The Posterior Cerebral Circulation

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Understanding of the correlation of structure and function in the occipital lobe probably exceeds that in any other area of the brain. By applying the information compiled from detailed studies of patients with localised brain wounds to patients with vascular disease it is possible to identify those areas most commonly affected by various types of vascular lesion and to shed some light on the disorders of the cerebral circulation that underlie them.

Although the projections of the retina on to the striate cortex have been studied extensively (Henschen, 1926; Holmes, 1931) there has been comparatively little interest in the blood supply to this area.

The extent of the territory of the posterior cerebral artery was first well shown by the injection studies of Beevor (1905). Using coloured media simultaneously to inject three or more arteries, he showed that the posterior cerebral artery usually supplied the medial surface of the hemisphere, posterior to the parieto-occipital sulcus, including the whole of the striate cortex, the splenium of the corpus callosum, the thalamus and the under surface of the temporal lobe. The border zone between middle and posterior territories

Fig. 1. Common distribution of posterior cerebral artery: (a) medial surface of hemisphere; (b) lateral surface of hemisphere. Note border zone close to posterior pole (Beevor, 1905).
is a U-shaped zone on the lateral surface of the hemisphere, the base of the U reaching to the posterior pole (Fig. 1a, b). Beevor remarked on the considerable individual variation in the extent of the main vascular territories and showed that sometimes the posterior cerebral artery might supply the whole of the lateral aspect of the parietal lobes as far forward as the central sulcus. He also showed that when the main trunk of any of the three cerebral arteries was occluded, and communication at the Circle of Willis was prevented, the territory could easily be filled from the other vascular fields by anastomoses on the surface of the brain.

'The communication between areas of two different arteries is well shown by the following observation, which I have repeated several times. If the anterior middle or posterior cerebral arteries be injected with three different colours at the same pressure until there is a well-marked line of demarcation, let us say between middle and posterior cerebral arteries, and if now the tube supplying the middle cerebral artery is collapsed it will be seen that the injection from the posterior cerebral artery will invade that of the middle cerebral artery and drive its colour out but on releasing the clamp the original border line is attained.'

Beevor also noticed that when a branch of a main cerebral artery is tied, its territory fills from that of the adjacent main artery rather than from other branches of the same vessel. Quite a different situation was found between arteries below the brain surface, for after stripping a portion of the pia or making a circular incision in the cortex, an area of unperfused brain was left, suggesting that deeply situated anastomoses do not play a significant role.

The physiological function of the pial anastomoses has also been studied experimentally, notably by Meyer and Denny-Brown (1954). They confirmed in vivo that, after vascular occlusion in animals, blood does flow into a low-pressure arterial bed from surrounding vascular fields, and that anastomotic arteries may show a rapid and striking enlargement to accommodate the increased blood flow. It is now known from studies of regional blood flow that the intrinsic autoregulatory function of cerebral arteries may be disturbed by ischaemia, so that the periphery of an ischaemic zone may actually receive more blood than it requires (Lassen, 1966). As might be expected, the area most affected by ischaemia is in the centre of the vascular field at a point farthest from the collaterals and, should collateral flow be inadequate, this point will show the earliest microscopic change of infarction.

It may be asked what relevance these anatomical and physiological principles have to occlusion of the posterior cerebral artery in man. Neuropathologists can measure the size and configuration of the infarct in fatal cases.
Fig. 2. Coronal sections of posterior 10 cm of brain to show: on left, distribution of the main cerebral arteries (AC, MC, PC); on right, the extent of infarction following occlusion of posterior cerebral artery at its origin (dark shaded area).
Figure 2 shows the average extent of infarction in twenty cases, where posterior cerebral artery occlusion was caused by compression of the artery at the tentorium. The infarcted zone is much smaller than the posterior cerebral territory and damage is maximal in the lingual and funiculate gyri and in the rostral part of the striate cortex, extending along the under surface of the temporal lobes. The border zone, including the posterior pole, escapes infarction. The damage is mostly in grey matter and the radiation tends to escape except at its most posterior part. Histology does not reveal how often the posterior cerebral artery is occluded without infarction, nor the state of the smaller blood vessels and anastomotic vessels. For this information we must turn to radiology.

