Hemiplegic Syndrome of the Posterior Cerebral Artery

BY D. FRANK BENSON, M.D., AND E. B. TOMLINSON, M.B., M.R.A.C.P.

Abstract: Hemiplegic Syndrome of the Posterior Cerebral Artery

An unusual syndrome is described consisting of right hemiplegia, right hemisensory loss, mild naming disturbance, and severe alexia coupled with normal expressive language and ability to write. The clinical syndrome of alexia without agraphia strongly suggests involvement of the left posterior cerebral artery. A study of the territory of distribution of the posterior cerebral artery would appear to confirm the possibility that occlusion of this vessel could produce the entire symptom picture. Of particular interest was the unusual hemiplegia, involving both limbs and face equally, but with a minimum of spasticity.

ADDITIONAL KEY WORDS: arterial occlusion, cerebral infarction, alexia, hemianesthesia, agraphia, homonymous hemianopia

Well-defined neurological syndromes produced by discrete infarctions of the nervous system were first described in the latter part of the nineteenth century. At that time Huebner's arteritis was common and most of these syndromes involved the brainstem. The unique combination of well-localized cranial nerve nuclei and well-defined fibers of passage enabled discrete damage involving both systems to be accurately localized clinically. Elsewhere within the central nervous system such precision is rarely possible.

The cerebral hemispheres have a much better collateral circulation, and less specific relationship between structure and function, than has the brain stem. Thus, a hemispheric vascular lesion may produce no change, or a variety of neurological disabilities of varying severity. Further, large-vessel disease may result in pathological lesions which, though extensive, appear clinically to be very limited. Both pathological studies and cerebral angiography have tended to create skepticism as to the precise causal relationship between specific vascular involvement and specific symptomatology. Nevertheless, several vascular syndromes have been recognized, particularly the occurrence of contralateral hemiplegia and aphasia following occlusion of the middle cerebral artery of the speech-dominant hemisphere.

We recently studied two patients who suffered the sudden onset of right hemiplegia, hemianesthesia and homonymous hemianopia together with an aphasic disability. In these patients the lesion appeared to be a left occipital infarction, resulting not from involvement of the middle cerebral artery but of the posterior cerebral artery.

Case 1

A 53-year-old dispatch clerk was admitted to the hospital directly from his place of employment following the onset of a rapidly progressive right hemiparesis. For four days prior to admission he had been aware of numbness involving the right face and had suffered one episode of transient syncope. There was a five-year history of known,
untreated hypertension and a longer history of excessive ethanol intake.

On admission he was conscious but disoriented and mildly aphasic. There was a flaccid right hemiplegia, a right visual field defect and anesthesia to all sensory modalities on the right side. Initial blood pressure was 140/85 but soon rose to 215/110. Tangent screen examination demonstrated a right homonymous hemianopia with macular sparing. Pinprick on the right side was reported as only a vague sensation; other sensory modalities were not appreciated at all on the right side. All sensory testing was normal on the left. The hemiplegia remained hypotonic with only slight flexor withdrawal from painful stimuli. The right tendon reflexes were brisker than the left and the right toe sign was extensor.

The initial disorientation cleared rapidly and multiple examinations of mental status through the remainder of the hospital stay revealed no abnormality. He was always fully alert and cooperative, aware of his hemiplegia although he appeared to underestimate the severity. He gave a good account of the illness and formal testing revealed normal memory function. Conversational speech was fluent with normal grammatical structure but showed evidence of word-finding problems. Comprehension of spoken language was easily within normal limits. He had great difficulty naming objects, even common ones, on confrontation but invariably demonstrated that he recognized the object by manually or verbally demonstrating its use. He could spell out loud and immediately recognized words spelled to him. The hemiplegia precluded use of the right hand but he produced legible writing with his left hand, both to command and dictation. In contrast, he totally failed to interpret written language; he could name a few individual letters but recognized no words, not even his own name. He could read numbers aloud and do arithmetic problems. Although he could draw, he showed considerable constructional disturbance, particularly with block designs. He had great difficulty with color names. While he could accurately match colored chips he could neither name a color nor point to the appropriate color when the name was offered by the examiner.

All blood, electrolyte, enzyme and liver function tests were consistently normal. X-rays of skull and chest were normal. EEG revealed diffuse left hemisphere slowing without focus. Radioisotope brain scan demonstrated increased uptake in the left parieto-occipital area, seen most clearly in the left midline aspect of the posterior view (fig. 1).

