Cerebellar Infarction
Clinical and Anatomic Observations in 66 Cases
Carlos S. Kase, MD; Bo Norrving, MD; Steven R. Levine, MD; Viken L. Babikian, MD; Eliot H. Chodosh, MD; Philip A. Wolf, MD; and Kenneth M.A. Welch, MD

Background and Purpose: Cerebellar infarction displays different clinical features, depending on the vascular territory involved. We studied patients with infarcts in the territories of the posterior inferior cerebellar artery or the superior cerebellar artery to compare their clinical presentation, course, and prognosis.

Methods: We retrospectively analyzed the clinical features, laboratory data, and imaging studies of 66 patients with cerebellar infarction collected consecutively at five institutions. All the cerebellar infarcts were documented on computed tomographic scan or magnetic resonance imaging.

Results: Two distinct profiles emerged, depending on the vascular territory involved. In 36 patients with posterior inferior cerebellar artery territory infarcts, a triad of vertigo, headache, and gait imbalance predominated at stroke onset. Computed tomography showed severe cerebellar mass effect in 11 cases (30%), with associated hydrocephalus in seven. In these seven patients (19%), postinfarct swelling led to brain stem compression that resulted in four deaths. In 30 patients with superior cerebellar artery infarcts, gait disturbance predominated at onset; vertigo and headache were significantly less common. The clinical course was usually benign. Computed tomography showed marked cerebellar mass effect, hydrocephalus, and brain stem compression in only two instances (7%). Presumed cerebral embolism was the predominant stroke mechanism in patients with superior cerebellar artery distribution infarcts, whereas in those with posterior inferior cerebellar artery distribution infarcts, the stroke mechanism was equally divided between cardiogenic embolism and posterior circulation arterial disease.

Conclusions: Cerebellar infarcts in the posterior inferior cerebellar artery and superior cerebellar artery distribution have different clinical presentations, course, and prognosis. These differences should help in the selection of appropriate monitoring and treatment strategies. (Stroke 1993;24:76–83)

KEY WORDS • cerebral arteries • cerebral infarction • prognosis • vertebrobasilar circulation

Infarction of the cerebellum is uncommon, representing approximately 1.5% of strokes. However, its clinical importance is because it may initially present as a “benign labyrinthine disorder” that can evolve into life-threatening brain stem compression from postinfarct edema. The recognition of the full spectrum of clinical presentation in cerebellar infarction has occurred since the advent of computed tomography (CT) and magnetic resonance imaging (MRI). These techniques have made possible the diagnosis of minimally symptomatic patients as well as the delineation of patterns of clinical-anatomic evolution.

We studied the clinical and radiological features of 66 patients with cerebellar infarction retrospectively analyzed at five institutions: Boston University Medical Center (seven patients); Lund (Sweden) University Hospital (26 patients); Henry Ford Hospital, Detroit, Mich. (24 patients); the Boston Veterans Administration Hospital (seven patients); and Wayne General Hospital, Wayne, N.J. (two patients). Patients selected for the study fit the following inclusion criteria: CT or MRI documentation of cerebellar infarction; and detailed clinical information on stroke risk factors, symptoms and signs at onset, subsequent clinical course, and investigation of stroke mechanism. Excluded were patients with cerebellar infarcts involving multiple vascular territories and those with bilateral infarcts. Five cases were excluded because of multiple and bilateral cerebellar infarcts. The patients included 36 with infarction in the territory of the PICA and 30 patients with
TABLE 1. Demographic Features and Vascular Risk Factors in 66 Patients With Cerebellar Infarction

<table>
<thead>
<tr>
<th>Vascular Risk Factors</th>
<th>Male</th>
<th>Female</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>32</td>
<td>15</td>
<td>48</td>
<td>23</td>
</tr>
<tr>
<td>Diabetes</td>
<td>9</td>
<td>4</td>
<td>14</td>
<td>6</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>24</td>
<td>1</td>
<td>36</td>
<td>2</td>
</tr>
<tr>
<td>Smoking history</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>CAD</td>
<td>5</td>
<td>2</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>AF</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>MVP</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>CHF</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>RHD with prosthetic valves</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>“Marantic” endocarditis</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>Antiphospholipid antibodies</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>Polycythemia vera</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>1.5</td>
</tr>
</tbody>
</table>

n, Number of patients. CAD, coronary artery disease; AF, atrial fibrillation; MVP, mitral valve prolapse; CHF, congestive heart failure; RHD, rheumatic heart disease; TIAs, transient ischemic attacks.

