THE SYNDROMES OF THE ARTERIES OF THE BRAIN AND SPINAL CORD

Part I

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Introduction

Little interest is shown at present in the syndromes of the arteries of the brain and spinal cord, and this perhaps is related to the tendency to minimize the importance of cerebral localization. Yet these syndromes are far from being fully elucidated and understood. It must be admitted that in many cases precise localization is often of little more than academic interest and provides nothing but a measure of personal satisfaction to the physician. But it is worth recalling that the detailed study of the distribution of the bronchi and of the pathological anatomy of congenital abnormalities of the heart, was similarly neglected until the prospect of therapeutic application demonstrated the fallacy of this attitude.

Cerebral thromboses are usually the expression of diffuse vascular disease and often it is neither possible nor profitable to implicate named vessels. Optimistic conclusions have often been expressed in the past regarding the specific effects of occlusion of one particular vessel, but are difficult to accept in view of the ultimate demonstration of multiple lesions. Cases of cerebral embolism would seem to provide a better prospect of accurate study but are less common; many vessels are seldom, if ever, involved and the opportunity of making pathological studies becomes very remote when the smaller—and in many respects more interesting—vessels are affected. Cerebral angiography may in the future offer an alternative to post-mortem examination and provide a chance of identifying the site of the primary occlusion at a time when the clinical picture remains uncomplicated. Such studies as that made by Ethelberg (1951) of the anterior cerebral artery, are likely to lead to a rapid expansion of our knowledge of these syndromes.

The varied effects of occlusion of any particular artery in different individuals depends on several factors. The extent of the territory supplied and the efficacy of the collateral circulation differ widely from person to person. The degree of

general circulatory efficiency at the time of the catastrophe is of the greatest importance. An occlusion, producing little effect in the presence of a normal blood pressure, may cause widespread pathological changes if hypotension co-exists.

A marked discrepancy has been noted between the effects of ligation and of spontaneous thrombosis of an artery. The former seldom produces ill effects whilst the latter frequently determines infarction. This is probably due to the tendency of a spontaneous thrombus to extend along the affected vessel, sealing its branches and blocking its collateral circulation, and to the fact that the arterial disease is so often generalized.

In this paper the various syndromes resulting from occlusion of the arteries of the brain and spinal cord will be described in the light of our present knowledge. The pathology of the occlusive process will not be considered, nor will the slowly developing syndromes due to diffuse cerebrovascular disease be discussed. The anatomy and distribution of the vessel in question will be outlined briefly before its syndromes are described.

Internal Carotid Artery

The internal carotid artery enters the skull through the carotid canal and runs forwards through the cavernous sinus to the medial aspect of the anterior clinoid process. Here it turns upwards and at the commencement of the Sylvian fissure divides into its terminal branches—the anterior and middle cerebral arteries.

The results of occlusion of the internal carotid artery are extremely varied. The condition is a chance finding at arteriography in a number of patients who have none of the anticipated symptoms or signs. Certainly surgical ligation of the vessel—if proper precautions are taken—rarely gives rise to any disability.

When symptoms do occur, the onset is often gradual. Persistent headache of a migrainous character on the same side as the thrombosis may

occur for years before other features develop. Transient attacks of focal weakness, of focal paraesthesiae or of dysphasia are common and rarely generalized convulsions may occur. The suggestion has been made that these transient disturbances result from spasm of the smaller cerebral vessels reflexly induced by impulses from nerve endings in the wall of the diseased internal carotid artery. In other cases an acute onset is seen. The left internal carotid is affected much more frequently than the right.

The full syndrome that might be expected on anatomical grounds is seen only occasionally. Ipsilateral anosmia is sometimes present. There is blindness of the ipsilateral eye—which may be transient—due to involvement of the ophthalmic artery, while the opposite eye shows a temporal field defect due to a lesion of the optic tract and radiation. Optic atrophy develops in the blind eye. A severe contralateral hemiplegia and hemianaesthesia are also present, together with a severe dysphasia if the major hemisphere is affected. Although mental changes of the organic reaction type have been attributed to occlusion of the internal carotid artery, their occurrence is so haphazard as to suggest that their presence is coincidental.

Cases of lesser severity are far more numerous. The most common picture is that of a moderately severe hemiplegia accompanied by a hemianaesthesia of lesser severity with astereognosis and some degree of dysphasia. It is obvious that there may be nothing characteristic about the condition and its differentiation, particularly from occlusion of the middle cerebral artery or one of its branches, can only be made by carotid arteriography.