**COURSE**

The blood pressure elevation was treated successfully. Physical rehabilitation measures were rapidly followed by walking, at first with cane and brace, but soon without either. Return of function to the arm was slower and less complete but, in contrast to many hemiplegics, showed return of considerable digital function and residual spasticity was very mild. Sensory appreciation improved slowly. If urged he could hold a cup or shave himself with the right hand, but if left to himself he invariably used the left for one-handed tasks. There was also some improvement in reading ability, although this remained limited. At discharge he could not return to work but was able to care for himself and an invalid wife.

**Case 2**

A 54-year-old itinerant alcoholic, found unconscious in a hotel room, was admitted to a local hospital where examination revealed a right hemiplegia and hemisensory defect. He rapidly regained consciousness, and additional examination disclosed a right visual field defect, skew deviation (right eye down and in) and mild word-finding difficulty when speaking.

![Radioisotope brain scan (case 1). Note increased activity on lower left of PA view and in lower posterior aspect of lateral view.](http://stroke.ahajournals.org/FIGURE_1)
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Ten days later he was transferred to the Boston VA Hospital, where general examination disclosed a thin, poorly nourished man with blood pressure of 160/80 and surgical scars on the abdomen and left leg (sympathectomy and left femoral artery bypass). Tangent screen examination demonstrated a dense right homonymous hemianopia with macular sparing. The skew deviation persisted with the right eye lagging behind on conjugate movements. There was distinct diminution of all sensory modalities on the right side although some pain appreciation was present. Paralysis was pronounced but with relatively little spasticity. The tendon reflexes were increased and the right toe sign was extensor.

Orientation was full; speech was fluent with excellent comprehension and repetition and only mild difficulty when naming to confrontation. Colors were consistently misnamed although the patient could match colors with ease. Reading was extremely poor, limited to a few letters and a few high-frequency words. In contrast he could write, spell aloud and recognize spelled words effortlessly.

Laboratory tests such as blood, urine, electrolyte, enzyme, liver function and spinal fluid evaluations were all normal. X-rays were noncontributory. EEG showed leftsided slowing but without specific focus. An isotope brain scan revealed increased activity in the posterior midline extending to the left (fig. 2) suggesting involvement of the medial aspect of the left occipital lobe.

**COURSE**

The hospital course was characterized by notable but incomplete improvement in all disabilities. Thus, the hemiplegia cleared leaving only a mildly spastic weakness in the right limbs. Sensory appreciation also improved but not to normal and was intermittently aggravated by painful dysesthesias involving much of the right side. This latter finding improved with Dilantin therapy. Eye movements also improved although the tendency for the right eye to lag remained. The hemianopia did not change but there was improvement in reading ability; he learned to read individual letters, spell words out loud and thus "read" short phrases or sentences. The entire course was complicated by severe ischemia in the right lower extremity, finally necessitating right femoral artery surgery and amputation of several toes.

**Discussion**

The first patient was originally thought to have an infarction involving a major portion of the dominant parietal lobe secondary to middle cerebral artery involvement. The diagnosis was based on the presence of fluent speech, gross disturbances in reading ability and word finding associated with a rightsided paresis, sensory loss and homonymous hemianopia. Repeated evaluations, however, demonstrated that he could recognize spelled words, could spell out loud and with help could spell with a pencil or anagram blocks but could not read. These features were even more fully developed in the second patient and suggested the syndrome of *alexia without agraphia*. Both patients, however, were hemiplegic and had a sensory loss, findings not classically associated with this syndrome.

*Alexia without agraphia* has long been recognized and, while not common, there is a remarkable consistency in both the clinical findings and the pathology. This syndrome typically has an acute onset with difficulty in reading, some unsteadiness and little else. Examination reveals a right homonymous hemianopia with macular sparing and total inability to read; in contrast there is an almost
normal ability to write, to spell out loud and to recognize spelled words. In addition, there is frequently an inability to name a color or to point to a color when the name is given, even though colors can be matched accurately. This has been called color agnosia. There are no other aphasic symptoms. The underlying pathology is infarction of the medial occipital structures (lingula, fusiform and calcarine cortex) plus infarction of the splenium of the corpus callosum. Most often this has followed occlusion of the left posterior cerebral artery. Our patients had the alexia without agraphia, color agnosia and right homonymous hemianopia, clinical findings similar to the reported cases.