infarction in the SCA distribution. The CT or MRI delineation of cerebellar infarcts in the PICA or SCA territory was performed following the radiological descriptions of Savoiardo et al.° and the anatomic diagrams of Amarenco and Hauw.°° Cases of infarct in the anterior inferior cerebellar artery (AICA) territory were not included in the analysis. All patients were clinically evaluated by one of the authors, and detailed clinical, laboratory, and radiological data were recorded. Statistical analyses were performed with likelihood ratio, two-sided, \( \chi^2 \) tests to compare features of the PICA and SCA territory infarcts. A value of \( p<0.05 \) was considered significant.

Results

The demographic features and vascular risk factors for the group are listed in Table 1. Hypertension was present in about one half of the patients, whereas diabetes mellitus and coronary artery disease affected approximately 25% of them. One third of the patients were current or former cigarette smokers. Two patients with artificial heart valves were adequately anticoagulated with warfarin at the time of onset of the cerebellar infarct. Vertebrobasilar territory transient ischemic attacks preceded the cerebellar infarct in two patients for periods of 12 and 18 months, respectively. One patient had episodes of vertigo, vomiting, and imbalance preceding a PICA distribution infarct, with lack of filling of the left PICA demonstrated on angiography. The other patient had episodic vertigo, diplopia, and diaphoresis before developing a SCA distribution infarct; angiography was not performed.

Headache, a common symptom at onset, was more frequently reported in the PICA (64%)) than in the SCA (40%) infarct patients, and two types of location predominated: cervical (either isolated or, more frequently, with occipital, periangular, or hemifacial-ocular radiation) and occipital. The unilateral headaches (in eight patients) were all ipsilateral to the cerebellar infarction. In the SCA group the reported headaches had a less well-defined pattern, and a frontal location was given in one half (six patients) of those reporting the symptom. The others were described as unilateral or bilateral occipital, “between the ears,” and cervical in location. Vertigo was always of sudden onset and was a more frequent and prominent feature in the PICA (78%) than in the SCA (37%) infarcts (difference significant at \( p=0.001 \)), with two thirds of the latter group denying this otherwise common symptom of acute posterior fossa disorders. Vomiting associated with vertigo was frequent in both groups (61% PICA, 40% SCA). However, it occurred in the absence of vertigo in seven of the 12 SCA patients who experienced vomiting at onset. Gait instability, manifested by sudden inability to walk or stand unassisted, occurred equally in both groups (75% PICA, 67% SCA). The gait disturbance was in most instances severe, despite preserved motor strength in the legs, leading to descriptions such as “had to lean against the wall to stay upright” and “had to crawl to the toilet.” Attempts at standing or walking led to falling in one direction; in 16 of 17 patients this direction was toward the side of the cerebellar lesion. The one exception in which falling occurred in the opposite direction was in a patient who in addition had a hemiparesis (from associated cerebral hemispheric infarct) that affected the limbs opposite to the cerebellar lesion.

The findings on neurological examination are listed in Table 2. Gait ataxia was a frequent finding in both groups, whereas limb ataxia was found more often in the SCA (73%) than in the PICA (50%) infarction patients. Signs of a lateral medullary syndrome coexisted in only one third of the PICA distribution cases. Of these 12 patients, six had a complete lateral medullary syndrome...
(i.e., ipsilateral limb ataxia, Horner's syndrome, palatal weakness, facial hypesthesia to pain and temperature, and contralateral hypesthesia to pain and temperature in the limbs and trunk), whereas the rest showed fractions of it, the most common being a combination of dysarthria with ipsilateral Horner's syndrome and facial dysesthesias. The associated brain stem signs in the SCA cases were not generally localized to a particular brain stem level. Those occurring most commonly were dysarthria (nine patients) and Horner's syndrome (six patients), with single instances each of diplopia, vertical gaze palsy, 6th nerve palsy, and internuclear ophthalmoplegia. Only one patient had a transient choreiform dyskinesia affecting the ataxic limbs ipsilateral to the cerebellar infarct; he also had sensory loss to pain and temperature in the contralateral limbs, trunk, and face. Nystagmus was a common feature in both types of cerebellar infarction, somewhat more frequent in the PICA distribution cases. In both groups the predominant form of nystagmus was horizontal, in the direction of the affected hemisphere in the PICA cases and either bilateral or toward the involved hemisphere in the SCA cases. No elements in the bedside analysis of the nystagmus allowed a distinction to be made between the two vascular territories.