Vertebral angiography has recently been refined by the subtraction and enlargement techniques. It is possible to see not only occlusion of the main trunk and three principal branches, posterior temporal, parieto-occipital and calcarine, but also the capillary bed. It is now known that total occlusion of the posterior cerebral artery near its origin is compatible with survival of the brain in the posterior cerebral artery territory and that an apparently avascular area of brain may be receiving an adequate blood flow from the middle cerebral artery by anastomoses (Fig. 3). For this reason it is often necessary to perform both carotid and vertebral angiograms in each patient. Even clipping the calcarine artery itself (Hoyt and Newton, 1970) is compatible with normal vision and full fields. Conversely, cases with a persistent visual field defect may show a normal angiogram, or occlusion of the main trunk, or single branch occlusion of the posterior or middle cerebral artery (Fig. 4) or, most commonly, multiple branch occlusions having the sharp cut-off typical of embolism (Fig. 5). As in the carotid circulation a most important factor is the time at which the investigation is done, and the shorter the interval between the stroke and the angiogram the higher the number of occlusions seen (Ring, 1966).

Experience of the clinical effects that follow infarction of this area of brain is drawn from elderly atheromatous patients in whom posterior cerebral artery occlusion occurs as a result of local thrombosis or embolism from a source of thrombus in the basilar or vertebral arteries. Other types of pathology include aneurysm with spasm, compression of the artery after brain swelling or arteritis of the vertebral arteries. In younger patients, occlusion may occur as a complication of migraine or the use of steroid contraceptives. In all these patients the first and most important clinical deficit is an isolated homonymous hemianopia of sudden onset without hemiparesis or sensory loss but with pain in the ipsilateral eye as a common accompaniment. The defect is usually absolute, includes the complete half field, has a steep vertical cut-off and, usually, but not invariably, spares the central 5–10° around the fixation point. It seems that there is infarction of the major part of
the striate cortex but that central sparing is due to the integrity of the posterior pole and the macular fibres of the radiation. Patients with a dense complete hemianopia of this kind, persisting for more than 24 hours after the onset, rarely show much recovery, but, even if crude visual perception is retained in the affected half field, considerable improvement may ensue, often in one quadrant and especially in young patients. In these cases recovery spreads
outwards from the centre and perception of form and colour and spatial arrangement in the affected field may be incomplete, suggesting residual damage to the parastriate area or connecting tracts. Some incongruity between the two eyes may be present.

A vascular hemianopia is a commonplace clinical event and it is not unusual to hear patients complain of complete blindness at the moment of onset, which clears in a few minutes on one side to leave a homonymous hemianopia.

Fig. 4. Vertebral angiogram (subtraction film) of young woman with persistent right homonymous hemianopia following an attack of migraine. The calcarine artery is occluded at its origin.
Simultaneous permanent loss of both halves of the visual field from vascular occlusion is much less common and is due to embolism. In these cases loss of vision may be complete but, in most instances, there is again sparing of the central few degrees of vision with an island of retained vision extending upwards or downwards along the vertical meridian (Fig. 6a) (Symonds and Mackenzie, 1957).

Pathological examination in these cases shows preservation of the posterior pole and sparing of some of the calcarine cortex on the lower or upper lip of the fissure at the farthest extent of the calcarine artery territory and at the point
nearest to the collaterals. The area of maximum damage is in the anterior part of the calcarine cortex, and grey matter appears to suffer more severely than white matter.

Homonymous hemianopia is the hallmark of posterior cerebral artery occlusion but it may be combined with other defects suggesting damage to more anteriorly situated parts of the territory. Dysphasia is well documented as occurring in left posterior cerebral artery occlusion. In personal cases this has been mild, transient, and has consisted of difficulty in recalling the names of objects and of discriminating colours. In an early case of this kind (Foix and Masson, 1923) damage was found in the lingual and fusiform gyri and to the cortex of the under surface of the left temporal lobe.

