Occlusive vascular disease of the rostral basilar artery frequently causes infarction of midbrain, thalamus, and portions of the temporal and occipital lobes fed by posterior communicating and posterior cerebral arterial tributaries of the basilar artery. Clinical signs include an array of visual, oculomotor, and behavioral abnormalities, usually without prominent motor dysfunction, which may confuse those inexperienced with these findings. Segarra used the term "the syndrome of the mesencephalic artery" to describe rostral paramedian brainstem infarction, and analyzed one type of behavioral manifestation ("somnolent mutism"). Others have described clinicopathologic aspects of isolated neuroophthalmologic and behavioral features. This report, based principally on experience from personally examined cases with autopsy, computed tomography (CT), or angiographic verification, reviews the major clinical features. Aspects of the syndrome considered in detail elsewhere (e.g., memory loss and alexia without agraphia) are given only passing attention. The "top of the basilar" syndrome is a recognizable subdivision that can be distinguished from the large group of heterogeneous conditions usually lumped together under the term "vertebrobasilar insufficiency." Infarction of rostral brainstem and posterior hemispheres often coexists because of the common vascular supply. However, because either may occur in isolation, it is appropriate to consider brainstem and hemisphere manifestations separately.

Part I. Rostral brainstem infarction. Visual defects. Disorders of ocular movement. Oculomotor dysfunction in patients with bilateral ischemia of rostral midbrain and posterior thalamus is indistinguishable from that in patients with thalamic hemorrhage. The signs include:

**Disorders of vertical gaze.** Voluntary or reflex vertical gaze (tested by oculocephalic and caloric maneuvers and Bell phenomenon) is often abolished. One or both eyes may rest in a downward position. Isolated paralysis of upward or downward gaze occurs less frequently. In human patients with vertical gaze paralysis due to vascular disease, bilateral lesions are found in the midbrain tegmentum. In monkeys and humans, lesions of the pretectum, in the region of the posterior commissure, are necessary to produce paralysis of upward gaze. Isolated downward gaze palsy is rare, and may be associated with lesions medial and dorsal to the red nucleus. Lesions causing paralysis of downward gaze are more ventral and caudal in the midbrain tegmentum than those responsible for deficits of upward gaze.

**Disorders of convergence.** One or both eyes may rest in an inward position. Hyperconvergence or convergence spasm may be observed when the patient attempts conjugate lateral or vertical gaze. Convergence retraction nystagmus, a rhythmic inward beating movement of the eyes, may be spontaneous, but is best elicited by having the patient fix on an optokinetic stimulus which is moving upward.

"Pseudosixth." This sign, described by Fisher, refers to failure of ocular abduction which is not due to dysfunction of the sixth nerve. The sign is frequently bilateral and is accompanied by "hyperconvergence." Failure of the eye to abduct is due to two mechanisms: (1) Fixation with the hyperconvergent eye (the right eye in figure 1). When the hyperconvergent eye is covered,
monocular fixation by the abducting eye on a distant object far to the side may elicit further abducting movements. (2) Convergence vectors are also present in the abducting eye and neutralize or counteract conjugate lateral movements. If the abducting eye is watched carefully, convergence or adducting jerks of the abducting eye are often present.

Elevation and retraction of the upper eyelids (Collier sign) may be unilateral or bilateral; retraction of one eyelid may contrast with a droop of the opposite lid.

Sudden darting or lightning-like oscillations of the eyes may complicate horizontal or vertical gaze.

Skew deviation (ocular divergence in the vertical plane) has been documented in lesions of the middle cerebellar peduncle and medulla. "Midbrain skew" has been inferred by Smith, David, and Klintworth because of accompanying signs of dysfunction of the pupils and third nerve believed clinically localized to the periaqueductal gray region of the midbrain. Autopsy verification of a rostral brainstem cause of skew deviation is uncommon, but is provided by cases 1 to 3 (described below).

