The Posterior Cerebral Artery Syndrome

C.M. Fisher

ABSTRACT: Embolic and thrombotic infarction in the territory of the posterior cerebral artery (PCA) is described with emphasis on the stroke and cerebrovascular features rather than special neurological syndromes. Of 47 cases of obstruction at the distal bifurcation of the basilar artery, 43 (95%) were consistent with embolism. The clinical categories and pathological findings are presented. Local embolism, vertebral distal-stump embolism, the dynamics of hemorrhagic infarction and embolus-in-transit are briefly described. The prodromal manifestations of PCA thrombotic occlusion include photopsias, hemianopic blackouts, headache, transient episodes of numbness, episodic lightheadedness, spells of bewilderment and rarely tinnitus. Recognition of these may allow prevention of a stroke. Prodromal photopsias did not closely resemble the scintillating displays of migraineurs. When the stroke occurred, visual complaints ususally predominated. A sensory deficit occurred in one-third of cases. In 25 cases of memory impairment the dominant hemisphere was involved in 24. The kinds of visual hallucinations, simple and formed, are described.

RÉSUMÉ: Le syndrome de l'artère cérébrale postérieure. L'infarctus embolique et thrombotique dans le territoire de l'artère cérébrale postérieure (ACP) est décrit en mettant l'emphase sur l'ictus et les manifestations cérébrovasculaires plutôt que sur des syndromes neurologiques particuliers. Parmi 47 cas d'obstruction au niveau de la bifurcation distale de l'artère basilaire, 43 (95%) étaient compatibles avec une embolie. Nous en présentons les catégories cliniques et les constatations anatomopathologiques. Nous décrivons brièvement l'embolie locale, l'embolie au niveau du moignon distal de l'artère vertébrale, les aspects dynamiques de l'infarctus hémorragique et de l'embole en transit. Les manifestations prodromiques de l'occlusion thrombotique de l'ACP incluent la photopsie, l'anopsie hémianopique, la céphalée, les épisodes transitoires d'engourdissement, les étourdissements épisodiques, les accès de désorientation et plus rarement le tinnitus. L'identification de ces symptômes peut permettre de prévenir un ictus. La photopsie éprouvée comme prodrome par ces patients ne ressemble pas aux scintillements perçus par les migraineux. Quant l'ictus survient, les symptômes visuels prédominaient habituellement. Un déficit sensitif était présent chez un tiers des cas. L'hémisphère dominant était impliqué dans 24 cas de déficit de la mémoire sur 25. Nous décrivons les types d'hallucinations visuelles, simples et organisées.

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The arteries of pre-eminent interest to the neuro-ophthalmologist are the ophthalmic and central retinal arteries anteriorly and the posterior cerebral artery posteriorly. While the former receive a good deal of clinical attention, the stroke profile of the posterior cerebral artery (PCA) is not often discussed. Specific aspects of the PCA syndrome are singled out for special study, for example, amnesia, mesencephalo-thalamic deficits and the visual and speech disorders of the temporo-occipital region. The more general features of PCA ischemia have been described in several reports over the years.[1-8] However, the literature contains no comprehensive description of *strokes* due to occlusive disease of the PCA. PCA strokes are rather common and my records contain notes on more than 370 cases which had been studied with variable degrees of thoroughness. The aim of this paper is to describe the clinical syndromes associated with ischemic events in the PCA territory, with emphasis on the neuro-ophthalmological aspects.

Vascular Anatomy

The PCA arises from the terminal basilar artery bilaterally in 71% of cases, mainly from the internal carotid artery (ICA) unilaterally in 22% of cases and bilaterally in 7%. In angiographic studies a vertebral injection fills both PCAs in almost 90% of cases. In embolism to the bifurcation of the basilar artery and the precommunal PCAs the size of the precommunal PCAs (the segment proximal to the posterior communicating artery) and the size of the posterior communicating arteries will be a factor in determining the availability of collateral flow.

The penetrating branches to the midbrain and the thalamus according to Percheron[9] are usually four in number and arise from the proximal few mm of the PCA. They fan out in the sagittal plane to supply a paramedian core of tissue extending in continuity through the entire vertical extent of the midbrain, subthalamus and posterior two-thirds of the thalamus (Figure 1). Their distribution is unusual in that a penetrator from one

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From the Neurology Service, Massachusetts General Hospital, Boston, Massachusetts.