(See Elvidge and Werner, 1951; Fisher, 1951.)

The Anterior Cerebral Artery

From its origin at the medial end of the Sylvian fissure, the anterior cerebral artery runs forwards and medially across the anterior perforated substance and above the optic nerve to the margin between the medial and orbital surfaces of the frontal lobe. At this point the vessels of either side are connected by the anterior communicating artery. Each then turns abruptly upwards to reach the genu of the corpus callosum and runs backwards in the depths of the longitudinal fissure, finally emerging from it to end in the quadrate lobe or the parieto-occipital fissure. Branches:

(a) Basal: (i) Several fine branches enter the anterior perforated substance and supply the head of the caudate nucleus. (ii) Recurrent artery (artery of Heubner). This pierces the lateral extremity of the anterior perforated substance and passes upwards and outwards to supply the anterior part of the caudate nucleus, the anterior

third of the putamen, the outermost part of the globus pallidus and the anterior limb of the internal capsule.

(b) Anterior communicating artery—which often gives off a few twigs to the optic chiasma and infundibular region (Rubenstein, 1944).

(c) Branches from the convexity: (i) Prefrontal or orbital branch supplying the medial aspect of the frontal lobe. (ii) Frontopolar branch—to the most anterior portion of the superior frontal gyrus. (iii) Callosal-marginal artery which divides into anterior, middle and posterior internal frontal branches supplying the medial portion of the convexity of the frontal lobe. (iv) Paracentral artery—to the paracentral lobule. (v) Superior parietal branch. (vi) Precuneal branch—to the region of the quadrate lobule. (vii) Parieto-occipital branch.

(d) Branches from the concavity: (i) Numerous twigs to the corpus callosum and to the anterior pillars of the fornix. (ii) Pericallosal artery which lies deep to the anterior cerebral artery and continues the line of this vessel.

Occasionally both anterior cerebral arteries arise from the same internal carotid and sometimes only one vessel is present.

Occlusion of the Main Trunk Proximal to Heubner's Artery

Much depends on the size of the anterior communicating artery. If this is large and not obstructed by the thrombus, occlusion of the main trunk of the anterior cerebral artery will either produce no ill effects or give rise to a syndrome similar to that following thrombosis of Heubner's artery. If the collateral circulation is inadequate the resulting syndrome is much more serious. In most cases consciousness is lost. Dandy (1930) as the result of some unfortunate experiences following the tying of the left anterior cerebral artery, maintained that total occlusion of this vessel always resulted in stupor or coma from which recovery did not occur. Many cases have since been recorded in which ligation produced no ill effects (Poppen, 1939). Major convulsions may occur and the eyes and head may be turned to the side of the lesion due to the unbalanced action of the opposite frontal eye field (area 8). When the patient is conscious it may also be found that he has some defect of lateral conjugate movement of his eyes to the opposite side because of the damage to the ipsilateral eye field. These features are short-lived and disappear in a few days or weeks.

Mental changes—including all the organic reaction types—may occur and are regarded by some authors as a specific part of the clinical picture, but this cannot be maintained. Patients showing these conditions are usually old and have diffuse

FIG. 1.—Medial aspect of right cerebral hemisphere showing distribution of cortical branches of anterior, middle and posterior cerebral arteries. (The paracentral branch in this case comes off the calloso-marginal artery.) (de Jong, 1950, 'The Neurologic Examination,' Cassel & Co., London.)

cerebrovascular disease. Of Ethelberg's 20 cases, seven only showed some memory disturbance and shallowness of affect. Bilateral occlusion of the anterior cerebral arteries is of course much more likely to be associated with dementia—usually accompanied by a placid euphoria—but diffuse arterial disease is invariably present too.

A severe hemiplegia results from the combined effects of lesions of the anterior limb of the internal capsule and of the paracentral lobule. The face and limbs are involved more or less in equal degree, though either limb may be differentially affected in some cases. After the initial period of 'shock' the hemiplegia becomes spastic. Vasomotor and trophic changes are commonly marked. The skin is cold and blue and chronic oedema may be present. Dystrophic changes in the skin, nails, subcutaneous tissues and joints may follow.

Sensory loss of a cortical type is present in the lower limb due to the lesion of the paracentral lobule and is most marked distally. Occasionally similar loss is present in the arm. Some urgency or frequency of micturition is common.

