A STUDY OF THE VARIATIONS IN CEREBRAL VESSELS IN RELATION TO THE OCCURRENCE OF THE SO-CALLED CONGENITAL ANEURYSM.

By
Fathy W. Tadros
M.B., Ch.B., D.P.M. (Cairo)
M.R.C.P. (Edinburgh)

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Due to a typographical error, pages 120 and 121 are missing.
In 1952 Hamby wrote in his informative monograph on Intracranial Aneurysms: "One may conclude that the origin of the small 'berry' aneurysm from which develop the majority of spontaneous subarachnoid haemorrhage is not yet definitely known..... Perhaps one day we may know the answer to this fascinating riddle."

Many theories have been suggested to explain the pathogenesis of these aneurysms. The most interesting of these are -

(a) That of Forbus (1930) who postulated that they are acquired lesions developing on the basis of congenital defects of the media. The validity of this factor as being solely responsible for the development of aneurysms has been investigated and criticised by many observers such as Glyn, Carmichael, Richardson, Hayland and others.

(b) Bremer and Padget consider that aneurysms develop from persistent remnants of the embryonic capillary network. Although this theory is intriguing, it does not explain the fact that aneurysms are very rare in childhood. The percentage of aneurysms found at routine autopsy of children is negligible compared with that found amongst adult autopsies.
(c) In 1920 Drennan put forward the theory that the origin of one or more small branches, from the angle of junction of the anterior cerebral and anterior communicating arteries, acts as a minor defect in the vascular 'plumbing' and precipitates the protrusion of an aneurysm. Since then this work has not been further investigated.

The fact that none of these theories alone has been generally accepted as the full explanation of the aetiology of these aneurysms and none has stood the validity of absolute proof, has stimulated me to delve further into the investigation of the aetiology of aneurysms.
INCIDENCE:

Between the years 1929 and 1952, 14,750 autopsies were performed in the Royal Infirmary, Edinburgh. Amongst these 160 cases of the so-called congenital aneurysms (1.09%) and 12 cases of proved mycotic aneurysms were found. In the year 1953 alone in 752 autopsies, 28 aneurysm cases (3.72%) were encountered. In all these series the presenting symptoms were those of rupture or local pressure. Routine examination of the cerebral arteries was not performed in the absence of a suggestive clinical history.

The apparent increase in the incidence of patients dying from complicated cerebral aneurysm has been confirmed by the statistics of Professor Dott’s neurosurgical department of the Royal Infirmary, Edinburgh. In the year 1949 among 542 admissions only 16 cases (2.95%) were proved to have a ruptured intracranial aneurysm. In 1953 on the other hand, 73 patients out of 657 admissions (11.1%) had aneurysms. The relative incidence has increased further since the beginning of 1954.

So far the incidence has been calculated from autopsy tables. Most of the figures published have shown a lower percentage of cases of aneurysms.
<table>
<thead>
<tr>
<th>Name</th>
<th>Dates of collection</th>
<th>No. of necropsies</th>
<th>Percentage of cases of aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author</td>
<td>1929-1952</td>
<td>14,750</td>
<td>1.09</td>
</tr>
<tr>
<td>Author</td>
<td>1953</td>
<td>752</td>
<td>3.72</td>
</tr>
<tr>
<td>Turnbull</td>
<td>1908-1913</td>
<td>6,751</td>
<td>0.62</td>
</tr>
<tr>
<td>Courville</td>
<td></td>
<td>30,000</td>
<td>0.32</td>
</tr>
<tr>
<td>Richardson &amp; Hayland</td>
<td></td>
<td>4,618</td>
<td>0.07</td>
</tr>
<tr>
<td>Mitchell &amp; Angrist</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In all these necropsy series the head was not examined in every case as a routine. They therefore do not represent an accurate or even an average figure of incidence of aneurysm.

In the files of the Neuropathology Department, Royal Infirmary, Edinburgh, amongst 6,612 necropsies performed between 1934 and 1953 where all the heads were examined, 112 cases of symptomatic aneurysms and 10 cases of mycotic aneurysms were detected. Excluding the mycotic ones, which are very rare recently, the percentage of cases of aneurysm was approximately 1.86%. Routine examination of the cerebral arteries was not done. In 1953 alone the percentage of cases of aneurysms was 6.16%.

It has been difficult up till now to calculate
the incidence of aneurysms in the general population because:

(1) Many hospital patients die of cerebral apoplexy and no post-mortem is asked for.

(2) Many hospital cases of established subarachnoid haemorrhage and even with established diagnosis of aneurysm die in the general medical wards without autopsy being carried out.

(3) Some cases of intracranial haemorrhage die either in their homes or in nursing homes.

The apparent increase in incidence in the last two years can be accounted for by:

(1) Many general practitioners are now more aware of the diagnosis of subarachnoid haemorrhage and intracranial aneurysm than before.

(2) The establishment of the Neurosurgical Unit in the Royal Infirmary, Edinburgh under Professor H. Dott. It has recently been recognised that cases of intracranial aneurysms can be treated successfully by surgery. The increasing knowledge of this amongst the medical profession is the cause of referring more cases now to hospital and to the Neurosurgical Department.

(3) The profound interest of Professor Dott in the subject and his insistence on autopsy on all cases dying of subarachnoid haemorrhage.

The true incidence of intracranial aneurysm
can only be estimated if all autopsy cases were submitted to routine examination of the cerebral blood vessels as many cases have asymptomatic aneurysms.

Busse in 1921 was the first to direct the attention to aneurysms, giving no suggestive clinical data during life. He found 39 small aneurysms (10%) in 400 brains examined routinely as to the vascular supply.

Schmidt, K. in 1931 emphasized that smaller aneurysms are overlooked at autopsy and those causing no symptoms during life frequently are discovered only accidentally at autopsy. He estimated such aneurysms to occur in 0.5% of autopsies.

Popp, 1945, asserted that aneurysms are found by looking for them; in his surgical series of 110 patients with intracranial aneurysms, only 9 were encountered in a 10 year period, while 101 were found in the next 4 years, chiefly because of an awareness that such a condition might be present.

Rizza and Rinn, 1943, found 131 aneurysms in 1,437 routine examinations of the Circle of Willis (9%). All were related to arterial branching.

In a period of six months I did a routine examination of 300 brains, of which only 7 had died of signs of subarachnoid haemorrhage. In this series
I encountered 38 cases showing small aneurysms of the Circle of Willis, of which 7 had ruptured (12.6%).

Although this series was small the percentage was near those of Busse and Riggs & Rupp. These cases will be described later in detail, and the relation between the ruptured and unruptured will be commented on.

As regards incidence among children many writers agree that they are very rare. Riggs and Rupp in 1943 were not able to find a single case among 102 children's brains under the age of 10.

In the files of the Pathology Department of the Royal Hospital for Sick Children, Edinburgh, amongst 3,960 autopsies performed between 1929 and 1953 only 2 cases died of ruptured aneurysms. One was a case of a boy aged 4½ years published by Herman and McGregor (1949); another was a boy aged 10 years. The aneurysms in both cases were not found in the Circle of Willis or its major branches, but on small perforating branches in the brain substance.
AGE AND SEX:—

The majority of the 188 cases encountered among post-mortem reports of the Royal Infirmary, Edinburgh, in the years 1929 to 1953 were between the ages of 40 - 60 years. They comprised 94 cases. 48 cases were between 20 - 39 years and 26 cases between 60 - 70. 8 were between 15 - 20, and 9 were above 70 years.

These figures agree with Dandy’s generalisation of the age incidence. He said that a few cases are found below the age of 20; thereafter the decades contain about equal numbers until the age of 70, after which they decline, but remain as frequent as between 10 - 20 years of life.

It was only the 2 cases encountered from the Royal Hospital for Sick Children, Edinburgh that were at or below 10 years.

I was able to find only 14 non-embolic cases in the literature that were at or below the age of 10. They were reported by Dandy, Dial & Maurer, Weinberg, Hearne, Ingraham & Cobb, Pitt, Forster & Alnara, Monschel, Herzer, Ortman, Talbot, Bailo & Levy, Von Hoffmann and Herzan & Nazragor.

As regards the sex incidence of the same 188 cases, 95 were females and 93 were males.
Gower & Gull found the lesions more in men than women. Gower put the ratio as male: female as 3:2.

<table>
<thead>
<tr>
<th>Name</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author</td>
<td>93</td>
<td>95</td>
</tr>
<tr>
<td>Gull</td>
<td>35</td>
<td>23</td>
</tr>
<tr>
<td>Schmidt, M.</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>McDonald &amp; Korb</td>
<td>519</td>
<td>574</td>
</tr>
<tr>
<td>Poppen</td>
<td>38</td>
<td>72</td>
</tr>
<tr>
<td>Jaeger</td>
<td>4</td>
<td>27</td>
</tr>
<tr>
<td>Hamby</td>
<td>41</td>
<td>50</td>
</tr>
<tr>
<td>Richardson &amp; Hayland</td>
<td>18</td>
<td>22</td>
</tr>
</tbody>
</table>

It can be concluded, especially from the larger series, that the lesion is slightly more prevalent in women than men.
Site of Aneurysms:

The distribution of the 222 aneurysms encountered in 188 autopsies of the Royal Infirmary, Edinburgh, was as follows:

(1) Aneurysms were much more frequent on the carotid half of the Circle of Willis than on the vertebral portion. This also agrees with the figures given by Gull (1859), Gowers (1888), Martland (1939), McDonald & Korb (1939), and Riggs & Rupp.

<table>
<thead>
<tr>
<th>Name</th>
<th>Internal Carotid Supply</th>
<th>Vertebral Portion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author</td>
<td>90.1%</td>
<td>9.9%</td>
</tr>
<tr>
<td>Gull</td>
<td>55%</td>
<td>45%</td>
</tr>
<tr>
<td>Gower</td>
<td>58%</td>
<td>42%</td>
</tr>
<tr>
<td>Martland</td>
<td>71%</td>
<td>29%</td>
</tr>
<tr>
<td>McDonald &amp; Korb</td>
<td>83%</td>
<td>17%</td>
</tr>
<tr>
<td>Riggs &amp; Rupp</td>
<td>88%</td>
<td>12%</td>
</tr>
</tbody>
</table>
11.

(2) As regards the sites of predilection of this series of 222 aneurysms, 85 were found at the junction of the anterior cerebral and anterior communicating arteries (38%); 66 at the first or second bifurcation of either middle cerebral artery (about 30.8%); 40 at the junction of the internal carotid and posterior communicating arteries (17.2%); 7 at the bifurcation of the basilar artery, and 10 on the stem of the basilar artery; 4 at the bifurcation of the internal carotid artery into its 2 divisions, the anterior and middle cerebral arteries, and 2 on the posterior cerebral artery; 1 at the junction of posterior cerebral and posterior communicating arteries; 3 at a bifurcation of the anterior cerebral artery distal to the anterior communicating artery; 1 on the anterior cerebral artery proximal to the anterior communicating artery; and 1 on the internal carotid artery before it divides; 1 on the posterior inferior cerebellar artery; 1 on the vertebral artery.

# This includes the number of all aneurysms found in the Circle of Willis of all the 188 cases. See also page 17.
Distribution of aneurysms of the author's series.
The site of development of aneurysms on the Circle of Willis as reported by other observers is shown in the following table:

<table>
<thead>
<tr>
<th>Author</th>
<th>Junction of ant. cerebral &amp; ant. communicating arteries</th>
<th>Internal carotid at or near its branching</th>
<th>SuprACLinoID internal carotid artery before branching</th>
<th>Other sites</th>
</tr>
</thead>
<tbody>
<tr>
<td>222</td>
<td>38%</td>
<td>30.8%</td>
<td>17.2%</td>
<td>1.3%</td>
</tr>
<tr>
<td>Szekely</td>
<td>152</td>
<td>33.8%</td>
<td>27%</td>
<td>9%</td>
</tr>
<tr>
<td>Riggs &amp; Rupp</td>
<td>183</td>
<td>30%</td>
<td>21.3%</td>
<td>33%</td>
</tr>
<tr>
<td>Dandy</td>
<td>87</td>
<td>32%</td>
<td>24%</td>
<td>3.6%</td>
</tr>
<tr>
<td>Hamby</td>
<td>83</td>
<td>26.5%</td>
<td>18%</td>
<td>24%</td>
</tr>
<tr>
<td>Von Hoffmann</td>
<td>78</td>
<td>24.3%</td>
<td>28.2%</td>
<td>18%</td>
</tr>
<tr>
<td>Conway</td>
<td>44</td>
<td>50.3%</td>
<td></td>
<td>29.9%</td>
</tr>
<tr>
<td>Fearnside</td>
<td>28</td>
<td>35.8%</td>
<td>35.8%</td>
<td>21.4%</td>
</tr>
<tr>
<td>Wichern</td>
<td>21</td>
<td>13.8%</td>
<td>20%</td>
<td>4.8%</td>
</tr>
<tr>
<td>Brennan</td>
<td>8</td>
<td>100%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Busse</td>
<td>7</td>
<td>100%</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

13.
The incidence of the different sites in which aneurysms may be found suggested by the collection of McDonald & Korb of 1023 cases from the literature cannot be taken as representing the sites of predilection. It is neither a personal nor a consecutive series. It includes many single reported cases, probably published due to their uncommon site or uncommon pathology at the time. Individual series reported would give a more accurate information because all cases of aneurysms encountered would be included.

Conclusion.

The commonest site in which the so-called congenital aneurysms are found is the junction of the anterior cerebral and anterior communicating arteries. The second two common sites are at the first bifurcation of the middle cerebral artery and the junction of the internal carotid and posterior communicating arteries.

Other sites are comparatively much less frequent.
Distribution of the anesthetics encountered by Rigga and Supp.
Laterality:

Of the 222 aneurysms encountered, 107 were on the left side and 97 were on the right. Only 18 were midline, of which 17 were found on the basilar artery and one was at a point of fusion of 2 distal anterior cerebral arteries. The anterior communicating aneurysms are classified as Right or Left according to whether they arose from the angle of junction of the Right or the Left anterior cerebral arteries.

Cowers & Berger put the relative frequency of Left:Right as 4:3 and 7:4 respectively, and they both thought that this might be caused by the greater pressure in the left side since the left common carotid artery arises directly from the aortic arch.

Other series, however, show the following relationship:

<table>
<thead>
<tr>
<th>Name</th>
<th>No. of aneurysms</th>
<th>No. of aneurysms</th>
<th>No. of aneurysms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author</td>
<td>107</td>
<td>97</td>
<td>18</td>
</tr>
<tr>
<td>McDonald &amp; Korb</td>
<td>385</td>
<td>368</td>
<td>216</td>
</tr>
<tr>
<td>Riggs &amp; Rupp</td>
<td>68</td>
<td>75</td>
<td>39</td>
</tr>
<tr>
<td>Fearsides</td>
<td>24</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>Hamby</td>
<td>28</td>
<td>43</td>
<td>23</td>
</tr>
<tr>
<td>Poppen</td>
<td>63</td>
<td>47</td>
<td></td>
</tr>
</tbody>
</table>

The small difference on both sides is
Multiple Aneurysms:

Out of my series of 188 patients who died of ruptured intracranial aneurysms, 28 cases had multiple aneurysms; 26 of those had 2 aneurysms each. In one 3 aneurysms were encountered on both middle cerebral arteries, and in the 28th 5 aneurysms were encountered - one on the anterior communicating artery, two on the middle cerebral arteries and two unruptured ones on the internal carotid arteries at their junction with the posterior communicating arteries.

These make the total aneurysms encountered 222. The percentage of cases having multiple aneurysms is about 15%.

Dandy gave the same percentage on a study of 108 patients having 133 aneurysms.

Riggs & Rupp, however, in their series of 133 cases of aneurysms, reported in 1943, found out that 28 cases had multiple aneurysms or 21%. One patient had as many as 5 aneurysms.

Hamby reported that 8 patients of his 86 patients, i.e. 9.3%, had multiple aneurysms.

In the 300 consecutive autopsies in which I
examined the Circle of Willis I encountered 5 cases of the 31 asymptomatic aneurysms - showing multiple aneurysms. These will be described in detail later.

I agree with Tandy who thought that the actual percentage of multiple aneurysms would be near or about 15% of cases of aneurysms.
Since the publication of the first definite account of an intracranial aneurysm by Biomi of Milan in 1765 many observers have attempted to establish the cause or etiology of intracranial aneurysms.

Many theories have been suggested, and many varieties have been described. Embolism, arteriosclerosis, syphilis, trauma and congenital defects were among the most frequent causes met with in the literature.

A. Embolic or Mycotic aneurysms: Sandifort (1778) reported Biomi's case of aneurysm of the internal carotid and noted that the patient suffered from several severe rheumatic attacks. Quoted by Drennan.

Rokitansky (1844) recognised and described this variety. Koch (1851) described aneurysm secondary to ulcerative endocarditis.

Lebert (1866) was the first to notice the frequency of the association of intracranial aneurysms with cardiac disease, but did not trace any causative relationship between these conditions. It was Church (1870) who first showed the causative relationship between the formation of aneurysms and vegetative endocarditis. He reported 13 cases, 4 of his own of cerebral aneurysm in patients...
under 20 years of age being due to emboli arising from endocarditic vegetations. He was convinced that intracranial aneurysms in the young were nearly always due to embolism.

Porfik (1873) clearly demonstrated that intracranial aneurysms are caused by emboli in cases of progressive ulcerative endocarditis. He failed, however, to recognise the infective nature of the emboli and attempted to explain the formation of the aneurysms on a mechanical basis.

Peacock (1876) reported 3 new cases and collected 86 intracranial aneurysms from the literature. As to causation he remarked that "in some cases the disease seems to have originated in embolism."

Eslinger (1887) classified aneurysms into 4 groups. His third group were aneurysms which owe their origin to the local action of infective emboli. On the other hand, 3 years later Newton Pitt (1890) who examined 23 cases of intracranial aneurysm found amongst 9,000 autopsies at Guy's Hospital, believed that it was exceptional to find a cerebral aneurysm which is not associated with fungating endocarditis. He considered the sequence of events in the production of aneurysm as follows: (1) The lodging of a septic embolus is the starting
point of the changes which take place in the vessel.

(2) The clot disappears in most cases and the vessel wall dilates from the inflammatory process in its wall.

(3) An aneurysm may form and rupture in 3 weeks.

(4) The onset of embolism may not be marked by any symptoms.

Simonds (1901) described the minute pathological and bacteriological features which he had found in seven cases of aneurysm due to infective emboli. All his cases were examples of progressive, malignant endocarditis where the septicaemia was of coccal origin; they occurred in children or young adults who at autopsy showed vegetations on the valvular endocardium.

Gower (1893) and John Rose Bradford (1908) considered embolism as the second important causative factor of aneurysm especially in the young.

Since then all reports and papers on intracranial aneurysm described this variety of mycotic aneurysm as a special entity. Richardson & Eyland described them as being small, and saccular, with marked inflammatory changes, tending to occur far out on the branches of the Circle of Willis or in brain tissue. If the haemorrhage is not large enough to cause death, the organisms so released
may produce pyogenic cerebral lesions. The aneurysm or the resulting abscesses are often multiple.

This description fits exactly with the 12 cases of mycotic aneurysms I have encountered. They all occurred in young people between the ages of 12 - 30. They are rarely encountered nowadays, probably due to the use of antibiotics in the treatment of the primary condition.
B. Trauma as a cause of aneurysms:

In 1866 Lebert, and in 1897 Kiellian reported each a case of intracranial aneurysm with a history of trauma to the head.

Von Hoffmann (1894) found residues of old contusions in the meninges and brain substance surrounding an aneurysm of the internal carotid artery in a patient aged 26 years.

2 more cases associated with post-traumatic laceration, contusion or softening of the brain were reported by Geipel (1915) and Brandness (1923).

Hedinger (1917) and Harbitz (1933) described 2 cases of post-traumatic intracranial aneurysms. They believed that the connective tissue forming the wall of the aneurysm is actually 'healed ruptures' secondary to the effect of trauma which occurred some time ago.

Pawlowski (1924) reported 3 such cases, and Quest (1910) one case. All had a history of trauma. They believed that the trauma did not lead to rupture of the vessel at the time, but merely torn the elastica and media, and hence protrusion of an aneurysmal sac.

Menschel (1922) and Jung Michel (1932)
thought that the trauma stretching the vessels and the sudden rise of blood pressure at the time of the accident both contributed to the tear of the inner layers with the development of an aneurysm.

Kata & Mühe (1924) described one case with a history of trauma, but they believed that there was a congenital disposition of the vessel wall being prone to development of aneurysm. Trauma was only an additional factor.

Kahleu (1938) thought that indirect cerebral trauma may cause an aneurysm. He reported 2 such cases and summed up that to prove the traumatic aetiology (a) other causes must be excluded, and (b) signs or traces of trauma must be found in the brain or skull.

Schmidt, L.E. (1938) has listed trauma as a cause of aneurysm in single cases only. He thinks that it is very difficult to prove. As recently as 1948 Nachsler wrote that aneurysms may follow fractures of the base of the skull, especially of the orbital plate.

Hambry (1952) thought that trauma is a very rare cause of aneurysms of intracranial vessels. He quoted the case of Pireley & Trotter of post-traumatic aneurysm of the internal carotid artery in the cavernous sinus. "There is little doubt that
trauma causes aneurysms of the internal carotid artery - or even the artery itself - to rupture into the cavernous sinus, producing arterio-venous communications."

In all the cases I examined and looked up in Edinburgh, there was no evidence of trauma or even a history of trauma to the head before the rupture of the aneurysm.

I do not think that trauma has any relation to the causation of intracranial aneurysms, except possibly in the case of the infraclinoid aneurysms which occur in the cavernous sinus.
C. Syphilis as a cause of aneurysms:

One of the earliest writers to consider syphilis as a cause of intracranial aneurysms was Bartholow in 1872. After reporting one new personally observed and 6 newly collected cases, again reviewed the literature. From his studies of the records of 114 cases he concluded "there is much reason for believing that syphilis is the chief cause of formation of intracranial aneurysms."

