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CONTRIBUTION OF RADIOLOGY TO THE STUDY OF INTRACRANIAL ANEURYSMS*

BY

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[WITH SPECIAL PLATE]

When one considers how relatively common are aneurysms at the base of the brain it is somewhat surprising that neither Thomas Willis nor his distinguished medical colleague and anatomy artist, Christopher Wren, described one on the arterial circle which has made Willis's name immortal. Another century passed before any description of an intracranial aneurysm can be found in the literature (Biumi, 1765). Nearly a century later Brinton (1851) reported on 52 cerebral aneurysms, nearly all of which he had collected from the literature. A more detailed history of the recognition of these early aneurysms is described by Bull (1962). Collier (1922) and Symonds (1923) were in large measure responsible for the clinical recognition of ruptured intracranial aneurysms, but it was Moniz's (1927) introduction of cerebral angiography which led the way to a definitive diagnosis during life. However, only sporadic cases were reported until after the percutaneous technique of opacifying the cerebral vessels had been firmly established by Engeset (1944) and his colleagues.

Nowadays we no longer think in terms of a likely aneurysm at the base of the brain but rather try first to confirm its presence and then also to define its exact anatomical site, its size, shape, and position, and such complications that it may have set in train.

There are six types of aneurysm to consider: (1) Congenital or "berry"; (2) atherosclerotic; (3) dissecting; (4) those associated with angioma; (5) infective, (*a*) mycotic, (*b*) syphilitic; and (6) traumatic.

Pathogenesis

The congenital, or berry, aneurysm is by far the commonest type, and for some reason as yet not fully explained it is almost entirely confined to the cerebral vessels and largely to certain specific sites on or near the circle of Willis. Not only are these aneurysms found within very limited anatomical confines, but they develop almost without exception at an arterial bifurcation. Furthermore, the exact position in relation to the bifurcation is nearly constant. There does not appear to be an anatomical name for this site, so I have borrowed the botanical term "axil" to describe it. An axil is the upper angle between the branch and trunk of a tree.

Forbus (1930) showed rather convincingly that a mural weakness often exists at what I call the axil of many of the larger cerebral arteries. He also showed by experiment, using a number of branching glass tubes connected to a water hydrant, that the point of maximum pressure is always that corresponding to the point in the glass vessel wall where the longitudinal axis of the impinging column intersects the vessel wall. This point is in fact the axil, and the mural weakness consists in a very localized defect of the pars media of the artery. Attractive though Forbus's theory was, it has not received unanimous support by histologists. Thus Glynn (1940), while verifying Forbus's medial defects, pointed out that they were so common in cerebral vessels where aneurysms were absent that they cannot be regarded as a *locus minoris resistentiae*. He further observed that medial defects are seen in coronary and mesenteric arteries, but that aneurysms of those vessels are very rare. He took the view that aneurysms follow damage to the internal elastic lamina and that such damage is usually provoked by atherosclerosis.

Crawford (1959) expressed the opinion that a multiplicity of factors are involved in the pathogenesis of these aneurysms: the combination of an absent media, an internal elastic lamina damaged by atherosclerosis, a poorly developed or absent external elastic lamina, together with hypertension, all acting together. He produced statistical evidence to show that patients dying from ruptured cerebral aneurysms suffered from more severe atherosclerosis than controls. Crompton (personal communication, 1961) found that 62% of his large unpublished series of cases of ruptured aneurysms suffered from hypertension.

Although the large majority of berry aneurysms occur at well-recognized arterial bifurcations, a few do not. Padget (1948) made a study of the embryological development of the cerebral arterial tree and postulated that when aneurysms develop at a point other than a bifurcation an embryonic vessel existed at the site. She also found that aneurysms were twice as common in congenitally anomalous cerebral arterial trees as in normals.

Atherosclerotic aneurysms are second in frequency within the cranium, but they are not common. At necropsy Stehbens (1954) found only 1 in 182. The other types listed are relatively rare.

Radiology

My main purpose is to attempt to give a picture of aneurysms as we find them in the living. It is here that we must turn to radiology. The great majority of

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aneurysms present clinically when they rupture, and, although plain *x*-ray films of the skull seldom provide helpful information, the examination should never be omitted. Occasionally such aneurysms are calcified, but the vast majority bleed before this healing process takes place. On the other hand, the much rarer atherosclerotic aneurysms often produce plain *x*-ray changes.

When a ruptured aneurysm is suspected both carotid trees should always be investigated by angiography even if an aneurysm is found on the first carotid tree examined, because multiple aneurysms are often present. Before undertaking this procedure it is important to remember that digital carotid compression, first on one side and then on the other, should be applied for about 10 minutes to ensure that there is an adequate anastomosis across the circle of Willis. If neurological signs develop during compression it can be assumed that it will be dangerous to ligate the carotid artery, a surgical procedure undertaken particularly in the treatment of bleeding posterior communicating aneurysms. In such circumstances we usually postpone the angiographic investigation.

It is well known that the vast majority of berry aneurysms lie on the carotid trees. When no aneurysm is shown on either carotid tree, vertebral angiography should be performed after an interval of about 48 hours.

Incidence of Congenital Aneurysms

Many pathologists have given figures for the incidence of intracranial aneurysms found at necropsy, usually varying between 0.5 and 1.5%. In recent years interest in this subject has been enhanced and perhaps more careful searching of the cerebral vascular trees has been made. Thus Stehbens, whose work has already been mentioned, found 182 cases with intracranial aneurysms in 2,800 consecutive necropsies (3.7%), a considerably higher figure than that recorded by pathologists previously. He also gave a high figure for multiple aneurysms (25.5%) and in one case in his series there were no fewer than seven aneurysms present. In one-third of his cases the aneurysm was unruptured and had caused no symptoms. Brolin and Hassler (1958) took 35 brains at random. They carefully examined the arteries forming the circle of Willis under a stereoscopic microscope, having first separated them from the brain and digested them in potassium hydroxide for three weeks; this removed most of the muscular and collagenous components. Although macroscopical examination did not reveal any aneurysms in these 35 cases, four were discovered by this special technique. Since then Hassler (1961) has published further data on minute aneurysms. He found them in as many as 17% of random brains, most commonly in elderly and middle-aged patients. In two cases subarachnoid haemorrhage was suspected to have been caused by these minute aneurysms.

Situation

Since the advent of percutaneous cerebral angiography and parallel advances in surgery, naturally much more stress has been placed on defining the precise site of aneurysms than previously. McDonald and Korb (1939) made such an analysis in 1,023 cases examined only at necropsy, but their collection was eclectic and was gathered from about a century of literature. Valuable though their collection was, it does not reflect a fair picture of the true incidence at various sites during life. Certainly angiography must be regarded as a crude method of obtaining accurate figures, particularly in the light of Hassler's recent work on minute aneurysms which are too small to be diagnosed radiologically. Nevertheless, we have no better means of diagnosis during life. Perrett and Bull (1959) attempted to assess the accuracy of angiography in demonstrating aneurysms. The angiographic accuracy in 210 cases all shown at necropsy to have ruptured aneurysms was 89%, and it was concluded that with improved technique, better contrast media which were then available, and more skilled interpretation the potential figure should be about 96%.

