THROMBOSIS OF THE INTERNAL CAROTID ARTERY

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Seen in long-term perspective the discovery of X-rays and their use as a diagnostic agent must surely rank as one of the major advances in medicine. Though of less immediate practical benefit than were either anaesthesia or antiseptics, the scientific importance was greater and even now, after more than fifty years, new techniques are still being evolved with resulting increases in knowledge.

Among the many sub-divisions of radiology the introduction of contrast radiography in exploring the vascular pattern of organs otherwise inaccessible, has been one of the more recent and most fruitful developments. A striking instance is the use of percutaneous carotid angiography with the consequent recognition of thrombosis of the internal carotid artery. No doubt from time to time an inquisitive morbid anatomist had inspected the carotid arteries in the neck and found one or other occluded, but the fact passed unrecorded and was doubtless regarded as nothing more than a pathological curiosity. It is interesting, however, to recall that Ramsay Hunt (1914), in discussing cerebral vascular disease referred to the possibility that some cases of hemiplegia might be the result of thrombosis in the carotid vessels and suggested that it would be wise for the clinician to examine the state of the carotid pulsation in all cases of sudden hemiplegia. His plea was either ignored or soon forgotten and we hear no more of the matter until Moniz and his associates (1937), rediscovered thrombosis of the internal carotid artery in the course of their pioneer studies in cerebral angiography. The word 'rediscovered' scarcely does justice, however, to their findings, for, in reporting four cases of carotid occlusion, they showed at once that the condition was one which could be recognised during life and which in all probability was by no means excessively rare. Since then a good deal has been learned confirming their pioneer observations and at the present day the general physician, just as much as the neurologist, has the possibility of carotid occlusion in mind whenever he is confronted with a case of sudden hemiplegia.

Valuable pathological evidence was brought

forward by Hultqvist (1942). He reported on a series of 3,500 autopsies in which he examined the carotid vessels and evidence of occlusion was found in no less than 91 instances. The majority occurred in the sixth and seventh decades and the condition was about twice as common in men as in women. These converging lines, radiological and pathological, indicated that occlusion in the carotid vessels was relatively common and that with an adequate radiological technique it could be clearly demonstrated. Clinical recognition during life is, however, another matter and even with the rapid gain in experience over the past few years the diagnosis still remains conjectural in the majority of cases. A large number of case reports have appeared in recent years, mostly of quite small series of cases. Special mention must, however, be made of the admirable review by Cloake (1954).

Before reviewing the symptomatology it will be advisable to say something of the pathology of the condition. It is now accepted that the great majority, at least two-thirds, are a primary thrombosis beginning in the carotid sinus, some 0.5 cm. distal to the origin of the internal carotid artery (Fig. 1). A very small number originate in the common carotid itself and a few at higher levels of the internal carotid, in some cases even as high as the intracranial portion of the vessel. Why the carotid sinus should be the site of predilection is obscure though Hultqvist (q.v.) showed that this region shares with the coronary vessels and the abdominal aorta an unusual tendency to develop atheroma and to develop it prematurely. In some cases no doubt the primary lesion in the vessel is an embolus though the relative incidence of this condition is unknown and Hultqvist himself recognised that even at autopsy it was sometimes difficult to determine this point with certainty.

Symptomatology

From the writer's personal experience and from a survey of the literature it would seem that in rather less than half of the cases the disease shows itself by a sudden and devastating hemiplegia.

FIG. 1.—Radiograph showing a very typical example of occlusion of the internal carotid artery.

Tracing of Fig. 1.

This form of the disease is the most immediately menacing for full half of these patients may be expected to die within a few days and the remainder are likely to be left with severe and disabling hemiplegia. In a smaller number of cases the paresis is less profound and often affects the limbs unequally, the arm being more severely affected than the leg. In a right-handed person some degree of dysphasia is to be expected. Hemianopia, either complete or incomplete is, on the whole, rare. On the whole motor symptoms greatly predominate over sensory. An interesting point is that in a fair proportion of cases the onset of the attack is heralded or even preceded by hours or days, by a certain degree of headache, localized about the temple on the affected side or more diffusely over the side of the head. It must be emphasised, however, that the hemiplegia of itself does not show any striking characteristics or anything which would differentiate it from a classical hemiplegia resulting from occlusion of the middle cerebral artery.