Peacock (1876) after analysing the pathological findings in 89 cases of intracranial aneurysm remarked that "not infrequently the aneurysms seem to be caused by syphilis."

Covers (1893) amongst causes of intracranial discussed syphilis and stated that "an aneurysm has been frequently met with in cases of constitutional syphilis in young adults in whom no other cause of aneurysm was discoverable." He found it "well established" that aneurysms of the larger arteries of the brain frequently were due to syphilitic disease.

Earlier writers as Eppinger (1887) and Lebert (1866) dismissed syphilis as a cause of intracranial aneurysms. Lebert considered that the few cases which had occurred in patients giving
a history of syphilitic infection did not support the hypothesis that this condition bears any direct relationship to the formation of cerebral aneurysms.

In the early 20th century Bradford (1908) and Burns (1908) considered that the formation of a cerebral aneurysm is due to local atheromatous disease of the artery wall. They both admitted that syphilis is no doubt the cause of this atheroma.

The concomitant presence of syphilitic infection in the body, the positive Wassermann reaction and the presence of pathological evidence of syphilitic endarteritis of the cerebral vessels have induced many writers to report cases of intracranial aneurysms labelled as syphilitic among their series.

Thus, Richert (1911) reported 2 such cases. Ledinger (1917) thought that aneurysms are caused by inflammation and that this inflammation is due to syphilis. Shore (1929) reported a case and interpreted the type of cellular reaction found in the walls of the aneurysms as being the result of syphilitic arterial disease. Globus (1936), Holtman & Shelden (1927) and Baker & Shelden (1936) reported syphilis as the cause of aneurysms of the basilar and vertebral arteries. Hasson (1939) thought that while syphilis is a rare cause of the
common small aneurysms of the Circle of Willis, it is less rare cause of the more infrequent large aneurysms of the basilar and vertebral arteries. Hanss (1937) regarded syphilis as the main cause in her 8 reported cases of intracranial aneurysms. She thought that the gaps in the elastica were due to scarring secondary to syphilitic infection.

Syphilis as an aetiological agent of intracranial aneurysms was challenged and denied by many writers.

Turnbull (1915) and Fearnside (1916) amongst 10,500 necropsies did not find a single case of cerebral aneurysm due to syphilis. Turnbull did not see any evidence to show that syphilis induces atheroma. In the small muscular and elastic arteries, the group to which cerebral arteries belong, syphilis induces endarteritis obliterans and not aneurysms. Russe (1921), Symonds (1923), Parker (1926), Pollock (1927), Sanda (1929) and Schmidt (1931) dismiss syphilis as being a cause. Schmidt said that the histology of cerebral aneurysms does not prove their luetic origin and that he had seen no reference to finding spirochetes in the aneurysmal wall.

All modern writers do not consider syphilis at all as a cause of intracranial aneurysms.
Richardson & Hayland (1941) found none in their series. Since 1940 reports such as those of Forster & Alpers, Bremer, Glynn, Carmichael, Dandy & Hamby did not describe or regard syphilis as having anything to do with the aetiology of these aneurysms.

In the cases I have encountered, as well as those I have examined histologically, there was no suggestive evidence of syphilis either pathologically or serologically. I examined one case of diffuse aneurysm of the basilar and one case of vertebral artery aneurysm histologically as well, and there was not any suggestive picture of syphilitic infection.

Syphilis need hardly be considered as a cause of intracranial aneurysms.
D. Atherosclerosis in relation to etiology of cerebral aneurysms:

The observers on the subject in the late 19th century did not consider atheroma as an important causative factor.

Gowers in 1893 noted that "primary degeneration is an occasional cause of aneurysm in the second half of life ..... Atheroma seldom leads to simple dilatation."

Von Hoffmann (1894) concluded that neither atheroma, syphilis or trauma are common direct causes of the formation of aneurysms. Atheroma and a high blood pressure are, however, important factors in the causation of rupture.

It was in the early 20th century that Bradford (1903) thought that aneurysms are a manifestation of a patchy local disease which in a large proportion of cases is atheroma; and Burns (1908) said that "In the majority of cases the aneurysms are due to arteriosclerosis and atheroma of the intima." But as has been said before, they both thought that atheroma was due to syphilis.

In 1911, Wiggers classified 2 cases of his series of 22 cerebral aneurysms as "atheromatous." He used the term atheroma to cover changes of both the intima and media of the arterial wall. Both
cases occurred in old people. Yet later he suggested it is probable that even cases of old people were congenital with superimposed arteriosclerosis on top due to old age.

**Turnbull (1915)** describes atheroma as a degeneration which affects and is almost confined to the intima. He does not believe that there is any evidence that syphilis induces atheroma. Atheroma, according to him, assumes the forms of (a) fatty degeneration, (b) calcification associated with fatty degeneration, (c) mucous degeneration, and (d) medial fibrosis. He found that in 21 cases out of 30 aneurysms atheroma was absent or slight. Saccular aneurysms are due to medial degeneration. Many of these aneurysmal cases contrast sharply with those of direct rupture of the intracerebral vessels which are associated with atheroma. "If, therefore, these cerebral aneurysms were entirely the result of excess blood pressure and arterial degeneration, they would precede and be more common than direct rupture ...." which is not the case.

Since then there has been more argument concerning the part played by arteriosclerosis in the production of aneurysms. Some tended to believe that it is responsible for all aneurysms. Thus, **Strauss & Globus (1932)** in 11 autopsies of patients dying of subarachnoid haemorrhage, found evidence
in all, of generalised or focal arteriosclerosis of cerebral blood vessels. In 3 cases they discovered arteriosclerotic changes in the aneurysmal walls and in the vessels adjacent to the aneurysms. In 4 the lesions were microscopic. Tuthill (1933) reported 5 cases of saccular aneurysm of the larger cerebral vessels having various stages of arteriosclerotic changes. She considered the true nature of arteriosclerosis as thickness chiefly in the connective tissue collagen of the wall. This is followed by fat absorption which destroys both the collagen and elastica and so precipitate to the bulging of an aneurysm. Neither age nor wear and tear are responsible for such a change. The lesions in the media are apparently secondary to changes in the intima. She also remarked that there were "various interpretations by different observers" of the nature of arteriosclerosis.

Other writers considered only some aneurysms as arteriosclerotic on the basis of the association of marked arteriosclerosis of the cerebral vessels, and if the patients were of the older age group. Thus, Pial & Maurox (1937) classified 7 of their 12 cases and Berger (1923) 15 out of 25 as arteriosclerotic on this basis. Yet Berger goes on to say that even the presence of arteriosclerosis is
not a proof that it is the only cause of aneurysms above the age of 35. Many people show arteriosclerosis with no aneurysm. Arteriosclerosis as the main cause of aneurysm is only considered by excluding the presence of other factors. Mitchell & Angrist (1943) reported 3 cases as arteriosclerotic aneurysms. They said "In this paper such instances are listed as arteriosclerosis if marked arteriosclerosis of the cerebral vessels was present. " But " once the aneurysmal pouch is formed - in healthy vessels - local vascular disease of atheroma, sclerosis or actual necrosis seem to predominate in the defective part of the vessel."

In 572 cases of intracranial aneurysms collected from the literature McDonald & Kern (1939) give the following figures as regards arteriosclerosis:

Normal arteries in 32.7% of cases.
Arteriosclerosis 49.5%.

On the other hand, other observers considered it hard to prove that arteriosclerosis was responsible for development of aneurysms. Schmidt, M. (1931) said, after encountering a group of 19 out of 23 cases with a series of arteriosclerotic changes, that it is difficult to be sure whether the aneurysms or the arteriosclerosis was primary. He saw no reason why an
aneurysm should not be exposed to or undergo arteriosclerotic changes.

Richardson & Hayland (1941) did not accept arteriosclerosis as a cause because some cases occur in healthy arteries.

Carmichael (1950) wrote "The arteriosclerotic origin of aneurysms is inadequate in view of the prevalence of degenerative changes in cerebral arteries without aneurysms. Moreover, aneurysms develop on arteries which appear healthy on naked eye. The gross findings in cerebral arteries are obviously inconstant and too unreliable to serve as a guide to the aetiology of aneurysms."

There is, however, less controversy as regards the role of arteriosclerosis in the production of the fusiform aneurysms of the intracranial internal carotid, vertebral or basilar arteries.

Richardson & Hayland (1941) found 2 such cases in their series of 40.

Courville (1945) identified 96 aneurysms, 12 of which were arteriosclerotic fusiform aneurysms. Pandy (1945) also considered such aneurysms as arteriosclerotic. Hemby (1952) had only one case in his series. It was in the internal carotid artery. He concluded that "it would seem that if arteriosclerosis were the chief cause of intracranial
aneurysms, the preponderance of the lesions should be found located on the vertebral-basilar segment of the Circle of Willis, since in practically all reported series these vessels are the most heavily involved by arteriosclerosis. Precisely the contrary is true; hence it is likely that some factor other than arteriosclerosis is responsible for the majority of intracranial aneurysms."

There were 10 cases of fusiform aneurysms among the 188 cases I encountered. In the last 2 years since I started collecting and examining the histological pictures of aneurysms, there was only one such case among the 37 cases on which this work is based.

These aneurysms are rare. They differ from the common type of intracranial "berry" aneurysms both in their histological structure and their behaviour. The cases I encountered as well as all reported cases have not ruptured to produce subarachnoid haemorrhage. They produce signs of either local pressure or thrombosis with ultimate softening of the brain tissue supplied by their branches. Courville described them as enlargement and tortuosity of the affected vessel as a result of diffuse atheromatous change. I wonder if the application of the term "aneurysm" to such cases would be wrong and confusing.
In the 37 reported cases in this work 20 cases showed varying degrees of atheroma in their cerebral vessels. In the remaining 17 cases the cerebral vessels appeared healthy. All but 2 cases were above the age of 35 years. Yet, irrespective of the condition of the cerebral arteries in all aneurysms that have ruptured as well as in some unruptured aneurysms patches of thickened intima were encountered. These patches show changes which are described as atheroma on grounds that they show thickening of the intima and multiplication or splitting of the elastica. But the nature of atherosclerosis, as Tuthill has put it, has been interpreted differently by various observers. Hall (1948) in Textbook of Pathology, edited by Anderson, describes atherosclerosis as intimal fibrosis and lipoidosis. "It is agreed that the primary change is in the intima, but precisely what the initial change is has not been established."

To suggest that these solitary patches localised to the wall of the aneurysm were the primary causative factor in its development is improbable. Atherosclerosis is a generalised arterial disease. It is always widely distributed either in a diffuse or patchy form in the whole arterial system. If it were responsible for the causation of aneurysms one would expect that all patients showing patchy or diffuse arteriosclerosis of the cerebral vessels
would have their Circle of Willis riddled with aneurysms. This is definitely not the case. It is more plausible to believe that these patches which are so-called atheroma are liable to occur in the affected part of the vessel, both in patients showing healthy vessels as well as those showing generalised atherosclerosis.

The other question that arises is what is the aetiological factor in atherosclerosis? This is not definitely known and remains a mystery. Is it due to hypercholestrolaemia with deposition of cholesterol in the intima? Klots (1915) & Leary (1941) put the hypothesis that lipids are carried to the arteries by macrophages rather than in solution or colloidal suspension.

Another theory was that of Winternitz (1938) who postulated that increased vascularity and haemorrhage in the vessel wall is the cause of the sclerosis.

The most intriguing and plausible is the latest theory of Lucull (1949), who demonstrates that patches of atheroma were actually thrombi which have formed in the lumen of the vessel and became later covered by overgrowing endothelium. These thrombi so covered may undergo fatty degeneration, hyalinisation or fibrosis. Would this theory explain the solitary patches found in
aneurysms developing on otherwise healthy arteries? As the aneurysm is the only abnormal part of the healthy arteries, this explanation seems reasonable and probable.
E. Theories of a congenital origin of aneurysms:

Ennenger (1887) was one of the earliest observers to note the congenital origin of some intracranial aneurysms. Since then the advocates of the congenital theory have based their proofs on one of the following data:—

(a) The co-existence of cerebral aneurysms with other congenital anomalies;
(b) That aneurysms develop from persistent remnants of embryonic capillary network;
(c) That they are acquired lesions developing on a congenital basis, which is congenital absence of the media and a congenital weakness of the elastica.

(a) The associated congenital anomalies:

The co-existence of cerebral aneurysms with conditions like polycystic kidneys, other congenital abnormalities of the kidneys, coarctation of the aorta, abnormalities in the structure of Circle of Willis and other congenital abnormalities suggests to some writers the possibility of a congenital origin of such aneurysms.

Of my series of 188 cases one case was associated with congenital polycystic kidneys, and another one with a congenitally small kidney and hypertension. There have been many reports in
the literature of a more frequent association of such anomalies. These are summarised in the following table:

<table>
<thead>
<tr>
<th>Name</th>
<th>No. of Cases</th>
<th>No. of aneurysmal cases associated with polycystic kidney or other congenital abnormality of kidney.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitchell &amp; Angrist 1943</td>
<td>42</td>
<td>1</td>
</tr>
<tr>
<td>Sütter 1949</td>
<td>35</td>
<td>5</td>
</tr>
<tr>
<td>Sahs 1950</td>
<td>60</td>
<td>4</td>
</tr>
<tr>
<td>Hamby 1952</td>
<td>86</td>
<td>4</td>
</tr>
<tr>
<td>Ask-Upmark &amp; Ingvar 1949</td>
<td>47</td>
<td>9</td>
</tr>
<tr>
<td>Author</td>
<td>188</td>
<td>2</td>
</tr>
</tbody>
</table>

Sütter thought that this association was more than a coincidence, and inferred that there was some relation between polycystic disease of the kidneys and the development of cerebral aneurysms. Sahs believed the cerebral aneurysms are more common in patients with polycystic kidney than in the general population.

The co-existence of cerebral aneurysms with cases of coarctation or atresia of the aorta was
first noted by Egler (1888). Since then, odd cases have been reported by Parker (1926), Holtman & Sheldon (1927), Abbot (1928), Baker & Sheldon (1936), O'Teilly & Chapman (1943), Hearne (1945) and Fandy (1945). Abbot concluded that 12% of the deaths of patients of coarctation of the aorta were due to intracranial haemorrhage. Baker & Sheldon go so far as to say that a cerebral vascular accident in a patient with coarctation of the aorta should suggest the possibility of a ruptured intracranial aneurysm. No such case was present in my series.

The association of such congenital anomalies with intracranial aneurysms is not so frequent as to warrant consideration. I agree with Hamby as he has put it - "that this convocation of the congenital factor must not be uncritical, however; one is not justified in so explaining a goitre that occurs in a patient who happens to have a cleft palate."

It might be possible that the consequent high blood pressure associated with polycystic kidneys and coarctation of the aorta may enhance the rupture of a co-existing cerebral aneurysm.

The relationship of aneurysmal development with the abnormalities of the structure of the
Circle of Willis was first put forward by Lebert in 1866. He observed the frequency of gross developmental anomalies of the vessels at the base of the brain to the formation of aneurysm of the basilar artery. This has been noted and investigated by many observers, such as Russe (1921), Jacques (1926), Berger (1923), Dial & Maurer (1937), Von Hoffmann (1894), Turner (1946), and Sizas & Runn (1953) through a personal communication. They thought that these abnormalities which were mainly differences in size between the right and left vessels, anomalous formation of the anterior communicating artery or one or both posterior communicating arteries would provide a potential source of hydraulic imbalance. Turner (1941) carried out an experiment on a set of freely moveable elastic tubes to simulate the physical forces responsible for development and rupture of aneurysms. It is suggested that the malformation in the Circle of Willis would produce local alterations in intravascular dynamics, to provide a mechanical basis for the development of aneurysms in congenitally weak parts of the vessel walls.

It seems that anomalous formation of the Circle of Willis is very common. Many cases of abnormal structure of the Circle were reported with no association of aneurysms. Mindle (1888), Mitchet (1889), Henle, Wybrow & Parnisetti (quoted
by Rothman, 1904), De Vries (1905) and Russe (1920) have reported all sorts of such abnormalities without aneurysms being discovered.

This subject was fully investigated by Riggs & Bunn in 1952, and no conclusive correlation was reached as regards the association of aneurysms with abnormal structures of the Circle of Willis. Dr. Helena Riggs was so kind as to send me a resume from her manuscript dealing with analysis of the anomalies of the Circle found with aneurysms, which I quote here:

"Associated Anomalies.

Anomalous formation of the Circle of Willis in the presence of cerebral aneurysm, however, was present in 118 of the 124 cases in which complete description was available. Because of the relatively few cases involving the posterior half of the Circle of Willis, analysis of the intracranial anomalies associated with leaking aneurysms is limited to 119 cases where the aneurysmal lesion involved the internal carotid or its branches. Essentially, the anomalous formation resulted from hypoplasia of one or more of the component vessels, reversion to an embryonic stem of origin, or a combination of these factors. Eight basic patterns could be identified.

1. Normally formed Circle of Willis - 10 cases.
2. Hypoplasia of all stem vessels which are relatively equal in size (fetal type) - 6 cases.
3. Hypoplasia of the first portion of the anterior cerebral artery on one side - 46 cases.
4. Hypoplasia of both divisional branches of the basilar artery; both posterior cerebral arteries from the carotid stem - 11 cases.
5. Hypoplasia of the divisional branch of the basilar artery on one side, the posterior cerebral artery on the same side from the carotid stem - 20 cases.
6. Hypoplasia of the first portion of the anterior cerebral artery and the divisional basilar artery on one side, the posterior cerebral artery of the same side from the carotid stem - 15 cases.
7. Hypoplasia of the first portion of the anterior cerebral artery of one side and the divisional branch of the basilar on the opposite side; the posterior cerebral artery from the internal carotid on the side of the hypoplastic divisional artery - 6 cases.
8. Hypoplasia of the first portion of one anterior cerebral artery and of both divisional branches of the basilar, both posterior cerebral arteries from the carotid stem - 5 cases.

No absolute correlation of aneurysms of a particular location with specific anomalous formation was found although 85 per cent of the 40 aneurysms of the anterior communicating artery were associated with hypoplasia of the first portion of one anterior cerebral artery, and the great majority of the aneurysms present lay in locations where circulation would be influenced by focal increase of resistance or altered field of supply."

I have encountered many such varieties of anomalies in the Circles of Willis that I have examined routinely. Some were associated with aneurysms and others were not. I have observed, however, other sorts of anomalies which have a great bearing on development of aneurysms. They were anomalies of branching of the arteries. These were only observed by Brennan (1921). I would rather prefer to call them a peculiarity of branching - rather than anomalies - which are an...
important aetiological factor in aneurysmal formation. They will be detailed later on.

(b) **Persistent embryonic capillary remnants:**

Bremer (1943) was the first to call attention to the many microscopic arteries and pericapillaries which persist after the development of the adult cerebral arteries from the original embryonic capillary plexus. These arise from the main arteries as endothelial tubes and some may acquire a thin muscular coat. He postulates that they remain as outpouchings which might dilate later to form aneurysms. To explain the absence of both media and elastica which are the rule in all ruptured aneurysms he quotes Roux's (1910) postulation that the type of vessel wall depends on the presence of pulsations in the lumen. As these outpouchings transmit no pulsations, there is no need for muscle or elastica to develop at all or if the elastica having once been there, may have disappeared.

Padget (1945) in her chapter of Dandy's book reported an extensive survey she made of the material of the Carnegie Embryological Collection. She was impressed with the transitory arterial plexuses from which develop the arteries of the Circle of Willis. As the possible origin of congenital aneurysms she found that reported
aneurysms that had not developed at arterial bifurcations occurred at the sites of temporary embryonic branches. She suggests "that an incomplete involution or atrophy of these temporary arteries or of an artery normally present in the adult form, may be related to the formation of congenital aneurysm."

**Bassetti** (1949) reported 5 cases of congenital aneurysms with abnormally small branches. He thought the presence of these vessels is a support of the idea that the origin of aneurysms is from unresolved vestiges of a primitively normal circulatory system.

**Suver. O.** (1951) maintains the congenital origin of aneurysms from consideration of the embryologic features of the vessels of the brain. According to Congdon & Padget the basilar artery is formed from the two longitudinal neural arteries under the neural tube. "At the line of fusion one might expect medial defects to occur, e.g. the case of a child of 13 weeks reported by Forster & Alpers and the case of the embryo of 42 weeks reported by Bremer. Since these long vessels fuse by growing towards one another and by capillary connections which later disappear, it would not be difficult to assume that relics of these vessels could expand into other basilar aneurysms, not
only at the bifurcation."

He goes on to say that the persistence of part of another primitive vessel - "the vertebral basilar anastomosis" - might well account for occurrence of aneurysms on one side of the basilar. While in the place of the primitive "trigeminal artery" - the earliest communication between the basilar and carotid systems - may be responsible for aneurysms on the cavernous portion of the internal carotid artery.

Remnants of "primitive ventral and dorsal ophthalmic arteries" may be responsible for lateral and medial aneurysms of the internal carotid in the area between the ophthalmic and posterior communicating branches of the adult. He actually renounces the arteriosclerotic origin of the elongated aneurysm in this area and attributes them to be produced by ruptures of the ends of vestigial vessels. According to this he sees no reason why aneurysms occurring in the Circle and at its periphery would not represent persistent embryonic remnants.

If this theory explains the development of all so-called congenital intracranial aneurysms one would expect these aneurysms to be found since childhood. It is well-established that aneurysms
in children are very rare. Even these rare cases reported were present either on the vertebral basilar portion, the internal carotid before its bifurcation or the intracerebral perforating branches. These are not the same as the common type of adult aneurysms present at the bifurcations of the Circle of Willis or its major branches. Examination of the cerebral vessels of 120 children by Riggs & Rupp and of 50 children by myself did not reveal any of the common type of ruptured or unruptured aneurysms. Furthermore, I have encountered in 6 cases of these children, such anomalies of branching in the cerebral vessels which were always accompanied by aneurysms in adults but not in the children examined. This is more in favour of the fact that the so-called congenital aneurysms develop later in life.