Reprint requests to: C. Miller Fisher, M.D., Neurology Service, Massachusetts General Hospital, Fruit Street, Boston, Mass., U.S.A. 02114

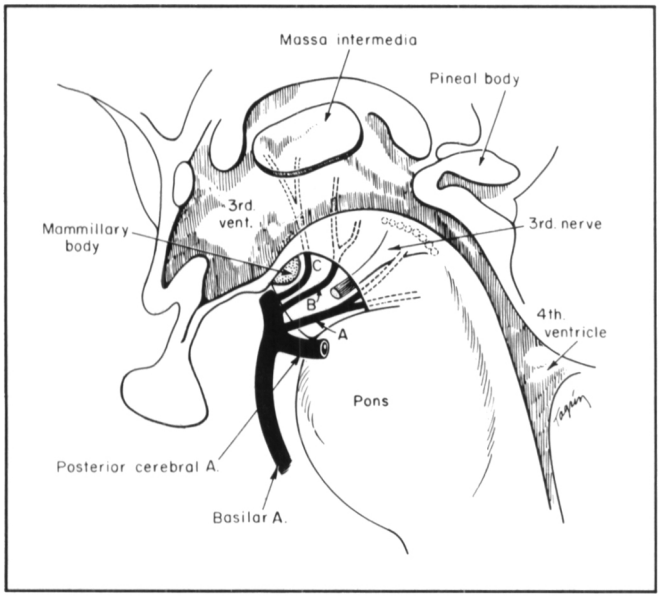

Figure 1 — Diagram of branches to mesencephalon and thalamus from PCA. A = Inferior and superior mesencephalic paramedian artery; B = Posterior subthalamo-thalamic paramedian artery; C = anterior subthalamo-thalamic paramedian artery.

PCA may divide to supply the medial part of the midbrain tegmentum bilaterally and the medial part of each thalamus bilaterally, occlusion producing a so-called butterfly infarct. In about 50% of cases the penetrating arteries arise predominantly from one PCA in the form of a pedicle giving rise to a leash of small vessels and the presence or not of this arrangement will again influence the effect of occlusion of the proximal PCA.

The structures supplied include in the midbrain the mesencephalic periaqueductal gray matter, nucleus and fibers of the third nerve, raphe nuclei, Edinger-Westphal nucleus, median reticular nuclei, medial longitudinal fasciculus, pars compacta of the substantia nigra, superior cerebellar peduncles and their decussation, and the medial third of the base of the peduncles. In thalamus and subthalamus the paramedian structures most often involved are the intralaminar and parafascicular nuclei, inferior median nucleus, medial anterior central nucleus and the superior internal pole of the red nucleus. The entire midbrain is supplied from the PCAs and not directly by branches from the basilar artery.

The long and short peduncular branches of the PCA — some six in number — supply the cerebral peduncles. More posteriorly there arise successively from the PCA, the posterior choroidal arteries, medial and lateral, running to the superior part of the thalami, the thalamogeniculate vessels to the posteroventral thalamus, including the sensory nucleus, the branches to the hippocampus, the anterior and posterior temporal branches to the inferior surface of the hemisphere, the parieto-occipital branch to the medial occipital surface, the splenial branch to the corpus callosum and finally the calcarine branch to the visual cortex and its association areas.

When an occluded PCA arises from the ICA, a lateral thalamic deficit occurs without the midbrain-medial thalamic syndrome while on the other hand, when a vestigial precommunal PCA unilateral or bilateral is occluded, the latter syndrome occurs in the absence of a hemianopia and other hemispheric signs.

Ischemia in the PCA territory is associated with a great many neurological abnormalities any one of which could in itself be the topic of a separate lecture. On this occasion our main interest is in the characteristics of the stroke process, rather than an analysis of complex neurological phenomena, and therefore, it must suffice to merely enumerate the various symptoms and signs from which the syndromes are derived (Table 1). The main feature, of course, is a hemianopia or other visual field defect which in most instances localizes the process and the other manifestations are largely subsidiary.

The Vascular Process

The two main vascular occlusive processes are *embolism* and *thrombosis*, just as they are in the other cerebral arteries. The PCA is unusual, however, in that it is the special site of another important process, namely *migraine*. In patients with classical migraine accompaniments, the visual system is involved in 90% of cases either by itself or along with other accompaniments (paresthesias, aphasia, etc.). So fundamental is the relationship of migraine to the PCA territory that one might say the PCA is the artery of migraine. The interpretation of visual symptoms, seemingly arising as the result of PCA ischemia, is regularly complicated by the possibility that the migraine process is operative; and this is not true to the same extent of the other cerebral arteries.

Postcommunal occlusion of the PCA in which the clinical picture is limited to deficits arising in the hemisphere and lateral thalamus is some seven times as frequent as precommunal occlusion in which the territory of the entire PCA is involved and midbrain and medial thalamic deficits are added to the postcommunal syndrome. The stupor coma or abulia of midbrain-medial thalamic origin so dominates the clinical picture that the hemispheral deficits may not be recognizable. In this regard the syndromes due to precommunal and postcommunal occlusion are really quite distinct and separate.

Embolism to the Posterior Cerebral Artery

Embolism is discussed first because it is pertinent to the aforementioned important matter of precommunal versus postcommunal occlusion. When an embolus is small, it enters one or other PCA and depending on its size is arrested somewhere along the course of the artery producing an infarct of corresponding size. When the embolus is large, it lodges at the top of the basilar artery, where it may block one or both PCAs in their precommunal segment. Differences in the size of the two PCAs along with the configuration of the bifurcation will help to determine the path, the final resting place and the effect of the embolus. In this so-called "top o' the basilar syndrome," all deficits are usually confined to PCA territory, but occasionally extend slightly into the uppermost pons or the adjacent cerebellum since paramedian arteries to the pons and the superior cerebellar arteries arise only 1 or 2 mm away.

