Basilar Branch Pontine Infarction
With Prominent Sensory Signs

Cathy M. Helgason, MD, and Andrew C. Wilbur, MD

We identified 10 patients with acute pontine infarction and specific sensory findings. Two patients had pure sensory symptoms, two had sensory complaints of the hand and mouth, and the other six had hemisensory loss referable to medial lemniscal or spinothalamic tract dysfunction but localized to one limb, to an arm and leg, or to the face, characteristic of stroke localized to the cerebral hemisphere. All patients had magnetic resonance imaging showing infarction of the medial or lateral pontine tegmentum and a patent basilar artery. No definite source for cardiogenic thromboembolism was found. Infarcts in the midline extending from the base of the pons posteriorly into the tegmentum suggested basilar branch occlusion, while infarcts involving only part of the tegmentum probably resulted from small penetrator branch occlusion. Vertigo, light-headedness, or cranial nerve dysfunction suggested a pontine location of neurological dysfunction in these patients, but the nature of the sensory findings did not always predict the lateral, medial, inferior, or superior extent of tegmental infarction. (Stroke 1991;22:1129–1136)

Isolated reports suggest that a pure sensory syndrome can be caused by pontine hemorrhage or infarction. We describe 10 patients with pontine infarction who presented with sensory complaints. All patients had ischemic changes localized to the pontine tegmentum by magnetic resonance imaging (MRI). Analysis of the symptoms and signs should aid in the diagnosis of basilar branch disease and provide insight into the afferent sensory system.

Subjects and Methods

Between June 1989 and June 1990 we identified 10 consecutive patients who had pontine infarction and acute sensory findings. Each patient was examined by one neurologist through the Stroke Service at one of the University of Illinois College of Medicine at Peoria hospitals, St. Francis Medical Center or Methodist Medical Center. All MRI scans were interpreted by a radiologist without knowledge of the clinical findings.

All patients had high-resolution cranial MRI with a 1.5-T superconducting unit, using a 5-mm slice thickness and T2-weighted axial spin/echo imaging with repetition times ranging from 2,000 to 3,000 msec, first echo times of 20 or 25 msec, and second echo times of 80 or 90 msec. Six patients also had T2-weighted coronal MRI scans with similar pulse sequence parameters.

All patients underwent neurological examination. Sensory testing included pinprick, touch, vibration, temperature, proprioception, and stereognosis. Only the abnormalities detected on examination are described in each case.

Case 1. A 53-year-old white man had the sudden onset of right leg tingling and numbness and an inability to place his foot on the ground correctly during ambulation. He became light-headed and had difficulty writing, with tingling and numbness of his right hand. On examination, bilateral carotid bruits were noted. His blood pressure was 192/100 mm Hg, and his pulse was regular. There was right-sided hyperreflexia and a right Babinski's sign. Clumsy, rapid, alternating movements of his right hand, intention tremor on right heel-to-shin testing, a wide-based gait, and veering to the right were noted. He had difficulty placing his right foot where he wanted. Perception of pinprick was decreased on his right leg below the knee. A left hemisphere infarct was suspected. The MRI and test results are summarized in Table 1 and Figures 1 and 2. His gait improved.

Case 2. A 60-year-old white man had the sudden onset of numbness in his left arm and leg on a background of congenital spina bifida, arrested hydrocephalus, paraparesis, and incontinence. Examination revealed a blood pressure of 128/86 mm Hg and a regular pulse. Bilateral muscle wasting below the knees was present, with clubbing deformity of both feet and no motion at the ankles or toes. Sensation was absent...
TABLE 1. Test Results and Risk Factors for Stroke in 6 Patients With Pontine Basilar Branch Occlusion