However, I encountered in my series 2 cases which had 4 small "nubbin" aneurysms on the internal carotid artery before its bifurcation. These are a very rare kind of aneurysm and contrast with the common type of aneurysms which always occur at the bifurcation. They also behave differently in that none was recorded as having ruptured and so presumably they do not endanger the life of the patient. I think this rare type of aneurysm has developed at the site of temporary embryonic branches, as
Padget postulated.

(c) **Congenital defect of the arterial wall:**

Enniger (1887) was the first to elaborate on the idea that a congenital weakness or defect in one or more of the muscle coats was responsible for development of what he called congenital aneurysms. He quoted Korsagni, Munro, Hunter & Palletta who believed that aneurysms are formed as a result of rupture of the inner layer or intima and the consequent bulging of the outer wall. Vorstal said that it need not rupture, but there was weakening of the elasticity of the inner wall, either due to disease or to a congenital anomaly. Gruveilhier was also quoted as suggesting that the important factor was congenital hypoplasia of the media. He then concluded that aneurysms were congenital because: (1) They occurred in young people; (2) The small calibre arteries were affected; (3) The elastica of these arteries is simple, consisting of one layer between the intima and muscularis and it is easy to see it here and there imperfectly developed; (4) Aneurysms are found at points of bifurcation. These sites are under normal circumstances rejuvenating. Therefore if the elastica is generally hypoplastic, it would be more so at the points of bifurcation; (5) They are multiple, and at all points where
they emerge the elastica is hypoplastic; (6) The presence of hypoplasia of all the vessel coats is a possibility. He put the sequence of events in the development of aneurysms as follows:-
The congenitally hypoplastic elastica first gives way. The intima, media and adventitia then bulge. The media gradually atrophies and disappears.

Von Hoffmann (1894) observed that the favourite sites of aneurysms were (a) at the bifurcation of the larger vessels, and (b) the points of division of their branches. He concluded that neither atheroma, syphilis nor embolism are direct causes of formation and that they must be due to congenital weakness of the walls.

Turnbull in 1914-15 wrote "There appears, therefore, to be an additional factor in the formation of these cerebral aneurysms. This factor is probably an inherent weakness due to a congenital abnormality in the structure of the arteries at their points of junction."

Freeman (1921) described the histological picture of 8 non-embolic aneurysms. All were found at points of bifurcation. He noticed the presence of small branches arising from the aneurysmal sac. He concluded that the presence of small branches at the angles of bifurcation is a developmental
anomaly, and that the whole force upon this area acts as a weak point, causing bulging of the angle and the development of an aneurysm.

**Berger (1923)** considered aneurysms discovered before the age of 30 as congenital. He attributes their presence to the congenital weakness of the wall of the vessels which he calls a constitutional inferiority of the vessel wall that may be due to inheritance of weak elastica.

**Green (1927-28)** reported 12 cases of congenital aneurysms. He noted the absence of media and elastica in them. He said "the congenital defect in the contractile tissue of the vessel wall still offers the most likely explanation of their occurrence ....... it is possible that the fragmentation of the elastic layer may result from impairment of nutrition of this part of the wall due to hyperplastic thickening of the intima ...... and partly because the blood in the lumen is separated from the sac wall by a mass of laminated blood clot."

To explain the defects in the media he postulated that a functionally weak point existed in the wall of the parent vessel at birth which is due to a change of direction of the muscle fibres of the parent vessel as they pass out in a branch.
Under the strain of blood pressure the medial defect widened and gave rise to a saccular aneurysm.

Forbus (1930) found that development of aneurysms was associated with a muscular (tunica media) defect in the part of the wall forming the angle of bifurcation of the vessel. With glass tube models he measured the pressure of fluids flowing through branching tubes, and found that the point of greatest pressure to correspond to the location of the muscle defect in arterial bifurcation. He decided that the point of minimal resistance in the arteries (area of muscle defect) thus is subjected to the greatest blood pressure thrust. The muscular defect is the ground work for aneurysmal formation. He came across one or two micro aneurysms with slightly thick intima and degenerating elastica which he took to be the precursors of aneurysms of larger size. Therefore, there is over-distension of arterial wall at the site of defect followed by reactive proliferation of intima and degeneration of elastica with persistence of saccular aneurysms. Minute sacs formed in this way might either grow into big aneurysms or be obliterated early by thrombosis and subsequent organisation. He based his conclusions on the facts that aneurysms always develop at angles of bifurcations where the media is defective, and that in none of the aneurysms could muscularis be found.
From his embryological studies he deduced that the media of the parent vessel and that of the branch develop independently only to fuse later. If this fusion fails, medial defects occur. Although he noted that medial defect was a common arrangement of arterial coats and exists in cerebral arteries without aneurysms as well as extracranial arteries, especially the coronaries and mesenterics, he did not comment on why aneurysms should develop in some and not in all such defects.

Nevin & Williams (1937) and L.E.C. Schmidt confirmed Forbus' observations. The first observed medial defects in two reported cases, one with multiple cerebral aneurysms associated with a ruptured splenic aneurysm. They also found characteristic medial defects in the bifurcation of cerebral, lung and kidney vessels. The second explained the medial defects to be the combined action of developmental and mechanical factors. He comments that the frequency of medial defect does not give the whole clue to aneurysmal development. The size of the gap is an important factor.

The hypothesis of medial defect at bifurcations was severely attacked and criticised by Tuthill, Glynn, Richardson & Hayland, Forster & Alpers and Carmichael. They based their arguments on the fact that medial defects at the angles of bifurcation which are the usual site of aneurysms
are more common and so frequently found that if it were the sole cause all Circles of Willis should be studded with aneurysms: That they occur at all ages yet aneurysms are common in adult life only: That in most cases aneurysms are solitary, while these defects are widespread; and that medial defects are found in other arteries than the cerebral vessels and yet aneurysms there are very rare.

Glynn believed that the internal elastic lamina is solely responsible for preventing undue expansion.

Richardson & Hayland postulated a superadded acquired lesion, which, added to the medial defect, would precipitate the development of aneurysms. They, however, could not define such a lesion.

Carmichael (1950) explains this additional factor as follows:

The aneurysm is evidently formed by protrusion of a pouch lined with thickened and degenerate intima through a gap in media. The elastic membrane has lost its cohesion as a result of degenerative changes and is gradually disrupted as the sac expands. The medial gap is also due in part to degenerative changes which are superimposed upon a primary aplasia or hypoplasia of media, which explains the virtually constant relationship of aneurysm to arterial bifurcation where alone
These developmental defects occur ....... Hence distinction between developmental (so-called congenital) and arteriosclerotic aneurysm is completely invalid. The medial and intimal lesions are not only completely independent in their origin, but affect the same part of the arterial wall only by chance, hence the relatively low incidence of aneurysm.

In cases of advanced atheroma the intimal surface at the bifurcation may be almost completely covered by atheroma in confluent plaques which often overlie relatively large medial gaps; yet in these circumstances aneurysms rarely develop. The reason is sought in the well-known difference in constitution and mode of development of individual atheromatous plaques.

The elastica may survive until the thickened fibrous intima has built up strength enough to withstand the arterial pressure, or alternatively be degenerated early. Perhaps only plaques showing precarious degeneration of the elastica are likely to yield.
ROUTINE EXAMINATION OF CEREbral VESSELS

The fact that almost all of the so-called congenital or "berry" aneurysms have the same histological structure and that all develop at points of bifurcation suggested that a single aetiological factor must be common to all of them, and that this factor might be absent in cerebral vessels not affected by aneurysms.

I started by undertaking routine consecutive examination of the cerebral vessels from each case coming to autopsy in this hospital for a period of six months. During this time I examined 300 brains. The ages of these cases varied from between 35 - 75 years; one case only was 23 years.

The first 150 brains were injected with Neoprene Latex. The apparatus used for the injection was the same as that used by Trueta (1939) for the injection of the renal vessels, but the method was different. Many difficulties arose, however, in injecting the cerebral vessels, especially after the brain had been removed from the skull because of the multiple leakages which occurred from the torn meningeal vessels. The leaking neoprene tended to accumulate in the interpeduncular fossa and when it became set after the addition of concentrated HCl, it obliterated the anatomical
features of the vessels at the base of the brain. Another difficulty was that it was impossible to get a complete cast of the cerebral vessels after injection because of the inability to find a corrosive solution which would dissolve the brain tissue and at the same time keep the cast intact. Treating the brain by placing it in concentrated 50% NaOH solution for 4 days and then in concentrated HCl. for 5 days did dissolve the brain tissue, but the cast became very soft and easily torn. To overcome these two difficulties the following method is adopted:

The brain is carefully dissected out of the skull to avoid undue tearing of the smaller cerebral vessels. Relatively long stumps of the internal arteries are also removed.

A glass cannula is tied to one vertebral artery or to the basilar artery if the former is of a small calibre. By means of a 75 c.c. metal syringe attached to the cannula by a rubber tube, the cerebral arteries are flushed through with tap water. The maximum pressure that can be applied manually is used to wash out any blood clots. The large clots will escape from the internal carotid arteries. When the arteries look apparently empty of blood, the two internal carotid arteries and the other vertebral artery - if necessary - are ligatured. The vessels are again washed 2-3 times to break
down small clots in the smaller branches. Minor leakages are ignored, but if any major ones are noticed the artery is clamped. The brain is then placed - base up - in a colander over a sink. The cannula in the basilar or vertebral artery is connected to the neoprene apparatus by means of the rubber tube. The neoprene is then injected at a pressure of 50 mm/Hg. Meanwhile a gentle stream of tap water is directed on to the base of the brain to wash away immediately any neoprene which may escape from minor openings in the arteries. Water does not set neoprene.

The injection is carried on until the fine cortical branches in all lobes are filled. This usually takes from 3-5 minutes; the stream of water is run over the surface of the brain throughout the period of injection. The injection is then stopped by clamping the rubber tube near the cannula and 500 c.c. of 20% HCl. in 15% formalin solution is slowly poured over the base of the brain. This partially sets the neoprene and seals all minor leaking points. The cannula is disconnected and the brain is placed in 15% formalin for one week. The cannula and its rubber tube are then disconnected from the apparatus and tap water syphoned through it to make it ready for the next injection.

By the end of one week, the neoprene is
completely set and the brain partially fixed. It is then easy to dissect out the injected vessels together with their small branches. As the neoprene has an elastic consistency it will withstand some degree of stretching, and this permits the small vessels to be removed without tearing.

As my interest was confined to the study of the major branches of the Circle of Willis and their immediate sub-divisions, the dissection proved sufficient for my requirements, for the purpose of the neoprene injection was not to obtain arterial casts, but to detect and preserve the smaller branches. The information gained by examining the 150 brains by the injection technique enabled me to undertake the dissection of the vessels from the remainder of the series without previous injection.

In 38 cases out of the total 300 brains aneurysms were found, of which 7 had ruptured to cause either a subarachnoid or both subarachnoid and intracerebral haemorrhages. The rest were asymptomatic.

I was impressed by the number of variations in the arrangement of the Circle of Willis and its major branches. These included variations in structure and number of the anterior communicating artery (some are shown in fig. 1 m.), variations
in size and occasionally number of the posterior communicating, anterior cerebral, posterior cerebral and middle cerebral arteries, whether or not these variations were associated with aneurysms. These anomalies have been reported and commented on by many writers (see chapter on review of the literature).

The most significant and interesting observation noted in this study, as well as in the study of the cases of ruptured aneurysm which will be reported later in this work, was the peculiar method of branching of the arteries. This mode of branching differed significantly and constantly in those cases in which aneurysms were found from those without aneurysms.

For example:
(a) Anterior communicating artery branches:—I noted the constant presence of one or more small branches arising from the anterior communicating artery to supply the corpus callosum. These have been described and called by De Vriese "The arteries of the corpus callosum." There may be one slender artery which later divides or there may be two or more fine twigs given off the parent vessel. They also vary in calibre, and occasionally may be quite large. But what appears to be important is the site of origin of this artery or arteries.
In cases in which no aneurysms were found: these branches arise either from any point on the anterior communicating artery, anteriorly, posteriorly, ventrally or dorsally. They may also arise at the point of junction of the anterior communicating artery with either anterior cerebral arteries. If they arise at the latter site then they do so either from the posterior, dorsal or ventral angles, but never from the anterior angle upon which the pulsating column of blood impinges.

(b) Branching of the middle cerebral artery: In almost all cases without aneurysms each major artery divided into two main branches of approximately equal size. The division of these main branches followed the same pattern, for they too divided into two sub-branches. Occasionally a small twig arose from the point of bifurcation to one or other side, but never did it arise from the anterior distal angle of the bifurcation. In only one case, that of a patient aged 23 years, the second division of one main branch of the right middle cerebral artery produced three sub-branches of equal size without an associated aneurysm.

(c) The branching of the anterior cerebral arteries distal to the anterior communicating arteries followed the same usual pattern of division as did the middle cerebral artery.
(d) Junction of internal carotid and posterior communicating arteries:

In all cases without aneurysms no small arterial branch or branches were found arising from the distal angle, i.e. the angle opposite the column of blood flowing through the internal carotid artery. The perforating branches of the posterior communicating artery arose from the main stem distal to the angle of junction, or very occasionally from the proximal angle, which does not receive the thrust of the blood column.

(e) The anterior angle of bifurcation of the basilar artery into the posterior cerebral arteries was always free from branches. Perforating branches of the posterior cerebral arteries arose from points on either side distal to this angle.

Examples of this mode of branching are shown in the following Figure 1, in which 12 drawings were made from the injection specimens.
In Fig. 1 a, b, & c. the artery or arteries of the corpus callosum are seen to arise either from the ventral or the posterior surface of the main stem of the anterior communicating artery.

In Fig. 1 d. & e. they arise from the plexiform formation of the anterior communicating artery. In Fig. 1 f. & g. one branch arises from the anterior, the other from the posterior surface of the main stem of the anterior communicating artery, while in Fig. 1 h. i. & j. they arise from the anterior surface.

In Fig. 1 k. these branches arise from the posterior angle of the junction of the anterior cerebral and anterior communicating arteries, and in Fig. 1 l. one branch arises from the posterior and the other from the dorsal angle of junction.

The pattern of branching of the middle cerebral and anterior cerebral arteries as described before is also well shown in all these figures. The distal angle of junction of the internal carotid and the posterior communicating arteries as well as the angle of bifurcation of the basilar artery are devoid of branches in all figures. Fig 1 h. shows that there are two main left middle cerebral arteries. The disparity in size of each pair of posterior communicating arteries and the proximal parts of the posterior cerebral arteries is shown
in most of the cases, especially in Fig. 1 b. c. d. f. g. & i.

The remainder of the 262 arterial circles which bear no aneurysms, show the same branching pattern.
The Circles of Willis upon which aneurysms were found showed a different and very characteristic mode of branching. This was constantly shown in the 31 Circles bearing 36 unruptured aneurysms, in the 7 cases showing ruptured aneurysms and in all the subsequently selected cases showing ruptured aneurysms reported in this work. The symptomatic ruptured aneurysms will be considered later. The unruptured aneurysms found incidentally during routine autopsy were always seen to arise in association with a certain type of branching occurring at the angles of junction or bifurcation. If small branches are present at the anterior angle of bifurcation, namely that angle which is exposed to the constant thrust of the column of blood, they are almost always associated with an aneurysm or a bulge at this site. This was the rule in all adult patients examined above the age of 22 years. Another feature of note is that apart from the site of origin of these branches, they are almost always smaller in calibre than the two main divisions. In some cases they may be only minute twigs. Their number also varies - there may be one small branch or several forming a sort of leash.

If this type of branching occurs in more than one situation there will be more than one aneurysm. I have encountered 5 cases, each having
more than one aneurysm among the 31 Circles bearing unruptured aneurysms.

Expressed in another way, I have found that:
(a) all aneurysms arose at angles of junction or bifurcation, which are opposite to the impinging column of blood. They were never found on any of the other sides of the angles nor in other situations.
(b) that whenever an aneurysm was present, one or more small branches were seen to arise from the summit of the bulge.

12 examples are shown in Fig. 2.
Fig. 2 a. b. c. & d. show small aneurysms arising at the anterior angle of the junction of the anterior cerebral and anterior communicating arteries, with one or more of these small branches arising at the summit of the aneurysm. The other parts of the Circle of Willis and its main branches do not show any fine branches arising from its distal angles.

Fig. 2 e. shows an aneurysm at the anterior distal angle of the first abnormal bifurcation of the left middle cerebral artery, and no other abnormal modes of branching are detected.

Fig. 2 f. g. & h. each show an aneurysm arising at the lateral distal angle of the junction of the internal carotid and posterior communicating arteries with the small branch or branches at the summit of the bulge.

Fig. 2 i. j. k. & l. each show two sites of abnormal branching, each carrying an aneurysm.

In Fig. 2 i. the aneurysms are situated one at the junction of the anterior cerebral and anterior communicating arteries, the other at the first bifurcation of the left middle cerebral artery.

In Fig. 2 j. & k. the first is on the bifurcation of the middle cerebral artery, and the
second at the distal angle of junction of the internal carotid and the posterior communicating arteries; while in Fig. 21, one is at the junction of the left anterior cerebral and anterior communicating arteries, and the other at the junction of the internal carotid and posterior communicating arteries.

Figure 3 is a photograph of a later case in which two aneurysms were also present.

It shows the small branches at the fundus of the unruptured right middle cerebral aneurysm and at the base of the ruptured anterior communicating aneurysm. As the vessels in this case had not been previously injected with neoprene they were readily displayed for photography. The injected cases proved more difficult to manipulate because of their elastic consistence and photographed poorly.
Fig. 4 illustrates diagrammatically all the 36 unruptured aneurysms encountered in the series. It shows the constant association of the small branches with each case.
The next problem to be decided was whether the small unruptured aneurysms were present since birth, i.e. were they congenital? To determine this I examined the Circle of Willis from 50 infants and children aged from a few days after birth up till 9 years, and looked for such small bulgings. This represents the total number of cases available to me for examination during a period of 1½ years.

Riggs & Rupp examined 120 children's brains and could not find a single case of aneurysm, ruptured or unruptured. In my small collection I also failed to find any aneurysms. The most pertinent observation, however, was that 6 cases of the 50, i.e. 12%, showed the same branching abnormality which was found associated with aneurysms in the adults, yet no aneurysms were present in any of these 6 cases either.

The following Figure 5 shows these anomalous branchings.
In 4 of them the artery of the corpus callosum arose from a point on the anterior (distal) angle of junction of the anterior cerebral and anterior communicating arteries. In one case a smaller branch arose from the distal angle of bifurcation of the right middle cerebral artery, and in the 6th case the anomalous branching was seen in 2 positions, one at the junction of the anterior communicating artery and anterior cerebral artery, and the other on the left middle cerebral artery.

Figure 6 is a photograph of one of these cases; the anomalous site of the artery of the corpus callosum is clearly seen.
HISTOLOGICAL EXAMINATION OF THE UNRUPTURED ANEURYSMS

The aneurysm with its accompanying vessels was fixed in formalin for at least a week. It was then post-fixed in Helly's fluid for 24 hours, washed, dehydrated and embedded in paraffin in the usual manner. Serial sections were cut at a thickness of 8μm. Every tenth section was mounted primarily, and where it proved desirable intermediate sections were mounted. Some cases were stained with Hart's elastic stain and counterstained with Van Gieson solution, others with Hart's elastic and Masson trichrome connective tissue stain.

Serial sections were essential for the complete study of the histological and topographical relationships of all parts of the wall of the aneurysm and all vessels related to them.

The histological structure and pattern of branching of all unruptured aneurysms were identical. It will, therefore, suffice to give two illustrative examples. Details of other similar cases are given in the appendix.
Case 1. Male, 46 years of age. Death due to a generalised peritonitis. No present or past history nor signs and symptoms suggestive of aneurysm given.

Naked eye examination revealed an aneurysm arising at the angle of junction of the right anterior cerebral (A) and anterior communicating arteries (B). A small branch (C) arose from the summit of the sac.

Some of the serial sections from this case are illustrated in Plate 1, figures 1 - 4. Figs. 1 & 2 show the aneurysmal sac (O.An.) at the junction of A & B. A cross section of the small branch C. appears near the top of the bulge. Figs. 3 & 4 at a higher magnification show the aneurysmal sac (O.An.) and the point of origin of branch C. The rest of the anterior communicating artery B. has joined the contralateral anterior cerebral artery, A₁.
The histological structure of the wall of the aneurysm is practically identical to the appearance of the wall of a normal artery. In some parts the intima, internal elastica, media and adventitia are well seen. In other parts, notably at the distal angle of junction, the media is hypoplastic. This confirms the well-known finding of medial hypoplasia occurring at sites of junction and bifurcation to which attention was drawn by Forbus. The internal elastica, however, is intact throughout the aneurysmal sac and is continuous with the elastica of the two main branches as well as with the thin elastic layer of the small branch C. There are, however, small patches of intimal thickening (figs. 1 & 3) which take up the connective tissue stain. Underneath one of these patches the elastica is relatively thin (fig. 3). Elsewhere in the main vessels the intima and elastica appear healthy.
Case 2. Male, 39 years of age. Death due to tetanus. No previous or present evidence suggestive of cerebral aneurysm elucidated. Examination of the cerebral vessels showed 2 small, unruptured aneurysms. One was situated at the junction of the right anterior cerebral and anterior communicating arteries, and the other at the first bifurcation of the left middle cerebral artery.

**Anterior communicating aneurysm**

Naked eye appearance.

The aneurysm, which measured 0.2 x 0.25 cm, arose at the junction of the anterior cerebral artery (A) and anterior communicating artery (B). It bulged from the distal angle upon which the column of blood flowing through A impinges. One sizeable branch (C) and 2 other small branches (D & E) were seen arising from the summit of the aneurysm. A is the contralateral anterior cerebral artery.