In all the cases in this series, the cerebellar infarction was documented by CT or MRI. Among patients with PICA distribution infarcts, 27 (75%) had lesions that involved only a fraction of that vascular territory, the most commonly affected area being the basal medial aspect of the cerebellar hemisphere (Figure 1). Only three of these subtotal infarcts had minimal mass effect in the form of effacement and displacement of the 4th ventricle; obstructive hydrocephalus was not present. The remaining nine patients (25%) had infarcts involving the full cerebellar territory of the PICA, including the whole inferior posterior surface of the hemisphere and tonsil and the adjacent inferior vermis (Figure 2). In eight of the patients there was marked mass effect, with displacement and obliteration of the 4th ventricle; seven had obstructive hydrocephalus. In all six patients with an associated complete lateral medullary syndrome, the cerebellar infarcts were of the partial variety, involving the medial basal aspect of the hemisphere. All 30 SCA infarcts, on the other hand, involved only portions of that vascular territory, corresponding most commonly to the vermian or hemispheric branches of the SCA (Figure 3). They had slight local mass effect, with effacement of the adjacent ambient cistern (in nine instances), rarely associated with obliteration of the quadrigeminal cistern (two cases) but without displacement of the 4th ventricle. The latter only occurred in two instances (7%), both corresponding to large infarcts involving most of the cerebellar white matter.

The mechanism of cerebellar infarction could be determined in 28 of the 36 patients with PICA distribution infarcts (Table 3). In the eight remaining patients (22%) the cause was unknown, as they had no cardiac risk factors for stroke, and cerebral angiograms were either normal (three patients) or not performed (five patients). Two of the seven patients with vertebral artery occlusion had angiographic evidence of fibromuscular dysplasia at a site of arterial dissection. In the single case of presumed vasculitis, angiography was not performed; the diagnosis was based only on the presence of fever, anemia, eosinophilia, and elevated sedimentation rate. In the SCA cases, the mechanism of infarction could be defined in 20 instances. In 10 patients the mechanism was unknown, as there were no cardiac risk factors for stroke, and cerebral angiograms were either normal (five patients) or not performed (five patients). The frequency of embolic mechanism (70%) was higher than that of local arterial disease (30%) as the cause of the SCA distribution infarcts, but the difference did not reach statistical significance ($p=0.115$, calculated as exact probability value testing for 50% probability using binomial distribution). The former group included two patients with SCA embolism from documented distal vertebral artery dissection (one of whom has been previously reported by two of us11) and one instance of SCA occlusion during vertebral angiography. Posterior fossa angiography, performed in 14 of the 30 patients with SCA distribution infarcts, showed normal results in five. The patient with "marantic" endocarditis had metastatic prostatic cancer with autopsy-documented mitral valve vegetations, multiple hemorrhagic infarcts of the cerebral hemispheres, and densely hemorrhagic infarction of the upper surface of the right cerebellar hemisphere.

The clinical courses of the patients showed differences depending on the vascular territory involved. In
After 18 and two patients, neurological deficits remained. The patient died without regaining consciousness after posterior fossa decompression with resection of infarcted cerebellar tissue. The other patient, a 19-year-old male with mitral valve prolapse and a history of drug abuse, developed a left SCA distribution infarct affecting the deep white matter of the cerebellar hemisphere, with associated 4th ventricular compression and hydrocephalus. Posterior fossa decompression with removal of necrotic cerebellar tissue was followed by survival and no neurological impairment. Mortality for the 30 patients with SCA distribution infarcts was 7%, and only one of the 28 survivors had persistent residual neurological deficits. The majority of these patients were either minimally disabled (20) or neurologically intact (seven).

