Relationship between visual field defect and arterial occlusion in the posterior cerebral circulation

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SYNOPSIS (1) The extent and severity of visual field loss has been compared in a series of 14 patients with occlusions of the posterior cerebral artery or its branches, all verified angiographically. Atheroma, embolism, and migraine were the commonest types of underlying vascular disease. (2) Occlusion of the main trunk of the artery was associated with severe and permanent field loss usually with some sparing of the central area and, in one case, of some field adjacent to the vertical meridian. It is suggested that this is due to collateral blood flow reaching the margins of the posterior cerebral territory from the adjacent middle cerebral territory via pial anastomoses. (3) Single or multiple occlusions of the main branches of the posterior cerebral artery gave variable amounts of field loss with considerable recovery in some cases. Collateral blood flow from the middle cerebral territory and from other branches of the posterior cerebral artery was demonstrated and the variation may be due to the size and number of pial anastomoses and to systemic factors such as blood pressure and vascular reactivity. (4) Occlusion of smaller branches of the calcarine artery produced localized zones of capillary underperfusion near the posterior cerebral pole. These corresponded to scotomatous paracentral defects in the visual field which were often permanent and showed no central sparing. The potential capacity of the collateral system may be limited by occlusion of intra-cerebral arteries or by involvement of segments of the pial arteries in the disease process.

The detailed clinicopathological studies of Holmes (1931) and Henschen (1926) in patients with defect of the visual fields after trauma allow us to delineate lesions of the occipital lobes with considerable accuracy, although it may not be possible to distinguish a lesion of the calcarine cortex from one involving the optic radiation.

More recently, improved methods of cerebral angiography have been applied to patients with vascular lesions in the circulation of the posterior cerebral artery and the introduction of selective catheter techniques combined with subtraction, colour subtraction, and radiographic magnification have enabled occlusion of smaller branches and regions of delayed or diminished capillary perfusion to be identified (Hoyt and Newton, 1970).

The relevant anatomy of the posterior cerebral artery and its branches may be summarized as follows: The posterior cerebral artery originates from the terminal bifurcation of the basilar

artery; its circum-mesencephalic portion passes around the midbrain immediately below the tentorium cerebelli and becomes the internal occipital segment as it crosses the free edge of the tentorial notch to lie on the medial aspect of the occipital lobe. It divides into terminal parieto-occipital and calcarine branches at a variable point along its course. The named branches are the thalamo-perforate, posterior communicating, medial and lateral posterior choroidal, splenial and anterior, middle, and posterior temporal arteries.

Passing from its origin in the interpeduncular cistern around the cerebral peduncle and through the crural cistern, the circum-mesencephalic segment curves backward with a downward convexity around the midbrain. In the ambient cistern it lies medial to the hippocampal gyrus, above and slightly lateral to the superior cerebellar artery. As it reaches the quadrigeminal cistern it courses medially and upwards through

the tentorial notch towards the calcarine fissure below the splenium of the corpus callosum and medial to the anterosuperior portion of the lingual gyrus. It may bifurcate into its terminal branches at any point beyond the crural cistern, often near its passage supratentorially.

In 80% of cases the posterior temporal artery arises as a single trunk, usually from the ambient segment of the posterior cerebral artery, occasionally from the quadrigeminal segment. In about 20%, however, there are two or more trunks of origin (Margolis *et al.*, 1971). The posterior temporal artery or arteries then course

posteriorly, laterally, and slightly downward along the parahippocampal gyrus to the lingual gyrus, anastomosing with the posterior temporal branch of the middle cerebral artery laterally and inferior branches of the calcarine artery posteriorly.

The parieto-occipital artery, the larger of the two terminal branches of the posterior cerebral artery, arises from its internal occipital segment in 40% of cases (Margolis *et al.*, 1971). In the remainder the bifurcation moves proximally and there is then no internal occipital segment. When it occurs the internal occipital artery passes posteriorly and upward over the cingulate gyrus, terminating in branches to the precuneus, parieto-occipital fissure, and cuneus. It anastomoses with the pericallosal branch of the anterior cerebral artery and with the calcarine artery.

The calcarine artery arises as one or two trunks, usually, but not invariably, on the lateral side of the parieto-occipital branch. The calcarine artery or arteries then cross the parieto-occipital on a posteromedial course to enter the calcarine fissure. It is no longer possible to support the contention of Shellshear (1927) that the calcarine artery exclusively supplies that area of brain which functions as the primary visual receptive field. In fact, that striate area itself often receives a contribution from the posterior temporal or parieto-occipital branches (Abbie, 1938; Smith and Richardson, 1966).

In patients with vascular occlusion, it is of interest to relate the site of blockage in the arterial tree to the type of visual field defect because it is possible to deduce the extent of the infarcted region of brain and the adequacy of collateral blood supply as well as other circulatory factors influencing survival of focal areas of cerebral ischaemia.

