Strokes—What is Wanted of Radiology?

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When considering the radiological investigation of strokes, first concern must be for transient ischaemic attacks (TIAs). These are defined as focal disturbances of neurological function of less than 24 hours duration. Once a completed stroke has occurred, brain damage has been done. To prevent this by detecting, investigating and treating TIAs must be the physician’s primary endeavour.

Not all TIAs require radiological study. Those associated with anaemia, polycythaemia, hypotensive episodes, hypertensive crises and episodic cardiac dysrhythmia are clearly not candidates for radiological investigation. But these causes together account for the minority of TIAs; radiology is concerned with the majority.

CAROTID TRANSIENT ISCHAEMIC ATTACKS

It is important to distinguish between carotid and vertebrobasilar TIAs, for the risk of a completed stroke is ten times greater after carotid TIAs than after vertebrobasilar TIAs (Whisnant et al., 1972). When a patient presents with carotid TIAs, the physician, having eliminated the causes listed above, should next decide whether or not the patient is hypertensive. For this purpose a diastolic pressure of 110 mm Hg or more is a useful dividing line. Patients with this degree of hypertension should not, in general, be subjected to angiography, partly because of increased risk of morbidity from the procedure and partly because the chance of finding occlusive-stenotic disease is not high (Prineas and Marshall, 1966). Exception must be made if evidence such as a carotid bruit indicates that a stenosis is present.

The patient without hypertension, whether or not he has a bruit, should be referred for angiography. The great majority of TIAs in this group are due to emboli; a small minority are due to reduction of blood-flow. In our experience, relevant lesions will be found in 50 per cent of cases and 20 per cent will come to surgery (Marshall, 1971). The questions to the radiologist are—
1. Has the patient a lesion relevant to his symptoms which could be a source of emboli or reduce flow?
2. If so, is it surgically accessible?

Emboli may arise from vessels within the cranium, especially from the intracavernous segment of the internal carotid, as well as from vessels in the neck and thorax, so that search for a potential site should include selective pictures of the intracranial arteries. In the case of TIAs it is also comforting if films show specific changes of occlusion such as small branch obstruction and collateral circulation or the non-specific changes of capillary blush and localised early venous filling which clinch the clinical diagnosis. The commonest surgically accessible sites of atheroma are in the neck near the origin of the internal carotid artery. It is important to identify small plaques here, and to assess their surfaces for irregularity due to ulceration or accretion of thrombus which points to their significance as a source of embolism; the significance of smoothly surfaced plaques, even if they are causing some narrowing, is much less certain. Such changes can only be consistently shown or most reliably excluded by selective angiography with profile views of the lesions, which involves filming in at least two planes at right angles (Fig. 1).

How selective angiography is to be performed depends on the specific problem to be solved and on factors such as the apparatus available and the skill of the radiologist. It is the latter that determines the morbidity. In expert hands, direct puncture of the central segment of the common carotid artery, which is not commonly affected by atheroma, is both simple and safe, carrying a serious morbidity of less than 0.5 per cent (Allen et al., 1965; Dayon and Ramée, 1972) and is an excellent method despite the fact that it does not show the proximal segment of the common carotid.

Selective catheterisation has advantages in many cases of carotid TIAs. It is performed from a femoral artery as this allows direct access to all the major vessels and is itself a large vessel, not prone to spasm like the brachial, or close to large nerve trunks like the axillary. It is rare to have trouble with passage of the catheter due to atheroma in the iliac arteries or aorta, but if there is clinical evidence of this it may be wise to consider other approaches. In skilled hands the serious morbidity, due usually to cerebral emboli, has been reduced to under 1.0 per cent (Meaney et al., 1973).

In thrombo-embolic and occlusive vascular disease selective catheterisation is preceded by arch angiography to assess the proximal part of the artery for ulceration, local thrombus, or gross stenosis, which would make it unwise to proceed by that method. The arch catheter is replaced by one with similar proximal size but with a long terminal narrow segment for the selective study.
Arch angiography demonstrates the thoracic and cervical segments of the cerebral vessels by serial filming after rapid injection of a concentrated bolus of contrast agent into the ascending aorta. It is the best way of showing the origins of the branches of the aorta and the only way of diagnosing occlusions at this site.

**VERTEBROBASILAR TRANSIENT ISCHAEMIC ATTACKS**

The approach to vertebrobasilar TIAs differs considerably from that to carotid TIAs. There are two reasons for this: firstly, the risk of progression to a completed stroke is very much less, particularly when the patient is experiencing only ‘nuclear’ symptoms such as diplopia, vertigo, facial paraesthesiae, dysarthria and drop attacks (Marshall, 1964); secondly, the opportunity for effective surgical intervention is restricted.