Vertical nystagmus, conjugate horizontal nystagmus, and ocular bobbing are not associated with high brainstem infarction. When infarction is more caudal and includes the midbrain tegmentum ventral to the aqueduct, internuclear ophthalmoplegia or third-nerve palsies are present. Bilateral infarction of the third nerve nucleus and adjacent reticular formation causes hypersomnolence and third nerve palsies, difficult to distinguish clinically from the brainstem dysfunction of transtentorial herniation. The sudden onset and the absence of severe headache, vomiting, or hemiplegia prior to the appearance of stupor are characteristics of primary midbrain infarction which help to distinguish the two entities. Computerized tomography (CT) documenting the absence of a supratentorial space-taking lesion is often a necessary corroborative diagnostic procedure. A unilateral lesion of the third nerve nucleus may cause severe bilateral ptosis. Pupils. Diencephalic dysfunction may interrupt the afferent limb of the pupillary light reflex arc. Bilateral sympathetic dysfunction usually accompanies the lesion, so that the pupils are small and the reaction to light is often transient and of small magnitude. A magnifying glass may be needed to separate the tiny, poorly reactive pupils of thalamic disease from small, reactive "pontine pupils." With lesions more caudal in the midbrain, large or midposition fixed pupils are caused by dysfunction of the Edinger-Westphal nucleus or its fibers.

The pupil can quickly assume an eccentric position in the iris, a phenomenon called "corectopia iridis." The pupil may shift from a central position to an eccentric one intermittently, a sign characteristic of midbrain lesions.

Three examples of rostral brainstem infarction illustrate the neuroophthalmologic findings.

Case 1. An elderly diabetic woman suddenly became sleepy and unresponsive. Her left eye was deviated laterally and the left pupil was midposition and fixed. The right eye was deviated down and in, and had frequent convergent inward movements. Neither eye moved vertically (either voluntarily or reflexly). On attempted conjugate lateral gaze, each eye could abduct but did not reach the lateral canthus. The right pupil was 2 mm and had a transient and minimal reaction to light. Right hemiplegia, hypesthesia to pinprick on the right half of the body, and bilateral Babinski signs were present.

Postmortem examination revealed a fresh organizing myocardial infarction. There was an old cystic infarction in the right caudate nucleus, putamen, and internal capsule. A fresh necrotic lesion (figure 2) due to embolic infarction involved the distribution of the left superior cerebellar and left posterior cerebral arteries. The region of the left third-nerve nucleus in the midbrain, periaqueductal gray region, red nucleus and cerebral peduncle were infarcted. The left superior cerebellar surface was also infarcted. There was a tiny infarction in the left ventrolateral thalamus.

Case 2. An 81-year-old woman suddenly believed the lights had been shut off while she was reading. There was complete bilateral ptosis; she could not open either eye. Pupils were 2 mm, and pupillary light reaction was slight, delayed, and transient. The eyes were deviated conjugately to the right with slight downward positioning of the left eye. Right conjugate gaze was full; on left gaze, there was slight adduction of the right eye and only minimal abduction of the left eye. There was no vertical gaze. She was hypersomnolent for 1 week and exhibited transient right visual inattention.

Postmortem examination in another hospital years after the stroke revealed a cavitary lesion of the mid-brain.
Case 3. After 4 days of dizziness and unsteady gait, an 80-year-old woman suddenly collapsed. The right pupil was 2 mm and fixed. The eyes did not move past the midline to the right on oculocephalic or caloric stimuli, but these maneuvers elicited full left gaze. Vertical gaze could be elicited by oculocephalic reflexes. There was a right hemiplegia, right hemisensory loss and right hemianopia. She remained mute. Subsequently, the right eye moved to a down and in position and, on right gaze, the right eye did not abduct as well as the left adducted, but conjugate gaze to the right was possible. Postmortem examination revealed occlusion of the top of the basilar artery and a paramedian infarction in the midbrain and anterior thalamus. Castaigne and associates described a patient with bilateral third nerve palsies who remained in a sleeplike state for 3 years. Postmortem examination revealed occlusion of the rostral basilar artery with thalamic and midbrain infarction and bilateral posterior cerebral artery territory infarction, more extensive on the left.

