Secondly, an example of the collateral circulation, which is known to exist between the external and internal carotid arteries. Fig. 2 shows contrast medium passing from the maxillary artery to the orbit, and from there the ophthalmic artery and carotid siphon are outlined. A by-pass round the blocked internal carotid artery can thus exist or become established.

The third example (Fig. 3 *a, b*) is of a block in the carotid siphon, in this case just beyond the origin of the anterior choroidal artery. The ophthalmic and posterior cerebral arteries are also outlined.

Emphasis was also laid on the importance of obtaining films both of the stump of the blocked artery and also of the needle point, before making a confident diagnosis of carotid thrombosis. Confusion can otherwise easily arise.

Some Aspects of the Pathology of Thrombosis of the Internal Carotid Artery

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Thrombosis of the internal carotid artery is more common than is usually expected, for Hultquist (1942), investigating the carotid arterial system in 1,300 routine autopsies, found a thrombosed internal carotid artery in 4·4% of the cases.

In the 4 cases under consideration the thrombosed artery was the site of marked atheromatous change as were the other arteries in the body.

Atherosclerotic deposits are often found in the internal carotid artery (Dow, 1925; Keele, 1933) and they are usually situated in the region of the carotid sinus and in the carotid siphon which are also the most frequent sites for thrombosis (Hultquist, 1942).

In all the present cases the initial site for thrombus formation was in the region of the carotid sinus. In one there was also old thrombus in the carotid siphon.

The frequency of demonstrable cerebral complications varies with the site and extent of thrombus formation. With thrombosis at the proximal end of the artery Hultquist found cerebral lesions in one-third of the cases. With thrombosis at the more distal level the incidence was much higher.

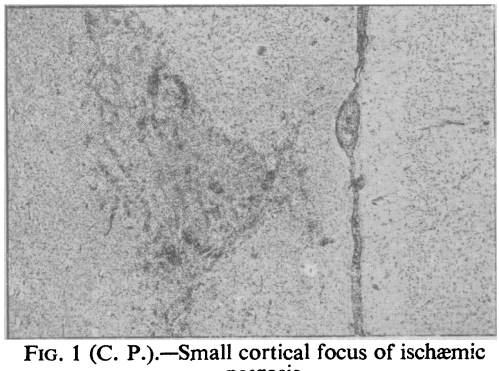

Fig. 1 (C. P.).—Small cortical focus of ischæmic necrosis.

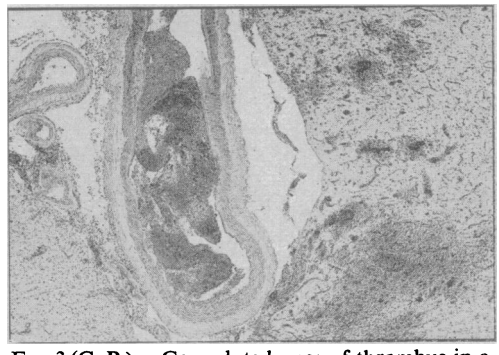

Fig. 3 (C. P.).—Convoluted mass of thrombus in a pial artery. Adjacent brain shows recent ischæmic necrosis.

In the present cases the cerebral changes ranged from ischæmic damage to the cortical neurones to foci of ischæmic necrosis of varying age and size.

In one patient (F. S., No. 1484) the internal carotid artery was partially occluded at its lower end by old organized thrombus and beyond this by more recent thrombus which extended as far as the origin of the middle cerebral artery. In the cerebrum there was massive old ischæmic necrosis in the region of distribution of this vessel. The heart showed an old myocardial infarct with an adherent laminated thrombus in the left ventricle. Emboli from the left ventricle may, therefore, have been wholly or partly responsible for both the carotid and the cerebral lesions.

In 2 cases (S. H., No. 37477 and C. P., No. 24452) thrombosis of the internal carotid artery and discrete foci of ischæmic necrosis in the brain were the principal autopsy findings (Fig. 1). The cerebral damage was almost entirely confined to the distribution of the middle cerebral artery on the same side as the thrombosed internal carotid artery (Fig. 2).

In the second of the 2 cases, close to several of the more recent lesions small cerebral arteries were partially blocked by convoluted masses of recent thrombus (Fig. 3). The walls of these arteries showed no evidence of arterial disease.

Other small arteries in this hemisphere were occluded by older organized thrombi.

The appearance of these thrombi was compatible with their being of embolic origin but it is not possible on histological grounds to exclude their being primary thrombi laid down at these points.

It is suggested that masses of thrombus material may have been dislodged from the thrombus in the internal carotid artery and lodged in the smaller cerebral vessels.