The significant serial sections of the aneurysm are shown in Plate 2, figs. 1 - 3. Fig. 1 shows the bulge of the aneurysm (0.An.) at the junction of A & B. It also demonstrates the origin of branch C from the sac. Fig. 2: The small branch (D) is seen arising from the aneurysm (0.An.). Fig. 3 shows branch E at its origin from the aneurysm.
Histological study of this aneurysm shows that its wall resembles the wall of an artery, i.e. it consists of intima with continuous elastica, media and adventitia. The media in some parts is hypoplastic. A small patch of intimal thickening with reduplication of the elastica is seen in fig. 2. There is no breach in the continuity of the elastica anywhere throughout the wall of the aneurysm. The intima in all the branches both proximal and distal to the aneurysm appear healthy.

**Middle cerebral aneurysm**

Naked eye appearance.

The aneurysm originated from the distal angle of bifurcation of the middle cerebral artery (M) into its main branches A & B, i.e. the angle which receives the full impact of the blood column flowing through M. Two small branches C & D were seen arising from the summit of the sac. (The arrow indicates the direction of the blood flow).

Serial sections of the aneurysm are shown in figs. 1 & 2, Plate 3.
Fig. 1 shows the bulge of the sac at the distal angle of bifurcation of M into A & B. Portions of branches C & D are seen lying tangential to the summit of the sac.

Fig. 2 shows the sites of origins of branches C & D from the aneurysmal sac.
Histological study of this aneurysm shows an intact intima and elastica. The elastica is continuous with that of the two small branches. The media is aplastic. This is not an uncommon finding at points of bifurcation. The intima looks healthy in almost all parts of the sac except for one patch of thickening at the mouth of the sac. The arterial walls of the main branches away from the site of the sac are healthy.

To avoid needless repetition other examples of such accidentally discovered aneurysms as well as other unruptured aneurysms accompanying cases of ruptured ones are tabled in the appendix. All without exception showed a similar pattern to what has just been described.
The following facts have been established:
(1) that all the common types of the so-called congenital ("berry") aneurysms occur at points of branching or bifurcations;
(2) that all ruptured, as well as unruptured aneurysms (as will be shown later) are developed on the anterior or distal part of the angle, i.e. that part of the angle which is exposed to the impact of the column of blood flowing through the main artery;
(3) that they never bulge in a direction opposite to the side of the direction of the blood flow.

If, therefore, this is a point of arterial bifurcation, the arrow showing the direction of the blood flow, and should an aneurysm develop it would always bulge in the direction shown in the diagram,

and it would never protrude in such situations as:
This study has shown so far that the presence of small branches arising in this vulnerable angle determines the development of aneurysms. These small branches were first observed in a series of cases published by Drennan. The constant presence of these branches in this situation in all cases accompanied by aneurysms and their constant absence in otherwise normal cerebral vessels which I have examined, emphasises their importance in aneurysmal development.

The presence of defects in the media, which is not an uncommon finding at bifurcations, whether or not associated with aneurysms, cannot be taken as the sole etiological factor responsible for development of such lesions. I agree with Richardson & Hayland in their rationalisation that there must be a superadded factor which, together with the medial defect, precipitates the development of aneurysms and Glyn's belief that the internal elastic lamina is solely responsible for preventing undue expansion of the artery wall.

The question which arises is how would the origin of such small branches at the vulnerable angles of bifurcation affect the integrity of the internal elastic lamina? In the absence of these small branches the anterior angle of bifurcation is protected by a continuous sheet of reasonably
thick elastica. This can be diagrammatically shown as follows:

This sizeable sheet of elastica acts as a protective barrier against the protrusion of the angle.

If small branches are present, this sheet of elastica would be interrupted at their orifices, and around each orifice it would be continuous with the thin lining of elastica which constitutes the internal lining of these branches. This is shown diagrammatically below:

This arrangement creates multiple small, relatively weak spear-heads of elastica against the impact of a strong column of blood. The proximal parts of the arteries forming the Circle of Willis are the common sites of aneurysm - as has been already noted - because they are relatively large, and the column of blood passing through them therefore has a substantial systolic thrust.

I have also noted, as other observers e.g. Jacques and Riggs & Rupp, that in some cases in
which there is a disparity in the size of the arteries on either side of the Circle of Willis the aneurysm develops on that side of the Circle which has the larger-sized arteries. But it is important to stress that primarily it is the presence of the small branches at the angles of junction which is the most important determining factor because in their absence the unilateral discrepancy in the size alone was not accompanied by the development of aneurysms.

The analogy of the protrusion of the bulge can be found in the study of pyelograms of cases of hydronephrosis. The normal pyelogram shows the following appearance:

with the pyramids of the kidney protruding as spear-heads.

In the early stages of hydronephrosis we get the following picture:

with flattening of the apices of the pyramids,

and later the following picture develops,

with invagination of the apices of the pyramids.
The same sequence of events happens to the spear-heads of the angles of bifurcation of the small branches. A bulging of a small sac with the small branches appearing to arise from its summit would be the ultimate result of the thrust of the blood column on such a vulnerable angle of bifurcation. The analogy is true, but with one difference; that in the case of the kidney there is a normal arrangement, but an abnormal pressure, while in the case of aneurysm there is an abnormal arrangement with a normal pressure.

The relation between the clinical condition of hypertension and the development of aneurysms is not established. Many cases that I have seen, as well as cases reported, were not known to have hypertension. The presence of high blood pressure in patients who have ruptured aneurysms is actually due to the increased intracranial tension from the presence of an intracerebral or subarachnoid haemorrhage.

From what has been said, one could postulate the sequence of events in the development of aneurysms in examples one and two just described, as follows:
Case 1.
Arrangement of vessels at bifurcation during infancy.

Bulging of the angles of bifurcation with the production of aneurysms later in adult life.

Case 2.
Lastly, it must be stressed that the bulging of such small aneurysms per se does not mean that the structure of the elastica has been altered. In fact the elastica was intact throughout. It is undoubtedly stretched, but this does not mean that it has lost its properties of protecting the vessel wall. Actually, as long as the elastica is intact there is no fear of rupture or the development of symptoms. All unruptured aneurysms had intact elastica throughout the wall.

Once the elastic lamina, which is the main protective factor of the integrity of the vessel, gives way through disease, or otherwise, the fear of complications arises.

How this happens and why, should an aneurysm rupture will be discussed in the next chapter.
This work is based on the histological examination of 37 cases of either subarachnoid and/or intracerebral haemorrhages due to rupture of an intracranial aneurysm. 7 of these cases were encountered during the routine examination of 300 successive brains from the autopsy room; some were old cases which had been preserved in the Pathology Department, and the remainder were collected during the last 2 years.

12 of these cases had multiple aneurysms. One had 5, 2 had 3, and the remaining 9 had 2 aneurysms, making the total number of aneurysms available for study 54, all of which were sectioned. Of these 54 aneurysms 4 appeared to belong to a different category. These were knob-like protrusions on the supraclinoid portion of the internal carotid artery proximal to its bifurcation. They seem to be rare because no similar aneurysms were encountered in the 188 cases of aneurysms examined in the Royal Infirmary, Edinburgh, from 1929-1953. They will be described and discussed in a later chapter.

15 cases were injected with neoprene latex, using the method already described. The rest were dissected and examined without previous neoprene injection. All of them were then fixed, embedded
in paraffin and serially sectioned. Every tenth section was mounted. Some were stained with Hart's and Van Gieson, and others with Hart and Masson. A few sections were also stained with Haematoxylin & Eosin from each case.

Of the 50 so-called congenital or "berry" aneurysms, 45 developed in one or other of the more common situations, namely: The junction of the anterior cerebral and anterior communicating arteries, first or occasionally second bifurcation of the middle cerebral artery and junction of the internal carotid and posterior communicating arteries.

One aneurysm was found at the terminal bifurcation of the basilar artery, 2 at the first major bifurcation of the anterior cerebral artery distal to the anterior communicating artery, one at the stem of the vertebral artery, and one was a diffuse dilatation of the left internal carotid artery.

The topographical and histological appearances of all cases were similar and only a few examples are given here to avoid repetition. The rest of the cases are detailed in the Appendix.

As a rule, every ruptured aneurysm was
situated at the anterior distal angle of bifurcation of one, or the junction of two major vessels. The base of the aneurysm was formed by a bulge of this angle, and it involved the sites of origin of the two main arteries as well as the origin of one or more smaller arteries arising from this angle. This basal bulge, which is equivalent to the small unruptured aneurysms already described, is referred to as the basal sac or the original aneurysm (O. An.) in the microphotographs. The main sac of the aneurysm which has ruptured is referred to as the distal sac (An.). The constriction at the junction of these two sacs is that part of the aneurysm which was called "the neck" by previous observers. This was the common structure observed in most of the ruptured aneurysms, except for one case in which only the basal sac was present. This will be described and commented on in detail presently.

When unruptured aneurysms were also present some of these had the same structure as those described in the previous chapter, i.e. they consisted of a basal sac only, while others showed a basal sac and a small distal sac which could only be differentiated microscopically.
The following is a description of 11 cases of ruptured aneurysms.

A. **Aneurysms at the junction of anterior cerebral and anterior communicating arteries.**

**Case 1.**

A male, aged 36 years. Death due to subarachnoid and intracerebral haemorrhages.

The aneurysm:

![Diagram of aneurysm](image)

The macroscopical examination shows that the aneurysm consists of a bilocular sac. Its long axis is in the direction of the blood flow in the anterior cerebral artery (A). The basal locule or sac gives origin to these small branches C, D, and E in addition to the distal sac. B is the anterior communicating artery. The rest of the Circle of Willis and its major branches appear normal in structure.

Significant serial sections through the aneurysm are shown in Plate 4, figs. 1 - 7.

**Fig. 1:** A is the anterior cerebral artery proximal as well as distal to the point of origin of the aneurysm. C and D are the origins of two small branches at the level of the aneurysm.

**Fig. 2:** Here the dilated sites of origin of C and D contribute with the distended lumen of A to form the basal sac of the aneurysm. At the right upper corner of the figure the distal part of the anterior cerebral artery A is seen.

**Fig. 3:** The basal sac of the aneurysm with the contributing branches has not yet joined the distal sac. The latter appears at the left upper part of the figure.
Plate 4.
Fig. 4: Shows the origin of the third small branch Dl. from the basal sac. It also shows the site of communication between the basal and distal sacs.

Fig. 5: Shows the dilated origin of the anterior communicating artery B. which contributes to the formation of the basal sac (O.An.). The origin of branch D. and the distal sac (An.) are also shown.

Fig. 6: Shows the anterior communicating artery B. joining the basal sac (O.An.). The two arrows point to what is usually referred to as the neck of the aneurysm, but which is actually the junction between the basal and distal sacs.

The histological structure of the two sacs is markedly different. The basal sac consists of intima, elastica and adventitia. The intima is in some places thickened, especially near the origin of the distal sac. In other places it is practically normal. The elastica, though intact, varies in structure. In some areas it is reuplicated, in others is normal, while in still other areas it is atrophic. Both intima and elastica of the anterior cerebral and anterior communicating arteries at a distance from the basal sac are normal in structure. The media of the basal sac also shows wide variation from complete normality to aplasia, especially at the origin of the small branches. In some areas it shows fibrosis or hypoplasia.

In contrast the distal sac consists only of fibrous tissue. The sharp transition between the histological structure of the two sacs is seen in Fig. 7, which is taken at their junction. Here the fibrous wall of the distal sac appears to arise from the adventitial layer of the basal sac. In some parts the wall consists of one layer of dense acellular fibrous tissue; in others the inner part of the wall shows newly formed cellular fibrous tissue.

Therefore, the sequence of events in evolution of such an aneurysm is as follows:-

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.
2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.

**Case 2.**

A male, aged 59 years. Death due to both subarachnoid and intracerebral haemorrhages.

**The aneurysm:**

Macrosopic examination shows that the aneurysm has developed at the distal angle of junction of the right anterior cerebral artery (A) and the anterior communicating artery (B). It consists of a large basal sac (C.An.) and two distal sacs (An.), one of which has ruptured and led to the death of the patient. Two small branches (C. & D.) arise from the basal sac.

The significant serial sections are shown in Plate 5, figs. 1 - 6.

Figs. 1, 2 & 3: Show the basal sac (C.An.) in the angle between A. and B. It demonstrates the site of origin of branch C.
Plate 5.
Fig. 4: Shows the sites of protrusion of the two distal sacs. One of these - on the left upper corner of the photograph - has ruptured.

Fig. 5: Shows the other small branch (D) communicating with the basal sac (O.A_n.). A cross section of the unruptured distal sac is also seen. It demonstrates the junction of B. and A_1.

Fig. 6: Shows the origin of branch D. and the ruptured distal sac.

It is noted that while the basal sac consists of intima, elastica and in some parts a hypoplastic media, the wall of the distal sac consists only of a thin layer of fibrous tissue.

Patches of intimal thickening with thinning of the elastica are seen in the basal sac, especially near the sites of protrusion of the distal sacs.

The sequence of events in the development of this aneurysm can be deduced as follows:

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
Case 3.

A male, aged 34 years. Death due to subarachnoid haemorrhage.

The aneurysm:

Macroscopical examination shows that the aneurysm appears to have projected laterally and slightly upwards. The anterior communicating artery is extremely short, so that the anterior cerebral arteries form a X. Short though it is the anterior communicating artery has participated in the formation of the aneurysm.

The aneurysm, which is bilocular, shows around its base a leash of small arteries (C, D & E). The left anterior cerebral artery (A1.) approaching the aneurysm expands slightly into the base of the aneurysm and beyond it; this artery is smaller in calibre than the right anterior cerebral artery (A.).

The significant serial sections of the aneurysm are shown in Plate 6, figs. 1 - 6.

Fig. 1: Shows the distended anterior communicating artery forming the base of the aneurysm between both anterior cerebral arteries (A. & A1.). E. is the origin of a small branch arising from the basal sac. An. is the distal sac of the aneurysm.

Fig. 2: Shows the distended base of the aneurysm. D. & E. are the origins of small branches from it. The origin of the distal part of the left anterior cerebral artery (A1.) is also seen.

Fig. 3: Shows the basal sac (O.An.) with the origins of the small branches (C. & D.).

Fig. 4: Shows the origin of the branches E. & C. and the distal branches of D.

Fig. 5: Shows the basal sac (O.An.) with the protrusion of the distal sac from it.
Plate 6.
Fig. 6: Shows the communication between the basal and distal sacs of the aneurysm. The distal parts of the branches E. & D. are also seen.

The histological structure of the basal sac here shows 3 well-formed arterial coats. The media is aplastic at the origins of the small branches. Elsewhere it shows varying degrees of hypoplasia or fibrosis. The intima and elastica appear healthy in most parts of the basal sac as well as in the proximal and distal portions of the branches contributing to the formation of the aneurysm. Only in scattered areas in the basal sac, and especially near the origin of the distal sac does the intima show degenerative thickening and the elastica fragmentation and thinning.

In contrast the wall of the distal sac consists only of fibrous tissue. In some parts it is thin and formed by old acellular fibrous tissue. In others it thickens to form what appear to be two layers - an inner layer of newly-formed cellular fibrous tissue, and an outer of

At the junction of the distal and the basal sacs, there is an abrupt termination of all the arterial coats except for the fibrous adventitial layer which is continued into the wall of the distal sac.

Therefore, the sequence of events deduced from these sections would appear to be as follows:-

1. Is the initial structure at the junction, with a leash of vessels facing the column of blood coming from the wide bore artery A.

2. Shows the primary bulging at the junction due to the flattening of the angles or origins of the small branches.
3. Is the end result after protrusion of the distal fibrous sac.

B. Aneurysm at a major bifurcation of the anterior cerebral artery distal to the anterior communicating artery.

Case 4.

A male, aged 63 years. Death due to subarachnoid haemorrhage and haematoma in left frontal lobe due to ruptured aneurysm at a point of division of the left anterior cerebral distal to the anterior communicating artery.

The aneurysm:

Macroscopical examination shows that the left anterior cerebral artery (A) has divided into 3 branches, B., C., & D. The point of division was distended, forming a basal bulge (C.An.) from which the distal aneurysmal sac (An.) has protruded to one side. The latter, which is connected to the basal bulge by a constriction or neck, has ruptured and caused the death of the patient.

Significant serial sections of the aneurysm and its branches are seen in Plate 7, figs. 1 - 3.

Fig. 1: Shows the basal bulge (C.An.) formed at the bifurcation of A. into branches B. & C. Cross sections of the distal sac (An.) and the 3 subdivisions (D.) also appear.
Fig. 2: Shows the origin of An. from the basal bulge (O.An.).

Fig. 3: Shows the origin of branch D. from the basal bulge (O.An.). It also demonstrates part of the distal sac (An.).

The contrast between the structure of O.An. and An. is well demonstrated and follows the same pattern. Patches of intimal thickening is only seen in the basal sac. The intima of the arteries furthest from the aneurysm appears healthy. The sequence of events was as follows:

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
C. Aneurysm at an abnormal bifurcation of the anterior cerebral artery proximal to the origin of the anterior communicating artery.

Case 5.

A female, aged 65 years. Death due to subarachnoid haemorrhage. Definitely no past history of headaches or similar attacks.

Aneurysm at an abnormal bifurcation of the right anterior cerebral artery.

The aneurysm:

[Diagram of the cerebral artery]

The proximal part of the right anterior cerebral artery shows an abnormality. Before joining the anterior communicating artery, it has divided into two main branches. These were united again into a single branch just proximal to the site of origin of the anterior communicating artery. At the angle of bifurcation, on which the column of blood strikes, a small aneurysm was found. The diameter of its base was 2 mm. and its height was 1 mm. Two small branches were arising at the bifurcation opposite the point of impact of blood and were contributing to the formation of the tiny aneurysm. The latter had ruptured to cause a massive subarachnoid haemorrhage.

Significant serial sections of the aneurysm are shown in Plate 8, figs. 1 - 3.

Fig. 1: Shows the right anterior cerebral artery (M.) at its bifurcation into the two main branches, A. & B. From the angle of bifurcation, which is distended, the origin of the two small branches C. & D. is demonstrated.
Fig. 2: Shows the same arrangement of vessels. The site of rupture of the aneurysm is shown by the arrow.

Fig. 3: Is a deeper level showing the aneurysm at the bifurcation as well as the whole arrangement of both right and left anterior cerebral arteries (R.A.C. & L.A.C.) joined by the anterior communicating artery (Com.).

The histological structure of the aneurysm shows that it consists of intima, elastica and adventitia. The media is aplastic at the bifurcation. Patches of intimal thickening were present in the sac, especially near the site of rupture. At the point of rupture the elastica has broken and it looked as if the thin layer of adventitia ruptured under the pressure of blood, with resulting haemorrhage.

This case represents conditions in which there was death due to subarachnoid haemorrhage, and no aneurysm was detected on preliminary examination of the Circle of Willis. Only during the injection of the Circle with neoprene was the site of this tiny aneurysm detected.

This aneurysm belongs to the accidentally found unruptured aneurysm described in the previous section. It shows no distal fibrous sac. The patient here did not experience any previous attacks of headache and she died immediately after the first rupture.

It is, therefore, concluded that this case shows the link between the first group of aneurysms - which consist of all coats of an artery except the media - and the second group which ruptured and showed a basal sac like the first plus a distal fibrous sac. In this case the bleeding was so severe and the adventitia was thin and weak that it could not check the haemorrhage and form a distal sac.
D. Aneurysms at the first bifurcation of the middle cerebral artery.

Case 6.

A female, aged 46 years. Death due to both intracerebral and subarachnoid haemorrhages.

Aneurysm at the first bifurcation of the middle cerebral artery.

The aneurysm:

![Diagram](image)

Macroscopical examination shows the aneurysm to consist of a bilocular sac. The basal sac, or locule, is situated at the bifurcation of the main artery M. into its two main branches, A. & B. Two small branches C. & D. are seen arising from the basal sac.

The whole aneurysm protrudes in the direction of the column of blood coming from M.

Significant serial sections of the aneurysm are shown in Plate 9, figs. 1 - 5.

**Fig. 1**: Shows the main stem of the middle cerebral artery (M.), with one of the principal branches (A.) arising from it. The bilocular character of the aneurysm is well seen. The origin of one of the bifurcation branches (C.) is seen connected with the basal locule of the aneurysm.

**Fig. 2**: Shows M. communicating with the basal sac of the aneurysm. The dilated origin of C. forming part of the basal sac is well seen. The origin of branch A. is dilated.

**Fig. 3**: Illustrates the basal sac of the aneurysm (O./n.) formed by the dilated origins of branches A., B. & C. It also shows that the aneurysm has arisen in the direction of the blood flow in M., the main trunk of the middle cerebral artery (see also Figs. 1 & 2).
Plate 9.
The distal sac (An.) is seen arising from the basal sac.

Fig. 4: Shows the part of the basal sac formed by the dilated origin of A. The origin of the other small branch D. is seen. Portions of C. & B. appear to the right side of the figure.

The histological structure of M., A., B. & D. at a distance from the aneurysm shows no notable abnormality. That portion of branch C. appearing in the figures shows changes resembling atherosclerotic thickening of the intima and splitting of the elastica.

In the basal sac, the media in some places appears normal, and in others it is aplastic, hypoplastic or fibroid. Near the origin of the distal sac there is intimal thickening with atrophy and splitting of the elastica. The important feature of the structure of the basal sac is that it shows all coats of an artery, though these may show various degenerative changes.

In contrast the distal sac consists of one layer of fibrous tissue which is not well formed.

Fig. 5: Illustrates the junction of basal and distal sacs. The main thickness of the wall of the distal sac appears to be formed by a continuation of the adventitia of the basal sac, the media and elastica having stopped abruptly.

Therefore, the sequence of events in the development of this aneurysm was as follows:

1. Was the original appearance at the bifurcation.

2. Is the development of the basal aneurysmal sac due to the impact of the column of blood flattening the angles of junction of the different branches.
3. Shows the protrusion of the distal sac from the basal, owing to the giving way of the media and elastica.

Case 7(a).

A male, aged 60 years. Death due to subarachnoid and intracerebral haemorrhages. Past history of two spontaneous attacks of headaches.

Aneurysm at the first bifurcation of the middle cerebral artery.