These cases necessarily form a highly selected group since not all cases of vascular hemianopia are subject to angiography. For instance, many patients who sustain only temporary visual field defects may not be included since they may not be referred to hospital. Patients of advanced age and those with widespread vascular or cardiac disease or with severe hypertension are also under-represented, since angiography carries a higher morbidity in this group. Similarly, in cases of embolism where there is a cardiac source for embolus, angiography may not be justified.

FIG. 1. J.P., a patient aged 64 years who wakened one morning with a headache and generally impaired vision. After four days the visual defect had become confined to the left field and proved to be a homonymous hemianopia sparing the macula. He suffered from severe hypertension and generalized arterial disease. The hemianopia improved and this vertebral angiogram, performed three months after the onset of the disturbance, shows almost complete occlusion of the right posterior cerebral artery about 1.0 cm from its origin.

(a)

(b)

FIG. 2. *P.W.*, a woman aged 42 years with a history suggesting episodic vertebrobasilar ischaemia who developed a transient right hemianopia which later returned and became permanent. There was central sparing. (a) The vertebral angiogram, A.P. view, shows occlusion of the calcarine artery and narrowing of the parieto-occipital, perhaps because it has recanalized. (b) The carotid angiogram, lateral view, shows collateral flow from the posterior temporal branches of the middle cerebral artery into the calcarine region.

A number of additional patients were examined; they had suffered an ischaemic episode and had a residual field defect but had no vascular occlusion on angiography; these patients have not been included in this report.

RESULTS

OCCCLUSION OF MAIN TRUNK OF POSTERIOR CEREBRAL ARTERY (Table 1) There were four patients who had occlusion of the main trunk of the vessel, usually at a point one to two centimetres from the origin (Fig. 1). Three of these had evidence of severe atheroma with diabetes or hypertension. The fourth patient had a history of recurrent migraine.

In two patients the hemianopia was dense and included the whole of the half-field with about 10° of central sparing. In the other two patients hemianopia was incomplete, with sparing of the central area and a portion of field adjacent to the

vertical meridian (Fig. 1). All these patients complained of headache at the onset. In two patients there was evidence of mild motor or sensory impairment on the affected side; one had spontaneous pain. Two patients with occlusion of the left posterior cerebral artery showed transient amnesia and mild dysphasia. The route of collateral blood supply was seen in one case to be from the posterior temporal branch of the middle cerebral artery.

There was a single case of bilateral posterior cerebral occlusion; he had evidence of additional upper brain-stem damage and died rapidly.

MAIN BRANCH OCCCLUSION (Table 2) Patients having occlusion of one or more of the three principal branches of the posterior cerebral artery included two with occlusion of the calcarine artery. In both the aetiology was atheroma and one had previous attacks of vertebrobasilar ischaemia. In both cases there was a dense and

TABLE 1
OCCLUSION OF MAIN TRUNK POSTERIOR CEREBRAL ARTERY

Sex, age (yr)			Aetiology	Associated features	Field defect		Outcome
Unilateral	1	M 38	Atheroma Diabetes Embolism	Headache			No change
	2	M 64	Atheroma Hypertension	Headache			Improved VBI
	3	F 42	Migraine	Hemianaesthesia Spontaneous pain Transient amnesia Dysphasia			Migraine continues
	4	M 62	Atheroma Hypertension	Hemiparesis Dyspraxia Transient amnesia Dysphasia			Unchanged
	5	M 55	Atheroma Embolism	Quadriplegia			Coma Death

permanent loss of a complete half field but with central sparing of about 10° and normal visual acuity. In one of these patients carotid arteriography showed collateral flow into the posterior cerebral territory from the posterior temporal branch of the middle cerebral artery (Fig. 2a, b).

There were three examples of multiple branch occlusion with diverse aetiology. One was a young woman six to seven months pregnant who developed a fluctuating visual defect of quadrantic type, most marked in the ipsilateral eye. The extent and severity of the field loss could be increased by hyperventilation and sometimes by eating. There was occlusion of the parieto-

occipital, calcarine, and posterior temporal branches (Fig. 3). The second case was a man aged 40 years with a history of vertebrobasilar insufficiency; both calcarine and parieto-occipital branches were occluded. The field defect was a transient incomplete hemianopia which recovered completely in a few weeks. The third case was a man aged 64 years with giant-cell arteritis of the vertebral arteries. Angiography showed multiple occlusions involving the left posterior temporal, left calcarine, and right calcarine arteries. Field defects again showed considerable fluctuation but a central 10° of vision was constantly retained.