Material

The 1,769 intracranial aneurysms collected by McKissock in the 11 years 1950–60 inclusive have been analysed. Female patients outnumbered males in the ratio 3:2. In 95% of the cases the aneurysm has bled, and in over half of the remaining 5% the aneurysm lay on the internal carotid artery at the origin of the posterior communicating artery. In this group the most frequent presentation was the onset of a third-nerve palsy. Table I shows the main aneurysmal sites in McKissock's series, together with Greitz and Lindgren's (1961) cases.

TABLE I

	McKissock		Greitz and Lindgren (1961)	
	No.	%	No.	%
Anterior cerebral—anterior communicating artery	485	27·4	85	35
*Internal carotid—posterior communicating artery	467	26·4	67	27
Middle cerebral artery (first bifurcation)	348	19·7	67	27
Bifurcation (termination) carotid	110	6·2	11	4·5
Distal anterior cerebral	48	2·7	11	4·5
Multiple	247	14·0	?	?
Vertebral artery tree	64	3·6	—	—
Others	—	—	5	2·0
Total	1,769	100·0	246	100·0

* Anterior choroidal and ophthalmic aneurysms were included in this group for surgical reasons.

It is apparent from Table I that 94% of these aneurysms were situated either on the intracerebral portion of the internal carotid artery or on one of its two main branches just after bifurcation. Furthermore less than 4% lay on the vertebral tree.

The only other figures I have been able to find in the literature concerned with living patients are those of Greitz and Lindgren (1961) (see Table I). They recorded the carotid angiographic findings in 238 consecutive cases, in which 246 aneurysms were found; they made no mention of the vertebral arterial tree, but the incidence of aneurysms at the various sites on the carotid tree is very similar in the two series. Table I is oversimplified in one respect. All aneurysms on the cerebral portion of the internal carotid artery—that is, distal to the anterior clinoid process at which point it pierces the dura—have been grouped together in our surgical records.

The reason for this grouping was that such aneurysms were usually treated by carotid ligation. It is a fact that the great majority occurred at the site of origin of the posterior communicating artery; but there are two other quite large branches given off from the cerebral portion of the internal carotid artery—ophthalmic and the anterior choroidal—from the origin of which aneurysms occasionally arise. The ophthalmic artery is given off anteriorly just after the carotid artery pierces the dura medial to the anterior clinoid process. The anterior

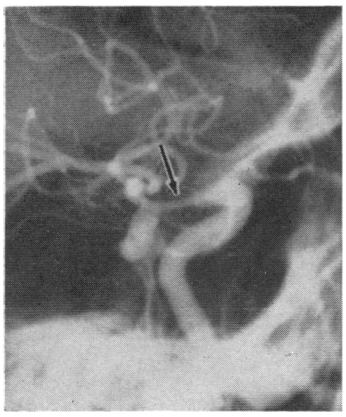

FIG. 1

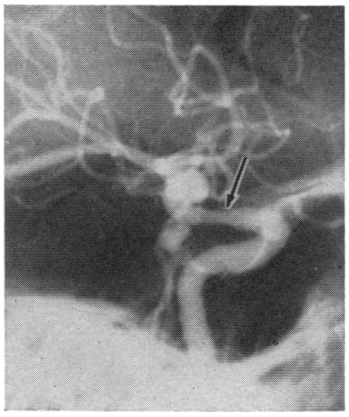

FIG. 2

FIG. 1.—To show posterior communicating aneurysm and associated spasm (see Arrow) of cerebral portion of internal carotid artery four days after haemorrhage from aneurysm.

FIG. 2.—Spasm almost disappeared (see Arrow) 13 days later.

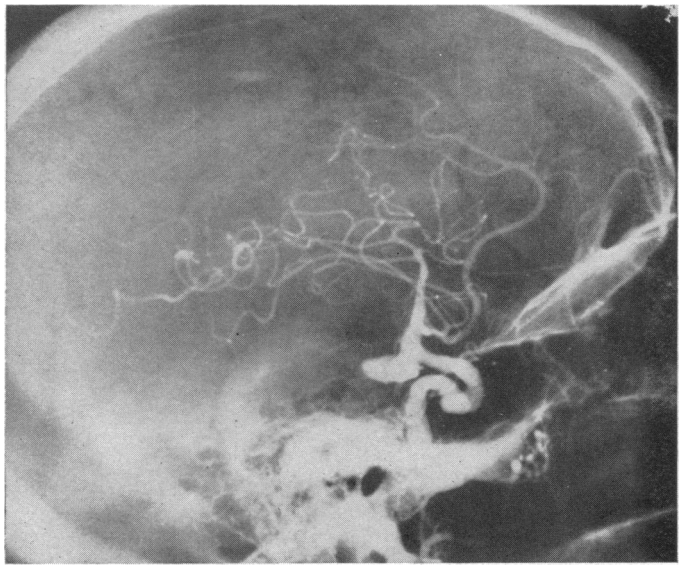

FIG. 3

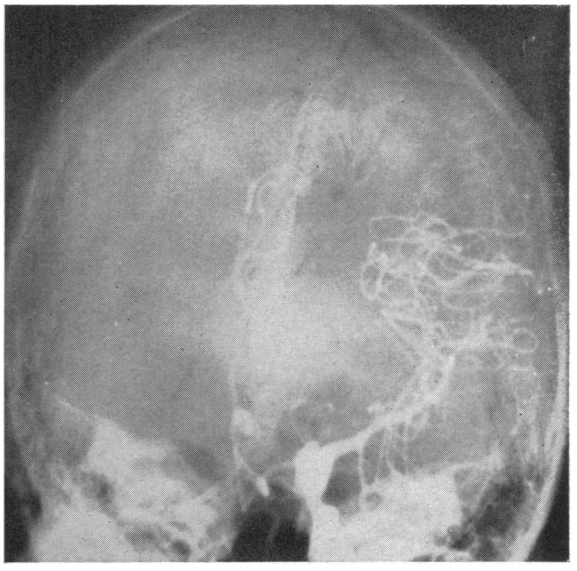

FIG. 4

FIG. 3. — Posterior communicating aneurysm; gross elevation of Sylvian vessels, indicating presence of large temporal mass. (Necropsy revealed subarachnoid haematoma.)

FIG. 4.—Same case as Fig. 3; deviation of anterior cerebral vessels and elevation of Sylvian vessels.