Other findings in these cases, however, are atypical and need further consideration. Hemiparesis is almost never reported in cases of alexia without agraphia. This hemiparesis differed from that typically seen after hemispheric infarction in that the entire side was involved (face, arm and leg) and the proximal limb was more impaired than the distal limb. The degree of spasticity was exceptionally mild, much less than usually recorded following either cortical or capsular infarction. Involvement of the pyramidal tracts at the peduncular level has been shown to produce an almost nonspastic hemiplegia, probably because at this point pyramidal and extrapyramidal pathways are separate. Thus, a high brain stem or diencephalic lesion would be consistent with the hemiplegia in these cases.

Hemisensory loss is not usually reported in alexia without agraphia. Originally, both of our patients suffered profound sensory loss involving all modalities including pain. The distinct loss of pain suggested a deeply seated lesion, at the level of the thalamus or below. Therefore both the motor and sensory disturbances could be caused by selective infarction in the diencephalic and mesencephalic regions which are fed by branches of the posterior cerebral artery. This would also be consistent with the usual etiology of alexia without agraphia.

Some of the other clinical observations are more difficult to explain. The first patient had a marked word-finding defect. Anomia (word-finding defect) is not readily localized in the cortex, and in clinical practice an aphasic syndrome consisting only of word-finding difficulty cannot be localized. Among the many cortical areas occasionally linked with word-finding defect, one lies in the posterior and inferior temporal region, Brodmann's area 37. This area may receive blood from two sources, the posterior and the middle cerebral arteries. It seems possible that the posterior cerebral artery could have provided most of the circulation for this area and that occlusion produced infarction which in turn affected word finding.

Constructional disturbance usually indicates parietal abnormality and is most severe with biparietal involvement. Our patients had no firm evidence of such lesions and other explanations can be considered. There are reports of patients without parietal abnormality who had definite constructional disturbance. These are the split brain patients where constructional performance was well below normal, actually on the level of parietally damaged patients. Is it possible that the callosal damage postulated in our patients, infarction of the splenium, disturbed constructional capabilities by separating the two parietal areas? This can only be conjectured.

FIGURE 3
Schematic view to emphasize some of the significant proximal branches of the posterior cerebral artery.
Radioisotope brain scans were performed to confirm the suspected localization. In our institution these studies have been used extensively and, when positive, are considered good indicators of the site of cerebral lesions. The scans were positive in both patients (figs. 1 and 2), demonstrating increased isotope activity in the medial aspect of the occipital lobe and thus in agreement with the clinical localization. EEGs were performed on both patients. They did not show focal cortical changes but did demonstrate slow wave activity involving the entire left hemisphere, possibly an indication of more centrally located lesion.

The known distribution area of the posterior cerebral artery is consistent with the conjectured localization. Just after formation of the posterior cerebals from the basilar artery a number of fine branches enter the anterolateral mesencephalon, particularly the area of the cerebral peduncles. A number of additional branches from this area enter the thalamus (thalamo-perforating, thalamo-geniculate, posterior choroidal) and provide circulation to the posterior thalamus (fig. 3). Beyond this level a branch proceeds upward and medially to the splenium of the corpus callosum. A series of branches arch downward and then forward on the medial and inferior surface of the hippocampus (the hippocampal branches) (figs. 3 and 4) and the main trunk proceeds posteriorly as the calcarine artery. Thus, occlusion of the left posterior cerebral artery just beyond the upper limits of the basilar artery could produce the hemiplegia (peduncular branches), the sensory loss (thalamic branches), the homonymous hemianopia (calcarine branch), the alexia without agraphia (calcarine branch plus splenial branch), the memory loss and anomia (hippocampal branch).

The literature offers some support for this conjecture. Foix and Masson postulated two syndromes resulting from posterior cerebral occlusion: (1) a posterior syndrome with homonymous hemianopia and alexia, and (2) an anterior syndrome with thalamic involvement producing a paresis, a profound sensory loss, and, if on the dominant side, a disturbance in naming. They presented a single case with pathological evidence of both anterior and posterior involvement but rather inadequate clinical studies. They also mentioned briefly a case report of Dejerine and Thomas with clinical findings similar to the present patients and pathology limited to the...
distribution area of the posterior cerebral artery.

Whether this syndrome is as rare as these few reported cases would suggest is questionable. It is altogether too easy to consider that the hemiparesis and hemianesthesia indicate a disturbance in the territory of the middle cerebral artery, thus neglecting the existence of a distinctly different syndrome. The desirability for a proper diagnosis lies in the much improved outlook for rehabilitation measures in the posterior cerebral cases. Motor and language function appears to respond better to retraining efforts. The failure to recover sensory function, however, may provide a major difficulty.

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