The general contra-indications to carotid angiography already listed also apply to the vertebrobasilar territory. In particular, hypertension should be seen as an even stronger contra-indication because hypertensive patients who
present with vertebrobasilar TIAs run an appreciable risk of permanent ischaemic brain stem damage following angiography.

There are, however, two groups of vertebrobasilar TIAs in which angiography is indicated. The first is when a supraclavicular bruit, inequality of the radial pulses, and a difference in brachial blood pressures of 15 mm Hg or more suggest a subclavian stenosis, possibly causing a subclavian steal. Here the physician asks the radiologist two questions—

1. Is there a subclavian stenosis?
2. If there is, what is the haemodynamic situation in relation to it?

Arch angiography is the ideal method of demonstrating the haemodynamics of the brachial-cephalic circulation. It will show delayed or absent flow in any of the vessels and the presence of collaterals contributing to, or stealing from, the cerebral circulation. The demonstration of retrograde flow in the vertebral towards the arm indicates steal of blood from the brain. It should be borne in mind that balanced flow or delayed antero-grade flow may be reversed when the arm is exercised, a situation that could be proved by repeating the angiogram while exercising the arm or after inducing reactive hyperaemia in it.

All anatomical variations in the major arteries demonstrated by this method may assume importance in an individual case, but the most frequently significant is a left vertebral originating from the arch of the aorta which occurs in at least 5 per cent of all patients and precludes left arm steal.

The second situation demanding radiological help is when the vertebrobasilar TIAs are clearly related to turning of the neck. In this situation the physician wants to know—

1. Is there encroachment upon the vertebral artery?
2. If so, is it at one or several levels?
3. Is the degree of encroachment influenced by the position of the neck?

Arch angiography is again the best method of investigation. Accentuation of the common inequality of size of the vertebral arteries to the point where one is very small or absent is important, as persons with this anomaly are more vulnerable to a vertebral stenosis and are ideal subjects for ischaemia due to compression by articular or uncal osteophytes. If there is a history of attacks occurring only when the head is turned in a particular direction, an injection is made in the appropriate position. Preliminary films of the neck are taken in flexion and extension to show the position of maximum encroachment and an angiogram may also be taken in this position. Such additional angiograms are accomplished by selective injection of the subclavian or vertebral artery.
to avoid excess contrast or overlap of other vessels and to obtain better definition. It must be confessed that, although osteophytes often displace vertebral arteries, they rarely cause significant narrowing. None the less, if localised stenosis due to bony spurs can be established, it is curable by simple surgery.

**TRANSIENT ISCHAEMIC ATTACKS IN BOTH TERRITORIES**

There remains one further group of patients with TIAs in whom radiological help is often invaluable. These are the patients whose story suggests that attacks are occurring in both carotid and vertebrobasilar territories or in whom it is difficult to decide which of the two is involved. In this respect it is important to remember that severe bilateral carotid disease (bilateral occlusion, or occlusion on one side with severe stenosis on the other) may present with either carotid and vertebrobasilar TIAs or even with vertebrobasilar TIAs alone. This latter presentation is believed to be due to the carotid territory stealing blood from the vertebrobasilar. Angiography is clearly essential in this situation. The physician needs to know—

1. How many lesions are there?
2. Where are they situated?
3. How is each likely to be contributing to the clinical picture?

The frequency of multiple lesions and the diversion of flow through the intracranial anastomoses between different vascular territories may cause some neural deficit in regions served by relatively normal cervico-thoracic vessels. An arch study may help to explain the basis of such attacks involving more than one vascular distribution and may show how flow into the circle of Willis may be increased by carefully planned surgery.

Because of these advantages there has been a tendency, especially on the part of some vascular surgeons, to assess all their patients with suspected TIAs by using arch angiography alone. For several reasons this is not usually satisfactory. Firstly, as already stressed, most TIAs, especially those in the carotid territories, are due to emboli and such emboli frequently originate on small irregular plaques that can be consistently seen only with the higher definition of selective angiograms. Secondly, it is evident that in some arch studies performed on symptomatic patients the distribution of cervico-thoracic lesions does not account for the distribution of neural deficits. Sometimes the lack of correlation is due to intracranial steal, but at other times it is due to significant intracranial lesions that are not revealed on the arch study. Such lesions may equally well occur distally to potentially significant lesions in the neck. Cervico-thoracic atheroma has always to be viewed against the knowledge
gained from post-mortem studies performed on persons without cerebral abnormalities and from arch angiograms carried out on asymptomatic prisoners, which show a high incidence of atheromatous stenoses and occlusion of the major vessels. Thirdly, lack of filling of a vessel on an arch angiogram could very occasionally be caused by severe but operable stenosis that is usually revealed by selective study.