A fairly common mode of presentation is, however, by remittent attacks of paresis, either hemiparetic or of a more limited range, and in some cases these have extended over a period of years. Usually there has been an abrupt onset of paresis with or without sensory disturbances, lasting a few days or weeks and followed by a considerable degree of recovery. In a very small proportion of cases the disease has shown itself by a more gradually developing hemiplegia and in two of the author's cases there was a striking and predominant sensory disturbance of the arm and hand, slowly increasing over a period of weeks, and presenting a picture highly suggestive of a cerebral tumour.

It will be seen that there is no one clinical picture characteristic of thrombosis of the internal carotid artery. At one extreme the picture is that of an abrupt and devastating hemiplegia differing in no way from classical hemiplegia; at the other a slowly increasing weakness with or without sensory loss, involving one side of the body or even one limb, and simulating closely the picture of an invasive cerebral tumour. Nevertheless there is one symptom which deserves close attention for though on the whole rare, it is the sole symptom which when present enables the clinical diagnosis to be made with confidence. This is the occurrence of transitory loss of vision in the eye on the side of the thrombosed vessel. It is not a common symptom even when close enquiry is made, though it must be admitted that in some cases its appearance may have been entirely forgotten by the patient. These attacks of transitory blindness or clouding of vision may ante-date the onset of the more dramatic hemiparetic phenomena by weeks

FIG. 2.—Radiograph from a case of occlusion of the internal carotid artery showing the collateral circulation through the ophthalmic artery and filling of the terminal part of the internal carotid. There is some filling of the middle cerebral and very good filling of the posterior cerebral artery.

or even by years. Its importance lies in the fact that it is indicative of a transitory ischaemia of the retina, and so far as our knowledge extends at present the only common cause of such a transitory and repeated retinal ischaemia is obstruction in the ipsilateral internal carotid artery. It is very interesting to observe that permanent loss or impairment of vision in the ipsilateral eye (that is, the eye on the side of the thrombosed vessel), is extremely rare. Indeed at first it seemed as if permanent blindness never occurred, but larger experience has shown that very occasionally the degree of ischaemia is such that permanent retinal degeneration and optic atrophy ensue. That the circulation to the eye so frequently escapes permanent damage must depend on the very adequate collateral circulation which is established by way of the external carotid, the facial artery and the various ocular branches of the ophthalmic artery (Fig. 2).

Tracing of Fig. 2.

The early reports of thrombosis of the internal carotid artery contained reference to diminution of the carotid pulse on the affected side, and it was suggested that clinical evidence of the occlusion might be afforded by noting a diminution or obliteration of the pulse, as indeed Ramsay Hunt had originally suggested. Whilst it is true that this may have been a valid observation it can be stated quite confidently that this procedure is of very little value and a moment's reflection will show why this is so. The internal and external carotid arteries lie in such close proximity in the neck that occlusion of one can scarcely affect the total pulsation. In those few cases where a definite diminution of the pulse has been found it is indeed probable that the common carotid artery was occluded.

Diagnosis

Not much remains to be said under this heading for the special features of the disease have been outlined in discussing the symptomatology. It will be apparent that in the great majority the clinical diagnosis is conjectural. When attacks of uni-ocular amblyopia have preceded the hemiplegia the diagnosis may be made with a high degree of confidence but in other cases, and they constitute the great majority, ultimate confirmation depends on angiography with demonstration of the actual occlusion. It may be mentioned at this stage that other ancillary modes of investigation have not yielded information of decisive value. No characteristic picture is afforded by the electroencephalogram and investigations of the cerebrospinal fluid, both as regards hydrodynamics and chemistry do not show any constant deviation from normal.

It is not within the scope of this paper to deal in any detail with the purely radiological aspects of the subject. Provided the radiological technique is adequate and correctly carried out the method is almost infallible, though now and again even a skilled and experienced radiologist may run into difficulties. Sometimes instead of entering the lumen the contrast medium becomes arrested in the media or in the sub-intimal space, producing what is in effect a dissecting aneurysm. In such cases the appearances may be suggestive of an obliteration of the lumen and consequently an incorrect diagnosis of thrombosis made. An experienced radiologist, however, will seldom be in any doubt as to the true state of affairs. The appearances which may result from such a faulty injection of the contrast medium are well shown in Fig. 3. At this point it may be well to give a word of caution against too prolonged attempts at inserting the needle into the carotid artery, or what is equally,

FIG. 3.—The contrast medium has passed into the sub-intimal space and is elevating the intima.

if not more, important, repeating the attempt within a few days. The writer has seen several cases where a repeated attempt at angiography, carried out about a week after the first, was followed by sudden and severe extension of cerebral damage. Whether this is brought about by dislodgement of clot from the site of the initial puncture with consequent embolism, or by intense cerebral ischaemia, the result of cerebral arterial spasm, is by no means clear. The practical implication is, however, plain and if angiography has failed for technical reasons then a period of at least ten days should be allowed to elapse before a further attempt is made.