<table>
<thead>
<tr>
<th>Case</th>
<th>WM</th>
<th>BG</th>
<th>Patent BA</th>
<th>Pontine infarct location</th>
<th>Echocardiogram, chest roentgenogram</th>
<th>Carotid duplex sonogram</th>
<th>Cerebral angiogram</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>L sup post tegmentum</td>
<td>↓ LV compliance, global LV hypokinesis</td>
<td>L ICA b moderate irregularity</td>
<td>ICA siphon irregularity, patent L VA</td>
<td>HTN; peripheral vascular disease; tobacco, alcohol use; ↑ chol</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>R post lat low tegmentum</td>
<td></td>
<td>Normal</td>
<td>. .</td>
<td>HTN; old myocardial infarct; hx LOC; ↑ chol</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>L lat mid sup tegmentum, R med low &amp; mid tegmentum</td>
<td>LV apical hypokinesis, cardiomegaly</td>
<td>Tight R ICA b, mild L ICA b, stenosis</td>
<td>L ICA b mild stenosis (MRI angiography)</td>
<td>HTN; CABG; hx R ICA occlusion, R middle cerebral artery infarct; ↑ chol, ↑ TG</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>L mid sup base &amp; tegmentum</td>
<td>Severe mitral anulus calcification, AV calcification, stenosis &amp; regurgitation, LV hypertrophy</td>
<td>Normal</td>
<td>. .</td>
<td>HTN; congestive heart failure; hx LOC</td>
</tr>
<tr>
<td>5</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>L mid sup base &amp; med tegmentum</td>
<td>Normal</td>
<td>Normal</td>
<td>L ICA b occlusion, reconstituted via thyrrocervical trunk</td>
<td>HTN; ↑ chol, TG; tobacco use; ↑ platelets</td>
</tr>
<tr>
<td>6</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>R high mid tegmentum</td>
<td>Global LV, inferior wall hypokinesis, aortic insufficiency, left atrial enlargement</td>
<td>L ICA b moderate stenosis</td>
<td>. .</td>
<td>CABG; HTN; ↑ chol, ↑ LDL, ↓ HDL</td>
</tr>
<tr>
<td>7</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>L mid base &amp; med tegmentum, R low mid base &amp; med tegmentum</td>
<td>Normal</td>
<td>Normal</td>
<td>L ICA b moderate stenosis; patent VA &amp; BA</td>
<td>HTN; tobacco use</td>
</tr>
<tr>
<td>8</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>L mid low med tegmentum</td>
<td>Normal</td>
<td>Normal</td>
<td>. .</td>
<td>↑ chol; HTN</td>
</tr>
<tr>
<td>9</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>Central &amp; med mid sup tegmentum</td>
<td>Normal</td>
<td>Mild bilateral ICA b stenosis</td>
<td>Anterior communicating artery aneurysm</td>
<td>Supraventricular tachycardia; ↑ chol</td>
</tr>
<tr>
<td>10</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>R low &amp; mid low base &amp; med tegmentum</td>
<td>Normal (transesophageal echocardiography)</td>
<td>Normal</td>
<td>Normal VA &amp; BA</td>
<td>Tobacco, drug abuse; ↑ cerebrospinal fluid IgG &amp; protein</td>
</tr>
</tbody>
</table>

MRI, magnetic resonance imaging; WM, white matter; BG, basal ganglia; BA, basilar artery; L, left; R, right; sup, superior; post, posterior; lat, lateral; med, medial; LV, left ventricle; AV, aortic valve; ICA, internal carotid artery; b, bifurcation; VA, vertebral artery; HTN, hypertension; chol, cholesterol; hx, history of; LOC, loss of consciousness; CABG, coronary artery bypass graft; TG, triglycerides; LDL, low density lipoprotein; HDL, high density lipoprotein.

below the knees. He had a moderate intention tremor on finger-to-nose maneuver on the left. Marked decreased perception of pinprick and vibration was noted on his left arm from the elbow to the dorsum of the hand, especially on the fourth and fifth digits. A right thalamic infarct was suspected. The MRI and laboratory results are given in Table 1 and Figures 1 and 3. His hospital course was unremarkable.

Case 3. A 59-year-old white man had a 3-week history of intermittent right arm and leg numbness and weakness, a feeling of right arm puffiness, and right leg tingling. On examination, his blood pressure was 180/100 mm Hg and his pulse was regular. There was constructional apraxia, left spastic hemiparesis, and depressed perception of pinprick in his left arm. Surprisingly, no right body signs were present. A left hemisphere infarct was suspected. The MRI and test results are given in Table 1 and Figure 1. The patient ceased to have right body symptoms.

Case 4. An 80-year-old white woman lost consciousness for a brief period. When she regained consciousness she could not ambulate and she veered to the right, slurred her speech, and described numbness in her right hand. On examination, her blood pressure was 180/100 mm Hg and her pulse was regular. She had left facial weakness. Her deep
tendon reflexes were brisk throughout. She veered to the right on walking and had depressed fine motor motion of her right hand and bilateral intention tremors on finger-to-nose testing (left limbs worse than right). Sensory examination was normal. The MRI and test results are given in Table 1 and Figure 1. Her right hand control improved.