The association of a more generalised amnesic syndrome with right homonymous hemianopia, but without hemiparesis or sensory loss, is important since such patients are often admitted to psychiatric hospitals, where the hemianopia may be overlooked. The amnesia is usually temporary, but persisting amnesic defect has occasionally been reported in left-sided occlusions, probably due to left temporal infarction extending to the hippocampus or interrupting the tracts linking parastriate and anterior temporal areas. A well-studied case (Mohr et al., 1971) showed persisting memory defect and a curiously circumscribed difficulty of matching colours with the colour name. Autopsy revealed infarction in the lingual gyrus and in the hippocampus. The isolated defect of facial recognition, known as prosopagnosia, is always found in association with a visual field defect. This is usually bilateral but in some cases it is the upper left visual field that is involved (Meadows, J. C., 1972, personal communication).
The syndrome of pure dyslexia without dysgraphia has been known since Dejerine’s time and still excites interest (Dejerine and Vailet, 1893). These patients have a dense right homonymous hemianopia and are unable to read in the remaining half field, although retaining normal expressive language. They can recognise words verbally spelled out and can write spontaneously but cannot read, not even the words they themselves have just written. The lesion in such cases has been shown to be in the splenium of the corpus callosum, interrupting the connection between the intact right visual cortex and the region of the angular gyrus on the opposite side.

In rare instances there is clinical evidence of infarction of the anterior part of the posterior communicating artery (syndrome cerebello-thalamique of Foix). The patient experiences sudden hemianesthesia that affects all sensory modalities, accompanied by hemianopia and by a mild hemiparesis. The hemiparesis, which is not spastic in type, clears in a few days, leaving a persistent hemisensory disturbance. Severe spontaneous pain and intention tremor may appear after an interval. The symptomatology in these cases points to lesions of the thalamus, lateral geniculate body, subthalamic nuclei and the upper mid-brain and is presumed to be due to occlusion of proximal branches of the posterior cerebral artery (Foix and Masson, 1923). The full syndrome is unusual, but minor signs of mid-brain damage such as unreactive pupils, vertical gaze palsy or an internuclear ophthalmoplegia may be encountered in some cases of posterior cerebral artery occlusion.
In contrast to these large and disabling lesions are minute ischaemic lesions of the striate cortex produced by occlusion of smaller branches. They provide an elegant confirmation of the localisation of visual field defects deduced from war wounds. The patients are hypertensive, diabetic, have valvular heart disease, or migraine. Small softenings of the upper and lower lips of the calcarine cortex produce scotomatous defects that are strictly congruous and extend right up to the fixation point, the opposite of central sparing (Fig. 6b). A well-studied case of localised infarction occurring during a migrainous attack is recorded by Poliak (1957). The patient, the celebrated pathologist, Dr Mallory, directed that his brain be examined after death. The field defect, which was scotomatous, quadrantic and extended for 20°, corresponded to a triangular infarction of the lower calcarine lip near the posterior pole. An even smaller softening (Henschen, 1926) in the depth of the calcarine fissure produced a cigar-shaped scotoma along the horizontal meridian, confirming the localisation of this area in the depth of the fissure. In these cases even when the defect splits the macula, central visual acuity may be well preserved and patients learn to compensate for their defect by looking a little to one side. However, bilateral small lesions at the posterior pole can produce irregular central or paracentral scotomas that are very disabling; they result from multiple embolism either in the middle or posterior cerebral artery.

It is of interest to consider how the vascular anastomoses that protect against occlusion of major vessels cannot function when vessels of a smaller calibre are involved. In the case of these small softenings it is possible that the origin of the penetrating arteries themselves are blocked since, as Beevor showed, there are no effective anastomoses below the surface. A further important explanation lies in the simultaneous occlusion of a number of arteries by emboli, some of them the anastomotic vessels themselves. Lodgement of emboli simultaneously in both posterior poles might seem an unlikely event to explain the appearance of bilateral central scotomas sometimes seen in cases of fat embolism until it is remembered that the surface border zones at the farthest extent of the vascular tree are also the favoured site of lodgement of such embolic particles under experimental conditions.