FIG. 3. *W.M.*, a woman aged 30 years, who, when six to seven months pregnant, suddenly developed a left-sided visual disturbance which later proved to be a quadrantic type of hemianopia. She also complained of transient left-sided numbness. This Towne's view of a vertebral angiogram carried out eight months later shows occlusion of parts of the parieto-occipital and calcarine arteries and also non-filling of the posterior temporal branch.

SMALL VESSEL OCCLUSION (Table 3) In this group there were three men and one woman, ages ranging from 29 to 52 years. Underlying factors thought to predispose to occlusion were migraine in three and the puerperium in one. Angiography showed normal main vessels but absence of branches of the calcarine artery and areas of unperfused capillary circulation, best shown on the subtraction film (Fig. 4). Field defects in all cases were similar and consisted of a scotomatous hemianopia, quadrantic in shape and extending to within 2° of the fixation point. There was no evidence of central sparing. Evidence of collateral blood flow was seen in only one case from the parieto-occipital branch of the posterior cerebral artery.

DISCUSSION

Beevor (1909), in a classical study of the distribution of cerebral arteries by the method of post-

FIG. 4. *G.D.*, a man aged 52 years who suffered from migraine and developed a right quadrantic visual field defect with no central sparing. On this subtracted Towne's view of the vertebral angiogram, the small branches of the left calcarine artery are difficult to see and there is substantially less capillary blush on this side. Colour subtraction highlighted the slow and poor capillary filling of the left calcarine cortex.

mortem injection, was the first to show the potential value of pial anastomosis. He noted that when the posterior cerebral artery was occluded its territory could easily be filled from the adjacent middle cerebral field by the border-zone anastomoses which in most subjects were found in the U-shaped region on the inferolateral aspect of the cerebral hemisphere, the base of the U being near the occipital pole. That the anastomoses have a function during life is known from the finding that in posterior cerebral artery occlusion the area of infarction is normally much smaller than the anatomical territory of the vessel. Ischaemic damage is most severe in the middle of the posterior cerebral arterial field (the anterior part of the striate area) and the border zones are spared (Zülch and Behrend, 1961).

The results of the present study, which attempts to correlate the site of posterior cerebral occlusion with the type and extent of visual field defect, provide further confirmation of Beevor's views. Our four cases of occlusion of the main

TABLE 2
OCCLUSION OF CALCARINE, POSTERIOR TEMPORAL, PARIETO-OCCIPITAL ARTERIES

Sex, age (yr)			Aetiology	Associated features	Field defect		Outcome
Calcarine 1	F	42	Atheroma Embolism	VBI			Unchanged
2	M	52	Atheroma	Nil			Unchanged
Multiple, unilateral 3	F	30	Pregnancy Embolism	Hemianaesthesia			3 main branches Fluctuating defect Full recovery
4	M	40	Atheroma	VBI			Calcarine and parieto-occipital Full recovery
Multiple, bilateral 5	M	64	Giant cell arteritis	Nil			L posterior temporal L calcarine complete R calcarine partial Partial recovery

trunk of the artery had severe permanent visual field defects but showed at least 10° central sparing and, in one case, sparing of a further vertical strip. In anatomical terms this implies intact macular projections to the posterior pole and, in addition, survival of an area in the periphery of the striate cortex. This area is furthest from the middle of the posterior cerebral field and nearest to the surrounding vascular territories. Conversely, that part of the visual field most consistently lost was the area adjacent to the horizontal meridian which corresponds to the depths of the calcarine fissure in the middle of the posterior cerebral territory.

There was also radiological confirmation that the route of collateral blood supply was via the posterior temporal and angular branches of the middle cerebral artery. In fact, posterior cerebral artery occlusion may sometimes be diagnosed by carotid angiography when in the late arterial phase retrograde filling of posterior cerebral branches is seen.

Radiological/clinical correlation is less easy in the case of branch occlusions since there is considerable variation in the number, size, and distribution of the three main branches—posterior temporal, parieto-occipital, and calcarine. In general, the calcarine artery is the

TABLE 3
OCCLUSION OF SMALL BRANCHES, DELAYED CAPILLARY PERFUSION

	Sex, age (yr)	Aetiology	Associated features	Field defect		Outcome
1	M 45	Migraine	Nil			Unchanged
2	F 29	Puerperium	Nil			Unchanged
3	M 52	Migraine	Nil			Unchanged
4	M 36	Migraine ?Polyarteritis	Nil Nil			Unchanged

main blood supply to the striate cortex but there are also contributions from the posterior temporal and/or parieto-occipital branches in over 50% of brains. Our cases of branch occlusion showed no constant relationship between severity and extent of visual field loss and the type of arterial occlusion. On the one hand there were two patients with unilateral calcarine artery occlusion who had permanent loss of all but the central 5–10° of visual field; on the other were three cases with multiple branch occlusions possibly embolic in origin, all of whom showed an incomplete hemianopia, fluctuating in extent and eventually recovering to a substantial degree.