The evidence supporting this proposition is, firstly, that the appearance of these masses is compatible with their being of embolic origin; secondly the distribution of the lesions is in accord with the known distribution of emboli entering the carotid circulation; thirdly the

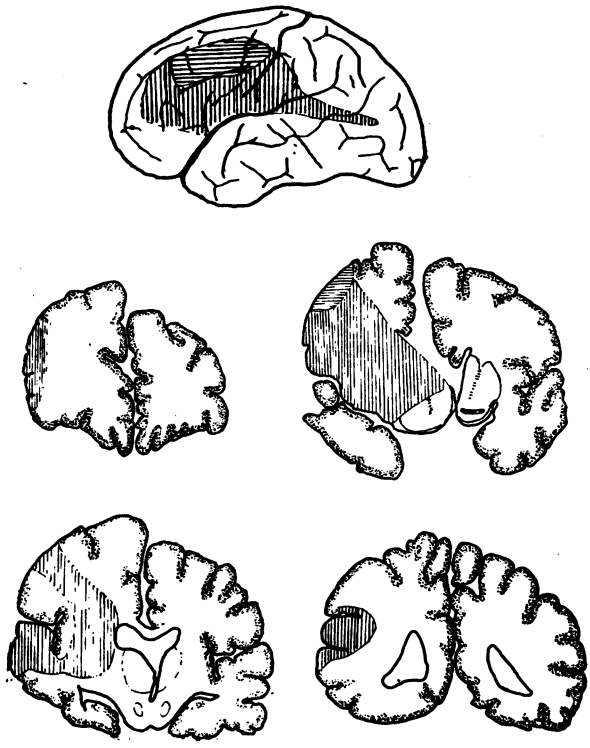

FIG. 2 (C. P.).—Diagram of the extent of the cerebral damage. Horizontal hatching represents recent and vertical hatching older regions of ischæmic necrosis.

discrete focal nature of the lesions with varying degrees of repair and organization; and lastly the episodic clinical course of these cases.

In the fourth case (E. B., No. 36501) there had been a sudden onset of cerebral symptoms and only a short interval between the onset and death. The internal carotid artery was occluded by recent thrombus and in addition the circle of Willis was very atherosclerotic. There was an extensive ischæmic lesion in the region supplied by the middle cerebral artery on the same side as the thrombosed internal carotid artery but, in the sections examined, occluded cerebral vessels were not found. It appeared that the circle of Willis was unable to provide an adequate collateral circulation and accordingly the territory of the middle cerebral artery suffered from an acute ischæmia.

Summary.—It is believed that atherosclerosis is an important factor in the pathogenesis of thrombosis of the internal carotid artery. Emboli arising from the thrombus and lodging in smaller cerebral arteries are a factor in the production of the cerebral complications of some of these cases.

My thanks are due to Sir Charles Symonds, Dr. E. A. Carmichael, Dr. Purdon Martin, Dr. S. P. Meadows and the Medical Committee of the National Hospital, Queen Square, for permission to utilize the case records, and to Dr. W. Blackwood for placing the pathological material at my disposal.

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Cerebral Pathology following Carotid Spasm

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Vasospasm is not a condition which persists after death and is not therefore a fact observable by morbid anatomists. Experimental observations in animals (Kinmonth, 1952) and the experiences of surgeons suggest that prolonged spasm of arteries is not easily produced except by direct trauma to the vessel wall.

An exception to this appears to be the well-known work of Villaret and Cachera (1939) who observed, through cranial windows, the lodgment of solid emboli in vessels of the pia-arachnoid. They noted brisk contractions of the walls not only of the vessel occluded but also of neighbouring arteries. In a few such instances these contractions might be prolonged. Such a phenomenon has often been suggested as the cause of the temporary nature of the neurological disorders which may occur in cases of embolization where the cerebral arteries are otherwise healthy.

A feature of the lesions of atherosclerosis is the presence of thin-walled capillaries and sinusoids in the atheromatous plaques and in the wall of the artery beneath the plaques. Occasionally these rupture, allowing hæmorrhage into the muscular wall of the vessel. I believe such a naturally occurring injury may produce arterial spasm, which, if short-lived, will give rise to fleeting neurological signs and, if prolonged, offers an explanation of infarcted cerebral tissues which at autopsy are seen to be supplied by arteries not thrombosed and only slightly occluded by atheroma.

The damage caused to the carotid artery by angiographic needling which may be insignificant in a healthy vessel may cause a hæmatoma and even extensive dissection of the wall in atheromatous arteries predisposed to such a lesion. Any such lesion may stimulate prolonged spasm of the vessel.

Death within a few hours of angiography sometimes occurs, especially in patients suffering from atherosclerosis, but in these cases time does not allow development of the picture of ischæmic damage which may, in any case, be confused by the primary disease.

A case which I think is significant is that of a man aged 63 who had suffered for two years principally from dysphasia. Left-sided percutaneous carotid angiography was performed apparently satisfactorily, though the films showed that little contrast medium had passed up the internal carotid tree. During the examination he became unconscious and remained so until his death eighteen days later. At autopsy there was slight evidence of bruising of the common carotid artery. Both the common and the internal carotid arteries showed plaques of atheroma but nowhere was there occlusion or any thrombosis. Examination of the brain showed a widespread atherosclerosis of a moderate degree but no obstructed vessels could be found. There

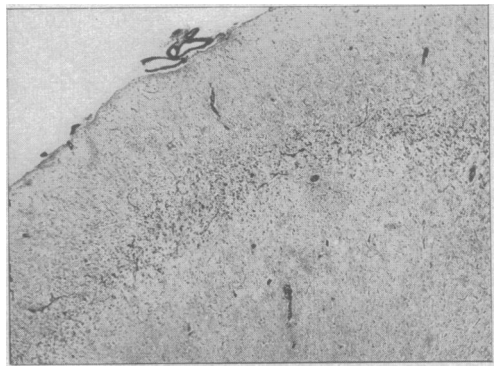

Fig. 1.—Cerebral cortex showing laminar necrosis.