Selective studies of the affected region are therefore necessary to show a large number of the lesions responsible for thrombo-embolism and to show up further related or relevant intracranial lesions, while the arch study in such cases shows the haemodynamic background and presence or absence of gross lesions of other vessels, especially of a contra-lateral carotid that may influence the mode of surgery and determine the safety of certain surgical methods. The arch study should be performed first on such patients, if selective catheter studies are intended. If this suggests that catheterisation through the origin of a vessel would carry increased risk, needle puncture of the common carotid or retrograde subclavian, or vertebral catheterisation from the axilla, is substituted.

In vertebrobasilar ischaemic disease transfemoral catheterisation is the best approach and, because of the relatively high incidence of atheroma at the origin of the vertebral, semiselective vertebral studies performed by injecting the proximal subclavian arteries are usually accepted. On the other hand, in carotid disease, puncture of the common carotid low in the neck allows a simple, quick and detailed assessment of the regions affected and of the intracranial vessels. Failure to show the common carotid below the needle will miss only very few lesions treatable by more difficult surgery. If a surgically amendable lesion is shown, a simple arch angiogram may then be performed to show any gross lesion that may modify the approach. To some extent the choice of method depends on the particular skills of the radiologist.

**Completed Strokes**

The scope for radiology in the investigation of the acute stage of completed strokes is very considerably less than in TIAs. This is not because of radiological limitations but because advances in the management of the acute stage of the completed stroke have been very limited. The advances that have been made, such as the control of cerebral oedema by dexamethazone, do not require prior radiological investigation.

The limited indications that do exist arise firstly in relation to acute cerebellar haemorrhage. This presents with occipital pain, vomiting and loss of consciousness suggesting a haemorrhage in the cerebral hemisphere but examination fails to reveal the expected hemiplegia, the signs, if any, indicating
brain stem compression. If this condition is to be dealt with successfully, urgent radiological investigation followed by surgical evacuation of the clot is required.

While vertebral angiography is the method of choice for investigating vascular lesions in the posterior fossa, considerable expertise both in performing the angiogram and interpreting the films is required. As this degree of skill may not always be available, acute cerebellar haemorrhage is often managed best by ventricular drainage through a burr-hole followed by Myodil ventriculography to reveal displacement of the IVth ventricle.

A second indication for radiological investigation is the rare haematoma that is subcortical instead of deep capsular. This type of lesion may be suspected when the patient experiences a stroke onset highly suggestive of intracerebral haemorrhage, but after his clinical condition has stabilised for 48 hours or so he begins to deteriorate gradually; the level of consciousness falls and the focal deficit increases. If a subcortical haematoma can be localised, evacuation is often life-saving.

Superficial cerebral haematomas are easily recognised by conventional carotid angiography. Angiography with magnification that shows vessels as small as 200 μm may be necessary for the more precise localisation of deep haematomas. The treatable external capsular and lentiform nuclear bleeds displace the lenticulo-striate arteries medially while the internal capsular bleeds disrupt them.

Selective serial angiography of the appropriate vessel is the only method that allows detailed search for any underlying pathology such as angioma or aneurysm, and should such a lesion be shown, it may be important to inject other vessels to show further feeders, or to reveal other segments of the circle of Willis.

The third indication arises not at the onset of a completed stroke but after some days and stems from the fact that not all strokes are due to vascular lesions. In most general hospital series between 3 and 5 per cent of patients admitted as strokes are ultimately shown to have neoplasms. While this possibility should never be forgotten, it does not call for the radiological investigation of every acute stroke. It is the subsequent course of the illness that usually raises doubt about the diagnosis, at which stage investigation is called for to decide whether the acute stroke was caused by a cerebrovascular accident or a neoplasm.

The development of a hemiplegia in a patient with subarachnoid haemorrhage may give rise to the question whether it is caused by a subarachnoid haemorrhage from a ruptured aneurysm, the hemiplegia being due either to a haematoma or to spasm, or by a primary intracerebral haemorrhage from
which blood has escaped into the subarachnoid space. Although the age distribution curves of these two conditions show considerable overlap, the clinical pictures are sufficiently different to make the angiographic investigation of all patients with intracranial haemorrhage unnecessary. However, the development of the EMI scan (computerised transverse axial tomography) promises to revolutionise the approach to subarachnoid haemorrhage and all acute cerebrovascular accidents.