FIG. 5.—Air encephalogram to show filling defect in right cerebello-pontine-angle cistern (see Arrow).

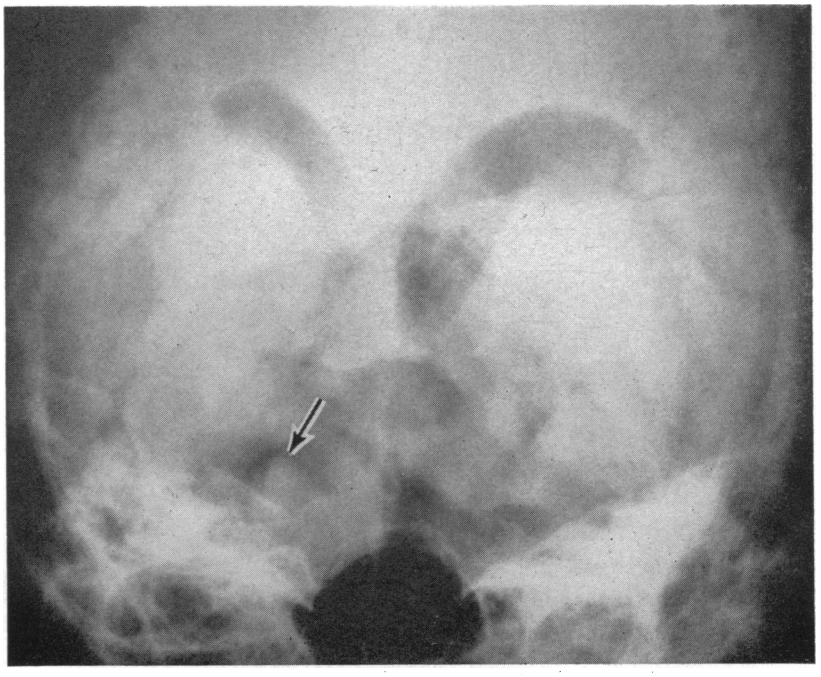

FIG. 5

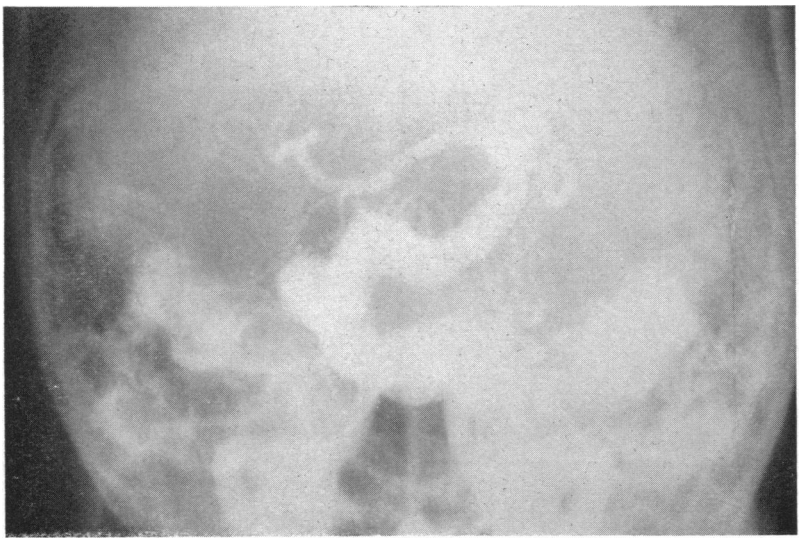

FIG. 6.—Same case as Fig. 5. Vertebral arteriogram to show fusiform aneurysm of right vertebral artery (occupying cerebello-pontine-angle cistern) and of left vertebral and basilar artery (see text for case history).

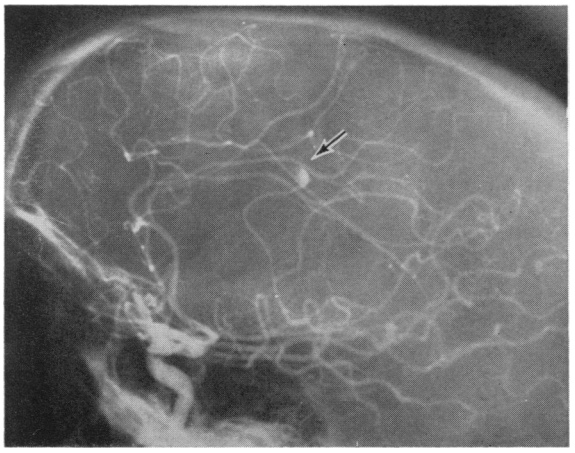

FIG. 7.—Mycotic aneurysm situated distally on branch of middle cerebral artery (see Arrow).

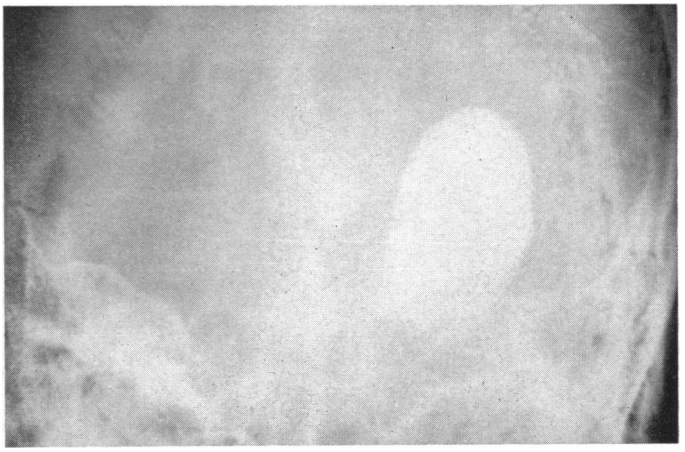

FIG. 8.—Large unruptured aneurysm at termination of internal carotid artery.

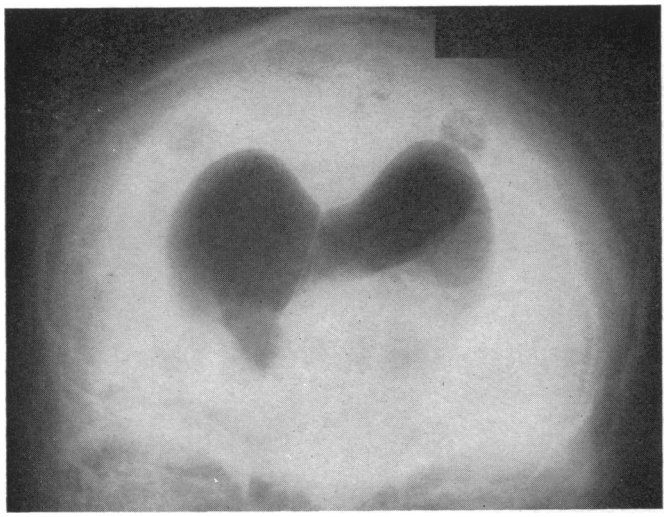

FIG. 9.—Same case as Fig. 8. Ventriculogram showing marked hydrocephalus with gross filling defect in floor of left lateral ventricle and obstruction of interventricular foramina. (Case history in text.)