The aneurysm:

![Diagram of aneurysm]

Macroscopical examination showed that the aneurysm arose at the angle of bifurcation of the main trunk of middle cerebral artery (A.) into branches A₁, B. & C. It is in the direction of the column of blood flowing into A. Its base is formed by the dilated ends of origin of the 3 branches.

Significant serial sections of the aneurysm are shown in Plate 10, figs. 1 - 6.

Fig. 1: Shows the main trunk A. opening into the sac. The dilated origin of B. is also demonstrated.

Fig. 2: Shows branch B. communicating with the aneurysm. The end of A. is also seen.

Figs. 3 & 4: Show the junction of branches A₁. & C. Both communicated with the aneurysm.

Fig. 5: Shows the distal part of branch A₁. further
away from the aneurysmal sac. (An.)

The histological structure of the aneurysm is analogous to previous ones. Here the base of the sac which is formed by the dilated origins of the 3 branches consists of the 3 coats of An. artery in various degrees of degeneration. The remainder of the sac consists of fibrous tissue.

Fig. 6: Shows the site of origin of the sac from its basal part where the intima and media end abruptly and only adventitia is continued as the wall of the sac.

Here the demarcation between basal and distal sac is not well-defined, yet there is no neck to the aneurysm. The diameter of the summit of the sac is less than the diameter of its base. It is believed that a large area of the original basal sac was degenerated and gave way, not allowing for a constriction or neck between it and the distal one.

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
Case 7(b).

An aneurysm at the first bifurcation of the middle cerebral artery.

The aneurysm:

This aneurysm was present in association with the previous case, on the contra-lateral middle cerebral artery (M). It is situated opposite the column of blood passing through M. and impingng at the bifurcation of the main trunk into 3 large branches (A, B, & C) and 2 small branches (D & E). The aneurysm has not ruptured.

Significant serial sections of the aneurysm are shown in Plate 11, figs. 1 - 4.

**Fig. 1:** Shows the aneurysmal dilatation at the end of branch M. The dilated origins of branches A, B, C & D which contributed to the formation of the aneurysm are well illustrated.

**Fig. 2:** Shows part of the aneurysm with the origins of branches A, B, C, D & E.

**Fig. 3:** Shows an extension of the middle cerebral artery (M) opening into the aneurysmal sac (O.Am.) Parts of branches E & A are seen further along. It also demonstrates the origins of branches B, C & D from the sac.

**Fig. 4:** Shows part of the sac consisting only of fibrous tissue, with no elastica marked (An.).

The histological structure of most of the aneurysm is similar to basal sac of all ruptured aneurysms showing all 3 coats of an artery. Only part of the wall between branches D & E consists mainly of fibrous tissue with no differentiation into media and intima. No elastica is demonstrated.
This part represents a protrusion of a small distal sac.

The sequence of events is as follows:

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
E. Aneurysms at the junction of internal carotid artery and posterior communicating artery.

Case 8.

A female, aged 46 years. Death due to subarachnoid haemorrhage.

Aneurysm at the junction of internal carotid and posterior communicating arteries.

The aneurysm:

Macroscopical examination of this aneurysm shows that it arises in the direction of the blood flow at the junction of the internal carotid artery (A.) and the posterior communicating artery (B.). The neck of the distal aneurysmal sac is separated from the angle of junction of these 2 arteries by a dilated basal sac. Two small branches C. & D. are seen arising from the basal part.

Significant serial sections of the aneurysm are shown in Plate 12, figs. 1 - 6.

Fig. 1: Shows the proximal part of the internal carotid artery (A.) to the right of the photograph. The dilated end of the distal part of A. is seen on the left, communicating with the aneurysmal sac.

The dilated origin of the posterior communicating artery (B.) communicates with the aneurysm.

Fig. 2: The aneurysm is arising at the junction. B. is now seen in a deeper plane.
Plate 12.
Figs. 3 & 4: The origin of the small branch C. is seen. The dilated ends of A., B. & C. are contributing to the formation of the basal sac (C.f.n.) in fig. 4. The arrows point to the neck of the distal sac.

Fig. 5: Shows the origin of branch D. from the basal sac.

The histological structure of the basal sac consists of all 3 coats of an artery. The media is aplastic at the origin of the small branches. In other parts it shows a varying degree of hypoplasia or fibrosis.

The intima and the elastica appear healthy in most parts of the basal sac as well as in the proximal parts of the branches contributing to the formation of the aneurysm, i.e. the parts furthest from the aneurysm.

Only near the site of rupture of the distal sac do they both show degenerative changes.

In contrast, the wall of the distal aneurysmal sac consists only of a thin layer of fibrous tissue, except at its base near branch C. where the fibrous wall is much thicker. In this segment the inner part of the wall consists of newly formed cellular, fibrous tissue.

The site of protrusion of the aneurysmal fibrous sac from the basal sac is shown at a higher magnification in Fig. 6, where there is an abrupt termination of all the arterial coats, except for the fibrous adventitia, which is continuous with the wall of the distal sac.

The sequence of events which has led to the development of this aneurysm can be deduced as follows:

1. Shows the initial structure at the junction prior to the formation of the aneurysm.
2. Shows the original bulging at the junction of A. & B. which mimic the small unruptured aneurysms described in the last chapter.

3. Shows the end result after protrusion of the distal fibrous aneurysmal sac.

Case 9.

A female, aged 22 years. Death due to both subarachnoid and intracerebral haemorrhage.

The aneurysm:

The patient was the youngest in this series. There was clinical evidence over a period of 3 weeks of two major intracranial subarachnoid haemorrhages before the final rupture which caused death. The patient, however, gave a history of a short sharp attack of headache two months previously.

The aneurysm is seen to arise from the junction of the left internal carotid artery (A.) and the posterior communicating artery. On the lateral side of this aneurysm there is a firm mass of thrombus measuring approximately 1 cm. in diameter and on removing this, it is seen that all that remains of the aneurysm is a small proximal portion of the distal sac forming a shallow, saucer-shaped cavity communicating by means of a narrow central
opening with the basal sac. A smaller branch (C.) is seen to arise from the basal sac.

Significant serial sections of this aneurysm are seen in Plate 13, figs. 1 - 4.

**Fig. 1:** Shows the constriction or neck between the basal sac (O. An.) and the distal sac (A. n.).

**Figs. 2, 3 & 4:** Show the relations of branches A., B. & C. to the basal sac (O. An.). A small portion of the distal sac is seen at its site of origin. At this point the elastica terminates.

The basal sac consists of all 3 coats of an artery. There are patchy areas of intimal thickening which is more marked near the point of protrusion of the distal sac. The latter consists only of fibrous tissue, which in some parts forms one layer to which organised blood clot is adherent; in other parts it consists of 2 layers, the inner being newly-formed, cellular, fibrous tissue. At the point of origin of the distal sac, the elastica and media cease abruptly. The adventitial fibrous tissue appears to be continuous with the fibrous wall of the distal sac.

The sequence of events in the formation of this aneurysm is presumably as follows:

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
Plate 13.
Another small, unruptured aneurysm measuring 2 mm. in diameter is detected at the site of the second bifurcation of the right middle cerebral artery. This is seen in Plate 14, fig. 5 as a small bulge on the anterior angle, with 3 fine twigs arising from its fundus. The bulge has intact elastica all around it, and has a similar histological structure to the aneurysm described in the previous chapters.

Case 10(a).

A male, aged 67 years. Death due to subarachnoid haemorrhage.

The aneurysm:

![Diagram of aneurysm](image)

Macroscopical examination showed that the aneurysm arose at the angle of junction between the internal carotid artery (C.) and the posterior communication artery (P.), where the column of blood in C. impinged. The aneurysm showed a basal portion and a distal portion. A small branch (B.) arose from the basal portion. The cerebral vessels showed marked generalised arteriosclerotic change. There was diffuse dilatation of the contralateral internal carotid artery.

Significant serial sections of this case are shown in Plate 15, figs. 1 - 5.

Figs. 1, 2 & 3: Show both the basal portion of the aneurysm (C.Am.) with branches C., P. & B. opening into it, and the distal fibrous sac, which has ruptured. It also demonstrates the distal part of the internal carotid artery (C.) away from the site of aneurysm, with its main divisions into M. & A.
Plate 14.
Fig. 4: Shows both portions of the aneurysm occurring at the junction of C. & P.

Fig. 5: Shows the site of rupture of the distal sac.

The histological structure conforms with the usual pattern, except that there is marked arteriosclerotic change in the basal portion of the sac as well as in the branches contributing to its formation.

The sequence of events is as follows:

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
F. Aneurysm at the terminal bifurcation of the basilar artery.

Case II.

A female, aged 60 years. Death due to subarachnoid haemorrhage.

The aneurysm:

Macroscopical examination shows that the aneurysm is situated at the angle of bifurcation of the basilar artery (B.) into the right (P1.) and the left (P2.) posterior cerebral arteries. The left superior cerebellar artery (C.) arises in common with the left posterior cerebral artery (P2). Two other perforating branches (E. & D.) arise at the bifurcation, which is also unusual. The swollen basal sac of the aneurysm is formed by the distended origins of P1, P2, D, E, and C vessels. The blood flowing through the basilar artery was impinging on the angles of bifurcation of all these branches which arise at nearly the same level. The distal sac of the aneurysm is relatively small, and has ruptured to cause a subarachnoid haemorrhage.

Significant serial sections of the aneurysm are shown in Plate 15, figs. 1-6.

Fig. 1: Shows the origin of the left superior cerebellar artery (C.) from the terminal end of the basilar artery (B.). Part of the distal fibrous sac is seen towards the top of the figure.

Figs. 2 & 3: Show the dilated basal sac of the aneurysm formed by C., P2, and the origin of P1. The communication between the basal and the distal sacs is well illustrated. A small branch (E.) is seen arising from the proximal end of P2, in fig. 2.
Fig. 4 & 5: Show branches P₁. & D. arising from the basal sac. Part of the distal fibrous sac is communicating with the basal sac.

Fig. 6: Shows more distal cross sections of branches P₁. & P₂. with part of the distal sac of the aneurysm between. Two other small perforating branches are also shown in cross section; these arise from P₁. & P₂. distal to the point of bifurcation, and do not contribute to the aneurysmal dilatation.

The histological structure conforms with the previous pattern. The marked contrast between the thin, one-layer fibrous wall of the distal sac and the arterial-like wall of the basal sac is well demonstrated.

The mode of formation of the aneurysm would appear to have been as follows:

1. Shows the presumed structure at the junction before any aneurysmal dilatation occurred.

2. Shows the bulge at the angle of junction between A. & B. due to the impact of the blood column on the angles of origin of the small branches, causing them to flatten and then to bulge.

3. Shows the protrusion of the distal from the basal sac after media and elastica have given way.
CONCLUSIONS AND COMMENT.

From the histological study of serial sections of all the ruptured aneurysms the following observations can be made.

(1) All aneurysms developed at points of junction of two arteries, or at points of bifurcation.

(2) Like the unruptured aneurysms they always protruded from the anterior or distal angle of bifurcation, namely that part of the angle which was opposite the thrust of the blood column.

(3) Every aneurysm consisted of two parts - a basal bulge or sac, and a distal sac. The relative sizes of these two compartments varied. They might be of equal size and hence the aneurysm appeared bilocular as in Case 2. The distal sac might be smaller than the proximal one, as in Cases 3 and 11, or vice versa - see cases, 1, 6, 8, 9, and 10(a). The two sacs were usually separated by a constriction which was referred to as the neck of the aneurysm. Sometimes, however, there was no line of demarcation or neck between both sacs, e.g. Case 7(a).

The basal sac was always due to the distension of the angle of junction or bifurcation. It was semi-lunar in shape, with the widest circumference at its base. The distal sac protruded from any
part of its wall. If this point of origin was narrow there resulted a distal sac separated by a constriction or neck from the basal one, e.g.

On the other hand, the distal sac might have a wide neck of origin, and then there was no line of demarcation between the two sacs macroscopically, e.g.

In such cases what is usually called the neck of the aneurysm was its widest part. The distinction between the two sacs could only be identified histologically - see Case 7a.

These observations explain the fact that some aneurysms are bilocular and that others do not show a constricted neck.

(4) There was a marked discrepancy between the histological structure of the wall of each sac. The wall of the basal sac or bulge consisted of some or all of the coats of an artery, namely intima with elastica, media and adventitia. The media, however, might be completely absent or might show hypoplasia or fibrosis. This is not unexpected in view of the fact that the basal sac represents the arterial wall at the angle of junction or bifurcation. The intima was either normal in appearance, or more usually in the ruptured aneurysms it showed areas of thickening, with thinning or
reduplication of the elastica. These intimal changes were seen to be particularly marked at the site of origin of the distal sac.

The wall of the distal sac, on the other hand, had a totally different structure; it consisted only of fibrous tissue. At its site of origin from the basal sac there was an abrupt cessation of the intima, elastica and media (if present). One or sometimes more elastic fibres might, however, extend for a short distance into the wall of the distal sac; this was rarely seen. The only part of the arterial wall of the basal sac which appeared to be continued into the distal sac was the fibrous adventitia.

Another characteristic of the wall of the distal sac was its variable appearance. It might be a thin layer of fibrous tissue, e.g. Cases 6 and 11, or a thicker layer, the inner part of which consisted of newly-formed cellular fibrous tissue, which gave the impression of a second inner layer. The endothelial lining of the basal sac appeared in many instances to cover this inner surface of the distal sac. It was, however, difficult to prove that the inner lining of the distal sac, when present, was in fact endothelium. It always stained like fibrous tissue, when either Masson's or Van Gieson's counterstains were employed. In some cases, e.g. 1, 2 and 6 when the distal sac had an
inner lining resembling endothelium, it was not continuous with the endothelial layer of the basal sac.

(5) The basal sac had the same histological structure as the unruptured aneurysms described in the previous chapter. It had arisen in the anterior or distal vulnerable angle of junction or bifurcation; in every case one or more small branches were seen arising from it; its wall was arterial in structure. Therefore, it had the same mode of origin as the unruptured aneurysms.

Its constant presence - even if only microscopically - in all cases of ruptured aneurysms suggested that it was an essential precursor to the development of the aneurysm. It was actually the original aneurysmal sac (O.An.) from which arose the distal fibrous-walled sac.

(6) Many authors have postulated that the fibrous nature of the wall of an aneurysm was due to the gradual distension of the angle of bifurcation of an artery, followed by gradual atrophy of the elastica and media, which were replaced by fibrous tissue. The constant presence of at least two sacs of dissimilar structure in the series examined, refuted this theory, and in addition would seem to prove that originally the aneurysm consisted of the basal sac alone, and as long as the elastic lamina - which is the main safeguard
against any breach in continuity of the arterial wall - of this basal sac is intact, no rupture and no formation of a distal sac is probable.

If at some point, however, this elastic layer degenerates or is torn, nothing could check a free haemorrhage except the adventitial layer. The subsequent stretching and distension of the adventitia by the escaping blood would result in the formation of the distal fibrous sac.

The constant finding of haemosiderin, indicative of haemorrhage at some previous date in the wall of the aneurysm at the junction of basal and distal sacs in all of 9 cases in which sections were stained by Prussian Blue method, supports the theory that the distal sac develops by sudden eruption from the basal sac after the elastica of the latter has been torn at some point.
Fig. 7 taken from Case 2 illustrates this feature. It shows haemosiderin and R.B.Cs. in between the adventitia and the inner coats of the basal sac at the point of origin of the distal sac.

It is well established that the nervous plexus of the vasa nervosum is present mainly in the adventitia. Stretching of this by the formation of the distal sac is probably responsible for the paroxysmal attacks of headaches from which these patients usually suffer before the final rupture.

In the early stages of its formation, the wall of the fibrous sac is presumably a thin layer of well-formed fibrous tissue, being formed of stretched adventitia, which might resist tearing. Later granulation tissue forms in the inner surface of this layer in an attempt to close the cavity of the fibrous sac. This may succeed in preventing a final haemorrhage. The aneurysm would hence appear as a bilocular unruptured aneurysm which is partly fibrous and partly arterial in structure as in Case 7(b) and other similar examples in the Appendix.

The thin fibrous wall may, however, have insufficient time to proliferate and obliterate the cavity of the distal sac and hence any sudden increase in the force of pulsation of the blood column as a result of a physical or emotional strain may rupture the thin, fibrous wall with the development of a sudden
massive intracranial haemorrhage, which would lead to the patient's death. The resulting aneurysm here would be a thin-walled, fibrous distal sac, such as was found in Cases 2, 6 and 11.

If the final haemorrhage is delayed there is time for the fibrous sac to become thickened by the development of what appears to be an inner layer of cellular, newly-formed fibrous tissue, e.g. Case 1.

One meets, however, with aneurysms which are multilocular, showing a mulberry appearance. One trilocular anterior communicating aneurysm is shown in the Appendix. These aneurysms were basically formed of two sacs, a basal arterial and a distal fibrous. If the distal sac was allowed some time to develop a thick wall, a momentary rise in the pressure of the blood within the sac might only succeed in tearing an area of the inner layers of the distal sac. This would lead to a localised bulging of the intact outer layer of the sac at that area. Multiple areas of bossings on the surface of the aneurysm would be so developed and might ultimately be filled with adherent thrombi, or one may give way and lead to a profuse haemorrhage.

Another cause of the development of a multilocular aneurysm is the protrusion of more than one distal sac, as in Case 2.
The distal sac may not develop at all if the break in the elastica of the proximal sac is followed by a strong gush of blood which the adventitia is too weak to check. The patient would then die from intracranial subarachnoid haemorrhage without the development of any sizeable aneurysm. Case 5 is a typical example in which the basal sac, consisting of all the coats of an artery, was so small that it was only detected after neoprene injection of the cerebral vessels. This observation might explain the many cases of basal subarachnoid haemorrhage in which no aneurysm was detected on superficial examination of the cerebral vessels.

Mitchell & Angrist (1943) described two cases of subarachnoid haemorrhage where no aneurysm could be found macroscopically. The sites of rupture were shown by injecting the cerebral vessels, using Dr. Richard Griner's method. The site of rupture was demonstrated by the escape of a thin stream of water.

Five such cases were met with in the necropsy files of the Royal Infirmary, Edinburgh. A description of one case examination by Dr. Neil Maclean, Neuropathology Laboratory, Edinburgh, is cited as an example.
"A male, aged 24 years. Death due to both intracerebral and subarachnoid haemorrhage.

Macrosconic Examination: A moderate subarachnoid haemorrhage is present over the lower half of the brain stem. The vessels at the base are free from atheroma and no evidence of an aneurysm could be detected on superficial examination, but as will be described later, an aneurysm is present at the bifurcation of the middle and anterior cerebral arteries. Serial coronal section of the cerebrum reveals a recent haemorrhage lying in the lower part of the corpus striatum on the right side. Further dissection of the inferior part of the corpus striatum on the right side where the haemorrhage lies, shows that this haemorrhage communicates with the under aspect of the cerebrum, and on carefully removing the adherent blood clot from the region of origin of the right anterior and middle cerebral arteries, the ruptured remains of a sessile ovoid aneurysm, probably measuring 0.5 cm. in its long axis, was discovered, i.e. the source of haemorrhage would appear to be from this aneurysm.

On microscopical examination of a portion of "fibrous" tissue from the site of the aneurysm it is found to consist of leptomeninges showing the usual reaction to recent subarachnoid extravasation of blood, and numerous collagenous strands not of leptomeningeal origin. It would, therefore, appear that the aneurysmal sac, probably formed by adventitia only, was of recent formation and had been largely disrupted by the final haemorrhage. There is no evidence of a vascular inflammatory process."

Both this case and my Case 5 gave no past history of attacks of headaches and they suffered only from one attack of loss of consciousness which ultimately led to their death.
The question now arises as to why the elastic lamina of the basal sac should give way or degenerate and lead to a haemorrhage or to the formation of the distal sac.

The frequency of the presence of patches of intimal thickening with underlying thinning, atrophy or reduplication of the elastica in the basal sacs as well as in the unruptured aneurysms was very striking.

In cases where there is a generalised arteriosclerosis of the whole cerebral vasculature these patches could be explained as being arteriosclerotic in origin. The exact nature of arteriosclerosis is still not yet known. In cases showing no arteriosclerosis, however, it is inconceivable to attribute these localised patches of intimal thickening to such a generalised pathological condition.

The pooling of blood in such recesses as the unruptured aneurysm and the initial basal sac of the aneurysm which later ruptures is accompanied no doubt by slowing of the blood flow in these recesses as compared to the rate of blood flow past the normal sharp angles of bifurcation. This would probably entail an increased tendency for the blood to clot in these situations. Also it can be assumed that if there were an increased tendency
for intravascular coagulation it would occur primarily in such situations due to the abnormal stretching of their endothelial lining as well as to the slowing of the circulation.

Duguid in 1946 observed that "When a thrombus forms in an artery it adheres to the wall, the endothelium disappears and there is an invasion of the mass by connective tissue cells from the intima. These bring about a progressive transformation of the outer layers of the thrombus into fibrous tissue, so that an advancing zone of fibrosis is formed which over-runs and obliterates the original line of demarcation between thrombus and intima. The effect is an appearance of fibrous thickening. At the same time blood vessels penetrate into the interior of the thrombus accompanied by connective tissue cells which gradually transform it into fibrous tissue. Complete organisation may thus be effected. A new layer of endothelium readily grows over it."

These observations were also confirmed by Heard (1949) and by Crawford and Levene (1952).

The following photographs show some of these early intimal patches encountered in the basal sacs of certain aneurysms and they illustrate that they are actually thrombi undergoing organisation. They are similar to the microphotographs shown in the papers by Duguid, Heard and Crawford.
The mechanism of intravascular thrombosis is still unknown, and it is not the main concern of this work. A possible explanation was suggested by the studies of MacFarlane and his colleagues (1946) and Mole (1948). Mole believes that under normal circumstances fibrinolysin is formed by vascular endothelium and dissolves any fibrin clots that may be formed upon it, but that in infection and cachexia fibrinolysin formation is inhibited.