Discussion

Analysis of these cases highlights important clinical, radiological, and prognostic differences between PICA and SCA distribution infarcts. Patients with PICA territory infarcts frequently experience vertigo, vomiting, headache, and gait imbalance at onset, with horizontal nystagmus (in the direction of the affected hemisphere) and gait ataxia and less prominent ipsilateral limb ataxia. In the SCA territory cases, vestibular symptomatology and headache are less frequent, a common presentation being gait and limb ataxia with associated dysarthria and horizontal nystagmus. Guiang and El-lington pointed to the clinical value of disabling gait imbalance in the absence of vertigo for the diagnostic suspicion of cerebellar infarction. The relatively lower frequency of vertigo in the SCA cases has been related to the comparatively less vestibular connections of those portions of the cerebellum supplied by the SCA, in contrast to the rich connections of the flocculonodular lobe supplied by the PICA and AICA.10,13-15 The latter relates to the observation that patients with PICA (as well as AICA) distribution cerebellar infarcts can display a purely vestibular syndrome resembling “labyrinthitis,” whereas SCA cases do not. A clinical presentation that has not been described in this knowledge in cases of cerebellar infarction confined to the SCA territory. According to Amarenco et al., patients with PICA distribution infarction that exhibit a purely vestibular syndrome are likely to have an isolated cerebellar infarction in the distribution of the medial branch of that artery. That particular vascular territory, which encompasses the medullary aspect of the cerebellar hemisphere, accounted for the majority of our cases of partial infarction in the PICA distribution, probably explaining the relative prominence of vestibular symptomatology in our PICA distribution cases.

Among the physical findings characteristic of cerebellar infarction, nystagmus is described with frequencies ranging between 47% and 62%. We found it in 64% of the cases, somewhat more frequently in the PICA (75%) than SCA (50%) infarction patients. Although oculographic studies reported opposite effects on facilitation of ocular saccades in lateral medullary and rostral cerebellar lesions, no distinctive characteristics

the PICA infarction patients, seven of 36 (19%) developed severe mass effect in the posterior fossa with compression of the 4th ventricle and supratentorial hydrocephalus. All seven patients had infarcts affecting the full PICA cerebellar territory. Three of these patients died without surgical treatment; of the four patients subjected to either lateral ventriculostomy (one patient) or posterior fossa decompression (three patients), three survived and the other died 1 month after surgery without regaining consciousness after the procedure. One of the three survivors subsequently died from multiple cerebral embolic infarcts, whereas the others remained well, with minimal or no residual neurological deficits. The interval between stroke onset and signs of brain stem compression varied from 18 hours to 4 days (mean, 50 hours). The earliest sign of brain stem compression was lethargy (in six of the seven patients), accompanied by bilateral Babinski signs in two and severe headache in one. In one patient the onset of signs of brain stem compression was followed within 1 hour by coma with decerebrate posturing, irregular respirations, and absent oculocephalic and corneal reflexes. Mortality for the 36 patients with PICA distribution infarction was 17%, and one half of the survivors had neurological sequelae.

The SCA cases had a more benign course; only two of 30 patients (7%) developed posterior fossa mass effect and brain stem compression. In one instance, 48 hours after onset of vertigo, vomiting, and bifrontal headache, lethargy ensued, leading to coma over an 8-hour period. CT scan documented an area of low density deep in the upper portion of the right cerebellar hemisphere and vermis, with compression of the 4th ventricle and marked supratentorial hydrocephalus (Figure 4). The patient died without regaining consciousness after posterior fossa decompression with resection of infarcted cerebellar tissue. The other patient, a 19-year-old male with mitral valve prolapse and a history of drug abuse, developed a left SCA distribution infarct affecting the deep white matter of the cerebellar hemisphere, with associated 4th ventricular compression and hydrocephalus. Posterior fossa decompression with removal of necrotic cerebellar tissue was followed by survival and no neurological impairment. Mortality for the 30 patients with SCA distribution infarcts was 7%, and only one of the 28 survivors had persistent residual neurological deficits. The majority of these patients were either minimally disabled (20) or neurologically intact (seven)