When the major hemisphere is affected, some degree of dysphasia of an expressive type is

common initially but rarely persists for more than a few weeks. Ethelberg (1951) maintains that the speech disturbance is not a true dysphasia but an 'impairment of rapid sequential movements of the speech organs' and is therefore a cortically determined dysarthria. In these cases a dyspraxia—usually ideomotor in type—is found on the side opposite to the paralysis. Theoretically, the dyspraxia is bilateral but on the hemiplegic side is masked by the paralysis. It is supposedly due to infarction of the anterior portion of the corpus callosum which interrupts the commissural fibres linking the premotor and motor areas of the major to the minor hemisphere. This portion of the corpus callosum has, however, been divided surgically on many occasions without producing dyspraxia, and it was pointed out by Akelaitis *et al.* (1942) that this condition occurred only if the medial aspect of the dominant frontal lobe was also damaged. Ethelberg, though not denying the existence of a true ideomotor dyspraxia, pointed out that in his patients the disability was simply a clumsiness, particularly when performing rapid and complex movements. He felt therefore that it should be regarded as an 'impairment of rapid

FIG. 2.—Lateral aspect of left cerebral hemisphere showing distribution of the cortical branches of the anterior, middle and posterior cerebral arteries. (de Jong, 1950, 'The Neurologic Examination,' Cassel & Co., London).

off at right angles to the main vessel and penetrate the anterior perforated substance. They are said not to anastomose with one another. They vary in number and are arranged in two sets—the medial striate and lateral striate groups. Together these supply the lentiform nucleus, the caudate nucleus, the posterior portion of the anterior limb, the genu and the anterior third of the posterior limb of the internal capsule. The 'artery of cerebral haemorrhage' so much emphasized by Charcot, is a member of the lateral striate group and is sometimes termed the lenticulo-striate artery.

Cortical branches. The following vessels may be distinguished, though two or more frequently arise from the main vessel by a common stem:—

Anterior temporal artery—supplies the anterior third of the superior and middle temporal gyri and the insula.

Orbitofrontal artery—supplies the inferomedial and inferolateral aspects of the frontal lobe.

Prerolandic artery—to the lower part of the pre-central gyrus and adjacent frontal cortex.

Rolandic artery—to the precentral and post-central gyri.

Anterior parietal artery—to the postcentral gyrus and the adjacent parietal cortex.

Posterior parietal artery—to the posterior portion of the parietal lobe and the supramarginal gyrus.

Artery of the angular gyrus—continues the line of the main vessel and supplies the angular gyrus and the adjacent parietal cortex.

Posterior temporal artery—to the posterior two-thirds of the lateral surface of the temporal lobe.

These vessels form anastomoses with branches of the anterior and posterior cerebral arteries.

There is no doubt that occlusion in the territory of the middle cerebral artery may occur without producing symptoms or signs. In other cases transient headache, giddiness, confusion and focal signs result but disappear rapidly and leave no sequelae. In a number of cases focal or generalized fits are seen at the onset.

Occlusion of the Main Stem of the Middle Cerebral Artery

Occlusion of the first part of the middle cerebral artery results in profound coma which frequently proves terminal. The thrombosis may commence in one of its branches and extend back to the main artery, so that progression of symptoms occurs over a period of hours or days. When adequate examination is possible, a severe contralateral total hemiplegia quickly becoming spastic, a hemi-

anaesthesia and a homonymous hemianopia can be demonstrated. If the major hemisphere is affected a severe global aphasia is also present.

Anosognosia, in which the paralyzed body half is ignored and even disowned, is often to be observed during the period of recovering consciousness when the minor hemisphere is the site of the lesion.

In survivors the residual disability is severe and contractures develop. Some retrogression of the sensory loss may occur.

Perforating Branches

The medial and lateral striate vessels are probably more subject to occlusion than any of the other cerebral arteries. Consciousness is usually retained. A contralateral spastic hemiplegia is constant, the weakness being equal in the two limbs or occasionally more obvious in the leg. The severity of the hemiplegia varies widely from case to case. Disturbances of sensation in the affected body half are occasionally present but are seldom severe. A hemianopia due to infarction of the optic radiation at its commencement is rare and is, in fact, more commonly the result of occlusion of the superficial branches of the middle cerebral artery. When the major hemisphere is affected, a dysphasia—expressive or less frequently global in type—is often found and is attributed to oedema of the overlying convolutions (Davison *et al.*, 1933). In most cases considerable recovery of speech function ensues.

Bilateral occlusion of the perforating branches of the middle cerebral artery is the commonest cause of pseudobulbar palsy.