Behavioral abnormalities. Somnolence. Sleepiness, apathy, and lack of attention to the environment result from infarction of the rostral medial reticular formation due to occlusion of the mesencephalic artery (the proximal portion of the posterior cerebral artery) or its penetrating branches. Facon, Steriade, and Werthein described a patient with bilateral third nerve palsies who remained in a sleeplike state for 3 years. Postmortem examination revealed occlusion of the top of the basilar artery and a paramedian infarction in the midbrain and anterior thalamus. Castaigne and associates described a patient with a butterfly-shaped infarction destroying intralaminal nuclei, the medial part of the centrum medianum, third nerve nuclei and part of the brachium conjunctivum; hypersomnolence and ophthalmoplegia were the major findings. Segarra used the term "somnolent mutism" to characterize the behavior of patients with high medial brainstem infarction and contrasted it with the "coma vigil" of patients with bilateral cerebral lesions.

Peduncular hallucinosis. Hallucinations occur but are rare in patients with high brainstem infarction. They may occur without visual field defects. These hallucinations are usually vivid and well-formed. One patient with a clinically unilateral midbrain and thalamic infarction saw a colored parrot with beautiful plumage off to his right, and another patient with episodic posterior hemispheral ischemia awakened at night and saw...
pictures of his grandmother flashed on the wall to his left, as if projected in a home movie. Rarely, the same patient heard a knocking noise, as if rocks were in a car engine. Though vivid to the patients, the hallucinations were always recognized as somehow not "real."

The term "peduncular hallucinosis" was used in a review by van Bogaert to describe strange hallucinations, usually visual, in patients with midbrain lesions. (The term "pedonculaire," when used in this context, refers to the midbrain, not necessarily the cerebral peduncle.) Anatomic verification of this phenomenon is scanty. Lhermitte, who was the first to introduce this term, described a patient in detail. A 72-year-old woman complained of vertigo and subsequently developed headache, vomiting, and bilateral sixth nerve palsies. Left ophthalmoplegia, a left central scotoma, and intention tremor of both arms subsequently developed. She had vivid hallucinations of animals—cats and chickens, with strange appearances. She also saw children at play with toys. A child would suddenly change into an old woman. She would try to touch the images, but was aware that they were not real. She also had insomnia at night and slept a great deal during the day. Hallucinations occurred only during late daylight hours, especially at sundown. There was no anatomic verification, but the lesion was thought to be a vascular brainstem lesion. Alajouanine, Thurel, and Durupt described another patient, studied only clinically—a young man with a sudden hemiplegia and hemianesthesia following amputation of an infected limb. Nystagmus and ophthalmoplegia indicated a brainstem lesion. He saw blood and red hair descending toward the bed, and had vivid imaginings of being vertically placed on an ambulance bed amid animals. This was a febrile, ill patient, again without anatomic verification.

A single pathologically verified case of peduncular hallucinosis concerned a patient with a clinical lesion of the red nucleus who died 14 months later. A 59-year-old woman with rheumatic heart disease developed vertigo, double vision, and ataxia. The major findings were a right third nerve palsy, dysmetria of the left limbs, left hyperreflexia, and gait ataxia. From the onset, she had vivid hallucinations accompanied by severe agitation. The hallucinations always occurred in the evening; she remained calm and unaffected during the day and the remainder of the night. On the wall opposite her, she would see the head of a dog or an image of a horse or a green serpent against a red background. The images appeared and disappeared and were never fixed. Intricate lines, odd colors, and images persisted for 1 to 2 hours, and then ceased. At postmortem examination 14 months later, an infarct was seen in the left midbrain, primarily affecting the superior cerebellar peduncle, cerebral peduncle, substantia nigra, red nucleus, and periaqueductal gray region. Baruk and Reeves and Plum commented that other lesions along the base of the brain, affecting the diencephalon and midbrain, could precipitate hallucinations.