Precommunal occlusion of one or both PCAs is by no means rare and comes to the attention of the neuro-ophthalmologist because of pupillary abnormalities and disorders of eye movement. The typical upper brainstem lesion associated with precommunal occlusion is the butterfly infarct. For this presentation I reviewed 47 cases of obstruction in the region of the basilar bifurcation,

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Table 1: Signs and Symptoms of Ischemia in Posterior Cerebral Territory

	PROXIMAL	DISTAL
VISUAL	Third nerve palsy, Parinaud's syndrome, vertical gaze paresis, paralysis of downward gaze, bilateral ptosis, oval pupils, blepharospasm, retraction nystagmus, tucking of eyelids, anterior internuclear ophthalmoplegia, pseudo-paralysis of 6th nerve, decreased or absent spontaneous blinking with retained reflex blink to light and sound, absence of slow roving eye movements, absence of horizontal saccades, delayed eye closure on falling asleep, ocular tremor.	Hemianopia, unilateral and bilateral; Anton's syndrome, alexia without agraphia, color anomia, dyschromatopsia, hallucinations: simple, complex, formed and unformed. Central photophobia, visual perseveration, polyopia, tilted vision, visual spread. Migraine: scintillations, blindness and blurring. Prosopagnosia, topographic agnosia, visual agnosia, peduncular hallucinosis, checkerboard fields, metamorphopsia.
NON-VISUAL	Abulia, drowsiness, brief wake-sleep cycles, coma, dementia, tachyphemia, hemiparesis, decerebrate posture, Horner's syndrome, cerebellar ataxia, rubral tremor, Weber's syndrome, Claude's syndrome, Benedikt's syndrome, hemiballismus, asterixis, Cheyne-Stokes respiration.	Sensory deficit, Dejerine-Roussy thalamic pain, choreoathetosis, confusion, agitation, impairment of memory, anomia for proper names, isolated dysgraphia, unsteadiness or lightheadedness, hemorrhagic infarction secondary to brain herniation.

including 24 studied pathologically. In 44 of the 47 cases (94%) the evidence favored embolism as the vascular process rather than thrombosis. It can be asserted, therefore, that almost all strokes involving the bifurcation of the basilar artery are embolic. In this preliminary analysis, the pathological findings in 24 cases showed the midbrain involved in 19 cases and the distal PCA in 12 cases. The distribution of lesions is shown in Table 2. The broad clinical categories in the 47 cases are listed in Table 3. Stupor and coma occurred in 35 cases, the neurological deficits were minor in 3 cases and the remaining cases had deficits of intermediate severity.

Since a penetrating branch from one PCA may supply the midbrain and thalamus bilaterally it may be difficult to determine clinically if an embolus has blocked one or both PCAs precommunally. Rarely, a small embolus enters an artery of Percheron (0.3 to 0.5 mm in diameter) and gives rise to an isolated small infarct in the midbrain or thalamus.

Embolism to the postcommunal PCA accounts for at least half of all postcommunal infarcts, thrombosis and migraine accounting for the rest. The neurological deficits due to embolism are similar to those associated with thrombosis of the postcommunal

Table 2: Structures involved in embolism to distal bifurcation of the basilar artery

	Number of Cases
Midbrain, thalamus, peduncle, PCA occipital	5
Midbrain, thalamus, peduncle, PCA occipital, SCA	4
Midbrain, thalamus, peduncle, PCA occipital, pons	1
Midbrain, thalamus, peduncle, pons	1
Midbrain, thalamus, peduncle	1
Midbrain, thalamus, SCA	1
Midbrain, thalamus, SCA, pons	2
Midbrain, peduncle	1
Midbrain, SCA	1
Midbrain tegmentum	1
Thalamus	4
Thalamus, peduncle, PCA, SCA	1
PCA, pons	1
	24

Table 3: Clinical features associated with embolism to the distal bifurcation of the basilar artery

	Number of Cases
Rapid onset of coma	12
Minor neurological deficits	3
Stupor with neurological deficits intermediate in severity between 1 and 2	23
Cortical blindness	2
Amnesia	2
Alteration of Behavior	1
Deficits produced by embolus-in-transit	4
	47

PCA, a subject to be discussed under that heading. Small emboli may enter a PCA branch, either penetrator or superficial, causing a very limited deficit. Minute embolic particles may cause multiple microinfarcts in the calcarine cortex leading to vague unexplained visual symptoms.

The diagnosis of infarction in PCA territory was revolutionized by the invention of CT scanning; and magnetic resonance scanning is adding another whole dimension of sensitivity especially in studies of the brainstem and thalamus.

Clinically the diagnosis of embolism is favored when the stroke has occurred in the absence of prodromal TIAs and particularly in the presence of an embolic source such as atrial fibrillation. The possibility of migraine may be raised, but embolism never produces the visual display with "build-up" characteristic of migraine. An embolus typically undergoes migration and lysis and a patent artery by angiography signifies embolism — or migraine. However, angiography in the first 48 hours may show an embolic plug that will have disappeared later. Symptomatic thrombosis always has associated with it tight stenosis or occlusion.