Superficial Branches

Isolated occlusion of the individual superficial branches of the middle cerebral artery is relatively uncommon and it is more usual to find two or more affected, as might be expected from their collective mode of origin. To some—the anterior temporal artery for example—no syndrome can as yet be ascribed. The majority of these cases fall into one of two groups, those with involvement of the more anteriorly placed branches and those with involvement of the posterior branches. It is often unprofitable to attempt to analyze these cases further.

Anterior group. Occlusion of the anterior group of vessels gives rise to a contralateral hemiplegia affecting particularly the face and upper limb, the weakness in the latter being most marked distally. Frequently the hemiplegia remains flaccid. Some cortical sensory loss with astereognosis is also present in the upper limb. If the major hemisphere is involved, a dysphasia occurs which is usually expressive in type but may show mixed

features. It is commonly severe and enduring. severe irreversible dysphasia resulting from vascular occlusion is always highly suggestive of a lesion of the middle cerebral artery. The extent of recovery depends not only on the severity of the damage but on the degree of dominance of the major hemisphere and on the age of the patient. A lesion occurring before the age of five years seldom leads to a permanent dysphasia.

Occasionally if the paresis is slight, dyspractic phenomena of an ideomotor type can be detected but are short-lived.

Posterior group. The most striking feature due to occlusion of the posterior group of vessels is contralateral homonymous hemianopia. The field defect may at times be limited to the inferior quadrants. It is the result of a lesion of the optic radiation, for the superficial branches supply the subcortical white matter to a varying depth as well as the cortex itself. When the major hemisphere is affected the hemianopia is accompanied by bilateral ideomotor or ideational dyspraxia. The various disturbances of the body scheme discussed below under 'posterior parietal artery' are also likely to be present.

If the anterior parietal branch is also affected a contralateral hemiplegia of moderate degree again due to involvement of subcortical white matter—and some cortical sensory loss over the contralateral body half are also present.

It may be very difficult to distinguish between the effects of occlusion of the superficial and deeper branches of the middle cerebral artery. In the former the weakness is likely to be greater in the upper limb; particularly if it commences in the upper limb and spreads to the lower, a cortical origin is likely. If the hemiplegia remains persistently flaccid the occlusion is almost certainly superficial. Marked sensory changes and the presence of a field defect also favour a superficial occlusion.

The results of occlusion of the individual superficial branches of the middle cerebral artery will now be discussed, but it must be emphasized that some of the descriptions are based on rather meagre details.

Orbitofrontal artery. On the major side an expressive dysphasia without paresis may follow occlusion of this branch. Occasionally weakness of the opposite face and tongue may be demonstrable but this is apparent only, and is, in fact, dyspractic in nature. Such a patient may be unable to protrude his tongue or whistle to order. If the lesion affects the minor hemisphere no localizing signs result.

Pterolentic artery. Occlusion of this vessel gives rise to contralateral weakness of the lower face and tongue. The limbs as a rule are spared completely.

FIG. 3.—The arteries at the base of the brain.

AC Anterior cerebral artery.
aca Anterior chorioidal artery.
a.c.l.p. Anterior clinoid process.
acm Anterior communicating artery.
B Basilar artery.
IC Internal carotid artery.
MC Middle cerebral artery.
PC Posterior cerebral artery.

pca Posterior communicating artery.
sba Basal branches of anterior cerebral artery.
sbm Perforating branches of middle cerebral artery.
SC Superior cerebellar artery.
VA Vertebral artery.
(After Buchanan, 1948, 'Functional Neuro-anatomy,' Henry Kimpton, London.)

but weakness limited to the hand may sometimes be seen. An expressive dysphasia, varying greatly in intensity from case to case, is also present when the major hemisphere is affected.

Rolandic artery. Lesions due to isolated occlusion of this artery are rare because of the richness of the collateral irrigation of its territory. Paresis of the contralateral upper limb—and to a lesser extent of the face—develops, together with some degree of cortical sensory loss in the same distribution. A slight expressive dysphasia occurs with lesions of the major hemisphere.

Anterior parietal artery. Cortical sensory loss in the contralateral upper limb and face is the most striking feature following occlusion of this branch.

It is accompanied by astereognosis and a sensory ataxia. Pseudoathetotic movements sometimes occur in the affected limb when the eyes are closed. Signs of a slight pyramidal tract disturbance and a mild degree of dysphasia may be present.