The presence of thickened, fully organised fibrous patches in the intima may deprive the underlying elastic layer of its nutrition, by acting as a barrier between it and the nutritive blood in the lumen. This would lead to atrophy and degeneration of the distended elastica of the basal sac. The field would then be set for the formation of the distal aneurysmal sac.

The following photograph shows the same process of clot organisation taking place in an adherent clot on the fibrous wall of the distal sac of an aneurysm. It is, therefore, possible that the inner cellular, newly-formed, fibrous layer sometimes seen in the wall of the distal sac of an aneurysm is so developed.
On the other hand the vascularisation of the deeper part of the thrombus, as already stated, may provide a sufficient blood supply for the nutrition of the elastica. It is possible, therefore, that fracture of the elastica is the first incident, to be followed by thrombosis which is later organised. This fracture of the elastica may well be due to the overdistension produced by the development of the basal sac. This formation of sac due, as already shown, to the special origin of branches at the bifurcation and impulse of blood on these, would increase the stress on the elastica to a point when it eventually breaks.

This last argument does not explain why the elastica of many unruptured aneurysms does not give way during life, in many cases to produce a distal sac
even in quite elderly subjects. In fact the elastica is never seen to be atrophied or reduplicated unless it is underlying patches of fully organised intimal thickening. If the intima is normal in appearance so is always the underlying elastica. It has also been observed that at the site of origin of the distal sac the intima of the basal sac is always thickened.

It is, therefore, concluded that the elastica only degenerates secondary to these changes in the intima.

It is true that in the early stage of organisation of an adherent thrombus, capillaries infiltrate the blood clot. Whether these capillaries penetrate from the adventitial plexus, as has been thought by Gross, Epstein, Kugel & Geiringer, or whether they are only in communication with the lumen of the artery (Robertson, Winternitz, Thomas & Le Compte), the end result is complete fibrous organisation.

Ramsay (1936-37) has demonstrated that the inner third of the media and all of the normal intima derive their nutrition from the arterial lumen. It is known that most of the elastic fibres of the cerebral arteries are concentrated in the intima and hence are supplied from the vascular lumen.
Thoma quoted by Adami in the Principles of Pathology, 1910, has demonstrated that during life patches of intimal thickening do not actually project into the lumen, but are evaginated into the wall of the arteries being so compressed by the arterial blood pressure.

It is therefore assumed that whether these fully organised patches of intimal thickening act by depriving the underlying elastica of its nutrition from the lumen or whether they merely exert pressure on it, the latter would ultimately degenerate. This would mean that the factors responsible for the breaks in the elastica of the basal sac (which is followed by the formation of the distal sac) are those which determine the onset of intravascular clotting and its severity.
In conclusion, the mode of production of such ruptured aneurysms in my opinion is as follows:-

(1) Developmental presence of one or more small branches at the anterior or distal angle of an arterial bifurcation or junction present since birth.

(2) This developmental anomaly precipitates the gradual bulging of the angle which usually becomes apparent in early adult life. The bulge forms a small aneurysm which has the structure of an arterial wall with intact elastica. This is in fact the precursor or original aneurysm.

(3) Clotting of the blood in this aneurysm, whether due to slowing of the circulation, physiological alteration in the distended intima, or to other unestablished factors, produces a thrombus which adheres to the wall.

(4) Organisation and endothelialisation of the clot results in fibrous patches of intimal thickening.

(5) If this thickening is sufficient to affect the nutrition of the elastic lamina, the latter degenerates and breaks up.

(6) This causes rupture of the inner part of
the wall of the original aneurysm.

(7) One of two things may now happen.
(b) More usually, the escaping blood stretches and distends the overlying adventitia, with the production of headache and the formation of a thin-walled distal fibrous sac. Clotting of the blood in the latter is followed by invasion of the thrombus with granulation tissue in an attempt to obliterate the sac. The wall of the distal sac thus becomes thickened. Sudden periodic rises in the force of the blood pressure may cause rupture of this sac which is inelastic, either when it is newly-formed or at a later date. This results in the development of a subarachnoid or intracerebral haemorrhage or both.

If the fibrous sac is thick enough it may resist rupture for a long time and thus explains the incidental finding of non-fatal bilocular, partially fibrous aneurysms. (See Appendix).
(b) If the rupture of the original basal sac is followed by a strong gush of blood, the bleeding may be so severe as to tear through the adventitia at once and the patient suffers apoplexy and may die. This explains those cases of subarachnoid haemorrhage in which no aneurysm was encountered on superficial examination.

The inability to find such small original
bulges in children proves that aneurysms are acquired lesions which develop as a result of a developmental abnormality or peculiarity of branching at bifurcations or junctions.

The name I would suggest for such an aneurysm, whether comprising a basal sac alone, or a basal plus a distal sac, is a "Developmental bifurcation aneurysm" and not a "Congenital aneurysm" because, although it is based upon a congenital vascular anomaly, development of the actual aneurysm does not occur till later in life.
SACCULAR ANEURYSMS OF THE SUPRACLINOID PORTION OF
THE INTERNAL CAROTID ARTERY PROXIMAL TO THE
POSTERIOR COMMUNICATING ARTERY.

This is a very rare type of aneurysm. None was
encountered in the Pathology Department of Edinburgh
until 1953. I found only two cases, each showing
two such aneurysms and associated with the common
type of developmental aneurysms of the Circle of
Willis. In one case there were two saccular
aneurysms, one on each internal carotid artery, and
two developmental aneurysms, the first on the
anterior communicating artery, and the second on the
right middle cerebral artery.

In the other case the two saccular aneurysms
were both found on the right internal carotid artery
and there was also a developmental aneurysm on the
anterior communicating artery.

These saccular internal carotid aneurysms
appeared as small "nubbin" protrusions from the wall
of the artery. They were not related to any
point of branching or bifurcation and no small
branches were associated with them. Their site of
origin was also of interest. They arose from the
posterior wall of the curvature of the internal
carotid artery as it leaves the cavernous sinus, to
turn backwards before dividing into its terminal

A third case was encountered later in which a
supraclinoid left internal carotid aneurysm was an
isolated incidental finding. Death was not due to
a cerebral lesion, and no other aneurysms were found.
branches.

Diagrammatically:

A photograph of the second case showing this aneurysm is also shown.

It is noted, therefore, that they take origin from that side of the wall that is not exposed to the thrust of the blood column.

The aneurysms that I found varied from between 2 - 5 mm. in length and they were all about 2 - 3 mm. in diameter.
The 4 aneurysms did not rupture and did not show any evidence of previous rupture. They were also free from adhesions to surrounding structures.

Very few such aneurysms have been reported in the literature. Dandy (1944) was the first to draw attention to them; he reported 9 cases only. Careful analysis of their histories showed that none had ruptured to cause an intracranial haemorrhage before surgical interference. His Case number 13 died of intracranial haemorrhage, but in fig. 10 from his paper the aneurysm shows no evidence of rupture and there is nothing to indicate that this aneurysm was the source of the fatal haemorrhage. No details of the autopsy were given. Although the aneurysm in this Case pointed anteriorly it appears from the figure to have originated from the posterior wall of the curvature of the internal carotid artery.

Hemby described one such case which had not ruptured.

Albright (1929) reported 2 internal carotid artery aneurysms and collected other 30 cases from the literature. His Case 32 (McCordock) showed one such aneurysm in fig. 8(a). This aneurysm was associated with 2 developmental aneurysms. It had not ruptured and evidently did not give rise to symptoms.
Most other supraclinoid internal carotid aneurysms reported in the literature fall either under diffuse arteriosclerotic dilatations of the vessel, or under developmental aneurysms in relation to arterial bifurcation.

Professor Dott told me that he remembered noticing on the internal carotid artery during one of his operations a "nubbin" which he could press down and invaginate with forceps. But after a period of compression it always bulged out again. It had no relation to any branching or bifurcation.

Serial sections through each of the 4 saccular aneurysms which I encountered in the 2 cases already mentioned showed identical findings. An example is given in Case 1, Plate 17, figs. 1 & 2.
The patient was 58 years, and had marked cerebral arteriosclerotic changes. Death was due to rupture of a developmental anterior communicating aneurysm.

Figs. 1 & 2: Show that the aneurysmal wall is of the same structure as the wall of the internal carotid artery with the three coats. It actually appears as a branch of the artery with a closed end. Marked arteriosclerotic change is seen in both the aneurysm and the internal carotid artery, with thickening of the intima, and though the elastica is continuous all round the aneurysm, it shows splitting, thinning and reduplication, which are due to the arteriosclerosis. The media shows areas of fibrosis.

From their site, the absence of any relation to bifurcation, the absence of small branches, and their clinical behaviour, it is suggested that their aetiology is different from the common type of developmental aneurysm often encountered.

SACCULAR ANEURYSMS of the VERTEBRAL, BASILAR, and POSTERIOR INFERIOR CEREBELLAR ARTERIES UNRELATED to BIFURCATIONS:-

Bandy reported 6 such cases of his own, and he encountered only 12 more cases in the literature. It is interesting to note that most of his cases
were in the younger age group.

The majority of the 14 cases of aneurysm reported in children below the age of 10 years occurred on the vertebral, basilar or internal carotid arteries; only 2 were found on the intracerebral arteries. The youngest patient reported was aged 2.5 years, and had an aneurysm on the vertebral artery. (Dial & Maurer.)

Most of these cases presented as intracranial space occupying lesions. They attained a huge size, and rarely caused death by rupture.

I encountered only 2 such cases, one of which I was able to examine by microscopic serial sections. This was a large vertebral aneurysm encountered in a patient who had multiple developmental aneurysms, one of which had ruptured, and who also had 2 saccular internal carotid aneurysms. The other was a large aneurysm of the posterior inferior cerebellar artery which presented clinically as an acoustic neurinoma.
Plate 18.
The serial sections demonstrate that the aneurysm arises from the main trunk of the vertebral artery distal to the origin of the postero-inferior cerebellar artery, i.e. it is unrelated to any bifurcation. It springs from a dorsal slit on the stem of the vertebral artery and projects dorsally, being embedded in the cerebello-medullary angle.

Most of the wall consists of fibrous tissue, and the lumen contains a firm, organising blood clot. It is significant, however, that slender degenerated elastic fibres are scattered in different areas around the wall, as shown in Plate 18. This indicates that the wall of the sac was of arterial origin, and had undergone fibrosis and degeneration of the media and elastica, probably due to the organisation of the blood clot. There was nothing to indicate that it represented a distal sac, because in the latter no elastica can even be found at any point in its wall.

It is possible that these saccular internal carotid, vertebral and basilar aneurysms, as Padget & Sugar have suggested, represent vestiges of embryological capillary vessels, most probably of the primitive carotid: basilar system anastomosis and the primitive dorsal and ventral
ophthalmic vessels, as has been explained by Sugar.

It is probable that these aneurysms have not ruptured because they did not develop in areas of the vessel wall which are exposed to the thrust of the blood column.
Finally, a word should be said about the diffuse arteriosclerotic dilatation of the supraclinoid portion of the internal carotid and the basilar arteries.

This type is not commonly met with; I was only able to find 2 such cases over the last 2 years, both of which were examined histologically. The first case of diffuse dilatation of the internal carotid was met with in Case 10, page 128. The patient was a female, aged 67 years at the time of death. She died from a ruptured developmental aneurysm at the junction of the right internal carotid and posterior communicating arteries. The contralateral internal carotid artery showed diffuse arteriosclerotic dilatation. No history was obtained from the patient, because she was admitted in an unconscious state. At autopsy there was no indication that the dilatation of the left internal carotid artery had caused any symptoms during life. It had not ruptured.
Histological examination of serial sections of the wall of the aneurysm shows marked arteriosclerotic change. The intima is much thickened, and there was an accompanying partial occlusion of the artery by recanalised thrombosis. The elastica is in some parts reduplicated, in others thinned out and split. Secondary fibrotic changes are marked in the media and the adventitia is thickened. (see Fig. 9).

The second case was that of a female aged 63 years, who died as a result of thrombosis within a diffusely dilated basilar artery. The thrombus had propagated to occlude the posterior cerebral arteries and smaller branches of the basilar artery with consequent infarction of thalamic nuclei and midbrain.

The cerebral arteries were diffusely arteriosclerotic, and the basilar artery was dilated and tortuous, and its lumen was filled with a thrombus.

Fig. 10.
Serial sections show the same marked arteriosclerotic changes in the wall of the basilar artery. The lumen is almost completely filled with ante-mortem thrombus, reducing it to a mere slit. (See fig. 10).

These diffuse dilatations are relatively rare. Among the 138 cases encountered in Royal Infirmary, Edinburgh, there were only 8 cases: 7 were on the basilar artery, and 1 on the internal carotid artery. They were all reported to have been filled by ante-mortem thrombus, and the cerebral arteries showed diffuse arteriosclerosis. All patients were above the age of 55, and it is interesting to note that they all occurred in females.

Hamby in his series reported one case in the internal carotid artery of a male aged 72 years.

Dandy, among his 120 cases, had 6 patients with such lesions on the internal carotid artery; 3 were males and 3 females; and 11 patients with dilatations of the basilar artery; 6 were males and 5 females.

The characteristics of these diffuse dilatations are:
(1) They occur in elderly people, who always have severe diffuse cerebral arteriosclerosis.
(2) All the cases encountered in the Royal
Infirmary, Edinburgh, including the 2 cases personally examined, were almost completely filled with ante-mortem thrombus.

(3) They never rupture and only give rise to symptoms either from pressure on surrounding structures or from thrombosis.

The mechanism of production of such diffuse dilatations is the basic weakening of the arterial coats, especially the elastica, by arteriosclerosis.

The superadded thrombosis, causing partial occlusion, would mechanically cause a local rise of pressure. This would press the thrombus against the weakened arterial wall, which would yield by dilatation. In addition, the organisation of the thrombus is accompanied by further intimal thickening and elastic degeneration.
SUMMARY

188 cases of ruptured intracranial aneurysms of the Circle of Willis encountered in the Royal Infirmary, Edinburgh from the year 1929 to 1953 are analysed.

A routine examination of the cerebral vessels in 300 consecutive autopsies was undertaken. 150 brains of these were injected with neoprene latex. In 38 cases out of the total 300 brains, aneurysms were found of which 7 had ruptured. The rest were asymptomatic. 5 of the 31 asymptomatic cases had multiple aneurysms. The total number of asymptomatic aneurysms encountered was thus 36, all of which have been serially sectioned.

A routine examination of the cerebral arteries of the brains of 50 children ranging in age from a few days up to 9 years, was also made. No aneurysms were encountered in them.

37 cases of either subarachnoid and/or intracerebral haemorrhages due to rupture of an intracranial aneurysm were examined histologically by serial sectioning. 7 of these cases were encountered during the routine examination of the 300 consecutive brains from the autopsy room; some were old cases which had been preserved in the Pathology Department and the remainder were collected during the last two years. 12 of these cases had multiple aneurysms,
making the total number of aneurysms available for study 54, all of which were sectioned. Only the first 15 cases were injected with neoprene.

The following conclusions were drawn:

(a) Cerebral aneurysms are more frequent than is generally thought.
(b) The majority of the cases develop symptoms due to complications between the ages of 40 - 60 although some may do so in the early twenties and also above the age of 70. They are very rare in children below the age of 10, and those encountered appeared to be of a different type.
(c) The lesion is only slightly more prevalent in women than in men.
(d) The commonest site in which they are found is the junction of the anterior cerebral and anterior communicating arteries. The second two common sites are at the first bifurcation of the middle cerebral artery, and the junction of the internal carotid and posterior communicating arteries. Other sites are comparatively much less frequent. There is not a significant discrepancy between the right and left sides of the Circle of Willis.
(e) About 15% of cases have multiple aneurysms.

The evidence accumulated from the study of the brains of the 300 consecutive adult autopsies, the 50 children, as well as the subsequently selected cases showing ruptured aneurysms has shown the
following:

(a) The Circles of Willis upon which aneurysms — ruptured or unruptured — were found, showed a different and very characteristic mode of branching compared with those showing no aneurysms.

(b) If a smaller branch or branches are present at an anterior angle of junction or bifurcation of the major arteries of the Circle of Willis, namely that angle which is opposed to the constant thrust of the column of blood, they are almost always associated with an aneurysm or bulge at this site. This was the rule in all adult patients examined above the age of 22.

(c) If this type of branching occurs in more than one situation there will be more than one aneurysm.

(d) All cases of the usual type of aneurysm arose at angles of junction or bifurcation which were opposite to the impinging column of blood. They were never found on any of the other sides of the angles nor in other situations.

(e) The small branch or branches were usually found to arise from the summit of the unruptured aneurysms, and from the basal part of the ruptured ones.

(f) No such aneurysms were present in my series of children's brains nor in an other larger series examined routinely by Riggs and Rupp.
(g) 12\% of the Circles of Willis of the children examined showed the same branching abnormality which was found associated with aneurysms in adults. Yet no aneurysms were present in any of these cases.

(h) It is therefore concluded that the common type of aneurysm frequently encountered is actually an acquired lesion developing on a basis of a congenital developmental anomaly.

The name suggested is "Developmental bifurcation aneurysm".

(i) It is explained why a defect in the media alone at the angles of bifurcation cannot be the only cause for the protrusion of an aneurysm, and that the internal elastic lamina is the main barrier preventing the bulge of the artery.

(j) The mechanism by which the presence of small branch or branches, at the angles of bifurcation, weakens the elastica is discussed.

The histological examination of both ruptured and unruptured aneurysms has shown the following:

(a) That most of the unruptured aneurysms consisted of a distended arterial wall, i.e. their wall was composed of intima, internal elastic lamina, a hypoplastic or aplastic media and adventitia. Every coat was continuous with the corresponding coat of the main divisions of a bifurcation as well as with the coat of the smaller branches. The intima might
contain patches of thickening with underlying reduplication of the elastica. In cases showing no generalised, cerebral arteriosclerosis these patches were only localised to the aneurysm.

(b) All ruptured aneurysms as well as some unruptured ones, were noted to consist of a basal portion or sac identical in structure to the unruptured arterial ones - with one or more smaller branches arising from it - and of a distal sac consisting only of fibrous tissue. The latter was found to be continuous only with the adventitial layer of the basal sac.

(c) It is suggested that the basal sac which represents the arterial unruptured aneurysm is actually the original aneurysm from which protrudes the distal fibrous sac. The mechanism of the development of the distal sac which depends upon a degeneration of part of the distended elastica of the basal sac is discussed.

(d) It is explained how in some cases the original aneurysm or basal sac may rupture and lead to intracranial haemorrhage without the formation of a distal sac.

(e) Comment on how multilocular aneurysms are formed is also given.

A different type of aneurysm found on the supraclinoid portion of the internal carotid artery and on the vertebral or posterior inferior cerebellar
artery was encountered. These aneurysms were saccular; had not developed at sites of junction or bifurcation and had not protruded opposite the direction of the blood column. No small branches were associated with them and none had ruptured. Their wall was found to consist of all the coats of an artery. The elastica was present everywhere.

The fact that the majority of aneurysms reported in children below the age of 10 were found in such situations and that these aneurysms had developed at the sites of primitive vestigial vessels suggests that they are actually congenital aneurysms formed by the incomplete involution of the embryological vessels. This type of aneurysm is very rare.

The diffuse arteriosclerotic dilatations of the basilar and the internal carotid arteries are commented upon. It is explained why it is not advisable to consider such a diffuse dilatation and tortuosity under the term aneurysm.

Finally, the following classification is suggested for intracranial aneurysms:

1. Developmental bifurcation aneurysms. They form the great majority. They are acquired lesions developing on a congenital or developmental anomaly.
Other very rare types are:

2. Congenital saccular aneurysms of the supraclinoid portion of internal carotid arteries and the vertebral arterial system, not related to bifurcation.

3. Mycotic aneurysms mainly on the cortical vessels. These are now rarely encountered since the antibiotic era.

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References.


Appendix

Cases of Unruptured Asymptomatic Aneurysms

The following are some of the cases of aneurysms encountered during the routine examination of cerebral vessels. They gave rise to no symptoms or signs of their presence during life. Death in these cases was not due to rupture of a cerebral aneurysm.

Case 1. Female aged 64 yrs.

Asymptomatic aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

![Diagram of an aneurysm at the junction of anterior cerebral and anterior communicating arteries.]

Naked eye examination: The aneurysm originated at the junction of the left anterior cerebral artery (A) and anterior communicating artery (B). At this angle of junction and from the summit of the aneurysm three branches C, D & E arise. Branch (C) is of a sizeable calibre and forms what was called by De Vriese "the artery of the corpus callosum". It is almost as large as the two anterior cerebral arteries.

Serial sections of the aneurysm are seen in Plate I, figs. 1 - 4.

Fig. 1: Shows the aneurysm (An.) arising from the left anterior cerebral artery (A) at the origin of the anterior communicating artery. Part of the contralateral anterior cerebral artery (A₁) is also seen.
Fig. 2: Shows small branch (E) arising from the aneurysm.

Fig. 3: Shows the origin of branch (D) from the aneurysm. The lumen of A1 is also seen.

Fig. 4: Shows branch (C) arising from the aneurysmal dilatation. It demonstrates the anterior communicating artery (B) joining A1.

The histological structure of the wall of the sac follows the same pattern of unruptured aneurysms as well as that of the basal sac of the ruptured ones.

Case 2. Female aged 34 yrs.

Asymptomatic aneurysms in a specimen injected with neoprene.

(a) At the junction of anterior cerebral artery and anterior communicating artery.

The aneurysm:

Naked eye examination: The aneurysm was as a small bulge arising at the junction of the left anterior cerebral (A) and anterior communicating artery (B). The proximal part of the right anterior cerebral artery (A) is smaller in calibre and divided into two terminal branches before joining the anterior communicating. Three small branches arise at the angle from the summit of the sac.

The specimen unfortunately dried up before embedding and was brittle on cutting. A section of the aneurysm is seen in Plate II, fig. 1. to demonstrate its site of origin and the structure of its wall which consists of all coats of an artery with hypoplastic media but intact elastica.
Plate 2.
(b) At the first bifurcation of the left middle cerebral artery.