Two aspects of PCA embolism warrant further comment.

(1) ***Vertebral distal-stump embolism*** When a vertebral artery undergoes thrombosis, thrombus building up in the distal stump, may become disloged and pass along the basilar artery to enter one or other PCA.[10] Clinically, such cases are probably not infrequent, although proving the source of an embolus is always

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virtually impossible. The results of a pathological study of "local embolism"[11] (artery-to-artery embolism) in the basilar system are shown in Table 4. It will be noted that "local embolism" had occurred in 25% of vertebrobasilar cases and the infarcts were in the PCA territory in 17 of 18 cases making distal-stump embolism particularly relevant to our present subject.

In a recent case (Figure 2) a man aged 61 developed a left PCA syndrome including impaired gaze upwards, a slight right hemiparesis, right homonymous hemianopia and alexia without dysgraphia. He died suddenly 2½ months later and pathologically there was occlusion of the left vertebral artery with distal extension of the thrombus and infarcts in the territory of the left PCA and right superior cerebellar artery, both arteries being free of thrombus. In the absence of any other source of embolism it was inferred that emboli had arisen in the stump of the occluded left vertebral artery. It is conceivable that timely anticoagulant therapy would prevent such a complication.

Table 4: Characteristics of infarcts in 18 patients with local embolism in the vertebrobasilar systems

	Number of Cases
Vessel Involved	
Basilar	6
Bilateral vertebral	6
Unilateral vertebral	6
Regions of Infarction	
Calcarine unilateral	14
Calcarine bilateral	2
Superior cerebellar	1
Calcarine and cerebellar	1
Age of Infarcts	
Old	13
Recent	2
Old and recent	3

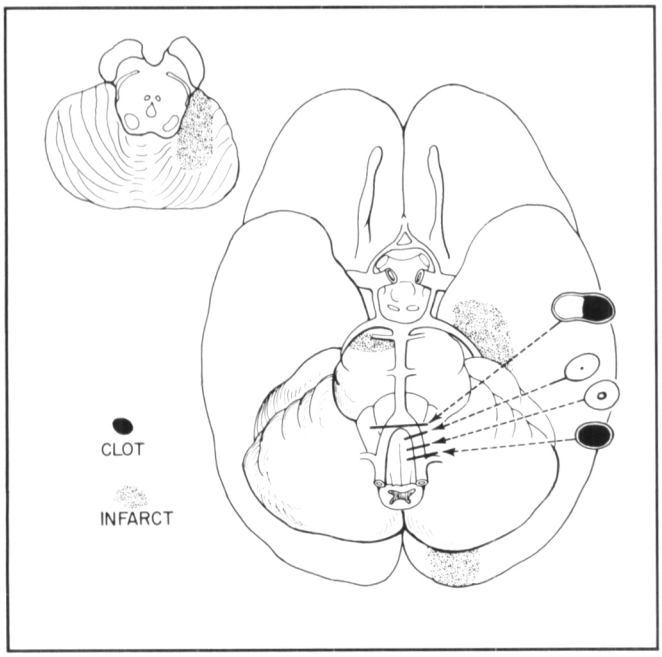

Figure 2 — Diagram of left verbetral artery occlusion with evidence of distal stump embolism to PCA and SCA.

(2) ***The dynamics of embolism and hemorrhagic infarction in the vertebrobasilar territory*** Embolic infarcts may be pale or hemorrhagic or mixed. Hemorrhagic infarcts are usually embolic and probably the result of the migration, fragmentation and lysis of emboli allowing the restoration of arterial flow to the infarcted tissue. Infarcts in which the embolus is found blocking the artery of supply, usually remain pale. When a large embolus passes along the vertebral artery and then along the basilar artery, it may leave in its wake hemorrhagic infarcts that are a sign of what is called an "embolus-in-transit". Figure 3 portrays the pathological findings in a case of embolism in which

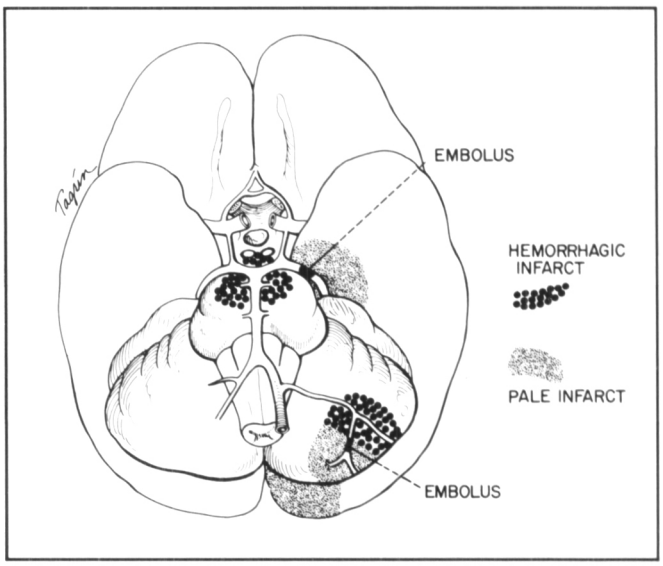

Figure 3 — Diagram of hemorrhagic and pale infarction left in wake of embolus passing through vertebral artery and basilar artery to reach left PCA where embolic plug was found in artery just proximal to pale infarct.

there were hemorrhagic infarcts in cerebellum, pons, midbrain and thalamus and the main embolus was found lodged in the left PCA proximal to a pale infarct. As the embolus moved along the basilar artery, it may have temporarily blocked the mouths of the penetrating arteries or given rise to a small embolic particle as it did in the posterior inferior cerebral artery. The PCA is a common site of hemorrhagic infarction (Figure 4).