Case 5. A 50-year-old white woman developed right face and hand numbness followed by slurred speech, the inability to grasp objects or write with her right hand, and loss of balance. On examination her blood pressure was 194/90 mm Hg and her pulse was regular. She had bilateral carotid artery and left vertebral artery bruits. She was dysarthric. She had mild weakness of her right face and a decrease in fine motor motion of her right hand. Dorsiflexion of her right foot was weak, and she fell to the right on tandem walk. Right hyperreflexia and Babinski's sign were present. The perception of pinprick was lost in her right hand. A left parietal infarct was suspected. The MRI and test results are given in Table 1 and Figure 1. Her symptoms improved.

Case 6. A 62-year-old white man reported sudden numbness and tingling in his arms and legs. He felt light-headed, stumbled, and dropped back into a chair. Afterward, he noted increased numbness in his right arm with fine motor difficulty in his right hand. His right hand and leg were clumsy. On examination, his blood pressure was 206/104 mm Hg and his pulse was regular. Mild left facial weakness and a right

FIGURE 1. Location of pontine infarcts as interpreted by magnetic resonance imaging for cases 1-10 (a through j), respectively.

FIGURE 2. Case 1. Axial (left) and sagittal (right) T2-weighted magnetic resonance images showing infarct in upper left pons (resonance time = 2,500 msec; echo time = 90 msec).
intention tremor on finger-to-nose testing were seen. He fell to the right when he ambulated. Perception of pinprick was diminished in his right leg, trunk, and arm; perception of vibration and proprioception were diminished in his right arm and leg. A left thalamic infarct was suspected. The MRI and test results are given in Table 1 and Figure 1. The patient's gait normalized, but he had residual tingling in his right limbs.

Case 7. A 57-year-old white woman noted right leg weakness. The next day, she slurred her speech and developed right hand, forearm, great toe, and dorsal pedal numbness. Examination revealed a blood pressure of 140/80 mm Hg and a regular pulse. Her tongue deviated to the right, and she had mild diffusely distributed arm and leg weakness. Her deep tendon reflexes were brisk, and there was a right Babinski's sign. Months later she noted increased dysarthria, difficulty swallowing, and left-sided clumsiness; her previous deficits had cleared. On examination, her blood pressure was 182/100 mm Hg and her pulse was regular. She had marked dysarthria, uncontrollable bouts of laughter and crying, difficulty handling her saliva, and weakness of her left face. Her reflexes were brisk. No Babinski's sign was present. Depressed perception of pinprick was found on her left leg. She had an MRI and other tests (see Table 1 and Figure 1). Over the following months, the patient remained pseudobulbar in affect. Her dysarthria and dysphagia cleared. She developed a prominent sensation of numbness in her fingertips and around her mouth. Repeat MRI showed no change.

Case 8. A 59-year-old black woman had lightheadedness and right cheek and left arm volar surface numbness, followed by a burning sensation and bilateral cheek and lip numbness. There was ringing in her ears. Neurological examination showed decreased perception of pinprick on her left face. Her blood pressure was 155/100 mm Hg, and her pulse was regular. The MRI and test results are given in Table 1 and Figure 1. Her symptoms remained unchanged.

Case 9. A 69-year-old white man developed left greater than right arm numbness. When he walked, he veered to the left. He felt light-headed. His symptoms lasted for several hours. Neurological examination was normal. His blood pressure was 145/80 mm Hg and his pulse was regular. His MRI and test results are given in Table 1 and Figure 1. He remained asymptomatic.

Case 10. A 39-year-old male intravenous drug abuser had the sudden onset of left-sided weakness and dysarthria followed by the inability to ambulate. On examination, his blood pressure was 160/100 mm Hg and his pulse was regular. He had right internuclear ophthalmoplegia, weakness of his left face, and nasal dysarthric speech. His limbs were weak distally. Mild intention tremors were present in his right arm and leg. There was left hyperreflexia and bilateral Babinski's signs. Sensory examination revealed extinction on the left side of his body to double simultaneous stimulation. His MRI and test results are given in Table 1 and Figure 1. He developed uncontrollable fits of laughter, and his gait improved.

Results

Ten patients, six men and four women, aged 39–80 years, had new sensory complaints and pontine infarction. The stroke was sudden and maximally manifest in four patients, progressed gradually in two patients, and progressed intermittently or stuttered in four. One patient had a significant residual neurological deficit, and the others either improved or were neurologically stable with minimal residual deficit. All patients were functioning independently at the time of discharge from the hospital.