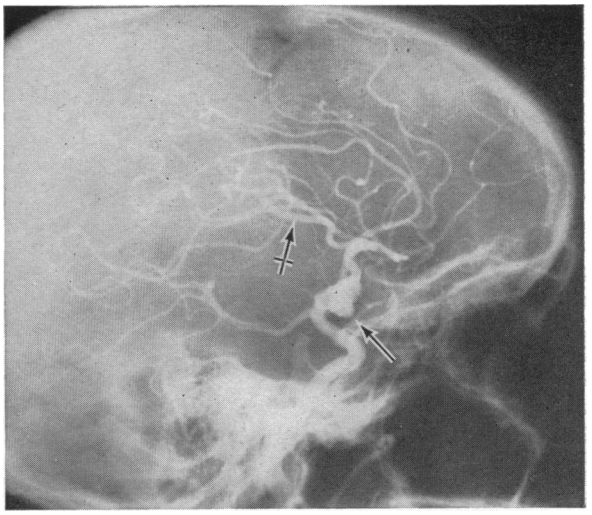

FIG. 10.—Middle cerebral aneurysm (see Arrow), probably largely thrombosed; note upward displacement of Sylvian vessels (see Arrow).

choroidal artery arises almost immediately distal to the posterior communicating artery, and both these vessels travel backwards roughly parallel on one another. It is often very difficult to determine on the angiogram from which of the two latter vessels the aneurysm takes origin, and sometimes the anterior choroidal artery is not seen at all. J. Ambrose (personal communication, 1961) analysed 185 of our aneurysms which had been previously grouped together under the general heading "posterior communicating" and found 10 at the origin of the ophthalmic artery and 9 at the origin of the anterior choroidal. Both these sites are rare as compared with all the others.

A small proportion of the aneurysms were found at a site where there is no known bifurcation, nearly always either on the cerebral portion of the internal carotid artery or on the proximal portions of the anterior and middle cerebral arteries before their main divisions. When Table I was compiled these aberrant aneurysms were listed under the adjacent main branches. Thus, for example, an aneurysm arising on the anterior cerebral artery 2–3 mm. proximal to the anterior communicating artery would be listed under the latter vessel. Aneurysmal sites on the carotid tree can be represented diagrammatically as shown in Fig. I.

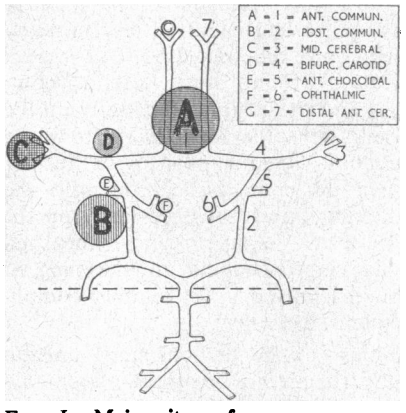

FIG. I.—Main sites of aneurysms on carotid tree.

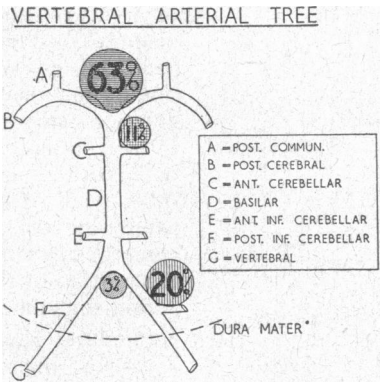

FIG. II.—Main sites of aneurysms on vertebral tree.

When multiple aneurysms were present, as they were in 14% of our angiographic examinations, they nearly always occupied the classical site or lay adjacent thereto as just described.

It has already been indicated that the incidence of aneurysms on the vertebral tree is very low (3.6%), a lower figure than that given by pathologists. This disparity can be explained partly by the fact that for one reason or another not all cases were submitted to vertebral angiography when no aneurysm was found on either carotid tree. Furthermore, when an aneurysm was present on one or both carotid trees vertebral angiography was *never* undertaken as no benefit would accrue from revealing possible further aneurysms. Lastly, as a general rule only one vertebral artery was investigated, a point which must be further elaborated.

Analysis of Aneurysms on Vertebral Artery Tree

Sixty-four aneurysms were found on the vertebral tree, and in no case were they multiple. Table II shows the sites.

Small though this series is, certain conclusions can be drawn from Table II. As on the carotid tree, the aneurysms tend to take origin from points of bifurcation of the main arteries (Fig. II). Ninety-seven per cent. arose from only four sites and nearly two-thirds arose at the termination of the basilar artery where it divides into the two posterior cerebral arteries. The only other group of significant size occurs at the junction of the vertebral and posterior inferior cerebellar arteries (20%). There

TABLE II.—*Posterior Fossa Aneurysms (Congenital Type)*

	No.	%
Termination basilar artery	40	63
Basilar and superior cerebellar arteries	7	11
Middle of basilar artery	1	1·5
Junction vertebral and basilar arteries	2	3·0
*Vertebral and posterior inferior cerebellar arteries	13	20
Distal posterior inferior cerebellar artery	1	1·5
	64	100

* As a rule only one vertebral artery was examined angiographically so this figure is too low.

is no doubt that the true figure here should be somewhat higher, as only one vertebral artery was examined in the great majority of cases. In a recent personal communication A. A. Donaldson, of Edinburgh, stated that he now investigates both vertebral trees if the two carotids and one vertebral are negative. In his first 25 cases he found four aneurysms on the second vertebral tree at the junction with the posterior inferior cerebellar artery. The origin of the posterior inferior cerebellar artery is an important site as it is readily accessible surgically.

Angiographic Features of Congenital Aneurysms

The angiographic appearances of the majority of berry aneurysms follow a fairly constant pattern: (1) they take origin at the axil of a branch; (2) they tend to emerge in one particular direction at each site; (3) they are usually circular to oval in shape; (4) they are often lobulated; (5) the diameter varies from about 2 to 20 mm. in the majority; (6) if recent rupture has occurred the supplying artery may be in spasm; and (7) arteries in the neighbourhood of the aneurysm may be displaced by complicating haematomas.

These features can be illustrated for the most part by discussing one common group—the posterior communicating artery aneurysm. Classically these aneurysms, which take origin at the junction of the internal carotid and posterior communicating arteries, present backwards. This is to be expected, since the axil lies on the posterior wall of the internal carotid artery. Table III shows the direction of presentation of 107 aneurysms.

