Intracranial berry aneurysms have occasioned much controversy for many years, and there is still no unanimity of opinion regarding the etiology. Two basic theories are current. The first states that these aneurysms are due to maldevelopment or some inherent hypothetical weakness of the arterial wall—a so-called congenital theory. The second contends the aneurysms are the result of acquired degenerative changes in the vessel wall—the degeneration theory. Proponents of a third theory attribute the aneurysms to a combination of developmental and degenerative changes. Crawford (40) alleged that developmental medial defects, atherosclerosis, and hypertension play roles of varying importance according to the age at which the aneurysm develops, the only criterion for differentiating aneurysms of different etiologic types being the age of the patient.

The congenital theory has dominated the field for many years. Lancisi in 1728 first suggested that an arterial aneurysm (extracranial) could be due to a congenital mural defect (111). In 1859 Gull (82) wrote that a cerebral aneurysm “may be a simple pouch of all the coats [of the vessel wall], the pouch portion being as transparent and normal in appearance as the rest of the wall [and] giving the impression that it might have been some original deformity.” At a loss to explain their occurrence in young people, in the absence of gross disease of the parent arteries, and probably relying on the macroscopic appearance of the aneurysm and the cerebral arteries, he postulated a congenital origin; but, as the aneurysmal walls do not consist of healthy intima, media, and adventitia, his conclusion was false. Eppinger (56) considered that aneurysms must be due to an inborn defect of the elastic properties of the arterial wall and, therefore, labeled them “congenital.” Thus was founded the congenital theory which, although based solely on this hypothetical congenital weakness of the arterial wall, has dominated medical thinking henceforth. The hypothesis was later bolstered by circumstantial evidence, most of which will not withstand scientific or statistical analysis. The varied factors often used to substantiate the congenital theory include age, multiplicity of aneurysms, variations of the circle of Willis, persistence of vestigial vessels, familial occurrence, medial gaps or raphés, associated congenital abnormalities, and associated cerebral arteriovenous malformations (AVM).

In any appraisal of the theories of the etiology of cerebral berry aneurysms, it is a fundamental necessity to evaluate all evidence used as substantiation for any of the hypotheses, taking into account the vagaries of aneurysmal distribution in nature and coexistence with certain diseases.
Distribution of Berry Aneurysms in Nature

Propensity for the Cerebral Circulation

The characteristic feature of berry aneurysms is that they are saccular aneurysms arising from the fork, or crotch, of an arterial bifurcation. They do not occur on the large elastic arteries but rather on the peripheral distributing muscular arteries, i.e., cerebral, spinal, and splanchnic arteries. By far the commonest site is the cerebral circulation. Other features are the rarity of rupture of berry aneurysms of splanchnic arteries and the infrequency of their discovery radiologically. The distribution suggests some singularity of the cerebral vasculature which predisposes to this type of aneurysm formation.

Macroscopically, the most notable and perhaps crucial architectural peculiarity of the cerebral arteries is the thinness of their walls and their lack of perivascular support. The main branches tend to be long and tenuous and do not arborize shortly. This factor may be of little significance, since most aneurysms arise from the circle of Willis and the proximal portion of the major arteries at the base of the brain, rather than on their peripheral ramifications.

Histologically, the cerebral arteries are muscular in type, with the media and adventitia considerably thinner than those of extracranial arteries of comparable size (185). The internal elastic lamina is slightly thicker than in extracranial arteries and there are a few fine elastic fibrils in the adventitia, but the media is virtually devoid of elastica and the external elastic lamina of extracranial arteries is absent. In the arterial walls of babies, children, and many young adults, no vasa vasorum are found. They appear later as a consequence of mural thickening due to the development of atherosclerosis.

This unique architecture of cerebral arteries has been attributed to the need to obviate the effect of systolic pulsations on the brain and its functions (185). Although the precise reason may be as yet uncertain, the structural peculiarity cannot in itself constitute a developmental defect responsible for the prevalence of cerebral berry aneurysms, because the cerebral arteries of all humans and all lower mammals exhibit this same architectural arrangement, and cerebral aneurysms are not ubiquitous in man and are extremely rare in lower animals (173,180). However, it seems highly likely that the architectural differences account for the greater frequency of berry aneurysms in the cerebral circulation than extracranially. The low frequency of these aneurysms on the spinal arteries is no doubt due to their small caliber (Chapter 11) and to the lesser severity of atherosclerosis.

The blood velocity in cerebral arteries is higher than that in branches of the external carotid artery. The possibility exists that systolic pressure accentuation, due to the summation effect of reflected pulse waves, and possibly some disadvantageous natural frequency of the cerebral arterial walls may each contribute to the prevalence of intracranial berry aneurysms. These factors require further study, but they are not in themselves causative.

Propensity for Man

There is ample evidence that berry aneurysms are far more frequent in man than in lower animals. This is substantiated by the rarity of subarachnoid hemorrhage, severe cerebral atherosclerosis, and even spontaneous intracranial hemorrhage in lower animals (183). Stehbens reported multiple berry aneurysms, subarachnoid hemorrhage, and moderate atherosclerosis in one chimpanzee and microevaginations in four cerebral arterial forks of a young gorilla (183). Two chimpanzees used for cholesterol-feeding experiments have been found with unruptured cerebral aneurysms not attributable to experimental procedure (7). Although an occasional aneurysm has been described in a few lower animals, the lack of pathologic detail and histologic confirmation raised doubt about their nature, particularly in view of the prevalence of parasitic aneurysms in many quadrupeds and their rarity in man.

Berry aneurysms in man occur mostly on the large cerebral arteries of high flow rates, and their occurrence on spinal arteries, when these are grossly enlarged in the presence of aortic coarctation or a spinal arteriovenous aneurysm (Chapter 11), indicates the relevance of arterial caliber to the pathogenesis. The human brain as a consequence of its comparatively large size, has large cerebral arteries. Man, with his relatively large cerebral arteries, can thus be expected to sustain more aneurysms than lower animals. Hypertension appears to be an aggra-