Posterior parietal artery and the artery of the angular gyrus. Occlusion of these vessels on either side may give rise to a contralateral homonymous or inferior quadrantic hemianopia. Disturbances of the body scheme are found, particularly when the major hemisphere is affected. The precise form of the disturbance varies greatly from patient to patient but the most common finding is a Gerstmann's syndrome comprising dysgraphia, dyscalculia, finger agnosia and confusion of

laterality (right-left disorientation). These phenomena may occur independently of one another. Other agnostic features may be present—a visual agnosia or an agnosia for colours—and the autotopagnosia, of which the finger agnosia is an expression, may be more profound so that the patient is unable to recognize any part of his body (Nielson, 1946). A bilateral ideational or ideomotor dyspraxia is also frequently present. A dysphasia, predominantly receptive in character, usually occurs and a dyslexia is its most prominent feature.

(See Foix and Lévy, 1927; Tichy, 1949.)

The Anterior Chorioidal Artery

The anterior chorioidal artery arises from the internal carotid artery near its termination. It runs backwards in close relationship with the optic tract between the uncus and the cerebral peduncle to the lower end of the chorioidal fissure. It supplies the uncus and the optic tract, the middle third of the cerebral peduncle, most of the globus pallidus, the posterior two-thirds of the inferior half of the posterior limb of the internal capsule, the anterior half of the subthalamic nucleus, the red nucleus and the lateral aspect of the external geniculate body.

Proven examples of occlusion of the anterior chorioidal artery are few. Loss of consciousness is unusual. A contralateral hemiplegia is constant and either affects the entire body half equally or the lower limb predominantly. In the former case it is probable that the lesion of the cerebral peduncle is pre-eminent whilst in the latter, damage to the internal capsule is mainly responsible. Contralateral impairment of all forms of sensation and a homonymous hemianopia are also present. When the hemianopia is complete it is said that the lesion responsible affects the internal capsule, while if it be a superior quadrantic defect, infarction of the lateral geniculate body is indicated (Abbie, 1933). Although considerable destruction of the caudate nucleus may occur, this produces no characteristic signs.

The combination of a hemiplegia, hemianaesthesia and hemianopia also occurs following occlusion of the trunk of the middle cerebral artery or a haemorrhage into the internal capsule. In such cases the result is far more grave. Deep coma will be present and the hemiplegia is very much more severe than that following occlusion of the anterior chorioidal artery.

(See also Steegman and Roberts, 1935.)

Posterior Communicating Artery

This vessel arises from the internal carotid artery a short distance before its termination and passes backwards to join the posterior cerebral

artery. Branches are given off to the optic chiasma, the tuber cinereum, the medial aspect of the thalamus, the anterior portion of the posterior limb of the internal capsule and the rostral third of the pes pedunculi.

The syndromes resulting from occlusion of this vessel have not been defined. Brock (1937) has reported that a contralateral mimetic palsy of the face may result.

The anterior lobe of the pituitary is supplied principally by the superior hypophyseal vessels which arise directly from the internal carotid arteries. Some twigs of supply come from the posterior communicating arteries. Occlusion of these vessels may give rise to Simmond's disease.

Posterior Cerebral Artery

Each posterior cerebral artery winds backwards round the cerebral peduncle and passes deep to the splenium of the corpus callosum to reach the medial border of the inferior surface of the temporal lobe where it divides into terminal or cortical branches. Developmentally, the posterior cerebral artery beyond its junction with the posterior communicating artery is derived from the internal carotid artery (Williams, 1936).

Branches: Paramedian group (retromamillary branches). There are about 12 of these. The anterior vessels—the thalamo-perforating or medial ganglionic branches—enter the posterior perforated substance and terminate in the medial portion of the thalamus; they also supply the mamillary body. The posterior vessels—peduncular branches—supply the cerebral peduncle, the substantia nigra, the third nerve nucleus and the posterior half of the subthalamic nucleus. There is little anastomosis between the vessels of the two sides.

Quadrigeminal artery. This is a small vessel which winds round the midbrain and supplies its lateral aspect and the superior and inferior colliculi.

Posterior chorioidal artery. This vessel winds round the midbrain and after running over the superior aspect of the thalamus, ends in the chorioid plexus of the third ventricle. Six or seven branches are given off to the cerebral peduncle and other twigs supply the superior colliculus and the internal and external geniculate bodies.

Geniculate arteries (thalamogeniculate arteries). These penetrate the external geniculate body supplying it and the superior colliculus before entering the thalamus. The territory of supply includes the posterior half of the thalamus and the posterior portion of the posterior limb of the internal capsule.