Of the 107 aneurysms, 54 (50%) were lobulated to some degree. Crawford (1959) drew attention to this sign and investigated the site of rupture of 163 aneurysms (Table IV).

He found that nearly two-thirds ruptured at the fundus. He also stated: "As the aneurysms grow, they

TABLE III.—*Posterior Communicating Aneurysms (Position in Relation to Internal Carotid Artery)*

Backwards	47
Backwards and downwards	33
Downwards	15
Pure lateral	10
Lateral, downwards, and forwards	1
Pure medial	1
	107

often become bilocular, but even those which remain grossly spherical or ovoid may show, on closer scrutiny, an appearance as of bubbles rupturing from the surface. It is evident that these 'bubbles' are extremely thin-walled."

The maximum diameter of the posterior communicating aneurysms was measured on the angiograms and was found to vary between 2 and 17 mm. (Fig. III a). (An aneurysm of less than 2 mm. diameter probably cannot be diagnosed angiographically with any certainty.) In posterior communicating aneurysms

TABLE IV.—*Site of Rupture of 163 Aneurysms (from Crawford, 1959)*

Site of Rupture	No. of Aneurysms
Fundus segment	105 (64%)
Lateral ,,	17 (10%)
Cervical ,,	3 (2%)
Undetermined	38 (24%)

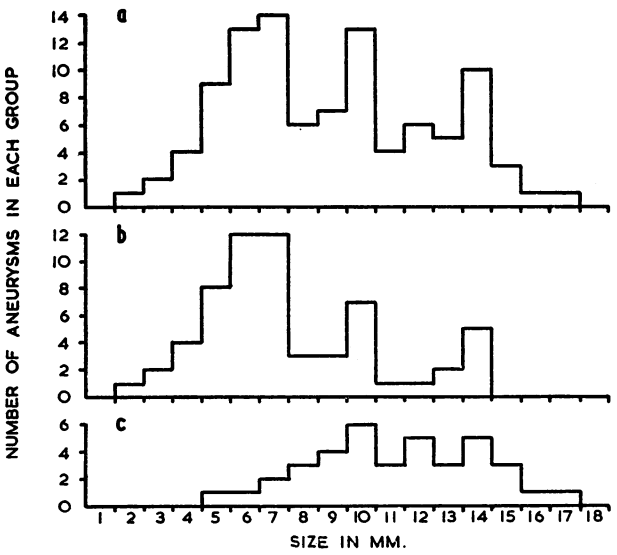

FIG. III.—99 Internal carotid-posterior communicating aneurysms to show: (*a*) general distribution of size; (*b*) size distribution of aneurysms *without* third-nerve palsies; (*c*) size distribution of aneurysms *with* third-nerve palsies.

palsy of the third nerve is not uncommon. The oculomotor nerve lies in close lateral relationship to the posterior communicating artery; if the aneurysm grows backwards and laterally and is of sufficient size it is likely to impinge on the third nerve. The relationship of aneurysm size to third nerve palsy was investigated. Eight out of the 107 cases had to be discarded because the physical signs were equivocal. The size distribution of the remaining 99 aneurysms was recorded (Fig. III a). Some degree of oculomotor paralysis was present in 38 out of the 99 examples (38%) (Fig. III c). A similar finding was made by Harris and Udvarhelyi (1957); in their series 40 out of 90 such aneurysms had affected the nerve clinically. It is possible to make a moderately accurate prediction of third-nerve involvement from the antero-posterior angiograms. At least two factors were involved: (1) the direction of the aneurysm and (2) its size. It will be seen from Fig. III, b and c, that the larger aneurysms tended to press upon the nerve. When a small aneurysm is seen on the angiogram to be directed away from the course of the third nerve and yet there is clinical evidence of partial or complete palsy, one may postulate that an adjacent subarachnoid haematoma (*q.v.*) is pressing upon the nerve.

The supplying artery is not infrequently found to be in spasm shortly after an aneurysm ruptures; this feature has often been confirmed by surgeons. Fig. 1 (Special Plate) illustrates this point. An angiogram performed 10 days after a first haemorrhage and four days after a probable second haemorrhage showed obvious spasm of the distal part of the intracerebral portion of the internal carotid artery (Special Plate, Fig. 1); a repeat angiogram undertaken 13 days later showed an absence of spasm (Special Plate, Fig. 2).

Arterial Displacement Caused by Complicating Haematomas

The great majority of aneurysms bleed primarily into the subarachnoid space, and the cortical, cisternal, and spinal subarachnoid pathways become bathed in blood, but sometimes a localized subarachnoid haematoma develops adjacent to the aneurysm. In 32 necropsies on cases of ruptured aneurysms Tomlinson (1959) found massive subarachnoid haematomas in 28%. They were situated at three main sites: (1) between the frontal lobes, (2) in the lips of the Sylvian fissure, and (3) in the depths of the sulci. Subarachnoid bleeding without complications was present in only three cases (10%).

Graeme Robertson (1949) and others have shown at necropsy that many aneurysms rupture partially or entirely into brain substance. He found that in 60% of his 93 cases such rupture had occurred and in many instances had extended into the ventricles. Crompton (personal communication) found that anterior communicating aneurysms and those situated distally on the anterior cerebral artery were the most likely to bleed intracerebrally. Subdural haemorrhage is a further complication and may occur in association with the other types of haemorrhage and not necessarily on the same side of the head as the aneurysm. Localized cerebral ischaemia leading to oedema and swelling of the portion of the brain involved is yet another complication sometimes found at necropsy.

Subdural haematomas can be demonstrated unequivocally by angiography, the picture being pathognomonic and easily recognized. The cortical arteries and veins are displaced away from the inner table of the skull, leaving a bare area devoid of vessels.

Subarachnoid haematomas, intracerebral haematomas, and ischaemic oedema manifest themselves by displacing the larger arteries, but unfortunately there is no certain means of differentiating these three conditions radiologically. Figs. 3 and 4 (Special Plate) illustrate this difficulty. The angiograms show a rather large posterior communicating aneurysm and gross upward and medial displacement of the Sylvian vessels, together with displacement of the anterior cerebral artery across the midline. Clearly a large mass is present in the temporal region. The patient died and at necropsy a large subarachnoid Sylvian haematoma was found, with no intracerebral haemorrhage.

The incidence of these complicating and usually very serious haemorrhages is as yet unknown. Pathologists see the brains of those who die only, and there is no doubt that these complications are a major factor in influencing the mortality and morbidity.

Atherosclerotic Aneurysms

In our experience these aneurysms are about fifty times less common than the berry type, but none the less they come second in order. We appear to encounter

them relatively more often than Stehbens (1954), who found only 1 in 182 cases at necropsy. They are quite different from congenital aneurysms in many respects. Atherosclerotic aneurysms very seldom rupture, they are not related to points of arterial bifurcation, they are seen almost entirely in the middle-aged and elderly, they frequently press upon cranial nerves, they tend to calcify and thus can often be recognized or suspected on plain *x*-ray films and sometimes they present clinically as tumour masses rather than aneurysms, and they tend to be large.