The patients had infarcts ranging in size from 1.5 to 1.8 cm³ and localized by MRI to the regions depicted in Table 1. Where the basis pontis was involved, the infarcts extended posteriorly into the tegmentum (Figure 1). All patients had a patent basilar artery defined by MRI.

Seven patients had hypertension prior to the onset of infarction. No patient had diabetes mellitus or a pathologically proven cardiogenic source for thromboembolism. All but one patient had echocardiogra-
phy and chest roentgenography performed. Four patients had evidence of cardiac disease. Findings suggestive of a cardiac source for thromboembolism included cardiomegaly, global hypokinesia, focal wall dyskinesia, and mitral anulus calcification. Five patients had cerebrovascular extracranial or intracranial circulation atheroma defined by duplex scanning or cerebral angiography. Cerebral angiography performed in three patients failed to reveal vertebral basilar artery abnormalities (Table 1).

Presenting symptoms included a light-headed sensation in four patients and loss of consciousness in one. Four patients had slurred speech, and one had diplopia. Ataxia was found in seven patients who veered when walking and had loss of balance or clumsiness. Hand and face numbness were the initial symptoms in three patients. Two patients had perioral as well as hand numbness, and seven patients either numbness or tingling in the arm and leg or in the arm or hand alone. One patient had tinnitus, one a puffy feeling in the arm, and another a perioral burning sensation. In all but two patients (cases 4 and 10), sensory complaints were either confirmed by examination of the patient or distinguished by the patient from motor signs at presentation.

Discussion

Lacunar-sized (≤1.5 cm in diameter) pontine infarction is associated with pure motor ataxic hemiparesis or the clumsy hand–dysarthria syndrome.1 Basilar branch occlusion or basilar artery thrombosis may produce larger infarcts with combined sensory, motor, and cranial nerve dysfunction.2–3 Sensory complaints, which occur alone or in a facial/brachial distribution, resemble those of hemispheric stroke of a thalamic or parietal location.4–6 Internal capsular posterior limb infarction may also cause hemisensory loss, and recent reports attribute pure sensory findings to pontine stroke.7–10 Hommel et al8 described an infarct in the right paramedian pons at the junction of the upper and lower two thirds of the tegmentum. This patient had a hemisensory complaint of numbness and tingling over the left side of the body exacerbated by touch or rub, and findings implicated medial lemniscus dysfunction and consisted of loss of joint and position sense in both limbs. Stereognosis, pain, and temperature perceptions were normal. Araga et al9 and Graveleau et al10 attributed pure sensory complaints to pontine hemorrhage localized to the dorsal lateral pontine tegmentum. Their patients had dyesthesias and decreased perceptions of touch, especially of the mouth and hand. Bilateral cheiro-oral syndrome due to unilateral paramedian tegmental pontine hemorrhage was described by Matsumoto et al.11 Usually the syndrome reflects a thalamic or parietal lesion. The pontine location for this symptom complex is unusual.12–14 Burning oral and midfacial pain has been attributed to dysfunction of the trigeminal nucleus or spinal tract after ventral pontine infarction.15

Hemorrhage or infarct localized to the paramedian or lateral tegmentum may be associated with mixed sensorimotor or combined sensory modality dysfunction of medial lemniscal or spinothalamic tract dysfunction. Caplan and Goodwin16 described three cases of lateral superior and inferior pontine tegmental hemorrhage associated with severe contralateral sensory deficits to pinprick, position, or all sensory modalities combined with motor and other neurological dysfunction. Kudo et al17 described a case of dorsal lateral tegmental hemorrhage in which asterixis accompanied by mild hemiparesis was overshadowed by profound contralateral hemisensory loss to all modalities and ataxia. A case of tetra-ataxic hemiparesis associated with posterior pontine midline base lacunar-sized infarction was characterized by a persistent and severe tingling sensation of both hands despite normal sensory findings on clinical examination by Van Gijn and Vermeulen.18 Only two patients of our series described pure sensory complaints (cases 2 and 8). Usually, sensory loss was a predominant component of a more complex array of neurological dysfunction that included ataxia, hemiparesis, reflex asymmetries, and dysarthria. One patient had transient extraocular motility dysfunction (case 10), and others had vertigo or light-headedness at the onset of their stroke. Cases 1, 4, and 5 resemble the ataxic hemiparesis or clumsy hand–dysarthria syndrome which, when localized to the pons, traditionally is not accompanied by sensory findings.19,20 Cases 4 and 6 had loss of consciousness and bilateral neurological signs suggesting dysfunction of the reticular activating system or bilateral brain stem dysfunction.