Treatment

Up to the present the great majority of cases have occurred in middle-aged and elderly patients and this, coupled with the frequency of evidence of more generalized cardiovascular disease has limited treatment to such conservative measures as would be proper to a cerebral thrombosis of the usual type. In other words treatment has consisted in general nursing care, in prevention of pulmonary and genito-urinary complications and in early physiotherapy and mobilization. Indeed in those cases, and they form the largest group, which present with an abrupt hemiplegia, it is difficult to see what else would avail, for cerebral softening must have already occurred with, as an inevitable consequence, some degree of permanent disability. Where the symptoms have been relatively slight or remittent the neuro-surgeons

have been tempted to employ more heroic measures. Stellate ganglionectomy and other measures designed to denervate the carotid vessels have been tried, but the general consensus of opinion remains sceptical. In a recent symposium on cerebral vascular disease, Bucy (1955) has expressed the view that the value of such measures remains quite unproved. Eastcott, Pickering and Rob (1954), have recently reported some success following resection of the thrombosed portion of the internal carotid artery but it is too early as yet to form any final opinion of the value of this method.

Discussion

As so often happens the solution of one problem or even the enlargement of one field of knowledge in a particular direction serves only to present further difficulties and problems and to pose further questions. In considering internal carotid thrombosis the fundamental question is why such an occlusion should produce symptoms at all for, as is well known, the carotid vessels may be tied by the surgeon with impunity. The incidence of any permanent degree of hemiplegia following a carotid ligation is certainly not greater than 5 per cent. It is clear then that in thrombosis of the internal carotid artery something more than the mere mechanical effect of occlusion has to be reckoned with. Consideration of this paradox should put us on our guard against the too ready acceptance of any purely mechanistic explanation. Preoccupation with the occlusion of the internal carotid artery must not lead us to ignore the rest of the cerebral circulation and, in particular, the state of the carotid vessels on the opposite side and of the Circle of Willis. It is possible that cerebral softening following on occlusion of the internal carotid artery occurs when there is insufficiency of the circulation through the apparently intact carotid vessels of the opposite side. Some confirmation of this hypothesis is afforded by the observation which has been made on several occasions of ischaemic softenings in both cerebral hemispheres in what was apparently a strictly unilateral internal carotid thrombosis. In such instances it seems reasonable to suppose that the relatively intact carotid vessels were unable to maintain an adequate circulation not only to the opposite cerebral hemisphere via the Circle of Willis but to their own more particular territory.

In concluding this article it is pertinent to say something of the general indications for the

employment of angiography and more particularly in cases of supposed or suspected thrombosis of the internal carotid artery. Since in any given case it may be impossible to decide whether a hemiplegia of vascular origin is the result of occlusion within the middle cerebral artery or the internal carotid artery it would seem on theoretical grounds that angiography might well be employed in all. Nevertheless despite the relative safety of the procedure it is doubtful if it should be employed indiscriminately, for there is no doubt that every now and then, even with the most careful technique, unfortunate accidents happen. There is all the greater danger of such accidents when dealing with elderly patients suffering from advanced cardiovascular degeneration. In the author's view the method should not be used merely to satisfy curiosity but should be reserved for those cases where real doubt exists in the diagnosis and, above all, where there is the possibility of some other condition possibly amenable to radical treatment. In this Centre we have more particularly tended to use angiography in those cases where the possibility of a cerebral tumour arose. We have been loath to submit patients to angiography where from the history it seemed perfectly plain that the disease was a vascular accident, always excepting, of course, those cases where the presence of intracranial haemorrhage, pain and the like suggested the possibility of an intracranial aneurysm. It must not be forgotten that hemiplegia of extremely sudden onset and without prior symptoms and without the occurrence of headache or alteration of consciousness is by no means a great rarity in young adults, and there is no doubt that in some of these cases the hemiplegia is the consequence of a precocious primary internal carotid occlusion. Such attacks, therefore, in young people certainly merit the most careful investigation and it would seem that cerebral angiography should form a necessary part in their investigation.

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