It is often difficult to state dogmatically at what stage a vessel becomes aneurysmal. When an artery has a calibre obviously greater than normal, but not grossly so, it is usually described as an ectasia. Ectasias can develop into fusiform aneurysms, but sometimes the expansion is saccular. Intracranially the three largest arteries—the internal carotid, the basilar, and the vertebral—are almost exclusively involved.

Internal Carotid Artery

For both anatomical and clinical reasons these atherosclerotic aneurysms fall into two groups: those occurring (*a*) in the cavernous or third portion of the artery, and (*b*) in the cerebral or fourth or terminal portion of the artery.

Carotid Cavernous Aneurysms

They are the only intracranial aneurysms outside the subarachnoid space. When small they cause no symptoms or signs, but as they grow they inevitably involve certain cranial nerves. Not only is the artery here ensheathed by the cavernous sinus, but this structure embraces the third, fourth, fifth (first and second divisions), and the sixth cranial nerves, and the optic nerve lies adjacent. If rupture occurs, which is not common, a carotico-cavernous fistula develops.

McKinney *et al.* (1936) drew attention to this group, and Jefferson (1938) and Meadows (1951) greatly extended our knowledge of the clinical picture. The disease occurs almost exclusively in middle-aged and elderly women. If the aneurysm is small there will be no bony erosion and no nerve compression and probably no symptoms. However, as soon as it reaches a certain size it will inexorably erode any bony structures with which it comes in contact. This can be clearly displayed radiologically. Rischbieth and Bull (1958) found *x*-ray changes in the bones in 21 out of 23 such aneurysms. Sometimes the wall of the aneurysm calcifies in a curvilinear manner, but unless the calcification is seen with certainty the diagnosis is not absolute until angiography has been performed. Rather similar bony erosive changes can be produced by parasellar extensions of pituitary tumours, some meningiomas, certain orbital tumours, and chordomas. The diagnosis is finally established or refuted by carotid angiography. The degree of bony destruction is probably proportional to the size of the aneurysm. One has reached this conclusion having followed some of these cases for over a decade and watched the aneurysm grow. An extreme example was seen recently.

A woman, aged 77, under the care of Dr. S. P. Meadows, had an intracavernous carotid aneurysm confirmed angiographically 12 years ago. In spite of carotid ligation, plain *x*-ray films taken recently indicated that it had continued to grow. One side of the sella, the anterior clinoid process, the optic foramen, the superior orbital fissure, the floor of the middle fossa, and much of the apex of the petrous bone (almost up to the semicircular canals) had all been eroded.

Very occasionally these aneurysms are bilateral.

Intracerebral Portion of Internal Carotid Artery

This terminal portion of the artery sometimes develops an ectasia if atherosclerosis is severe. If a frank aneurysm forms it can simulate a suprasellar mass. Signs of pituitary damage are usually absent, but, even so, a pituitary tumour cannot be excluded. Indeed, the sella turcica is often eroded in such a way that a pituitary tumour may be strongly suspected. It is therefore a wise precaution always to carry out carotid angiography prior to surgery or radiotherapy.

There is a subgroup described by Van 't Hoff *et al.* (1961). They had three angiographically confirmed cases of carotid aneurysm all with clinical and biochemical features of hypopituitarism; the sella was enlarged in each case. They were of the opinion that the hypopituitarism was due to interference of the blood supply of the hypothalmic nuclei which are now believed to regulate the secretion of the pituitary hormones.

Atherosclerotic Basilar Aneurysms

It is not uncommon to find the basilar artery lengthened in elderly persons, and when this occurs its termination presses upon the floor of the third ventricle deforming it in a characteristic manner. This in itself causes no symptoms or signs, but this characteristic feature should not be confused with the pressure effect of a tumour. Perhaps equally commonly the artery both lengthens and becomes tortuous and S-shaped; there may or may not be an associated ectasia, and, more rarely, definite fusiform aneurysm formation. These atherosclerotic aneurysms seldom rupture, but by virtue of their mass they act as space-occupying lesions and press upon the pons or lower cranial nerves on one side or the other. Dandy (1944) encountered an unduly high proportion on the basilar artery. When dividing the fifth nerve in the posterior fossa for trigeminal neuralgia he found 11 examples of basilar aneurysm pressing on the nerve, and he went so far as to say that this was the cause of the neuralgia, a view not generally accepted by neurologists.

Occasionally these aneurysms may closely simulate acoustic neuromas clinically. The following case, under the care of Dr. J. St. C. Elkington, will serve as an example.

A spinster aged 67 was admitted in August, 1959, having had bilateral deafness for many years, and severe occipital headaches and double vision for nine months. Recent gradual deterioration in her mental functions and memory had been noticed by her sister. Examination showed a right sixth palsy and slight lateral nystagmus both to right and to left. The right corneal reflex was impaired, there was slight right facial weakness and perceptive deafness, more in the right ear than the left; there was increased tone in all the limbs and the plantar responses were extensor. The heart was enlarged; the blood-pressure was 180/110, and atherosclerotic changes were present in the optic fundi. Neuro-otological examination by Mr. T. Cawthorne revealed perceptive deafness on both sides with bilateral brisk caloric responses, making the presence of an acoustic neuroma unlikely. *X*-ray films of the skull and petrous bones were normal. The cerebrospinal fluid protein was 100 mg. In spite of this finding, Dr. Elkington felt that the clinical picture could best be explained by a basilar aneurysm with arteriosclerotic changes elsewhere, but that

a space-occupying mass in the right cerebello-pontine angle should first be excluded, and therefore an air encephalogram was undertaken. This was performed with special reference to the cerebello-pontine-angle cisterns, which were well filled with air and a small space-occupying mass was demonstrated partially filling the right cistern (Special Plate, Fig. 5). This finding was in keeping with an acoustic neuroma and was supported by the raised C.S.F. protein, but an aneurysm could not be excluded as the cause. A vertebral angiogram which was undertaken showed tortuous, dilated vertebral arteries with gross dilatation of the basilar artery (Special Plate, Fig. 6). It was felt that the patient may well have had associated cerebral vascular disease elsewhere to explain the falling off of her mental functions. She died eight months later, but there was no necropsy. This case, diagnosed by Dr. Elkington, is of some historical interest as I am not aware that the condition has been confirmed prior to operation or necropsy in the past.