The pathologic anatomy and physiology of these hallucinations is not clear; they may be related to an abnormality of nonspecific cortical excitation (reticular formation), or abnormal stimulation or deafferentation of specialized thalamic nuclei (e.g., lateral geniculate). Similar visual hallucinations in the evening ("sundowning") are common in elderly patients without cerebrovascular disease.

Fischer-Perroudou, Mouret, and Jouvet reported a patient with distal limb pain and diarrhea in whom polygraphic EEG recordings confirmed total insomnia. Dramatic hallucinations occurred only between 9 and 11 P.M. The hallucinations disappeared after administration of 5-hydroxytryptophan had caused a return to normal sleep. No central nervous system lesions were found on postmortem examination.

An abnormality of sleep may be the essential factor, and was present in our patients with hallucinations and in those previously reported. More detailed clinicopathologic correlation is needed to verify the origin and nature of this phenomenon. The pathologic anatomy responsible for this type of hallucination is probably not limited to the midbrain alone.

"Unusual reports." Patients with rostral brainstem infarction may reply in a bizarre way to queries requiring orientation. For example, one bedridden patient, when asked her whereabouts, replied that she was on the beach at Nice sunning herself in a bikini. Another patient excused herself from replying to a question because she was speaking to some friends on the telephone, and proceeded to hold an imaginary phone before her as if to speak. These reports are similar to those given by patients with metabolic encephalopathy or frontal lobe disease.

The unusual reports have had the following characteristics: (1) They are influenced by stimuli, e.g., pictures or preceding conversation in a patient's room. A patient in whose room a native picture by Gauguin hung on the wall reported that she was in Tahiti. Another patient, when conversation nearby concerned travel, reported she was on an airplane. (2) They have no approximation to reality. Patients with amnestic disorders such as the Wernicke-Korsakoff syndrome are frequently not oriented exactly to place, but they generally give an approximate answer after looking about for clues. For example, in a hospital they will supply the name of a medical facility with which they are familiar. The patient with high brainstem disease frequently replies without exploring the en-
environment, and answers are bizarre. (3) Observations or questions of the interviewer are incorporated into the reply. For example, a patient reported that he was driving a car headed toward Beacon Street. When asked who the questioner was, he replied, "You are a policeman, and you must get out of the way of the car or you will be hit." Patients with unusual reports have all had disturbances of wakefulness, characterized by periods of sleep or drowsiness. Stuss and associates consider this an extraordinary form of confabulation; their patients all had frontal lobe disease and the confabulation was attributed to altered frontal lobe function.

Some patients with this phenomenon of "unusual reports" have commented that they "dream a lot" and often "cannot tell dreams from reality." Patients recovering from general anesthesia and normal individuals awakening from sleep frequently have difficulty determining if a mental concept arose from an actual event or from a dream. Even in the fully awake, normal individual, thoughts somewhat extraneous to the practical matters at hand are frequent distractions. The prose of James Joyce and Virginia Woolf contains easily recognizable examples of the stream of consciousness that is familiar to all of us.

The unusual reports and dreams are equally characterized by suggestibility from environmental factors and frequent absurdity. The anatomic substratum that helps us separate dreams or thoughts from reality is unknown. The appearance of dream confusion in sleeplike twilight states and anesthesia suggests a disturbance of nonspecific alerting systems, e.g., the reticular activating system and thalamic nuclei stimulating hemispheral regions. This could explain the presence of these reports in disease of either brainstem or cerebral hemispheres. Unusual reports, somnolence, and hallucinations may all be related signs of dysfunction of the rostral reticular formation of the brainstem.

Part II. Posterior cerebral artery territory hemisphere infarction. A. Unilateral infarction. Visual defects. Hemianopia. A homonymous field defect may result from a lesion anywhere in the visual radiation, from optic tract to calcarine cortex. Several features identified in 15 cases of CT-confirmed unilateral occipital infarction were correlated with lesions in or near the calcarine cortex within the posterior cerebral distribution, as opposed to more anteriorly placed lesions in the territory of the middle cerebral artery.