Seventy years ago a multitude of neurological syndromes were described, each supposedly corresponding to occlusion of a single artery or arterial branch. With increasing knowledge of the cerebral anastomoses and recognition of the role of the extracranial occlusions, these views lost favour, but now small vessel occlusions are seen more frequently, since the demonstration by refined angiography that multiple embolic occlusions are quite common (Ring, 1966) and that emboli can dissolve in the circulation, having originated from thrombotic lesions on the walls of the heart or large arteries (Gunning et al., 340
1964). These views are now widely accepted for the carotid circulation but they may be no less important for the vertebral basilar territory.

The detailed correlation between clinical deficit and ischaemic brain lesions is unfortunately not matched by knowledge of the state of the vessels themselves. It is sufficient, however, to support the general contention that, in occlusions of the posterior cerebral artery, the zone of maximum ischaemia is in the middle of the vascular field and that sparing of border zones occurs because of collateral flow from surrounding territories, notably the middle cerebral (Zulch and Behrend, 1961). The extent of permanent damage depends partly on anatomical factors such as size and number of pial anastomoses and partly on physiological factors such as the height of the systemic blood pressure, gas tensions, and the state of contraction of vascular smooth muscle. Under exceptionally favourable conditions the whole of the posterior cerebral territory may be adequately perfused after occlusion but, under unfavourable conditions, practically the whole of the territory may be infarcted. Under average circumstances, infarction is limited to the cortex of the calcarine area, except in its posterior pole. Sometimes the situation may be unstable immediately after occlusion and the patient may have a fluctuating field defect. An exceptional case of this kind has a small residual lower quadrantic defect that expanded during overbreathing to become a complete hemianopia (Fig. 7) (Kremer, M., 1972, personal communication).

Patients suffering from a generalised reduction in cerebral perfusion present an entirely different problem, but one which also has important effects on the posterior cerebral circulation. Circulatory arrest for longer than a very few minutes results in death of the whole brain, but under less drastic circumstances where there is a drop in perfusion pressure lasting some hours or days, or when the heart stops or fibrillates and is then quickly re-started, the patient may survive with residual cerebral damage. Often this takes the form of generalised damage to both hemispheres in which the occipital lobes share, but which may spare the brain stem to an extent compatible with a vegetative existence (Brierley, 1964). At other times, especially when there have been repeated short-lived falls in perfusion, there may be evidence of selective damage to the border zone territories of the hemispheres between the major cerebral vessels (Romanul and Abramowicz, 1964). The parieto-occipital area and posterior pole constitute such an area on the border between posterior and middle territories; lesions in these areas have been produced experimentally by hypotension in primates (Brierley and Excell, 1966).

The clinical features of these lesions have received little attention. Vascular insults of this kind most commonly affect elderly patients with pre-existing occlusive disease in the neck arteries and are provoked by transient cardiac
dysrhythmia or fall in cardiac output after myocardial infarction. However, the syndrome is increasingly seen in intensive care units after cardiac operations or suicidal attempts in patients with previously normal brains. A very
similar clinical picture may also occur in a reversible form during hypertensive encephalopathy, where it is presumed that generalised vasospasm affects much of the cerebral vasculature, or following vertebral angiography, where a similar though less widespread condition prevails.

The clinical picture varies with the amount of brain damage. In its mildest form with a short period of diminished perfusion, the patient may experience transient attacks of blindness on sudden standing or on exercise, during which the pupillary responses are preserved; recovery is rapid and complete. Unfortunately, we more commonly see seriously affected cases with longer periods of disturbed circulation where the patient is initially in a state of decortication. There is general unresponsiveness to visual, auditory and tactile stimuli, together with motor responses of a primitive kind to pain. Brain-stem reflexes are preserved and, although the patient appears quite blind, the pupils react briskly to light. The legs are usually held extended, with exaggerated reflexes and extensor plantar responses. Although this state may be permanent, the clinical state presented by some patients during recovery is both interesting and unusual. After emerging from a state of complete decortication, the patient passes into one of restless delirium with recovery of some cortical function. He is noisy, moves all limbs, is resentful of interference, hallucinated and still appears blind. There is no blinking to menace although he may blink to a hand clap. Spoken commands of a simple kind may be carried out. The optokinetic responses are absent and no alpha rhythm is found on the EEG. Under favourable conditions recovery proceeds and it becomes clear that visual perception of a kind is returning. The patient may attempt to follow an object moved before his face and may turn his head to look at a visitor. He may appear to have a defect of visual fixation, looking to one side rather than at the face of the person speaking to him. He may also have difficulty in moving his eyes from one object to another, as though he can see but not look.