Atherosclerotic Vertebral Aneurysms

These aneurysms occur in the terminal portion of the vertebral artery where it enters the subarachnoid space; they are very rare, are difficult to diagnose clinically, and are seldom suspected. The signs consist of compression of the lower brain stem, or even medullary-cord junction and may suggest the presence of a tumour in this situation. The radiological approach to the investigation of these cases is usually by means of either pneumoencephalography or myelography. Since vertebral aneurysms are so rare and therefore not suspected, vertebral angiography is seldom undertaken.

Symonds *et al.* (1937) described six cases of tumours adjacent to the foramen magnum. One of the patients, a man aged 49, was shown at operation to have a large right vertebral aneurysm at the level of the foramen magnum, partly intradural and partly extradural. The aneurysm was excised, and at necropsy the late Dr. J. G. Greenfield found that the right side of the medulla had been severely eroded and there was a secondary syringomyelic cavity of the spinal cord extending down to the third cervical segment.

The syndrome of vertebro-basilar ectasia or aneurysm formation can vary very much, depending upon which nerves are involved. Furthermore, the precise level at which the brain stem suffers varies, and the degree to which it is indented depends upon the size of the aneurysm.

Dissecting Aneurysms

Shennan (1934) stated: "In most cases the aorta is involved, though dissecting aneurysms can form in the pulmonary artery and in all grades of arteries down to the small perforating arteries of the brain." Wolman (1959) described three cerebral examples at necropsy and found 14 more in the literature. The middle cerebral and basilar arteries were most commonly involved. Two of the 14 cases had angiograms (Poppen, 1951), and both aneurysms lay on the middle cerebral artery. We have had no experience of cerebral dissecting aneurysms demonstrated either angiographically or at necropsy.

Association of Aneurysms and Angiomas

In necropsies on nine patients with cerebral arteriovenous angiomas Anderson and Blackwood (1959) found saccular aneurysms of the berry type in five, and in all but one the aneurysms were multiple. Some were situated on arteries feeding the angioma, but not all.

Since the introduction of percutaneous cerebral angiography arteriovenous angiomas have been found relatively frequently. Thus Paterson and McKissock (1956) collected 110 of their own cases, nearly all seen since we began employing the percutaneous angiographic technique in 1947. Anderson and Blackwood's figures which showed aneurysms to be present in over half of their small series probably do not represent the true picture in life, for we believe that haemorrhage from an aneurysm tends to be fatal far more often than from an angioma. Three out of five of their patients, all of whom had aneurysms, died from a ruptured aneurysm and not from the angioma. In Paterson and McKissock's series of 110 patients with angiomas, aneurysms were recognized in only three cases. Many had undergone arteriography in the early days of the percutaneous technique and some of the radiographs were not of the quality expected now, with better contrast media available and better angiographic techniques. Furthermore, at the beginning of the period one was not conscious of the association of the two lesions, and the far more dramatic picture of an angioma may have removed one's visual attention from a small saccular aneurysm which was possibly present. The true incidence of the associated lesions in living persons must lie somewhere between Paterson and McKissock's figure of 3% and Anderson and Blackwood's 55%.

Infective Aneurysms

There are two types, the bacterial and the spirochaetal. The former are saccular and probably all rupture, while the latter are usually fusiform and tend not to rupture. The bacterial or mycotic type develop secondarily to infected emboli derived from the heart valves; the diagnosis can usually be made or strongly suspected on clinical grounds. We have had three examples of mycotic aneurysm all situated peripherally on the middle cerebral tree. The distal position of the aneurysm should make one suspicious of its true pathological nature if the diagnosis of the primary disease is not already known. Fig. 7 (Special Plate) illustrates a mycotic aneurysm lying distally on the middle cerebral tree.

Reference to the literature suggests that even when syphilis was rife it seldom attacked the large cerebral arteries. Mourgues (1954) described one case. The patient was a man aged 41 who at necropsy was found to have a basilar aneurysm of 5.5 cm. maximum diameter massively compressing the pons. Histology showed a round-cell infiltration of the vasa vasorum, and the Wassermann reaction of the cerebrospinal fluid had been positive shortly before death. Mourgues stated that sporadic cases of syphilitic aneurysms have been described in the literature, and they are nearly always situated on the basilar artery.

Traumatic Aneurysms

This type is extremely rare and we have had only one confirmed case. A man aged 39 suffered a craniocerebral gunshot wound eleven years before admission to hospital. He developed a left hemiparesis following the trauma and focal epilepsy during the next year. When admitted under Mr. McKissock he complained of malaise, headache, vomiting, and neck stiffness. The cerebrospinal fluid was heavily blood-stained and the left hemiparesis had become more pronounced. Angiography revealed an aneurysm situated distally on the

temporal branch of the right middle cerebral artery. The aneurysm was excised and at operation bone fragments were found adherent to it.

It seems likely that cerebral traumatic aneurysms only follow penetrating wounds. There is no evidence that the carotico-cavernous fistula which sometimes occurs after fracture of the skull base is secondary to an aneurysm. The fistula develops as the result of a tear in an otherwise healthy artery.

Aneurysms Simulating Tumours

Already some mention has been made of aneurysms which tend to simulate tumours in their clinical presentation. I will conclude by describing three atypical cases.

Aneurysms at the termination of an already lengthened atherosclerotic basilar artery may occlude the interventricular foramina and so lead to a pressure hydrocephalus of the lateral ventricles.

A woman aged 66 was admitted to Queen Square under Dr. M. J. F. McArdle. Eight years previously she was said to have suffered from meningitis, having had a bursting pain in her head, causing her to scream and to lose consciousness; she remained semiconscious for two weeks. (In retrospect it was clear that she had suffered a subarachnoid haemorrhage.) She now presented signs of an organic dementia and was unable to walk. An air encephalogram was attempted, but no air entered the ventricles, so ventriculography was then undertaken. The lateral ventricles were very enlarged and there was an obstruction at the foramina of Monro. A colloid cyst of the paraphysis could not be excluded, and Mr. McKissock explored the region. A blue-green tumour presented, looking like a colloid cyst not only in colour but also in size and shape. A needle was inserted and blood gushed out. It was in fact an aneurysm at the termination of a lengthened basilar artery.

Aneurysms on the carotid tree can also grow in such a way as to obstruct the interventricular foramina and lead to pressure hydrocephalus. The next case provides such an example.

A man aged 51 was first admitted under Dr. Denis Brinton with a history of four years' difficulty of speech and recent drowsiness, with episodes of loss of consciousness. Left carotid angiography showed a very large aneurysm near the termination of the internal carotid artery (Special Plate, Fig. 8). The patient was treated by common carotid ligation. Six months later he was readmitted with the same signs and symptoms but had in addition suffered recently from falling attacks. Examination now revealed severe bilateral papilloedema and extensive retinal haemorrhages. A ventriculogram (Special Plate, Fig. 9) showed marked hydrocephalus with a gross filling defect in the floor of the left lateral ventricle almost completely obstructing the interventricular foramina. This was clearly caused by the aneurysm, which had either grown recently or the true size was not revealed previously at angiography on account of a thick mural thrombus. Mr. McKissock excised the aneurysm and Professor Blackwood examined it histologically. It measured 5.2 by 4.2 by 3.8 cm. and section showed that it was almost filled with laminated thrombus of long standing. The patient was discharged from hospital but died shortly afterwards.

