SECTION I

Preliminary Remarks on Subarachnoid Hemorrhage

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Preface

The subject of vascular disease is attracting greater and greater attention. Vascular disease of the brain ranks third in the causes of death in the United States at the present time. It has been estimated that hypertensive intracerebral hemorrhage accounts for 20 per cent, cerebral embolism 15 to 20 per cent, and cerebral thrombosis 50 per cent of all strokes. These figures will vary depending upon the population from which patients are derived and the nearness to hospital facilities. The remaining 5 to 10 per cent represents subarachnoid hemorrhage (Stevenson '28; Ohler and Hurwitz '32; Falconer '50, '51; Merritt '63).

In their excellent treatises, Dandy '44, Hamby '52, Walton '56 and later, Pool and Potts '65, reviewed the literature in detail and made important contributions to our understanding of subarachnoid hemorrhage, aneurysms and arteriovenous malformations. It is interesting and encouraging to note the many advances which have been made in this field in the past decade. These are covered in the publication by Fields and Sahs '65. No apologies need be made for the fact that what appears to be a rather simple problem turns out to be an exceedingly intricate and difficult one, and that much work needs to be done to reduce the mortality and morbidity of this condition.

The authors hope that the review to follow will be a useful background for the material to be presented by the Central Registry of the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage. The number of references must be limited in the interest of saving space for the large amount of data to be presented. Thus it is not possible to cover the literature in great detail, but pertinent references for the interested reader are provided.

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Introduction

Today's clinical approach to the problem of spontaneous subarachnoid hemorrhage is a product of the remarkable advancement of medical knowledge in recent years, but interest in this subject is not new. Gull, in 1859, wrote, "Whenever young persons die with symptoms of ingravecent apoplexy, and after death large effusion of blood is found, especially if the effusion be over the surface of the brain in the meshes of the pia mater, the presence of an aneurism is probable."

Fearnside's '16 reported that the first definite account of an intracranial aneurism was published by Biumi of Milan 1763 and was reprinted by Sandifort at Leyden in 1778. Morgagni 1761 is also given credit as one of the first to describe an intracranial aneurysm. However, Walton '56 indicated that both of these authors were antedated by Pierre Dionis in 1718 who described an aneurism in detail. Duke and a Prince, both of whom probably died from subarachnoid hemorrhage. Pool and Potts '65 suggested that intracranial aneurysms were recognized as a cause of cerebral hemorrhage by Wiseman 1696.

Incidence of Aneurysms

Richardson and Hyland '41 found the incidence of intracranial aneurysms to be 0.87 per cent in an autopsy series, in which slightly less than half the brains were removed. They indicated the actual incidence may be considerably higher. In a total of 2,030 cases of sudden and unexpected natural death necropsied between 1937 and 1943, Helpern and Rabson '50 found 95 cases (4.7%) of spontaneous subarachnoid hemorrhage. This represented 25.7 per cent of the 370 fatalities from diseases of the nervous system. Housepian and Pool '58 reported an incidence of 2.1 per cent in the last portion of their autopsy series covering 1931 to 1958. Chason and Hindman '58 indicated that the incidence of aneurysms in their series was 4.9 per cent. This included 80 instances of unruptured aneurysms. The anterior portion of the circle of Willis was implicated nine times
more frequently than the posterior segment. The average size of the ruptured aneurysms was almost twice that of the unruptured ones. Stehbens '54 reported 182 cases of aneurysms in a large series of necropsies. He reported the incidence as 3.7 per cent.

**Location of Intracranial Aneurysms**

Bull '62 reviewed the 1,769 intracranial aneurysms collected by McKissock in the 11 years from 1950–60, inclusive. The locations were as follows:

<table>
<thead>
<tr>
<th>Location</th>
<th>No.</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior cerebral-anterior communicating</td>
<td>485</td>
<td>27.4</td>
</tr>
<tr>
<td>Internal carotid-posterior communicating</td>
<td>467</td>
<td>26.4</td>
</tr>
<tr>
<td>Middle cerebral artery (first bifurcation)</td>
<td>348</td>
<td>19.7</td>
</tr>
<tr>
<td>Bifurcation (termination) carotid</td>
<td>110</td>
<td>6.2</td>
</tr>
<tr>
<td>Distal anterior cerebral</td>
<td>48</td>
<td>2.7</td>
</tr>
<tr>
<td>Multiple</td>
<td>247</td>
<td>14.0</td>
</tr>
<tr>
<td>Vertebral artery tree</td>
<td>64</td>
<td>3.6</td>
</tr>
</tbody>
</table>

It should be noted that the percentage of intracranial aneurysms in the vertebral-basilar system is relatively low. Three- or four-vessel angiography would be expected to provide a higher yield of posterior fossa aneurysms.

**Pathology**

Pathologists have been making contributions to the study of aneurysms for many years. One of the pioneers in this field was Turnbull '18, who reported 33 examples of "medial degeneration following congenital developmental deficiency" in 29 cases. However, the classical work of Eppinger was published many years before, in 1887. He insisted that in the small and smallest arteries, like those forming the circle of Willis and its branches, many aneurysms have a congenital origin and are due to an inborn defect of the elastic properties of the vessel wall; in others, congenital defects with subsequent degeneration of atheromatous processes complicating the defect are the essential factors in causation. A major contribution was provided by Forbus '30, who emphasized the role of medial defects in the genesis of aneurysms. Since that time a great deal of controversy has developed as to whether saccular aneurysms of the circle of Willis are congenital, acquired or combinations of both. The role of atherosclerosis has been emphasized, especially by such investigators as Walker and Allègre, '53, '54.

**Clinical Aspects**

With the introduction of the spinal puncture by Quincke 1891, it became possible to establish the diagnosis of subarachnoid hemorrhage in life. It required many years, however, for physicians to develop the necessary sophistication to distinguish between bloody spinal fluid and the results of a traumatic puncture. Indeed, this differentiation is still an occasional problem.

The introduction of cerebral angiography by Egas Moniz in 1927 provided an important new diagnostic tool which is now an essential part of the work-up of patients with all types of subarachnoid hemorrhage. It is now possible to visualize the posterior part of the circle as well as the anterior portion of the circle of Willis. Unfortunately, angiography is not entirely without risk; skill is required in its performance and its possible dangers must be measured against the information to be sought.

Rupture of an aneurysm may occur into the subarachnoid space, brain substance, subdural space or cavernous sinus. Profound changes may take place in the intracranial circulation, especially in that portion distal to the aneurysm. The aneurysm will sometimes reach such a size that it will present as a space-occupying lesion. Finally, the extravasation of blood into the subarachnoid space will often result in obstruction of flow of the cerebrospinal fluid and impairment of absorption of cerebrospinal fluid.

The symptoms of intracranial aneurysms are variable and depend on the location of the aneurysm and the effect on the surrounding structures. An aneurysm may remain asymptomatic and be found incidentally at autopsy.

The symptoms of subarachnoid hemorrhage vary with the acuteness of the bleeding episode and the amount of blood extravasated into the subarachnoid space. In the classical case, the patient suddenly complains of a violent headache, with nausea, vomiting and prostration. Disturbance of consciousness is usually associated with the ictus, and the patient may have a convulsion. The neck becomes stiff and there is restriction of the leg-raising tests. In many instances there is little to be found in the way of focal neurological signs. More detailed accounts of the symptoms and signs can be found in textbooks by Merritt '63, Elliott