A lively topic of discussion is just when does an infarct become hemorrhagic if it is going to do so. It has been my impression that it occurs early, perhaps in the first 24 hours, but recently there came to my attention a case of PCA infarction in which the infarct was bland by CT on day 9 and very hemorrhagic on day 17 in the absence of therapy.

Thrombosis of the Posterior Cerebral Artery

Thrombosis of the PCA is usually the result of atherosclerosis, which has a predilection to form in the circumpeduncular or ambient segment of the postcommunal part of the artery rather than the precommunal. One can predict the site of thrombosis

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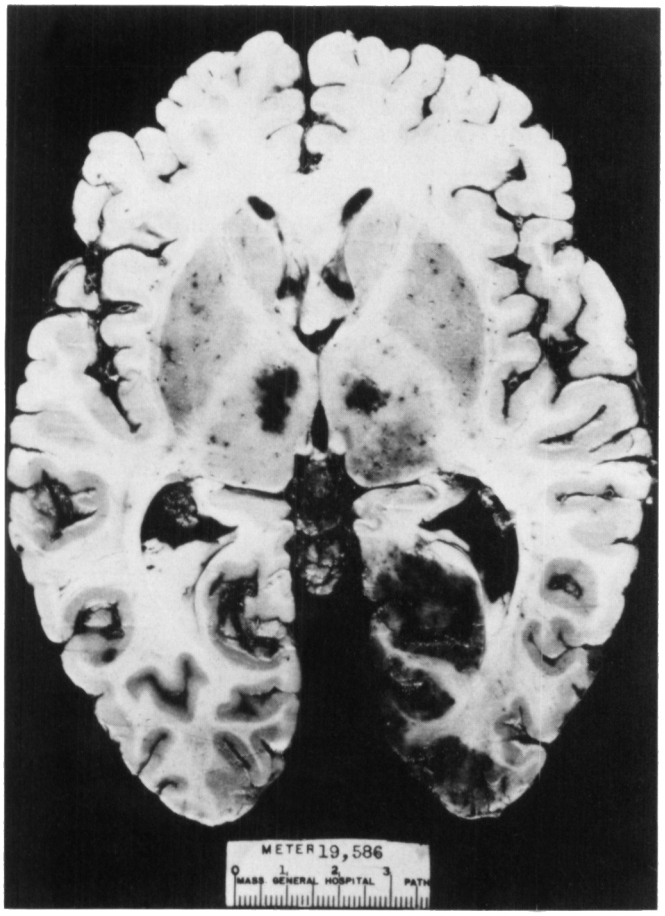

Figure 4 — Hemorrhagic infarction of the right PCA territory and both thalami in embolism.

there with some regularity and hence in thrombosis the clinical manifestations are almost always those of the postcommunal territory sparing the midbrain. (While midbrain-medial thalamic deficits are here being assigned almost automatically to embolism rather than thrombosis, it should be pointed out that there is no record of a detailed pathological study of Percheron's arteries in such a case.) The results of thrombotic occlusion are variable and unpredictable since collateral blood flow to the distal PCA from the middle cerebral artery and anterior cerebral artery via the meningeal anastomoses are different from patient to patient. Total occlusion is occasionally asymptomatic. Whether the blockage is precommunal or postcommunal, embolism occurs suddenly and usually without warning, whereas *thrombosis* is frequently associated with prodromal headache and transient ischemic attacks (TIAs) consisting of numbness of the face and arm, or face, arm and leg on the opposite side.

Prodromal TIAs

From the standpoint of the neuro-opthalmologist the occurrence of prodromal photopsias would be important since early diagnosis would provide an opportunity to intervene and prevent a full PCA stroke. However, such herald photopsias were uncommon and were recorded in only 5 of 150 cases.

Case 1 — Floaters, hexagons and octagons came and went in a flash and were seen far temporally in the right visual field. When the patient looked in their direction, they disappeared. After two weeks, there appeared a light chocolate-colored spot in central vision, also coming and going. In the next two weeks he had frequent TIAs consisting of numbness of the tongue, the right side of the chin and right hand lasting a few seconds. On Warfarin sodium the spells ceased.

Case 2 — A man aged 67 in one month had five spells of striped vision in the right visual field likened to looking through a venetian blind placed on its side. There were approximately 15 stripes. They lasted 10 to 15 seconds and seemed to occur when he looked at a bright landscape or seascape. Aspirin was prescribed. One year later he reported five spells of numbness of the right cheek, hand, forearm and leg, each spell lasting about four minutes. Angiography showed tight stenosis of the left PCA.