Very occasionally epilepsy may be the first manifestation of the presence of an aneurysm.

A married woman aged 41 was admitted under Dr. John Marshall in February, 1961, with a history that at the age of 15 she had a fairly severe head injury with loss of consciousness. For the last two years she had suffered from headaches, mainly occipital and right frontal, sometimes associated with vomiting. For the past 18 months she noticed a sensation of faintness followed by a dreamy state which lasted for a few seconds. She also admitted to unpleasant sensations of smell, which would come and go without any obvious cause. On December 10, 1960, she had a generalized convulsion without any localizing features. On examination there were no neurological signs. The blood-pressure was 170/100. An E.E.G. showed a slow-wave focal disturbance in the right temporal region which correlated well with her description of the attacks. A right carotid arteriogram showed a middle cerebral aneurysm measuring about 1.5 by 1 cm. (Special Plate, Fig. 10), with displacement of the Sylvian vessels quite out of proportion to the size of the aneurysm. An air encephalogram confirmed the presence of a right temporal space-occupying mass, and the degree of displacement of the right temporal horn confirmed that the lesion was very much larger than the aneurysm appeared to be. There is little doubt that the angiogram only outlined the core of the aneurysm and that the total mass was very much greater, as was so clearly demonstrated by Professor Blackwood in the previous case. As yet there is no confirmation, surgery not being considered justified at the time.

Summary

Short of operation or necropsy, angiography is the only means of diagnosing an intracranial aneurysm with any certainty. The majority (about 95%) present clinically after rupture. This paper is based on 1,769 confirmed intracranial aneurysms collected by McKissock in the 11-year period (1950–60). The importance of confirming the diagnosis angiographically is stressed: the site, size, shape, and possible multiplicity of aneurysms, and in many instances complications can be demonstrated by radiology. Intracerebral and subdural haemorrhages were easily shown if they were of moderate size. Localized subarachnoid haematomata could not be differentiated from intracerebral bleeds. Multiple aneurysms were found in 14% of cases, and the necessity of investigating the whole intracerebral arterial tree is discussed.

Pathologists have known for years that the vast majority of so-called congenital or "berry" aneurysms develop at sites of bifurcation on or near the circle of Willis. This fact is confirmed and detailed figures of sites are given. The relative rarity of aneurysms on the intracranial vertebrobasilar arterial tree is also shown.

Before rational treatment can be prescribed it is essential to obtain all possible information by angiography and this investigation should be undertaken as soon as possible after the tentative diagnosis has been made clinically.

Six types of aneurysm are discussed: (1) Congenital or "berry," which form the vast majority; (2) atherosclerotic; (3) dissecting; (4) those associated with angioma (arteriovenous malformation); (5) infective (*a*) mycotic and (*b*) syphilitic; and (6) traumatic. Examples of every type except dissecting and syphilitic aneurysms were demonstrated radiologically in this series.

Attention is drawn to that group of unruptured aneurysms which presents clinically as "tumour." Some of them grow to a very large size before causing symptoms or signs. They may press upon any of the twelve cranial nerves; they can obstruct the ventricular system and lead to hydrocephalus; and very occasionally the first and only symptom is epilepsy.

I would like to thank all my colleagues for allowing me to quote their cases and findings, and particularly Mr. Wylie McKissock for his large series of ruptured aneurysms. My thanks are also due to Mr. Prickett, head of the Photographic

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COMPARISON OF CORTICOSTEROID AND SULPHASALAZINE THERAPY IN ULCERATIVE COLITIS

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No perfect treatment of ulcerative colitis exists. Nevertheless a number of measures are known to be beneficial in an attack of the disease, and they fall into two main groups. On the one hand, certain general medical measures are plainly beneficial when circumstances demand them; the most important ones are correction of dehydration and electrolyte deficiencies, blood transfusions to combat loss of blood, a nutritious diet containing ample protein to minimize wasting, and vitamin supplements to guard against deficiencies. On the other hand, a large number of drugs have been employed because of some evidence that they may bring the attack swiftly to an end. We know of only two types of therapeutic agent for which there is strong evidence that they promote the chance of rapid termination of the attack. They are the corticosteroids and sulphasalazine respectively.

Corticosteroid Treatment

Shortly after the discovery by Hench *et al.* (1949) of the beneficial symptomatic actions of cortisone in rheumatoid arthritis, reports began to appear of the use of this agent and of A.C.T.H. in ulcerative colitis. The early reports were conflicting, but a large-scale controlled therapeutic trial showed that cortisone increased the chance of clinical remission within six weeks of starting medical treatment (Truelove and Witts, 1954, 1955). A second therapeutic trial showed that A.C.T.H. was similar to cortisone in the treatment of first attacks but was superior to cortisone in relapses of established disease, although at the price of more complications of therapy (Truelove and Witts, 1959). The newer corticosteroids have been extensively used in the treatment of ulcerative colitis, but it is questionable whether they are markedly superior to cortisone in equivalent doses, at any rate so far as can be judged from comparing published results (Watkinson, 1960).

Another way of using corticosteroids in this disease is to apply them topically to the colon. After preliminary studies had given encouraging results (Truelove, 1956, 1957) two independent controlled therapeutic trials employing a "double-blind" technique yielded unequivocal evidence that this form of treatment was beneficial (Truelove, 1958; Watkinson, 1958).

These two methods of using corticosteroids can be combined, and there is evidence that this enhances the therapeutic effect (Truelove, 1960).

Sulphasalazine Treatment

Sulphasalazine was first used for the treatment of ulcerative colitis by Svartz, who has written a number of articles on its use (Svartz, 1942, 1948, 1954, 1956, 1960). Another Scandinavian physician who has advocated its use is Lagercrantz (1949, 1955), who has employed it extensively in children with the disease. About 1950 the drug began to be used in America, under the name of "azopyrin," which was later changed to "asulfidine," and many favourable reports have come from physicians there (Morrison, 1952, 1953; Bargen, 1955; Moertel and Bargen, 1959).

Sulphasalazine ("salazopyrin") is an azo-compound of salicylic acid and sulphapyridine. Like other acid azo-compounds, it has a pronounced affinity for connective tissue, as has been shown by fluorescent microscopy (Svartz, 1960). It is a brown powder which is prescribed in the form of tablets, each containing 0.5 g., for oral use. For an acute attack of ulcerative colitis it is usually employed in a dose of 1–2 g. four

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