**FIGURE 2.** Coronal plane computed tomographic scan with infarction in entire territory of left posterior inferior cerebellar artery involving lower two thirds of cerebellar hemisphere.
of the nystagmus were observed on bedside testing in our patients. In particular, vertical, upbeat nystagmus in primary gaze was not a distinctive feature of SCA cases, in which frequent involvement of the superior vermis of the cerebellum might have been expected to produce this sign.\textsuperscript{24,25} Another common physical finding in these cases was dysarthria, a sign that is particularly frequent in SCA distribution infarcts,\textsuperscript{15,26} which on occasion can present with isolated cerebellar dysarthria in the setting of a unilateral paravermian infarct.\textsuperscript{27} This suggests that the dysarthria of SCA distribution infarcts can be due to the cerebellar infarct itself, especially in the absence of other clinical signs indicative of brain stem involvement.

\textbf{FIGURE 3.} Partial infarcts in superior cerebellar artery distribution involving lateral aspect of superior cerebellar surface (arrowheads, top left panel), superior vermis (long arrows, top right panel), medial portion of hemisphere (short arrows, postcontrast enhancement, bottom left panel), and cerebellar white matter (open arrows, bottom right panel).
TABLE 3. Mechanism of Infarction in PICA and SCA Distribution Cases

<table>
<thead>
<tr>
<th>Mechanism of infarction</th>
<th>Cerebellar infarct location</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PICA ($n=28$)</td>
</tr>
<tr>
<td>Cardiogenic embolism</td>
<td>14 (50)</td>
</tr>
<tr>
<td>AF</td>
<td>6</td>
</tr>
<tr>
<td>MI</td>
<td>3</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>4</td>
</tr>
<tr>
<td>Prosthetic valve</td>
<td>1</td>
</tr>
<tr>
<td>MVP</td>
<td>...</td>
</tr>
<tr>
<td>&quot;Marantic&quot; endocarditis</td>
<td>...</td>
</tr>
<tr>
<td>Artery-to-artery embolism</td>
<td>...</td>
</tr>
<tr>
<td>Local arterial disease</td>
<td>14 (50)</td>
</tr>
<tr>
<td>VA occlusion</td>
<td>7</td>
</tr>
<tr>
<td>VA stenosis</td>
<td>3</td>
</tr>
<tr>
<td>PICA occlusion</td>
<td>3</td>
</tr>
<tr>
<td>Distal BA stenosis</td>
<td>...</td>
</tr>
<tr>
<td>SCA occlusion</td>
<td>...</td>
</tr>
<tr>
<td>SCA stenosis</td>
<td>...</td>
</tr>
<tr>
<td>? Vasculitis</td>
<td>1</td>
</tr>
</tbody>
</table>

$n$, Number of patients. Values in parentheses are percent. PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery; AF, atrial fibrillation; MI, myocardial infarction; MVP, mitral valve prolapse; VA, vertebral artery; BA, basilar artery.

