Observations on the Pathology of Saccular Aneurysms

A. L. SAHS, M.D.*

Embryology

Very little has been added to the classical descriptions of the circle of Willis by Padget '44, '48. The circle of Willis is recognizable in a ventral view of the brain at an ovulation age of 52 ± 1 days. "Like vascular development elsewhere, that of the head arteries is characterized by a gradual dwindling or elimination of certain vessels, originally prominent, which serve temporary needs, paralleled by the elaboration of others."

Bassett '49 indicated that as early as the 3 mm. stage in the development of the fetus, vascular channels are well defined as such. They contain primitive nucleated erythrocytes. A single endothelial layer forms the walls of the vessels. "Vascularization occurs in response to metabolic demands from centers of proliferation. Early the primordial endothelial channels give rise to innumerable buds forming a plexiform germinal circulatory bed. This gradually differentiates into efferent and afferent structures forming a fine capillary mantle. Cleavage of vessel layers for skull, dura and brain next occurs, beginning in the basilar regions with multiple intricate anastomoses followed by adaptation to developmental alterations in form, size and rate of growth of the brain."

Evolution to the various components such as artery, vein, capillary and sinus types represents the final step in development, according to Bassett '49. Not until mural structure is well developed are the arteries physiologically and anatomically fixed as such, since there is much shifting about of arterial channels in young embryos. Realignment has progressed to identifiable anterior and posterior portions of the circle of Willis by the 9 mm. stage (4 weeks).

Large Arteries of the Base of the Brain

The subject of the large arteries of the circle of Willis has been reviewed in detail by Baker and Iannone '59, and the dimensions and dynamics by Murray '64. There are certain differences between the cerebral arteries and those of other portions of the body. In general, the cerebral arteries have a much thinner intima and media and a much more conspicuous internal elastic lamina than equivalent-sized vessels in other parts of the body. External to the media is a thin, loose adventitial layer. The adventitia and the intima apparently contribute very little to the strength of the vessel wall (Hassler '61).

The media is often thin or absent at bifurcations. These medial defects can be demonstrated with regularity in carefully-prepared specimens. As long as the elastic layer remains intact the medial defect is tolerated, and apparently nothing unusual takes place.

Variations in the Circle of Willis

Wilson et al. '54 in a study of 143 autopsied cases reported that an anomalous formation of the circle of Willis was noted in 118 of the 124 cases in which a complete description was available. Essentially, the anomalous formation resulted from hypoplasia of one or more of the component vessels, persistence of an embryonic stem of origin, or a combination of these factors. 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, 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.

Alpers et al. '59 reported comprehensively on the anatomical structure and variations in the circle of Willis. Eight hundred and thirty-seven brains were examined. Three hundred and fifty were selected for their survey. "Normal" circles of Willis were found in 52.3 per cent. The most striking anomaly was a filiform or string-like caliber of one of the component vessels in 27.5 per cent of the

* Professor and Head, Department of Neurology, University of Iowa, Iowa City, Iowa. Supported by USPHS Grant NB-02346.
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circles. This most frequently involved one or both posterior communicating arteries.

Alpers and Berry '63 reported that a comparative study of the circle of Willis in 91 cases of cerebral aneurysm revealed a high incidence of anomalies involving the posterior communicating arteries, the embryonic derivation of the posterior cerebral artery, and absent arteries.

Riggs and Rupp '63 reported that the configuration of the circle of Willis generally considered “typical” was found in only 192 of 994 cases (21 per cent) while “deformity” of the circle occurred in 802, or 79 per cent. Such structural defects would generally result in greater restriction of collateral flow than in the circle of Willis with the “typical” configuration.

Because of the possible importance of this area in the overall consideration of cerebrovascular disease, Pallie and Samarasinghe '62 have attempted to quantitate the various components of the circle of Willis.

Theories of Formation of Saccular Aneurysms

Hassler '61 reviewed the many theories of the etiology of intracranial arterial aneurysms. At the present time the etiological factors center around two causes, or combinations:

(1) The concept of congenital defects. This theory was suggested by Eppinger, 1887 who indicated that certain aneurysms occurred at the site of defects of the elastic layer of arteries, and that these breaches were inborn or congenital defects. He did, however, mention complicating degenerative or atheromatous processes in some instances. It remained for Forbus '30 to elaborate the concept of medial defects. Forbus believed that the intima bulged into the weak medial defect and that the elastica then underwent degeneration secondary to the strain produced by overdistention. Glynn '40, however, was unable to demonstrate evidence of bulging of the medial defect under a greatly increased pressure.

Forster and Alpers '45 stated that their observations conformed with the prevailing opinion that the defect in the media was congenital in type, and that the changes in the elastic membrane varied widely and were independent of degenerative changes elsewhere. These authors felt that the changes in the elastica were secondary to the formation of the aneurysm and minute classification on the basis of a variable anatomical feature did not seem warranted.

Bremer '43 favored the concept of an embryological defect as the cause of intracranial aneurysms by virtue of persistence of remnants of the embryonic vascular system. Bassett '49 also favored the idea that congenital aneurysms probably exist as unresolved vestiges of a primitively normal circulatory system. He stated that, "This primitive system has erred in its response to the evolutionary stimulus of resorption or modification for the pattern of normality as seen in the embryologically mature organism."

(2) The concept of postnatal changes in the arterial wall. Progressive fragmentation of the elastic layer was described in detail by Hackel '28. He described the internal elastic lamella as one of exceptional thickness and consisting, during the first two years of life, of a poorly stained middle layer with heavily stained borders. With increasing age there is a tendency for the inner layer to become raised. The space between it and the main lamella becomes occupied by collagen and fine elastic fibrils. During infancy this splitting and increase of elastic tissue is slight, is confined to the larger of the cerebral vessels and is most marked at points of branching and bifurcation. In older subjects these changes become exaggerated and begin to appear in the smaller vessels, but, as in the larger vessels, the condition is always more marked at the points of branching and bifurcation. These intimal hyperplasias are said to be independent of atheromatous degeneration. No fat is demonstrable within them by the usual staining methods.

Glynn '40 called attention to the fact that true saccular aneurysms of noninflammatory origin occurred more frequently upon the cerebral vessels than upon any other of the muscular arteries. He found medial defects in 80 per cent of the bifurcations examined. He proved that the unsupported elastic elements of the vessel wall could normally withstand pressures of 600 mm. Hg, without bulging. Glynn '40 concluded that, "the medial defect did not constitute a locus minoris resistentiae and could play no part as