Awareness of the visual deficit. Patients with posterior cerebral artery lesions often complain of a void or blackness to one side. In middle cerebral artery parietal lesions, the field deficit is usually accompanied by visual neglect, and the visual defect is usually not noticed or acknowledged by the patient.

Preservation of optokinetic nystagmus. Cogan's rule that optokinetic nystagmus is lost in an occipital lobe mass lesion but preserved if the occipital lobe lesion is vascular holds up well. Temporal or parietal lesions in middle cerebral artery territory are usually associated with loss of optokinetic nystagmus to the side of the hemianopia.

Partial vision within a hemianopic field. Patients with a calcarine infarction may, on occasion, identify the color, nature or size of an object within the "blind field"; in lesions of the middle cerebral artery territory that interrupt the visual radiation, vision is usually all-or-nothing.

Homonymous but differing involvement of superior and inferior quadrants in a hemianopic field. This implies unequal involvement of both banks of the calcarine fissure and is less common in patients with lesions of the optic radiations.

Scintillations at the edge of a hemianopic field. Patients with occipital lesions frequently note poorly formed scintillations in the hemianopic field as a presenting symptom of posterior cerebral artery occlusion. Scintillations also occur when the defect is clearing and usually involve the partially affected edge of the hemianopic field. They are so commonly seen (5/15 patients) that I usually warn the patient not to be concerned if they appear.

Visual perseverations. Perseverations may take one of several forms. (1) Seeing an object repeated toward the hemianopic side; a train of individuals may seem to be repeating within the affected field. (2) If the patient looks toward the hemianopic field, he may continue to see an image that had previously been in front of him. (3) Persistence of an image in the center of the field of vision after the image has moved. The first two types of visual perseveration have been seen in patients with posterior cerebral artery disease and are not a part of dysfunction due to lesions within the middle cerebral artery territory.

Absence of visual neglect. Patients with occipital infarction do not usually neglect the hemianopic field. They read a full paragraph or headline, copy a full diagram if given time, and do not neglect one side of space when asked to bisect lines. At the onset of the deficit, however, especially if objects are shown tachistoscopically and quickly, there may be a transient tendency to neglect part of the visual field.

Behavioral defects. Left occipital infarction may be accompanied by anomia,22 alexia without agraphia,22 a temporary Korsakoff-like amnestic syndrome,10.20.22 or visual agnosia.16.22.30 Right occipital infarction has been associated with the Charcot-Wilbrand syndrome of defective visualization and absence of visual dreaming and aprosopagnosia,23 though the latter is generally associated with bilateral infarction. These syn-
dromes have been reviewed elsewhere.19

B. Bilateral infarction. Visual defects. Cortical blindness. Cortical blindness is the most severe visual defect caused by bilateral occipital infarction. Symonds and Mackenzie 18 reviewed clinical and pathologic aspects of this syndrome and identified embolus as the most common vascular etiology.

The Balint syndrome. Elements of the Balint syndrome15-45 are frequently found in infarcts of the territory of both posterior cerebral arteries. The major characteristics are:

Asimultanagnosia, or difficulty viewing the whole visual field at once. Patients may see things piecemeal and identify a part instead of the whole. Useful techniques to elicit this phenomenon include asking the patient to: (1) enumerate the number of objects on a paper (letters, words, crosses, or circles), (2) identify a number of objects shown simultaneously, (3) explain the action in a cartoon or picture, and (4) read a paragraph. Patients with the Balint syndrome usually cannot read a paragraph because they omit words or whole lines. They can, however, read individual words and letters, in contrast to patients with alexia without agraphia. They may have considerable difficulty describing the action in a picture or cartoon, or comparing parts of a picture.