The aneurysm:

Naked eye examination: The aneurysm arose at the bifurcation of (M) into three branches A, B & C. (C) arises from the summit of the sac at the angle of bifurcation between A & B.

The specimen which dried up shows on section Plate II, fig. 2. the aneurysmal dilatation at the end of (M) found by the dilated origins of A, B & C.

Its wall shows that it consists of all coats of an artery with intact elastica of hypoplastic media.

Case 3. Female aged 51 yrs.

Asymptomatic aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:
Naked eye appearance: This is one of the specimens injected by Neoprene. The aneurysm has arisen at the junction of the left anterior cerebral artery (A) and anterior communicating artery (B). The beginning of the distal part of (A) as well as (B) were distended to contribute to the formation of the aneurysm. Two small branches (C) and (D) arising at the angle of junction between A & B are seen on the summit of the aneurysm.

Serial sections of this case are shown in Plate III, figs. 1 - 4.

**Fig. 1:** Shows the (H) shape appearance of A, B and A₁. The arrow points to the site of origin of the aneurysm.

**Fig. 2:** Shows the aneurysm bulging at the junction of A & B, and distending (B). The origin of the small branch (C) is illustrated.

**Fig. 3:** Shows the aneurysm with the origin of branch (C) from its summit.

**Fig. 4:** Shows the origin of branch (D) from the aneurysm. It also demonstrates a cross section of branch (C).

The histological structure of the wall shows that it consists of all coats of an artery with a few patches of intimal thickening. The elastica is intact all through. The material within the vessels is neoprene.

**Case 4:** Female aged 47 yrs.

Asymptomatic aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:
Plate 4.
Following the same pattern the aneurysm has arisen at the angle of junction of the two vessels A & B. A small branch (C) originally at the junction has contributed to the formation of the aneurysmal sac.

Serial sections are demonstrated in Plate IV, figs. 1 & 2.

Fig. 1: Shows the sac at the level of the anterior communicating artery. A cross section of the small branch (C) also appears.

Fig. 2: Shows the origin of (C) arising from the dilated lumen of (A) which is part of the aneurysm.

The histological structure of the wall is that of an artery wall, the elastic a being continuous all round.

Case 5. Male aged 39 yrs.

Asymptomatic aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye examination: The aneurysm was tiny and situated at the angle of junction. Two small vessels C & D were arising at the summit of the sac. A third small vessel was arising posteriorly from the anterior communicating artery (B). It did not contribute to the bulge.

Serial sections of the aneurysm are shown in Plate V, figs. 1 - 3.

Fig. 1: Shows the bulge between A & B as well as the origin of branch C.

Fig. 2: Shows part of the stem of (C) arising from the sac.
**Fig. 1**: Shows branch (D) arising from the sac. Another branch arises posterior to the anterior communicating artery (B). It has not contributed to the bulge.

The histological structure of the wall is that of an arterial wall with areas of medial aplasia and hypoplasia. The elastica is continuous. Branches C & D have contributed to the bulge of the aneurysmal sac because they are opposite the column of blood flowing into (A). The third posterior branch was not associated with any bulge being away from the head of the blood column.

**Case 6.**

Asymptomatic aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

![Diagram of aneurysm](image)

**Naked eye appearance**: The aneurysm is situated at the junction of anterior cerebral artery (A) and anterior communicating artery (B). (B) was directed forward and laterally as it passes from (A) to the contralateral anterior cerebral artery. Two small branches (C) and (D) were arising from the convexity of the aneurysm and their points of origin contributed to the formation of the sac.

Serial sections of the aneurysm are seen in Plate VI, figs. 1 - 2.

**Fig. 1**: Shows the aneurysm at the junction with the origins of branches C & D. The other end of (B) is also seen.

**Fig. 2**: In addition to the features in Fig. 1, it shows how branch B is directed forwards and laterally to join the contralateral anterior cerebral artery.
Similar to other asymptomatic aneurysms, the wall consists of all coats of an artery. Patchy intimal thickening and reduplicated elastica are also found.

Case 7. Male aged 31 yrs.

Bilateral asymptomatic small aneurysms at the junction of anterior cerebals and anterior communicating arteries.

The aneurysms:

Naked eye examination: Here there were two small aneurysms each arising at the junction of the anterior communicating artery and the corresponding anterior cerebral artery. The right measured 0.3 x 0.2 cm. and the left 0.2 x 0.2 cm. From the summit of each aneurysm at the angle of junction arises a small branch B & B₁.

Serial sections of the aneurysms are shown in Plate VII, figs. 1 - 3., which illustrate the site of the two aneurysms as well as the origins of the small branches B & B₁. The figures also show the cross sections of these branches further along.

The histological structure of the wall of the aneurysm shows that it consists of all three coats of an artery. There is small patchy intimal thickening. The media in some parts is hypoplastic, in others it is aplastic.
Case 8. Male aged 49 yrs.

A symptomatic aneurysm at the bifurcation of the internal carotid artery.

The aneurysm:

Naked eye appearance: This aneurysm occurred in the same Circle of Willis associated with another ruptured anterior communicating aneurysm. (Case 3). The internal carotid artery on the side of the aneurysm was larger than on the contralateral side. The other structural abnormality here was that the posterior communicating artery arose at the same level as the bifurcation of the internal carotid into anterior and middle cerebral arteries.

The aneurysmal sac arose opposite the column of blood flowing through the internal carotid artery (C). In addition to the anterior cerebral artery (A), the middle cerebral artery (M), and posterior communicating artery (P), two other small branches (B) and (D), arose and contributed to the formation of the aneurysm. Branch B arises at the junction of P & M., and branch (D) is at the junction between C & P.

Serial sections of the aneurysm are shown in Plate VIII, figs. 1-3. As the specimen was injected by neoprene the walls of the sac collapsed slightly during the sectioning.

Fig. 1: Shows the internal carotid artery (C) opening into the sac (O.An.). The origins of A, M & P are seen commencing from the sac.

Fig. 2: Shows the Sac (O.An.) with the dilated origins of the other two small branches B & D. Origins of P & C are seen in a deeper plane. The lumina of both anterior and middle cerebral arteries A & M are illustrated communicating with the aneurysm.
Plate 8.
Fig. 3: Shows the sac beyond the origins of P & B. Only branches D, A & M are well illustrated.

The histological structure of the sac consists of all coats of an artery. The elastica is intact all round. The media is deficient at the angles of bifurcation. The intima of the sac only shows patches of degenerative thickening - near origins of branches D & P.

Case 2. Female aged 64 yrs.

Unruptured aneurysm at the first bifurcation of the middle cerebral artery.

The aneurysm:

Naked eye examination: The aneurysm is a bulge that protruded at the bifurcation of the middle cerebral artery (M) into two large branches A & B and two small branches at the angle of bifurcation namely C & D.

It is situated opposite the direction of the column of blood when it impinges at the angles of bifurcation.

Serial sections of this aneurysm are shown in Plate IX, figs. 1 - 4.

Fig. 1: Shows the main bulge at the bifurcation of (M) into A & B. The origins of C & D are demonstrated from the angle between A & B.

Figs. 2 and 3: Show the aneurysm between A & B.

Fig. 4: Shows part of the aneurysm (An.), part of (M) and a cross section of branch (C) further along.

The histological structure of the aneurysm shows a continuous elastica with intima. The media is aplastic at the angle of bifurcation. The adventitia is also thinned out.
Case 10. Female aged 45 yrs.

A small unruptured symptomatic aneurysm was found at the first bifurcation of the left middle cerebral artery.

The aneurysm:

Naked eye appearance: The aneurysm is seen to arise at the anterior angle of bifurcation of the middle cerebral artery (M) into its two main branches A & B.

A smaller branch (C) is seen arising from the summit of the aneurysmal sac (O,An.)

Microscopically Plate XX - the aneurysmal sac (O,An.) is formed by the distension of the angles of junction between branches A, C & B. The wall of the aneurysm consists of all three coats of an artery with intact elastica all around.
Case 11. Female aged 51 yrs.

An aneurysm at the junction of the right anterior cerebral and anterior communicating arteries found accidentally during routine examination.

**The aneurysm:**

\[
\begin{align*}
A & \quad \text{Right anterior cerebral artery} \\
B & \quad \text{Anterior communicating artery} \\
C & \quad \text{Small branch}
\end{align*}
\]

**Naked eye examination:** The aneurysm is seen to arise at the anterior angle of junction of the right anterior cerebral artery \(A\) and anterior communicating artery \(B\). A small branch \(C\) is arising from the summit of the sac.

**Microscopically Plate XI:** the aneurysmal sac \(O, \text{An.}\) is seen in relation to branches \(A \& B\) and the origin of branch \(C\) is seen on the summit.

The wall of the sac consists of all coats of an artery with hypoplastic media. The elastica is intact all around the wall of the sac.
SYMPTOMATIC RUPTURED ANEURYSMS.
Symptomatic Ruptured Aneurysms.

Case 1. Male aged 63 yrs.

Massive subarachnoid haemorrhage two weeks before death. Death due to thalamic infarct after ligation of the right internal carotid artery.

The patient had five aneurysms, three congenital: one on each internal carotid artery and the third on the left vertebral artery (described in text), and two developmental bifurcation aneurysms, one at the junction of anterior cerebral artery and anterior communicating artery (ruptured) and one at the first bifurcation of the right middle cerebral artery. The last two are described here.

Aneurysm at junction of right anterior cerebral and anterior communicating arteries.

The aneurysm:

The aneurysm has arisen in the anterior (distal) angle of junction of the right anterior cerebral artery (A) and anterior communicating artery (B). It consists of a basal sac (0, An) from which arise two small branches C & D and a distal sac (An.) which has ruptured to cause subarachnoid haemorrhage. (A,) is the contra lateral anterior cerebral artery.

Serial sections of this aneurysm are shown in Plate XII, figs. 1 - 2.

Fig. 1: Shows the two sacs of the aneurysm 0, An. and An. A small branch (C) is seen arising from the basal sac. The aneurysm is shown to arise at the anterior angle of junction between A & B.
Fig. 2: Shows branch (D) arising from the basal sac C.An. (An.) is the contra lateral anterior cerebral artery seen to communicate with (B).

The histological structure of both distal and basal sacs conform with the usual pattern.

Right middle cerebral aneurysm:

The aneurysm:

It arises at the bifurcation of the right middle cerebral artery (M) into its main subdivisions A & B. It consists of a basal sac (C.An.) and two distal sacs (An.1) and (An.2.). Two smaller branches C & D arise from the basal sac.

Significant serial sections are shown in Plate XII, figs. 3 - 5.

Figs. 3 and 4: Show the basal sac (C.An.) formed by the bifurcation of (M). It demonstrates the two distal sacs consisting only of fibrous tissue at the points where the elastica of the basal sac has broken. (An.1) has developed between branches D & C and (An.2) between branches C & B.

Fig. 5: Shows both (An.1) and (An. 2) the latter is seen to communicate with the dilated origin of branch (B).

The histological structure of both basal and distal sacs follows the same pattern. The distal sacs have not ruptured to produce a subarachnoid haemorrhage.
Case 2.

Female aged 50 yrs.

Several attacks of headaches for the last two years before death. Some attacks were associated with neck stiffness and vomiting. Attacks of subarachnoid haemorrhage 6 months before death. Aneurysm localised and ligated. Death eight months later due to infarction in territory of both anterior cerebral arteries.

Aneurysm at the junction of the anterior cerebral and anterior communicating arteries.

The aneurysm:

The aneurysm was situated at the junction of the left anterior cerebral artery (A) and anterior communicating artery (B). It arose opposite the column of blood impinging at the junction. It had a basal portion and a ruptured distal portion.

Two small branches C & D arose from the base of the aneurysm. The aneurysm was successfully ligatured at operation. Four clips were applied: one, just proximal to the neck of the distal sac and it included the origin of branch (D); one at the stem of the branch (C) and other two unfortunately involved the distal parts of the two anterior cerebral arteries. This patient died from infarct involving the corpus callosum and areas of both frontal lobes due to the last two clips. The distal sac was atrophied and lay twisted at the posterior part of the specimen.

Serial sections of the aneurysm after removal of the ligatures are shown in Plate XIII, figs.1 - 3.

Fig. 11 Shows the basal part of the aneurysm at the junction of the anterior cerebral artery (A) and anterior communicating artery (B). Branch (C) is seen arising from and contributing to the formation of the basal sac.
Plate 13.
Fig. 2: Shows branch (D) arising at the junction of A & B from the basal sac. The arrow points to opening between the basal and distal sacs where the elastica is broken.

Fig. 3: Shows portion of the distal fibrous sac (An.) beside a cross section of branch (C) further along.

The histological structure conforms with previous cases.

Aneurysm at the first bifurcation of the right middle cerebral artery.

The aneurysm:

![Diagram of the aneurysm](image)

The aneurysm arose at the bifurcation of the right middle cerebral artery (M) into two main branches A & B and two small branches C & D. The bulging occurred where the column of blood impinged at the bifurcation. An early distal sac (An.) has protruded from the basal bulging.

Serial sections of the aneurysm are shown in Plate XIII, figs. 4 - 6.

Fig. 4: Shows the trunk of the middle cerebral artery (M) terminating into a bulge at its bifurcation into its two big branches A & B and the small branch (C). (B) appears at its origin and further along a cross section of it. A small distal fibrous sac (An.) is protruding from the bulge between A & C.

Figs. 5 and 6: Show in addition the origin of the other small branch (D) from the aneurysm.
The histological structure of the aneurysm shows all coats of an artery all through the main bulge except at the protrusion of the small distal sac where the intima and elastica cease and the wall consists only of fibrous tissue forming what appears to be two layers. The inner layer is newly formed fibrous tissue. Patches of intimal thickening are present near the origin of the aneurysm from (M) and at the origin of branch (D).

Case 3. Male aged 64 years.

Attack of unconsciousness two months before death. Aneurysm of anterior communicating artery identified by angiography. Clip applied at junction of distal and basal sac. Two months later death due to acute nephritis.

The aneurysm:

Naked eye examination: The left anterior cerebral artery is much larger than the right and, in the region normally occupied by the anterior communicating, this vessel divides into two branches; one continuing as the main stem of the left anterior cerebral, the other crossing the midline and joining the small right anterior cerebral and then continuing in the line of distribution of the right anterior cerebral artery. In the distal angle of this Y-shaped bifurcation of the left anterior cerebral the aneurysm arises. It is multilocular with a large basal sac (O, An.) formed by the bulging of the angle of bifurcation and a multilocular distal sac (An.) A clip was applied at the neck of the distal multilocular sac. A small branch (C) is seen to arise from the basal sac.

Relevant serial sections are seen in Plate XIV, figs. 1 - 4.
Fig. 1 and 2: Show the basal sac (O.An.) and the origin of branch (C). The distal part of (A) is seen on the right upper corner of the figures.

Figs. 3 and 4: Show (O.An.) communicating with multilocular distal sac (An.). The origin of branch (C) is also seen. (A,) is the contralateral anterior cerebral artery.

The histological structure of the walls of the distal and basal sacs is identical with previously described cases.

Case 4. Male aged 57 yrs.

Sudden attack of headache with drowsiness from which patient recovered. A second attack two weeks later which patient overcame. Final attack of convulsions and unconsciousness one week later. Death due to both intracerebral and subarachnoid haemorrhages.

Patient was found to have two aneurysms:
1. A ruptured anterior communicating aneurysm.
2. Unruptured left middle cerebral aneurysm.

1. The aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye examination: The aneurysm arises at the distal angle of junction of the left anterior cerebral artery (A) and the anterior communicating artery (B). It is bilocular with the basal sac (O.An.) larger than the distal (An.). A smaller branch (C) is seen arising from the basal sac. (A,) is the right anterior cerebral artery. (A) is larger in size than (A,). The rest of the circle of Willis shows slight arteriosclerotic change but no other sites of abnormal branching.

Relevant serial sections are shown in Plate XV, figs. 1-4.
Plate 15.
Figs. 1 and 2: Show the aneurysm arising at the junction of A & B. It demonstrates the large basal sac (O. An.) and the connection between it and the distal small sac (An.). The dilated origin of branch (C) from (O. An.) is also seen.

Fig. 3: Shows both locules of the aneurysm and branch (C).

Fig. 4: Shows a cross section of branch (C) in higher magnification.

The comparative structure of both distal and proximal sacs follows the same pattern described in other cases.

2. The middle cerebral aneurysm.

The aneurysm:

![Diagram of aneurysm]

Naked eye examination: This did not rupture and it is seen to arise at the first bifurcation of the left middle cerebral artery (M) into its main division A & B. Two small branches C & D were seen to arise from the summit of the aneurysm.

Relevant serial sections are seen in Plate XV, figs. 5 and 6. They show the bulge at the angle between A & B. The origins of C & D are also demonstrated.

The histological structure is similar to the unruptured aneurysms, i.e. its wall is arterial. No distal sac has developed.
Case 5. Female aged 62 yrs.

4 weeks before death an attack of headache and collapse from which she recovered. Four days later similar attack with recovery. Further headache and loss of consciousness ten days later and a fourth before death. Death due to both subarachnoid and intracerebral haemorrhages and spasm of anterior cerebral arteries with ischaemic nerve cell changes in their territory.

Ruptured aneurysm at the junction of left anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye examination: The aneurysm arose at the junction of A & B. The anterior communicating artery (B) and the origin of the distal part of the anterior cerebral artery (A) are distended to contribute to the formation of the basal portion of the aneurysm. The origin of a small branch (C) at the angle of junction also contributed to the formation of the basal portion of the aneurysm.

Serial sections of the case are demonstrated in Plate XVI, figs. 1 - 4.

Figs. 1 and 2: Show the origin of branch (C) as well as a cross section of it further along, as it arises at the angle between A & B. It contributed to the formation of the base of the sac.

Figs. 3 and 4: Show the distal sac arising from the basal portion formed by the dilated parts of A, B & C. The wall of the distal sac is thin and the rest of the rounded swelling consists merely of blood clot.

The histological structure of both the base and distal sacs conforms with the usual pattern.

History of unconsciousness one week before death from which patient recovered. Death due to massive subarachnoid haemorrhage a week later.

Aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye appearance: The aneurysm following the same pattern arises at the junction and opposite the column of blood flowing through anterior cerebral artery (A). There is the basal sac (O.An.) with a small branch (C). The origin of the latter contributed to its formation.

The distal sac (An.) is here bilocular.

Serial sections of this aneurysm are seen in Plate XVII, figs. 1 - 6.

Fig. 1: Shows branch (C) arising at the junction in the level of the aneurysm.

Fig. 2: Shows the basal sac (O.An.) and part of branch (C) adjacent to it.

Fig. 3 and 4: Show the two locules of the distal sac (An.1 and An.2) communicating with each other and with the basal sac as well. (B) is the continuation of the anterior communicating artery to the contralateral side.

Fig. 5: Shows similar appearances and illustrates the site of rupture of the distal sac.

The histological appearance of both basal and distal sacs is identical with already described cases.
The two locules of the distal sac consist only of fibrous tissue.

The point of protrusion of the distal from the basal sac is shown in Fig. 6, where both intima and media cease suddenly. A strip of thin broken elastica is continued for a short distance into the fibrous wall of the distal sac which appears to be a continuation of the adventitia of the basal one.

The bilocular character of the distal sac is consistent with the clinical picture of the patient who had two attacks of subarachnoid haemorrhage before he died.

Case 7. Male aged 44 yrs.

History of headache and drowsiness two weeks before death from which patient recovered. Death due to both subarachnoid and intracerebral haemorrhages.

**Aneurysm at the junction of anterior cerebral and anterior communicating arteries.**

**The aneurysm:**

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Naked eye appearance: The aneurysm is bilocular and is situated at the junction of the anterior cerebral artery (A) and anterior communicating artery (B). It is opposite the column of blood flowing through (A). The anterior communicating artery (B) and the distal part of the anterior cerebral artery (A) are distended at the aneurysm and appear to contribute to the formation of the basal locule or sac of the aneurysm (O.An.). A small branch (C) arises from the latter and it is related anteriorly to the distal locule or sac (An.).

Serial sections of this aneurysm are shown in Plate XVIII, figs. 1-5.
Plate 18.
Fig. 1: Shows the two anterior cerebrols connected by the anterior communicating artery (B). The latter is distended forming the basal locule of the aneurysm. The dilated origin of the branch (C) is also seen contributing to the formation of the basal locule.

Figs. 2 and 3: Show the basal locule with the origin of branch (C).

Fig. 4: Shows the protrusion of the distal locule (An.) from the proximal (O.An.)

Fig. 5: Shows the distal aneurysmal sac which has ruptured at its summit. In the lower part of the figure part of the basal sac appears in communication with the distal.

The histological structure of the basal locule and the distal fibrous one follow the general pattern described previously.

Case 6: Female aged 54 yrs.

Past history of attack, headache and fainting. Death a fortnight later due to both subarachnoid and intracerebral haemorrhages.

Aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye appearance: The aneurysm was a finger-like projection at the junction of the left anterior cerebral artery (A) and anterior communicating artery (B). The anterior communicating was swollen up and has contributed to the formation of the basal part of the aneurysm. A small branch (C) was seen arising from the base of the sac. The right anterior cerebral artery was slightly smaller in diameter than the left at its summit.
Plate 19.
The aneurysm has ruptured to cause both subarachnoid and intracerebral haemorrhage.

Serial sections of the aneurysm were not satisfactory and many of the sections were fragmented because the specimen had dried before embedding.

They are described in Plate XIX, figs. 1 and 2.

They show the small branch (C) arising at the junction from the base of the aneurysm. The distended anterior communicating artery (B) is forming the basal part of the aneurysm. (An.) is the finger-like fibrous sac of the aneurysm.

Histologically both base and distal sac of the aneurysm conform with the usual pattern.

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**Case 2. Male aged 54 yrs.**

History of two previous attacks of severe headache in past two years. Death due to massive subarachnoid haemorrhage.

Aneurysm at the junction of anterior cerebral and anterior communicating arteries.

