the location of the lesion with reference to the topography of the motor representation of the head, upper limb, trunk, and lower limb within the internal capsule.² Previous reports suggested that predominant facial weakness from capsular stroke was related to involvement of the most rostral part of the posterior limb in the internal capsule.³

We studied 6 patients with acute stroke limited to the genu of the internal capsule on one side, as identified on CT following templates developed by one of us.³ All of them had contralateral faciolingual—and to a lesser extent masseter-palatal-pharyngeal-laryngeal—weakness contrasting with limb involvement limited to only mild hand weakness.

Patients. There were 5 men and 1 woman, aged 43 to 84 years. The clinical and CT characteristics in these patients are summarized in the table. All patients had unilateral lower facial and tongue weakness, 5 had dysarthria, 3 had dysphagia, 3 had unilateral masseter weakness, 2 had unilateral palatal weakness, and 1 had unilateral vocal cord paresis. No patient had sternomastoid or trapezius weakness. The cranial nerve findings contrasted with only mild to moderate upper limb involvement in 4 patients, which was usually limited to unilateral fingers or hand weakness. Two patients had no dysfunction in the limbs. Standard neuropsychological examination findings were normal in all patients. Extracranial Doppler ultrasonography was unremarkable in all patients. All patients were discharged within 3 weeks of admission, after they had made a good to complete recovery.

Case reports. Patient 1. A 68-year-old man was admitted because of acute dysarthria with right facial drooling and weakness in the right hand. He smoked 12 cigarettes per day but had no medical history. On admission, blood pressure was 160/90 mm Hg with a regular pulse. On neurologic examination, he showed severe inferior facial weakness and moderate weakness of eye closure on the right. He had marked dysarthria with slurred speech. He complained of dysphagia, and masseter and posterior palatal weakness was present on the right. The tongue markedly deviated to the right on protrusion and it could not push against resistance toward the left side. Facial and buccal sensation were normal. Corneal reflex was symmetric. The rest of the cranial nerves were normal, including trapezius and sternomastoid contraction. No abnormality was present in the limbs except for moderate weakness of the fingers on the right, involving mainly flexion movements. Stylo-radial tendon reflex was slightly more brisk on the right than on the left. Standard neuropsychological examination findings were normal. Extracranial Doppler ultrasounds and ECG were normal. CT 3 days later showed a small infarct involving the genu of the internal capsule on the left. Recovery was complete within 2 weeks except for right facial asymmetry and slight deviation of the tongue to the right on protrusion.

Patient 2. A 63-year-old man with intermittently treated arterial hypertension was admitted after he acutely experienced severe dysarthria and dysphagia. On examination, he showed moderate facial paresis on the right predominating in the inferior part, and had a tendency to drool. The speech was so slurred that only a few words could be understood. Closure of the mouth and masseter contraction were weak on the right. There was a slight right posterior palatal ptosis with hypomotility. The tongue deviated markedly to the right on protrusion attempts. Laryngeal examination showed hypotonia and

From the Department of Neurology, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.

Received January 26, 1990. Accepted for publication in final form March 29, 1990.

Address correspondence and reprint requests to Dr. J. Bogousslavsky, Department of Neurology, CHUV, 1011 Lausanne, Switzerland.