Optical apraxia, or poor hand-eye coordination. Patients may do better with hand motions when not under visual control, e.g., touching the hand to the nose with eyes closed. Tracing a line diagram or pointing to a precise part of an object in a picture are useful in studying this problem.

Apraxia of gaze. These patients cannot look where they desire. Ask the patient to look at one object and then direct gaze to another. Watch the patient observe a picture or scene.

Balint syndrome may occur without major field defects on tangent screen or perimetry. The normal person perceives a central percept and then searches the visual environment to amplify information concerning that initial cue. This leads to further perception and further searching.33 Looking and seeing are related functions, but with different anatomies. In the patient with the Balint syndrome, the anatomic connections between occipital and parietal lobes are disrupted, impeding fine interactions between perception and looking.

Metamorphopsia. Alteration in size, shape, or angulation of objects is an infrequent feature of cerebrovascular lesions. When it occurs, it is nearly always associated with bilateral occipital or occipitotemporal lesions. Patients may complain of enlargement (macropsia) or diminution (micropsia) of objects. The size alteration may be limited to one half-field or quadrant, giving objects a grotesque appearance. Patients may be unable to recognize distance relationships of objects within the environment and may have difficulty comparing distant objects with respect to size and depth. Other patients have complained of sharp angulation of objects, with the room appearing turned or upside down. When not related to an ocular muscle disorder causing vertical diplopia, this always means a posterior hemispheric lesion.

Behavioral abnormalities. Memory. Defects in the acquisition of new information and memory occur in patients with bilateral infarction of the medial temporal lobes.20,24 In addition, a unilateral left temporal lesion may be responsible for a Korsakoff-like syndrome which may be temporary, lasting for hours or up to 6 months.10,20,22

Agitated delirium. Patients with bilateral lesions in the distribution of the posterior cerebral arteries occasionally appear agitated and hyperactive, a state resembling delirium tremens. I have seen agitated delirium associated with bilateral visual defects in several patients with established posterior cerebral artery infarction, and in patients with an adverse reaction to vertebral angiography. In the patients with angiographic reactions, the vertebral, basilar, and posterior cerebral arteries were widely patent and the agitated delirious state was accompanied by visual and memory defects. Within 24 hours, the entire syndrome cleared, leaving amnesia which extended retrograde to the period prior to the angiography and anterograde to the point of clearing.

Horenstein, Chamberlin, and Conomy26 described nine patients with infarction of the undersurface of the temporal and occipital lobes whose behavior included restlessness, agitation, forced crying out and easy distractibility with exaggerated responses to visual, auditory, or tactile stimuli. The infarction involved calcarine, fusiform, and lingual gyri in all patients and, in some, the lesion extended to the medial hippocampal complex; in six patients, the lesions were unilateral and, in three, bilateral. Medina and associates28,29 also described severe agitation in patients with visual field defects; the syndrome remitted within days to 2 months.

Motor and sensory defects. Sensory loss accompanying posterior cerebral artery territory infarction is often profound, with severe loss of touch, position, and pain appreciation. Despite somatosensory "deafferentation," the patients surprisingly retain ability to use the limbs and frequently walk well. Objects are usually dropped from the hand without the patient realizing the loss. Loss of proprioception makes voluntary movement variable; when strength is formally tested, the patient may fail to perform the movement requested, or fail to exert power against resistance. If the examiner is patient and awaits the desired movement, normal strength can often be established. When the patient is asked to hold the arm outstretched with eyes closed, the arm exhibi-
iting sensory loss commonly arises or levitates in contrast to the downward drift which accompanies pyramidal system weakness. Some patients have commented that the arm or leg seems to be "moving on its own," and it is occasionally perceived as dead or separate from the body. One patient with a large posterior cerebral artery territory infarction was surprised to learn that a blow to her face had been delivered by her own hand, "unwilled" and unrecognized.