Cortical branches: Anterior temporal, posterior temporal, calcarine and parieto-occipital.

As might be expected from its distribution, there are many syndromes associated with lesions of the posterior cerebral artery and its branches. When there is infarction of the cerebral hemisphere, focal or generalized convulsions may occur at the onset and sometimes recur. Visual auras are common.

Occlusion of the Main Trunk of the Posterior Cerebral Artery

The results of occlusion of the main stem of the posterior cerebral artery depend largely on the adequacy of the posterior communicating artery. No ill effects may follow if this latter vessel provides an adequate collateral circulation. When infarction does occur, consciousness is usually retained though occasionally persistent somnolent states result (Bacaloglu *et al.*, 1934) possibly due to damage to the reticular substance in the tegmentum of the midbrain. A contralateral hemiplegia is present but is moderate in degree. It is due to infarction of the cerebral peduncle. The most important and characteristic finding is a contralateral 'thalamic syndrome'—first described by Déjerine and Egger (1903) and again with pathological studies by Déjerine and Roussy (1906). Paroxysmal or constant pain is present in the affected body half including the face. As a rule it is severe and often has a burning quality. It usually occurs spontaneously but may be provoked by heat, cold, movement or deep pressure. Pricking or icy cold sensations may be experienced. Although commonly diffuse, the pain may sometimes be sufficiently localized to simulate abdominal disease. Rectal or vesical pain associated with tenesmus or strangury is not uncommon.

The objective sensory findings vary from case to case, and in general their severity is inversely proportional to the amount of pain. Typically there is impairment of the sense of passive movement, of deep sensation and vibration sense. Pain, touch and temperature sensations on casual examination appear to be unaffected and the occurrence of hyperaesthesia or hyperpathia may even suggest an increased sensitivity. More careful examination reveals that the threshold value of the stimulus is raised and its localization is poor. Sometimes the response to pleasurable stimuli is also exaggerated and such things as music which produce a general affective tone may evoke a reaction on the altered side so intensely pleasurable as to be unbearable. One of Head's patients became somewhat amorous after his ictus and remarked that his affected side had become more responsive and more desirous of solace. The essential lesion was shown by Roussy to affect the posterior third of the lateral nucleus of the thalamus.

The extremities on the affected side often become oedematous and cyanosed while trophic changes may develop in the finger nails. These changes, though associated with the hyperpathia, are not necessarily of equivalent intensity.

Because of the impaired joint sense the affected limbs show a sensory ataxia and astereognosis. The ataxia is accentuated if the superior cerebellar peduncle is damaged and contralateral cerebellar signs may be demonstrable.

Various features due to destruction of extrapyramidal structures or their connections—notably the substantia nigra, the red nucleus and the subthalamic nucleus—are frequently present. The limbs may show a plastic rigidity and the arm is held flexed at the elbow and a little flexed at the wrist, with fingers extended or hyperextended (thalamic hand). When walking, the arm may be held extended and internally rotated with the hand behind the back. Movements of a choreic or athetoid nature may be present and are accentuated by emotion and often by voluntary movements of the normal limb. In the latter case mirror movements may be seen. Tremor occurs rarely (Langworthy and Fox, 1937).

Other signs may result from midbrain damage. An ipsilateral third nerve palsy or a defect of superior conjugate ocular movement (Parinaud's syndrome) sometimes occurs. An ipsilateral Horner's syndrome is more common than either.

Lastly, there may be evidence of damage to the occipital and posterior parietal and temporal areas of cortex. A contralateral homonymous hemianopia which is usually complete occurs but sometimes there is apparent sparing of the maculae. There is no evidence to support the suggestion that the maculae are represented bilaterally, nor is the view tenable that the macular areas are supplied by the middle cerebral arteries. It is more probable that so-called macular sparing is an artefact due to shifting fixation by the patient.

If the lesion affects the major hemisphere a dysphasia is also present. In general this is receptive in type but it is rarely severe. Often reading is particularly affected so that the defect approaches closely to a pure alexia. Visual agnosia may also be present and very occasionally there is visual autotopagnosia, which is more often partial than complete.

The paramedian branches. Corresponding to the short circumferential branches at lower levels are twigs given off to the lateral aspect of the midbrain by the various branches of the posterior cerebral artery. They are not so clearly defined as the corresponding vessels in the lower brain stem and as yet it is not possible to ascribe any particular syndromes to their occlusion. It is probable that they play a part in some of the syndromes des-