Computerised transverse axial tomography is a new, safe and atraumatic imaging method developed in the EMI Laboratories by Hounsfield (1973) and clinically evaluated in the St George’s group of hospitals by Ambrose (1973). A series of contiguous horizontal sections of the head are scanned through their edges from a large number of sequential directions by a beam of X-rays 13 mm in thickness. The intensity of the incident and transmitted X-ray photons are measured in collimated scintillation detectors, so that the amounts of absorption can be calculated. A computer assembles the data from each slice and reconstructs it as a square matrix of 80 x 80 cells, each 3 mm square with a number approximating to the absorption coefficient of the tissue at that point printed in each cell, resulting in a format corresponding to the shape of the original section. A picture with corresponding points of variation of light intensity is also displayed on a cathode-ray tube and can be recorded on polaroid film.

The densest tissue normally encountered is compact bone, and the lightest is air in the sinuses and mastoids. These have been fixed as the ends of a scale of absorption and arbitrarily given values of plus and minus 500 respectively, being depicted as black and white on the oscilloscope. Water has a value of zero on this scale and most of the soft intracranial tissues fall into the narrow range of 0 to +20. Grey matter, with an average value of +18, can be distinguished from white matter at +12, and cerebrospinal fluid at +1 is shown, outlining the shape of the ventricles and the size of the cerebral sulci. Lesions can be shown by the alterations they cause in normal anatomy or as abnormalities in tissue density which may be greater or less than that of the adjacent normal structures.

Intracranial haematomas can be recognised as such with a high degree of accuracy (Fig. 2). Their position, site of nearest approach to the surface, amount of penetration of deep structures, including presence of leak into ventricles and of displacement of adjacent structures, is apparent. Infarcts, on the other hand, owing to the presence of oedema and breakdown products of cells, show as low density areas which may later progress to cyst formation and atrophy which can also be shown (Fig. 3). Tumours may be revealed as areas of either increased or decreased density or a combination of the two, due both
to tumour tissue itself and sometimes superimposed haemorrhage or infarction due to involvement of blood vessels which occurs in malignant tumours and in meningiomas. Although a tumour could be confused with a stroke on a single examination, follow-up study will usually leave little doubt about the nature of the pathology.

The EMI scan is thus the most useful of the atraumatic studies which, together with other clinical and laboratory investigations, give a very good idea of the localisation and type of pathology prior to angiography. Angiography can then be planned on an informed basis to answer questions of treatment and

Fig. 2. EMI scan showing large haematoma as a circumscribed white area in left occipitoparietal region.
prognosis. In certain cases the need for angiography may be abolished; for example, it may resolve the common problem of whether a neurological deficit, occurring in a patient who has sustained head trauma due to an unexplained fall, is the result of a stroke or an extracerebral haematoma which may be revealed as a surface lesion.

The EMI scan will undoubtedly influence our approach to the management of the acute stage of the completed stroke. It is well recognised that the ability of the physician on clinical grounds alone to distinguish an infarct from an intracerebral haematoma is not very great. Except in the case of the subcortical
haematoma this has not been of great import as specific treatment was lacking for both. If specific treatment becomes available, diagnostic inaccuracy would be a serious difficulty which the EMI scan will relieve. Paradoxically, the fact that the differential diagnosis between infarction and haematoma can now be made may provide the stimulus for a further search for therapy.

Similarly, the ability to demonstrate relatively small haematomas in patients presenting as subarachnoid haemorrhage without localising signs will modify the angiographic approach. The angiographer’s attention will be directed to detailed study of the vessels adjacent to the haematoma and to the factors that influence surgery of the lesion. If a bleeding site is not demonstrable in the region of the haematoma it will no longer be necessary to proceed with more extensive angiographic investigations. The relevance of an aneurysm in an appropriate situation will be evident, whereas it previously depended on angiographic criteria that were not constantly present, such as an adjacent mass lesion or localised spasm or the presence of more than one loculus in the aneurysm which meant that it had bled at some time. If such criteria were absent it was customary to proceed with selective injection of the other cerebral vessels in the hope of finding another aneurysm with positive angiographic evidence of recent bleeding.

Radiology, particularly with the advent of the EMI scan, has a great deal to offer in the management of strokes. Best use of its potential can be made only by close collaboration between physician and radiologist. Unless the physician asks pertinent questions, the radiologist cannot endeavour to supply relevant answers. The development of newer methods of study such as the EMI scan will make this more rather than less true. Given such collaboration, it should be possible, in cases of stroke, to achieve a high level of diagnostic accuracy on which to base appropriate treatment.

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