Case 3 — For six months the patient experienced TIAs consisting of bright, moving circles before her eyes lasting 5 to 10 seconds. They revolved counterclockwise and the patient called them "pieces of electricity". At first they occurred one or twice a month, increased to once or twice a week and on the day preceding the onset of a persistent left homonymous hemianopia, the display occurred three times in one day.

Case 4 — Fourteen days and eight days before the onset of a left hemianopia the patient lost vision for five minutes associated with a flash or ball of light "like the sun".

Case 5 — On each of the three days before the onset of a left hemianopia, a purple blob appeared in the left visual field associated with a lightning bolt of different colors shooting out of it like Halley's comet.

These brief descriptions illustrate the many variations in the photopsias reported by patients with disturbances of the visual system, such as transient monocular blindness with or without carotid disease, migraine, transient ischemic attacks in the PCA territory, and occipital lobe seizures. The number of different patterns that a few basic ingredients can assume is truly remarkable. Perhaps they are visualized away from central vision where they cannot be seen clearly, causing a vagueness in description. It seems to be impossible to develop an experience with them that is of practical use clinically. Perhaps if studied more diligently, they would make more diagnostic sense, but that day has not arrived. It is even conceivable that analysis would provide information about the nature of the underlying processes. The subject of photopsias has an additional unfortunate aspect, namely, the undue amount of time and space it takes to describe them. Worse still, there is no framework in which to classify them and one is unable to remember for long the patient's account.

As already mentioned, episodic photopsias were not common as TIAs in PCA thombosis. Two further patients had prodromal transient hemianopic blackouts.

Case 6 — For one week there occurred three to five spells daily of blank vision out to the right side lasting for a couple of minutes. On the seventh day a spell was combined with numbness of the right hand and angiography showed tight stenosis of the left PCA.

Case 7 — For twelve hours, while awake, the patient had blackouts of the central part of the right visual field interrupted by a return of vision for a second or two three times a minute. After 24 hours the seeing and non-seeing periods each lasted 10 seconds and were occurring three times a minute. There were no scintillations. There was a deep headache in the left forequarter of the cranium. The spells ceased four hours after Heparin therapy was begun.

More frequently, patients had less well-defined transient visual complaints in the day or two before the stroke — blurring of vision, trouble focusing, difficulty seeing words, or a vague interference with vision lasting a few seconds up to one hour.

It is surprising that there was no example of prodromal visual spells closely resembling the scintillating phenomena described by migraineurs. In none of the cases was there a shimmering brightness and it may be a useful rule that the scintillating visual

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aura of migraine is not the forerunner of atherothrombosis of the PCA. However, it will be recalled that two patients had prodromal episodes of a blank hemianopia and it is my experience that migraine can produce the same phenomenon. Another feature was the brevity of the episodes of photopsia which lasted 3 to 15 seconds unlike the usual migrainous spell. The question is sometimes raised as to whether stenosis of a cerebral artery associated with a low pressure distally predisposes to attacks of migraine. From this group of cases, the answer appears to be "no". An analogous question is whether migraine spells occurring distal to a stenosis precipitate infarction, and again the main evidence is in the negative. There was one unusual case, however, which speaks to this point. A man, aged 78, had had recurrent typical scintillating zigzags for at least 25 years associated with "build-up", occurring about once a month and lasting 15 to 30 minutes. One day the scintillations appeared, but this time continued, and after eight hours, the right side of the face and right hand began to tingle and he became confused. Within 24 hours he had developed a severe precommunal PCA stroke, including a right hemiplegia. The possibility existed that events might have represented a persistent migrainous deficit, but pathological examination showed atherosclerosis and superimposed thrombosis of the left PCA in the precommunal segment. This might well be an example of a migrainous spell precipitating infarction in a compromised territory distal to arterial stenosis, but when the patient was first seen, he could no longer give the details of the onset.

From this account, the opportunity will not often come to the neuro-ophthalmologist to recognize incipient occlusion of the PCA and possibly prevent a stroke.

The initial TIA symptoms in the PCA territory, were more often ***spells of numbness*** coming and going, in face and arm, arm and leg, or all three. Approximately one-third of the PCA thrombotic cases noted prodromal spells of numbness lasting for a few seconds up to 30 minutes, for one day, or for as long as several months before the next symptom. There were sometimes one or two spells, other times as many as 100 before the visual symptoms were added. Spells of isolated numbness of this type can arise as a result of atherosclerosis of the PCA itself or as a result of lipohyalinosis of the small penetrating artery branch supplying the posteroventral nucleus of the thalamus. Differentiating the two possibilities is at times impossible. It is our experience that in PCA cases before a persistent stroke occurs, there will always be transient symptoms of another type than sensory, for example, visual disturbance, headache or confusion to indicate that the true site of the disease is in the main PCA rather than a branch and that prophylactic therapy is in order.

Transient weakness or hemiparesis was rare as a TIA and reflected involvement of the precommunal segment of the artery. Weakness combined with sensory deficit may result from ischemia in the territory of the thalamo-geniculate penetrating artery (thalamo-capsular lacunar infarct) but no such case was recognized.