**The aneurysm:**

![Diagram](image)

**Naked eye examination:** This case shows two aneurysms, No.1 and 2.

Aneurysm No.1 is situated on the right anterior cerebral artery (B) just proximal to the anterior communicating artery (G). Two small branches E & F are seen arising from the sac.
Aneurysm No. 2. arises at the junction of the left anterior cerebral artery (A) and anterior communicating artery (G). It consists of a bilocular sac. Two small branches C & D are seen arising from the basal locule or sac. Artery (A) was definitely larger than artery (B).

Serial sections of this case are shown in Plate XX, figs. 1 - 7.

Fig. 1: Shows the basal part of aneurysm (2) with the dilated origin of the branch (C) which contributed to its formation.

Fig. 2: Shows the dilated origins of C & D from the basal sac of the aneurysm. (A) is less in direct communication with the sac. The latter is situated in the line of the column of blood flowing into (A).

Fig. 3: Shows branch (D) as well as the origin of the distal sac from the basal one.

Fig. 4: Shows one of the sites of rupture of the distal sac causing subarachnoid haemorrhage.

It also shows the dilated end of the right anterior cerebral artery (B) forming aneurysm No.1. The origins of two small branches E & F are seen arising from the latter.

Fig. 5: Shows the same structures as Fig. 4, but with another point of rupture of the distal sac of aneurysm No.2.

Fig. 6: Shows aneurysm No.1. The right anterior cerebral artery (B) is opening in it. This figure is very important because it shows an early protrusion of a distal sac (marked D.S.) from this aneurysm.

Fig. 7: Shows the full lumen of the right anterior cerebral artery (B) communicating with the aneurysmal sac No.1.

The histological structure of this aneurysm consists of all three coats of an artery in most parts of it. The elastica and intima are healthy except near the early protruding distal sac (D.S.) where it shows some degenerative thickening.

The small distal sac on the other hand consists mainly of thick fibrous tissue. No media or elastica or intima are seen.
From the appearances presented it appears obvious that aneurysm No.2. was the first to form and with its formation a larger volume of blood accumulated at the junction of the anterior communicating and right (B) cerebral arteries, so impeding the flow in (B) and increasing the stress on the two small branches E & F, with the usual result of their yielding. So commenced aneurysm No.1. It had only just begun to form its distal sac when No.2 ruptured.

**Case 10.**

Male aged 35 yrs.

Past history of several attacks of headache. Death due to subarachnoid and intracerebral haemorrhage.

An aneurysm at the first bifurcation of the middle cerebral artery.

**The aneurysm:**

![Diagram of blood vessels with labels An., B, C, D, E, F, M](image)

**Naked eye appearance:** The aneurysm originated at the bifurcation of the middle cerebral artery (M) into two big branches A & B and three small branches, C D & E. It has erupted where the column of blood flowing through (M) is impinging at the point of bifurcation. The aneurysm has ruptured and caused subarachnoid haemorrhage. The sac was very big. The aneurysm was multilocular.

Serial sections of the case are shown in Plate XXI, figs. 1 - 4.

**Fig. 11** Shows one of the branches (E) in front of the sac. The two cavities marked (An.) represent outpouchings of the main aneurysmal sac produced by a depressed groove (G) in the wall of the sac.
Plate 21.
Fig. 2 and 3: Show the sac arising at the bifurcation of (M) into branches, A, C, B & D. The origin of the front branch (E) is also shown.

All these branches communicate with the aneurysmal sac.

Fig. 4: Shows branch (E) further along. It also demonstrates the folds of the sac.

The histological structure of both the basal and distal part of the sac is identical to the other cases.

Case II.

Female aged 63 yrs.

Death due to massive subarachnoid haemorrhage.

Aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye appearance: The anterior communicating artery was so short that the two anterior cerebrials show an X formation. Both the anterior communicating artery and the distal part of the right anterior cerebral artery (A) are swollen at the site of the aneurysm and appear to contribute to the formation of its basal portion. A small branch (C) is seen arising from the basal portion of the sac (O, An.) The aneurysm has ruptured to cause both subarachnoid and intracerebral haemorrhages.

Serial sections of the aneurysm are shown in Plate XXII, figs. 1 - 3.
Fig. 1 and 2: Show the aneurysm arising at the junction of the distal part of (A) and anterior communicating artery (B); where the column of blood flowing through the proximal part of (A) is impinging. The dilated origin of the small branch (C) is illustrated at the base of the sac. (0, An.) in Fig. 2, is the basal portion of the sac.

Fig. 3: Shows branch (C) further along arising from the anterior communicating artery.

The basal part is labelled (0, An.) and the distal is labelled (An).

The histological structure of both basal and distal parts follow the usual pattern.

Case 12. Female aged 49 yrs.

Rupture of anterior communicating aneurysm. Subarachnoid haemorrhage breaking into right frontal lobe infarct in territory of left anterior cerebral aneurysm successfully ligatured. Death due to pulmonary embolism.

Aneurysm at junction of anterior cerebral and anterior communicating artery which was clipped surgically.

The aneurysm:

Naked eye appearance: The figure shows the aneurysm after removal of the clips. The sites of the clips are marked in red. A bulge at the angle of junction of the anterior cerebral and anterior communicating arteries is seen occurring in the direction of flow of the blood coming from (A). This bulge represents the basal portion or sac. The main or distal sac (in ink) was twisted posteriorly and clipped at its neck (red dotted line is site of clip). Two branches C & D are seen arising for the basal part.

Two clips were applied on the two ends of the anterior communicating artery (marked red). A fourth
Plate 23.
clip was applied at the origin of branch (C).

An arterial twig connects the distal part of the anterior cerebral artery (A) with branch (C). (A) has a larger calibre than the contra lateral anterior cerebral artery.

Serial sections of the aneurysm are shown in Plate XXIII, figs. 1 - 5.

Fig. 1: Shows the distended junction of anterior communicating artery (B) and anterior cerebral artery (A) forming the basal sac. Branch (D) is seen arising from the anterior communicating artery. Branch (C) is also seen coming out from the basal sac.

Fig. 2: Shows as well the basal sac, (C) is seen further along and so is the anterior communicating artery (B).

Fig. 3: Shows branch (C) further along with its communication with the distal part of the anterior cerebral artery (A).

Fig. 4: Also shows branch (C) and below it part of the fibrous distal sac.

Fig. 5: Shows the distal fibrous sac which has ruptured. In the lower part of the figure parts of the basal sac appear in communication with the distal.

The histological structure of both basal and distal sacs conforms to the appearances described before.

Case 13.

Female aged 50 years.


Aneurysm at the junction of anterior cerebral and anterior communicating arteries.
Plate 24.
The aneurysm:

Naked eye appearance of the aneurysm which has been surgically treated shows it to occur at the junction of anterior cerebral artery (A) and anterior communicating artery (B). It is in the direction of the column of blood coming through the proximal part of (A).

(C) is a small branch arising from the base of the aneurysm.

Two clips (in ink) on the two ends of the anterior communicating artery. They have successfully obliterated the basal part of the aneurysm which was the source of trouble.

Serial sections of the aneurysm are shown in Plate XXIV, figs. 1 - 3.

Fig. 1: Shows both anterior cerebrals and anterior communicating artery (B). The lumen of (B) appears to be almost completely occluded by the clips which were removed. Yet the basal part of the aneurysm is shown to be situated opposite the blood flow coming from (A). The origin of the small branch (C) is seen arising from it.

There is not an actual distal sac but the basal portion seems to have ruptured with severe hemorrhage pulling away a tag of adventitia on both sides.

Fig. 2: Shows a further part of branch (C) in between the distal parts of the two anterior cerebral arteries.

Fig. 3: Shows a still further part of (C) among the blood clot of the rupture. It also demonstrates the proximal part of (A).
The histological structure of the basal portion is the same as the previous cases and the distal sac is only represented by two tags of fibrous adventitia. Though the aneurysm was successfully tackled and its base was efficiently clipped yet the patient died from the first leak of blood causing both subarachnoid and intracerebral haemorrhage. There was no chance for the adventitia to check the haemorrhage and to form a distal sac because the first haemorrhage was presumably severe.

Case 14.

Male aged 39 yrs.

Death due to intracranial haemorrhage from a ruptured aneurysm.

Aneurysm at the junction of anterior cerebral and anterior communicating arteries.

The aneurysm:

Naked eye appearance: During dissection of the aneurysm the anterior communicating artery was torn off near the base of the aneurysm.

The aneurysm occurred opposite the column of blood coming from the proximal part of the anterior cerebral artery (A) and is situated at the junction of anterior cerebral artery and anterior communicating artery (B). It consisted of a bilocular sac. The basal locule or sac gives rise to a small branch (C).

Serial sections of this aneurysm are seen in Plate XXIV, figs. 1 and 2.

Fig. 1: Shows the anterior cerebral artery (A) both proximal and distal to the aneurysm. The opening to which the anterior communicating artery was attached is marked (B).

The dilated basal locule (O, An.) is communicating with the distal locule (An).
The origin of the small branch (C) is contributing to the formation of the basal locule.

Fig. 2: Shows the basal locule (O.An) with the dilated origin of branch (C) arising from and contributing to its formation.

The histological structure of both the proximal and distal locules follows the general pattern of those previously described. The origin of branch (C) shows degenerative thickening of the intima with split elastic.

Case 15. Female aged 53 yrs.

Death due to intracerebral haemorrhage: aneurysm localised by angiography: surgical ligature of aneurysm.

Aneurysm at major bifurcation of anterior cerebral distal to anterior communicating artery.

The aneurysm:

Naked eye appearance: The aneurysm is situated at the anterior angle of bifurcation of the anterior cerebral artery (A) into two main branches C & B. (A) is swollen at its point of bifurcation forming the basal sac of the aneurysm (O.An). A small branch (C) is seen to arise from this basal sac. (An.) is the distal sac which has ruptured. The surgical clip was at the point of origin of branch (B) as well as the neck of the distal sac.

Relevant serial sections of this aneurysm are seen in Plate XXVI, figs. 1 - 3.

Fig. 1 and 2: Show the bulging of the terminal end of (A) at the bifurcation to form the basal sac (O.An). It also demonstrates branch (B) as well as the origin of the small branch (C). Part of the distal sac (An.) is also seen at the bifurcation. An organised ante mortem clot blocks the lumen of (B).
Plate 26:
Fig. 3: Shows branch (C) arising from the basal sac (O. An.)

The histological structure of both distal and basal sacs follows the usual pattern of the ruptured aneurysms.

Case 16. Male aged 50 years.

Past history of attack of subarachnoid haemorrhage. Death a month later due to intracranial haemorrhage from a ruptured aneurysm.

Aneurysm at the first bifurcation of the middle cerebral artery.

The aneurysm:

Naked eye appearance: The aneurysm arose at the bifurcation of the middle cerebral artery (M) into two main branches A & B and other two smaller branches C & D. (C) runs in front of the sac and (D) at its back. A leash of small branches (E) is also seen arising at the point of bifurcation and communicating with the aneurysm.

Serial sections of the aneurysm are seen in Plate XXVII, figs. 1 - 9.

Figs. 1 and 2: Show branch (C) and part of the aneurysm (An.) (C) is communicating with the sac in Fig. 2. The distal part of branch (A) is also seen.

Fig. 3: Shows one of the main divisions (A) communicating with the aneurysm.

Fig. 4: Shows the main trunk of the middle cerebral artery (M) opening into the aneurysm.

Fig. 5: Here (M) and the other main division (B) are seen communicating with the aneurysmal sac. The origin of a small branch (E) is seen near the bifurcation connected with the aneurysm.
Figs. 6 and 7: Show cross sections of the leash of small vessels arising at the base of the aneurysm.

Fig. 8: Shows the opening of the posterior smaller branch (D) connected to the aneurysm (An.) (B) is the cross section of one of the main divisions further along.

Fig. 9: Shows the cross sections of branches B & D still further.

The histology of both the base and fibrous sac of the aneurysm follow the same pattern.

Case 17.  

Female aged 51 years.

Death due to subarachnoid haemorrhage and infarct of antero-lateral portion of the left frontal lobe; three aneurysms were found. One has ruptured.

(1) Aneurysm at the first bifurcation of the right middle cerebral artery.

The aneurysm:

![Diagram of aneurysm](image)

*Naked eye appearance:* The aneurysm arises at the bifurcation of the middle cerebral artery (M) into branches A, B & C. It consists of a basal bulge or sac (O.An) and a distal sac (An.) which has ruptured to cause both subarachnoid and intracerebral haemorrhages and caused the death of the patient.

Serial sections are illustrated in Plate XXVIII, figs. 1 - 4.

Fig. 1: Shows both the basal sac (O.An) and the distal sac (An) formed at the bifurcation of (M) into A & B. It also demonstrates the site of rupture of the distal sac (see arrow).
Plate 28.
Fig. 2: Shows the distal sac communicating with that part of the basal bulge formed by the dilated origin of (A).

Figs. 3 and 4: Show cross sections of branch (C) and the distal sac. The communication between these two is illustrated in Fig. 4.

The histological structure of both basal and distal sacs is following the usual pattern. In some parts the distal sac consists of only one thin layer of fibrous tissue (Fig. 1). In others it consists of what appears to be two layers (Fig. 3 and 4). The inner consisting of newly formed fibrous tissue and adherent blood clot.

(2) Aneurysm at the first bifurcation of left middle cerebral artery.

The aneurysm:

Naked eye appearance: The aneurysm is situated at the angle of bifurcation of the main stem of the middle cerebral artery (M) into four branches, B, A, C & D of which (A) is the largest. It consists of a bulge formed by the dilated origins of these branches at their angle of bifurcation where the column of blood flowing through (M) impinges. A small fibrous sac protrudes from the bulge between branches A & B.

Serial sections of the aneurysm are shown in Plate XXVIII, figs. 5 - 7.

Figs. 5 and 6: Show the basal bulging sac at the point of bifurcation of (M) into A & B. It also demonstrates the protrusion of the small distal sac from the base.

Fig. 7: Shows the basal sac formed by the points of origin of branches A, C & D dividing from (M). The distal sac is also seen.
The histological structure of the aneurysm as regards its two sacs conforms with previous pattern. There was haemosiderosis in the arachnoid around the aneurysm but not frank subarachnoid haemorrhage. It is therefore concluded that the rupture of the basal sac was not severe and the bleeding was checked by adventitia forming the distal sac.

(3) Aneurysm at the second bifurcation of the right middle cerebral artery.

The aneurysm:

Molecular examination: Here the aneurysm also arises at the point of bifurcation of (M) into branches A, B & C. It also consists of a basal bulge and a distal bulge (An).

Serial sections are shown in Plate XXVIII, figs. 8 and 9, which show the basal sac formed at the end of (M) and consists of the dilated ends of origins of branches A, C and B. It also demonstrates the protrusion of the distal sac from the basal one.

Here again the histological structure of the two sacs follows the same pattern. Organised blood clot was found in the subarachnoid space around the aneurysm. The distal sac has presumably checked a haemorrhage.

Case 18. Male aged 43 years.

Death due to both subarachnoid and intracerebral haemorrhages.

Aneurysm at the junction of the internal carotid artery and posterior communicating artery.
Plate 29.
The aneurysm:

Identified as an abnormality arising at the base of the internal carotid artery (A) and posterior communicating artery (C). The aneurysm was formed by the dilated origins of these two vessels as well as by the origins of other small branches B, D, E and F arising at the bifurcation. The main sac of the aneurysm (An.) was large and has ruptured to cause subarachnoid haemorrhage.

Serial sections of the aneurysm are shown in Plate XXII, figs. 1 - 7.

**Fig. 1:** Shows the posterior communicating artery (C) and part of the aneurysmal sac (An.)

**Fig. 2:** Shows one of the small branches (B) opening into the aneurysm (An.). The posterior communicating artery (C) is seen arising from the internal carotid artery (A).

**Fig. 3:** Shows the origin of another small branch (F) from the posterior communicating artery (C) near its origin from the internal carotid artery.

**Fig. 4:** Shows the site of communication of both the posterior communicating artery (C) and internal carotid artery (A) with the aneurysmal sac.

**Figs. 5 and 6:** Show the origin of one of the small branches (B) near the junction at the base of the aneurysm.

**Fig. 7:** Shows the origin of the small branch (D) from the basal part of the aneurysm formed by the dilated part of (A).

The histological structure of the base of the aneurysm where all the branches take origin, and the remainder of the fibrous sac follow the previously described pattern.
Case 12. Female aged 79 years.

Fell unconscious in street two weeks before death. Death a fortnight later due to subarachnoid haemorrhage.

Aneurysm at the junction of the internal carotid artery and posterior communicating artery.

The aneurysm:

Naked eye appearance: The aneurysm which has ruptured is situated at the angle of junction of the internal carotid artery (C) and posterior communicating artery (P). From the base of the aneurysm, i.e., the angle of junction, two small arteries A & B arise. Their dilated origins contributed to the formation of the base.

Serial sections of the aneurysm are shown in Plate XXX, figs. 1 - 3.

**Fig. 1:** Shows the proximal end of (C) which is distended. From it is seen the origin of the small branch (A). Above this are shown cross sections of the distal aneurysmal sac (An.) and the posterior communicating artery (P). To the left side is the cross section of the distal part of (C).

**Fig. 2:** Shows the point of junction between P & C well distended. The fibrous distal sac is seen arising at the angle. A cross section of branch (A) is seen further along.

**Fig. 3:** Shows the angle of junction with the origin of the other small branch (B) from it.

The histological structure of both the base and distal sac of the aneurysm follow the usual pattern.
Plate 30.
Case 26. Male aged 67 yrs.

History of repeated attacks of headaches for the last two years. Death due to subarachnoid haemorrhage. The cerebral vessels show marked diffuse arteriosclerosis. A ruptured aneurysm discovered at the junction of the right internal carotid artery and right posterior communicating artery. Another unruptured aneurysm discovered at the first bifurcation of the right middle cerebral artery.

The aneurysm:

Naked-eye examination: The aneurysm is seen to arise at the distal angle of junction of the internal carotid artery (C) and posterior communicating artery (P). It consists of a basal sac (0, An.) from which is seen to arise a small branch (A). The distal sac (An.) protrudes from the basal sac and it is large and multilocular.

Relevant serial sections are seen in Plate XXXI, figs. 1 - 3, which show the relation of vessels C, P & A to the basal sac (0, An.). The distal fibrous sac (An.) is seen communicating with the basal sac.

The histological structure of all parts of the aneurysm conform with the usual pattern. The vessels at, as well as further from, the aneurysm show marked arteriosclerosis.

The second middle cerebral aneurysm shows the same structure as the unruptured ones formed only of basal sac.
Case 21. Male aged 58 years.

Death due to both subarachnoid and intracerebral haemorrhages.

Plate XXXII.

- is a photograph of the Circle of Willis which shows three aneurysms.

(1) Developmental bifurcation aneurysm at the junction of the right anterior cerebral and anterior communicating arteries. It consists of a basal sac from which arise a small branch and a distal fibrous sac.

(2) Two small saccular supraclinoid congenital aneurysms arising from the internal carotid artery before its division. In the photograph the internal carotid was twisted to show both aneurysms clearly. They actually arise from the posterior wall of the arterial bend.

Microscopically all aneurysms show the proper pattern described in the text.
Case 22.  

Female aged 50 yrs.

History of two previous attacks of subarachnoid haemorrhage. Death due to a third attack. Blood extended into brain substance as well. The aneurysm was clipped before death.

Plate XXXIII.

- is a photograph of the Circle of Willis. The arteries comprising the circle show slight diffuse fibrous thickening but very few atheromatous plaques. There is a developmental bifurcation aneurysm arising at the distal angle of junction of the right anterior cerebral and anterior communicating arteries. On removing the clip a large portion of the distal sac was detached. The clip appears to have embraced only a diverticulum of the distal sac. A smaller branch is seen to arise from the basal sac of the aneurysm.

Microscopically the aneurysm shows the usual pattern.
Case 23.  Female aged 47 years.

History of four severe attacks of headache.  Death due to both subarachnoid and intracerebral haemorrhages.

Plate XXXIV.

The arteries comprising the Circle of Willis, Plate XXXIV, show slight diffuse fibrous thickening and, in addition, several plaques of atheroma. There are two anterior communicating arteries set close together. They spring side by side from the right anterior cerebral artery but diverge slightly as they pass to the left, where they are separated by a space of about 1 - 2 mm.

A small unruptured thin walled bifurcation aneurysm arises in the distal angle formed by the distal of the two anterior communicating arteries and the left anterior cerebral. It has a fairly wide basal portion from which springs an anomalous branch. The latter runs for a short distance and opens distally in the left anterior cerebral artery. Its origin is at the distal angle of junction.

The histological structure of this aneurysm shows a basal sac from which arises the anomalous branch and a smaller distal thick-walled fibrous sac.

The aneurysm which has ruptured is seen to arise at the first major bifurcation of the right middle cerebral artery. It consists - like the usual type - of a basal sac from which a smaller branch arises and a distal fibrous sac which has ruptured.
Case 24.  

Female aged 40 years.

Death due to rupture of an intracranial aneurysm both in the subarachnoid spaces and in brain tissue. The aneurysm clipped a month before death. History of three attacks of subarachnoid haemorrhage in the last three months before death.

Plate XXXV.

The aneurysm is seen to arise at the distal angle of junction of the left anterior cerebral and anterior communicating arteries. (Plate XXXV photographed from the dorsal aspect). Like all other aneurysms it consists of a basal arterial sac from which arise a small branch and a distal fibrous sac which has ruptured.
Case 25. Female aged 46 years.

Death due to intracerebral haemorrhage.

Plate XXXVI.

The source of the haemorrhage is an aneurysm arising from the distal angle of the first major bifurcation of the left middle cerebral artery (Plate XXXVI). The aneurysm which hangs downwards and outwards from its origin is composed of a small thin walled proximal sac from which arise two other small branches and two distal sacs. The larger of the distal sacs (separated out in photograph) is pear-shaped. Part of the wall has a white fibrous appearance, the remainder is formed by a layer of smooth thrombus continuous with the thrombus within its cavity.

The histological structure of the distal sacs and the basal sac conform with the usual pattern.

Case 26 is represented in Fig. 3 in the text.