The area of unperfused capillary bed shown on the vertebral angiogram of such patients is considerably greater than the anatomical extent of brain damage deduced from visual field studies (Hoyt and Newton, 1970). This is due to collateral supply from the carotid territory and can

be verified by performing a carotid angiogram when contrast medium can be seen reaching the area via the posterior temporal (and perhaps the angular) branches of the middle cerebral artery. In our cases of isolated calcarine artery occlusion there was also radiological evidence of collateral flow from the parieto-occipital branch of the posterior cerebral artery, although Beavor and later Van der Eecken and Adams (1953) showed by injection studies that the anastomoses between branches of the same artery were less well developed than those between adjacent main cerebral arteries.

The third group showing small branch occlusion or areas of capillary avascularity is of particular interest since this abnormality would probably escape detection by older techniques. The visual field defect in these cases was of a uniform type and comprised a scotomatous congruous hemianopic defect, triangular in

shape with the apex extending to the fixation point, and with no evidence of central sparing. This would correspond to a wedge-shaped area of striate cortex extending to the occipital pole and including either the upper or lower lips of the calcarine fissure (Fig. 4). An exactly similar case with pathological confirmation is recorded by Polyak (1957). The lack of sparing of the central visual field in these cases of branch occlusion has been discussed elsewhere (Russell, 1973). It may be explained by involvement of a length of a single vessel or by involvement of a number of vessels in the pathological process, thus limiting the potential collateral supply. Such conditions may obtain in multiple small emboli, propagating thrombosis, or vasospasm involving a segment of vessel.

AETIOLOGY OF VASCULAR OCCLUSION Our findings illustrate that posterior cerebral artery occlusion is not due to a single pathological process. The various types of pathology found should not be taken as a true representation since older patients or those with hypertensive cardiac disease or with a known source of embolization have been excluded. The number of cases with simultaneous bilateral or multiple occlusions suggests that embolism with fragmentation of emboli in the circulation is more common than is generally thought. Pathological studies have shown that the source of emboli in these cases may be mural thrombus of the vertebral or basilar arteries; there is usually extensive atheroma and sometimes aneurysmal dilatation of the basilar artery. Giant-cell arteritis of the vertebral arteries is another recognized cause (Wilkinson and Russell, 1972).

In younger patients the most important aetiological factor was a history of severe recurrent migraine. The preceding migraines in all cases were accompanied by prominent visual symptoms which were sometimes bilateral and the permanent hemianopia appeared in the course of a severe migraine attack. The site of occlusion was the main posterior cerebral trunk in one case and small branch vessels in three. It must be emphasized that vascular occlusion and prominent neurological deficit is exceptionally rare in migraine but in reported cases the posterior cerebral artery territory is more often affected than other areas (Pearce, 1968). The

fact that vascular occlusion can be demonstrated days or weeks after the onset of symptoms suggests that the condition is not simply due to an exceptionally severe episode of vascular spasm or oedema but probably represents a superadded thrombosis. Other aetiological factors in young patients included contraceptive medication and the puerperium, both recognized as predisposing to thrombosis (Bickerstaff and Holmes, 1967). Delayed endarteritis after radiotherapy may also affect arteries of this size (Kagan *et al.*, 1971).

The angiographic examinations were done at various intervals, after the onset of visual loss and the appearance on x-ray examination cannot be assumed to be constant throughout the illness. Pathological studies in cases of cerebral infarction have shown that the earlier the examination the higher the proportion of occlusions. Lysis and fragmentation of arterial emboli has been demonstrated by pathological and angiographic studies and by direct observations on the retina (Fisher and Adams, 1951; Russell, 1961; Dalal *et al.*, 1965). The impaction of an embolus at the bifurcation of the basilar artery and its subsequent displacement into the posterior cerebral artery on one side probably accounts for those patients who suffer from total loss of sight which clears in the space of minutes or hours to leave a unilateral defect (Symonds and Mackenzie, 1957). Simultaneous small vessel blockage, smooth regular vascular outlines up to the point of obstruction, and the visualization of the sharply defined profile of the proximal end of the thrombus lodged in the vascular lumen are further angiographic points in favour of embolism.

The time factor must also be taken into account in the comparison of visual fields. It has been found that complete and dense hemianopia to all visual stimuli which is present 48 hours after a stroke frequently persists indefinitely, but if a patient retains even vague perception of hand movements in the affected field then considerable recovery can be expected. This takes the form of gradual enlargement of the central spared area and extension of vision across the vertical meridian. Incongruity between the two eyes is a marked feature of these cases, the eye showing the temporal field loss usually being the more severely affected. In the final stage the defect may be demonstrated in this eye only.

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