Headache could be an important prodromal symptom and preceded the stroke by days or weeks. The headache of PCA thrombosis was most often situated above the ipsilateral eye or in the anterior forequarter of the cranium. There could also be associated pain in the occipital region. Occasionally the headache was referred solely to the upper parietal region ipsilaterally, a somewhat unexpected finding. The headache was throbbing or steady and several patients reported exacerbation of the headache on coughing or shaking the head.

Another prodromal symptom, not rare, was ***episodic lightheadedness***, giddiness, dizziness, or unsteadiness. It sometimes occurred alone, at other times it accompanied the visual symptoms. The patient was often at a loss to characterize the sensation accurately and the neurological nature of the symptom is unclear.

Occasionally, there was a prodromal transient brief ***spell of bewilderment***, confusion or "not knowing where I was for a moment", a symptom that was probably linked to involvement of the hippocampus of the dominant hemisphere.

Two patients reported hearing ***a sound*** at the onset of the stroke, possibly an indication of involvement of the ipsilateral medial geniculate body. In one, it was described as the hissing of escaping steam.

The Stroke

Now we turn to the neurological features of the stroke itself. Visual complaints predominated in the form of a full or partial hemianopia. A sensory deficit occurred in only one-third of cases. It is a good rule — although not an infallible one — that in a PCA stroke, the patient is aware of the field defect, whereas with an occipito-parietal lesion in the middle cerebral territory, the patient is unaware of the field defect. Also, in a left or non-dominant occipital hemianopia central vision is not shifted far to the right as in non-dominant parietal lesions. This abnormality is revealed by having a patient read a newspaper headline — a test which should be routine.

The ***PCA visual symptoms*** included: poor vision, not seeing out to one side, blurred vision, bumping into things, seeing half of things or only parts of words, difficulty seeing, focusing or reading and spots in front of the eye. In other words, a patient's symptoms directed attention to the true nature of the stroke. When the lesion was confined to the calcarine visual cortex, a hemianopia constituted the entire stroke. An occasional patient found later to have a hemianopia reported temporary blindness or dim vision for ½ to 4 hours at the onset of the stroke. This event which suggests that a sudden unilateral insult may have a bilateral effect occurred only in cases of embolism or migraine not thrombosis. Opticokinetic nystagmus is characteristically preserved in calcarine hemianopia. Rarely a hemianopia was due to a lesion of the lateral geniculate body,[12] in which case a Wernicke-type hemianopic pupil may be present.

The sensory deficit, when present, varied in severity from slight to virtually complete anesthesia of face, arm and leg, involving all modalities. There may be an anesthetic pseudoparalysis in which the limbs appear paralyzed, but move with good force when the paralyzed limb is held under visual control in the seeing half-field. Parietal lesions in the territory of the middle cerebral artery occasionally produce such severe sensory loss, but neighborhood signs such as aphasia, elements of Gerstmann's syndrome, or visuo-spatial disorders provide the clue to the correct localization.

Confusion and impaired memory occurred when the hippocampal region of the dominant hemisphere was involved.[13,14] In 70 PCA cases, most of them studied only clinically, there were 25 instances of memory impairment and in 24 the dominant hemisphere was involved. Of the 45 cases with preserved memory and orientation the dominant hemisphere was involved 13 times, the non-dominant hemisphere 32 times. From a clinical point of

view, it is certain that the hippocampal regions of the two hemispheres are not equivalent. When a PCA patient became agitated and delirious, it was almost always the dominant hemisphere that was affected. In dominant lesions, the clinical history may be unreliable because of the patient's memory impairment, which may be inobvious.

Depression and obsessive thinking were prominent in several patients with involvement of the dominant hemisphere. This may be a reaction to the greater functional deficit associated with dominant lesions compared with non-dominant, but search for a specific relationship is warranted.

Dyslexia without dysgraphia occurred with left hemispheric lesions.[12,15] ***Anomia*** for proper names and ***color anomia*** were present in dominant lesions.[12] ***Topographic agnosia*** has been reported with a lesion of the lingual and fusiform gyri of the non-dominant hemisphere.[16] ***Visual perseveration***[17] or "sticky vision" was common, as was ***metamorphopsia*** in which objects appeared misshapen.

Headache could be a prominent symptom and was localized as already described. It was related to involvement of the PCA in its proximal 3 to 4 cm but did not correlate with the presence or not of confusion or sensory deficit. Headache also occurred in 7 of 25 cases of PCA embolism, but was prominent in only 2 cases.