Hommel et al8 and others9,10 suggest that sensory loss in pontine infarction runs true to tract type. When correlated with the anatomic site of the infarct on MRI, the sensory findings of the patients in our series did not always support this theory (Table 2). When pure sensory symptoms were the presenting or enduring complaint, one patient had lateral paramedical and two had medial tegmental infarction (cases 2, 7, and 8). All three patients noted “numbness.” The patients with cheiro-oral complaints (cases 7 and 8) did not display findings or loss of proprioceptive or vibratory perception, even when the infarct on MRI appeared to involve the medial lemniscal tract. Case 2 complained of “numbness” and had a lower pontine lateral tegmental infarct where the spinothalamic tract courses but sensory loss suggesting both medial lemniscus as well as spinothalamic tract involvement. In some instances, however, sensory findings correlated nicely with the anatomic site of the infarct. Case 1 had decreased perception of pinprick consistent with spinothalamic dysfunction and difficulty placing the same leg when ambulating, suggesting ataxia due to proprioceptive loss as might be seen with medial lemniscal tract dysfunction. The medial lemniscal and spinothalamic sensory tracts approach one another anatomically as they ascend in the pons (Figure 4). Case 1’s infarct appropriately was in the superior lateral pontine tegmentum. Brodal21 suggests that the final definition of sensory representa-
TABLE 2. Location by MRI of Pontine Infarcts and Clinical Sensory Complaints and Findings of Spinothalamic or Medial Lemniscal Type in 10 Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Medial</th>
<th>Lateral</th>
<th>Inferior</th>
<th>Superior</th>
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<tbody>
<tr>
<td>1</td>
<td>Spinothalamic, medial lemniscal sensory type; ataxia, &quot;numbness,&quot; tingling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Spinothalamic, medial lemniscal sensory type; ataxia, &quot;numbness&quot;</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>3</td>
<td>Puffiness, &quot;numbness,&quot; tingling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Ataxia, &quot;numbness&quot;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Spinothalamic sensory type; ataxia, &quot;numbness&quot;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Spinothalamic, medial lemniscal sensory type; ataxia, &quot;numbness,&quot; tingling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Spinothalamic sensory type; &quot;numbness&quot;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Spinothalamic sensory type; &quot;numbness,&quot; burning, ringing in ears</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Ataxia, &quot;numbness&quot;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Ataxia, extinction</td>
<td></td>
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</tr>
</tbody>
</table>

The MRI-defined pontine infarcts in this series were lacunar in size. There was no evidence for basilar artery stenosis or occlusion. Biller et al.22 and Knepper et al.23 suggest that traditional axial and coronal T1- and T2-weighted MRI scans may be used reliably to detect basilar artery thrombosis or occlusion. Finer details of basilar artery irregularity or degree of stenosis would not be defined by this method. The etiologic role of thromboembolic or atheroembolic events with regard to small unilateral pontine infarcts has yet to be defined. Therefore, the significance of the presence of potential cardiac sources for thromboembolism in these patients is undefined. The near-universal presence of hypertension, unilaterality of small pontine infarcts, and absence of a definite thromboembolic source suggests the presence of intracranial atheromatous or lipohyalinotic disease of the paramedian, circumferential, superior cerebellar, or anterior inferior cerebellar branches of the basilar artery (Figure 4).24 The good prognosis of these patients argues against progressive vertebral or basilar artery thrombosis, but not necessarily against embolism, as the etiology of stroke.

As shown by examination and MRI scans of the present series, pontine infarction may present with pure sensory dysfunction but most often is accompanied by other findings. Sensory complaints may suggest medial, lateral, inferior, or superior tegmental involvement, but it may be difficult to distinguish the anatomic site of the infarct on clinical grounds alone.
Although pathological proof is lacking, the etiology of the infarcts appears to be related to intracranial vascular occlusion due to intrinsic vascular disease. The isolated findings of a monoappendicular or facial/brachial distribution of sensory loss suggest cerebral hemispheric localized dysfunction. These patients, who had good prognoses, must be distinguished clinically from those with infarction due to large-vessel occlusive or thromboembolic mechanisms. The capability of MRI to accurately detect and localize pontine infarction and suggest basilar artery patency may allow this distinction to be made.25

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References


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