Further improvement brings the patient to a state resembling that described as visual disorientation (Holmes, 1918). The size, shape and spatial relationships of objects are disordered. Visual stimuli may be perceived in all parts of the field but incorrectly localised. This difficulty affects both sagittal and coronal planes, so that the judgement of distance is grossly defective. The relative position in space of a number of objects may be confused so that the patient may be unable to identify the nearest or farthest away of a group of objects, though he can do so at once if he can touch them. Similarly, the visual judgement of length or of size or thickness may be grossly at fault. An inability to count a number of objects while looking at them is also a prominent feature in some of these patients and they seem unable to see more than one object at
a time. Thus, they may be unable to grasp the point of a composite picture although able to see its component parts quite easily. Holmes summed this up in the phrase ‘a defect in mental synthesis of elementary visual perception’.

We have had an opportunity of following a few of these patients for some weeks after cardiac arrest, as they progressed to complete recovery. Although all of them at one time showed features of diffuse brain damage, the abnormalities that persisted longest were those that demonstrate an inability to synthesise and retain visual spatial information. Thus, the patient may have difficulty with reading or calculating, may show a constructional apraxia, with inability to copy or complete designs, may have a defect in memory, particularly topographical memory, so that he may be unable to draw a map of the road he lives in, or to remember the arrangement of rooms in his house.

Visual field defects may or may not be present in these cases. When visual perception is just returning, it may be possible to demonstrate a field defect to confrontation. This may be altitudinal in type or may involve a general contraction of the visual fields. Occasionally, permanent defects in the field of vision may remain. In some reported cases (Hoyt and Walsh, 1958) and in three personally studied cases, the pattern of visual loss has been altitudinal, with retention of whole or part of the upper field (Fig. 8). In some cases visual

![Fig. 8. Border zone infarction. Extensive field loss with retention of small island of vision in upper field in a 9-year-old child. Severe hypotension due to haemorrhage following tonsillectomy.](image)

acuity in the retained field has been poor, but in other cases it has been normal.

Pathological examinations of these cases are not frequent. Fatal cases show very extensive lesions and milder cases do not often come to autopsy. An old case of Bramwell et al. (1915), with preservation of a tiny field but with poor visual acuity, had bilateral lesions of the parieto-occipital area involving much
of the radiation. The striate area itself was spared. Poliak’s case (1957) showed a symmetrical lesion of the parieto-occipital area and had a permanent altitudinal hemianopia following a period of collapse. Gilman’s (1965) case showed an isolated disturbance in reading, drawing and calculating, but with retention of the visual fields, and had a lesion in the left parieto-occipital cortex. Figure 9 is taken from the case of a man of 50 who suffered an episode of severe hypotension during laminectomy and survived for nine months with a left hemiparesis and hemianopia, visual disorientation in the remaining field, denial of symptoms and a gross memory disturbance. He showed lesions that were most severe in the parieto-occipital area, and particularly affected the border zones.

In general, the resemblance between these cases and the discrete brain wounds studied by Gordon Holmes, together with the pathology that is available, indicates that the area most vulnerable to vascular insults of this type is the parieto-occipital border zone on both sides (Zulch and Behrend, 1961). White matter tends to be involved in severe cases, involving the upper fibres of radiation. The posterior poles may be infarcted but in many cases

Fig. 9. Border zone infarction. Coronal slices of caudal 10 cm of brain from a 50-year-old patient who suffered severe hypotension in sitting position during cervical laminectomy. Death occurred nine months later. Extensive ischaemic damage (stippled areas) to both hemispheres, maximal in border zones and at posterior poles.
there is sparing of the primary visual area. Further study of these interesting patients will lead to a better understanding of cortical localisation and the haemodynamic events leading up to cerebral infarction.

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