Visual hallucinations or ***photopsias*** which were uncommon as a prodome were more frequent once the stroke occurred. The following are examples: flickering yellow lights in the affected visual field and dancing pinpoints of light sometimes moving in circles, more prominent with the eyes open; (in the following descriptions, the field referred to will be the hemianopic field unless stated); kaleidoscopic brilliant multicolored snowflakes or shaved icechips carried down like a frozen mist; flashing kaleidoscopic lights of beautiful colors in the upper quadrant and unformed patterns far out in the visual fields; gold and silver shooting stars in the half-field; a red ball and a blue ball drifted across the field of vision from left to right every minute (right hemianopia); shimmering in the half-field for several days; flickering or shimmering dots of lights; episodes of shimmering spots and wavy lines, sometimes colored, tending to pass in front from left to right diagonally downward (right hemianopia); lights like a pinwheel with firecrackers; a flash of lightning across vision at the onset; flickering and billowing waves of blurring; colors rotating before the eyes while looking at brightly colored objects; blurry, blinking waviness with a sort of linear movement in the upper part of the half-field; dancing brilliant multicolored scotomas in the half-field. Displays of this type usually ceased in about one week, but occasionally they lingered for one month.

Can these descriptions be arranged to fit into a simple format? Although it would be a gross simplification, the displays might be divided into two categories — discrete foci and lines. The foci were of various sizes — sparks, lights, spots, etc. The elements were bright and often colored. Flickering, shimmering, dancing, moving and waviness were common terms. In the photopsias just described colors were particularly prominent (8 of 14, 57%), which is in contrast with the photopsias associated with transient monocular blindness of which only 3 of 20 (15%) were multicolored.

Complex formed hallucinations are a well known phenomenon in PCA infarction[18,19] and are more common when the non-dominant hemisphere is involved. A few examples must suffice:

Case 1 — a well formed horse's head, brown, shiny with fine looking harness, many tall buildings, some of which were recognizable. The hallucinations were situated 18" from the eye on an angle of 30 degrees to the left of center.

Case 2 — colorful, well formed fish of all sizes, moving about, shiny, shimmering, dark red, green, brown, light blue, yellow, copper and gold. Also, clouds and trees, etc.

Case 3 — three banks of vivid vertical lines with illuminated pips running up the lines a variable distance and breaking into colors on coming to a halt — the highest became red while the lower sites of arrest were yellow, blue or green, in that order.

Case 4 — scenes consisting of streets, buildings, cars and children on looking to the left. The scenes constantly changed. When Phenytoin sodium was prescribed, the scenes continued in the upper part of the field, while in the lower part of the field jungle scenes appeared with alligators, panthers, tigers, snakes, etc.

Case 5 — a multicolored oriental rug hanging on the wall of the right side of the room.

Case 6 — various patterns (octagons, target rings) in many bright colors, orange, yellow, green. A transparent German shepherd dog walked across the room. The patient's father appeared as a figure 4 to 5" tall standing on a coffee table in the left visual field. A ghost stood in the library looking at a knitting book.

Case 7 — five to seven purple flowers resembling tulips.

Case 8 — all kinds of colored things and patterns moving in the visual fields — street scenes, cars falling into trenches, etc., no people or animals.

Case 9 — black and white zebra stripes on the floor and tufts of hair growing out of objects.

Case 10 — a little face like a smokescreen off to one side.

These hallucinations came and went all day long and were always recognized as unreal. There was no narrative action or emotional content such as might occur in a dream. The hallucinations usually persisted for 3 to 14 days, in one case for 7 weeks. The prominence of vivid colors was again notable. The right cerebral hemisphere was involved in ten of 14 adequately described cases, the left hemisphere in four.

The Prognosis

Full recovery from a hemianopia was rare, especially after the deficit had lasted two weeks. Often relatively better vision was restored to one quadrant, usually the inferior quadrant. A deficit in the inferior quadrant was a distinct handicap compared with a superior defect. Impairment of reading greatly curtailed the enjoyment of life. Persistent sensory symptoms were usually not troublesome, but in about 1% of cases a thalamic pain syndrome developed. Orientation and memory were nearly always restored, but damage to one hippocampus probably reduces the margin of safety as the deterioration of old age develops. Hallucinations, visual perseveration and metamorphopsia were temporary. Central glare was a rare persistent complaint.[20]

Further Aspects of PCA Ischemia

The subjects discussed so far include embolism to the distal basilar bifurcation, PCA embolism, local embolism, hemorrhagic infarction, and PCA thrombosis with its prodromal and stroke manifestations. There are a number of other aspects of PCA ischemia, which will be summarized briefly.

1. ***Migraine***. As already mentioned, the PCA is par excellence the artery of migraine. The visual accompaniments, transient, prolonged, and permanent, are of unending variation and are beyond the scope of this presentation. However, a small group of cases with unusual vascular events in the PCA suspected of

being migrainous and for which the term "catastrophic" or "cataclysmic" migraine has been suggested are reported separately.[21]

2. ***The amaurosis-amnesic syndrome following angiography***.[22,23] So regularly are the alarming deficits in this reaction reversible within 24 to 72 hours that it is probably premature to ascribe the symptoms to ischemia.

3. ***Compression of the PCA in temporal lobe herniation***. The manifestations are usually buried in the stupor and coma caused by brainstem compression. The resultant infarction is always hemorrhagic, a circumstance which should provide a clue to the mechanism of the process.

4. ***Bilateral PCA infarction***.[24,25,26] Several syndromes are recognized depending on the severity and extent of the damage to each PCA territory. When embolism to the distal bifurcation of the basilar artery is responsible, mesencephalo-thalamic symptomatology will dominate the picture, as previously described.

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