Lesions limited to the ventroposterior-lateral nucleus of the thalamus, as in pure sensory stroke, often cause "numbness" or paresthesias without important objectively demonstrable loss of perception. Lesions limited to the lateral thalamus in the distribution of the thalamogeniculate arteries cause unilateral limb ataxia, clumsiness, and chorea, in addition to variable sensory loss. The motor disorder is probably related to dysfunction of the ventral anterior and ventrolateral nuclei and their connections with efferents from the cerebellum and extrapyramidal systems.

Patients with lesions limited to the lateral thalamus usually do not have the severe deafferentation seen in patients with a larger posterior cerebral artery territory infarction or thalamic hemorrhage, lesions that interrupt the thalamoparietal radiations.

Motor paralysis is uncommon in patients with occlusion of the posterior cerebral artery. These patients usually retain their ability to make fine distal movements, and do not usually have hyperreflexia, clonus, or extensor plantar reflexes. However, facial weakness is common and may be related to decreased tone of the facial muscles. Occasional patients with involvement of the very proximal posterior cerebral artery may have an infarction of the cerebral peduncle, so that hemiplegia accompanies the usual hemianopia and hemisensory loss.

Discussion. This review has focused on the details of the neurologic abnormalities of patients with "top of the basilar" territory infarction. The locus of brain dysfunction was corroborated by CT scans, which showed radiolucent lesions in the medial occipital or inferior temporal lobes (15 unilateral, 5 bilateral), or by autopsy (5). Unfortunately, in our own series and in those described in the literature, the precise locus and mechanism of the vascular compromise is often uncertain. Most patients have not had full angiography. At postmortem examination, atherosclerosis of the vertebrobasilar system may be widespread, not allowing reconstruction of the exact pathophysiology of the infarction. Furthermore, an embolus present in life often lyses or moves far distally by the time of autopsy.

Foix and Hillemand studied the anatomy of small penetrating and circumferential branches of the distal basilar artery. Segarra elaborated on the anatomy of the perforating branches of the mesencephalic artery (the proximal portion of the posterior cerebral artery extending from the basilar bifurcation to the posterior communicating artery) and described two examples of infarction in the distribution of this vessel. However, in neither of Segarra's cases was a lesion identified in this vessel at postmortem. In his case 1, a right vertebral artery occlusion might have served as a nidus for distal embolization, producing the sudden onset of deficit. His case 2, another patient with a syndrome of abrupt onset, had a heart murmur and atrial fibrillation, but no lesion within the vertebrobasilar arteries at postmortem. Sieben, DeReuck, and Vander Eecken reported two patients with occlusion of the mesencephalic artery documented at autopsy. Atherosclerosis with occlusion of small basilar branches remains a hypothetical cause of high brainstem infarction, but this has been documented only in branch disease of the lower basilar artery.

The anatomic configuration of the basilar artery with two arterial vessels merging into a larger artery and then bifurcating is unique. Atherosclerosis is usually most severe at the origin of the vertebral artery in the neck, in the intracranial portion of the vertebral artery, and at the proximal end of the basilar artery. Castaigne and associates commented on the frequency of embolic material within the distal basilar distribution. The basilar artery is widest at its origin and tapers distally; an embolus small enough to traverse the vertebral artery would ordinarily not block the basilar artery except distally. Intrarterial emboli arise from atherosclerotic plaques in the carotid artery, and atherosclerotic foci, prominent in the proximal vertebral arteries, could serve as a source for distal emboli within the vertebrobasilar system. In case 8 of Caplan and Rosenbaum, embolization to the distal basilar artery arose from a unilateral vertebral occlusion. The sudden onset of stroke in our patients has led us to postulate an embolic mechanism (intraarterial or cardiac) of the vascular occlusion, but this was anatomically verifiable in only one case (case 1).

Clarification of the clinical syndrome and safer angiography may lead to further study and analysis of the spectrum of possible underlying vascular pathologies. Therapy will be possible only when there is a more thorough understanding of the vascular pathophysiology of the "top of the basilar" syndrome.

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"Top of the basilar" syndrome
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