The radiological aspects of our cases were remarkable for a predominance of partial infarcts in both vascular territories, especially in the SCA, in which no example of full infarction was recorded. This probably explains the rarity of the “classic” SCA syndrome characterized by ipsilateral limb ataxia, Horner’s syndrome, and choreic dyskinesia, with contralateral thremoanalgesia, that results from combined superior cerebellar and lateral tegmental pontine infarcts in proximal SCA trunk occlusion. The rarity of the classic clinical syndrome has also been observed in clinicopathologic series: in a group of 33 pathologically studied cases, Amarenco and Hauw found only one example of the classic syndrome, the others being variable presentations of either pure cerebellar infarcts or a combination of cerebellar and distal basilar artery distribution brain stem infarcts. Our PICA distribution infarcts, by contrast, involved the full cerebellar hemispheric territory in one fourth of the cases. However, none of these patients developed a lateral medullary syndrome, indicating that the completeness of the accompanying medullary infarction is not predictive of the size of the cerebellar infarction. This may be related to the frequently independent origin from the distal vertebral artery of the branches that supply the lateral medulla. In other instances, a lateral medullary syndrome with partial sparing of the cerebellar hemisphere in proximal PICA occlusions results from the effectiveness of collaterals, primarily from SCA to PICA territory, across the cerebellar surface. The observation of frequently partial infarcts in the SCA territory and the relatively higher frequency of complete infarcts in the PICA distribution may relate to differences in the mechanism of infarction in both vascular territories. The higher frequency of embolic mechanism in SCA of deep white matter of right cerebellar hemisphere, with effacement and displacement of 4th ventricle (left panel), dilatation of temporal horns of lateral ventricles (left and right panels) and 3rd ventricle (right panel), and extension of infarction into superior vermis (right panel).
distribution infarcts observed in this series and in that of Amarenco and Hauw\textsuperscript{32} may relate to the occurrence of partial infarcts along branches of the main SCA trunk, whereas the frequently proximal atherothrombotic occlusions of the vertebral artery or PICA in cases of PICA distribution infarction\textsuperscript{21,33} are more likely to produce cerebellar infarcts in the full territory of those arteries. The association between cardiogenic embolism and predilection for SCA territory cerebellar infarction has been further suggested by the pathological study of Amarenco et al.,\textsuperscript{34} which documented SCA infarction in 16 of 26 (62\%) cases of cardiac embolic source; only 31\% and 7\% of the infarcts occurred in the PICA and AICA territories, respectively.

Major differences in prognosis were documented between SCA and PICA infarcts in this series. Although rare instances of poor prognosis in SCA infarcts have been documented,\textsuperscript{12,15} they are generally due to either accompanying brain stem infarction resulting from distal basilar artery occlusion\textsuperscript{2,12,32} or the presence of multiple, bilateral cerebellar and supratentorial infarcts.\textsuperscript{35} The usually benign course of isolated, unilateral SCA distribution cerebellar infarcts in our series correlated with small partial lesions of the superior cerebellar surface with minimal or no mass effect. Similar observations were recently reported by Amarenco et al.\textsuperscript{16} in a series of nine cases of cerebellar infarction in the distribution of the lateral branch of the SCA. Six of the nine exhibited prominent unsteadiness of gait or falls, and only three patients (33\%) had vestibular symptomatology at onset, with the most common physical finding (in eight of the nine cases) being dysmetria of the ipsilateral limbs.

Our two SCA patients who developed cerebellar edema and brain stem compression had infarcts located in the deep hemispheric white matter and dentate nucleus. This observation suggests that documentation by MRI of large SCA cerebellar infarction with deep white matter involvement at presentation should identify the patient as one at risk for the development of brain stem compression secondary to edema. On the other hand, patients with imaging studies showing partial cortical surface infarction can be expected to follow a benign course and not require posterior fossa decompression. The PICA distribution infarcts have a higher tendency to lead to severe mass effect and brain stem compression by virtue of more common involvement of the entire cerebellar territory of that artery.\textsuperscript{35,36} Because the presence or absence of an associated lateral medullary syndrome does not appear to be predictive of cerebellar infarct size, only close clinical monitoring and serial CT or MRI examinations can be relied on for early detection of mass effect, brain stem compression, and obstructive hydrocephalus. The neurological monitoring during the early stages of cerebellar swelling should attempt to separate the physical findings indicative of associated brain stem infarction from those due to mechanical compression from the swelling lateral or bilateral hemiplegia suggests accompanying brain stem infarction,\textsuperscript{37} whereas onset of ipsilateral 6th nerve palsy or horizontal gaze palsy should alert the physician to the likely presence of direct lateral pontine compression by the adjacent swollen cerebellar hemisphere.

\textbf{Acknowledgments}

We are grateful to Dr. Pierre Amarenco and colleagues from the Laboratoire de Neuropathologie Raymond Escourolle, Hôpital de la Salpêtrière, Paris, France, for making available to us their manuscripts on cerebellar infarction that were still unpublished (in press) at the time of completion of our study.

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