Aneurysms of the Anterior Communicating Artery and Gross Anomalies of the Circle of Willis*

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The various theories of development of aneurysms of the intracranial vessels are well known. Although early investigators thought embolic and inflammatory reactions were important factors in their formation, later workers stressed the probable importance of developmental defects of the media at sites of branching, supplemented by degenerative changes of the internal elastic membrane. Turnbull suggested that cerebral aneurysms formed as a result of degeneration of the media at sites of inherent weakness of the media as at points of branching. Forbus concluded, however, that although a small diverticulum may occur at the site of a medial defect associated with the bifurcation of an artery, an "anatomical" aneurysm will not develop while the internal elastic membrane is still intact. He concluded further that miliary aneurysms as such are not congenital malformations but are acquired lesions arising from a combination of focal weakness in the vessel wall, secondary to a congenital defect of the media and degeneration of the internal elastic membrane, due to continued overstretching of this membrane. While some have, in general, supported this theory of the development of aneurysms, others have dissented. Bassett and Lemmen remarked that most aneurysms of the cerebral arteries represent vestiges of the primitive circulatory system.

It is now well known that the incidence of gross anatomical anomalies of the circle of Willis is high. Approximately 60 years ago, however, Blackburn finding at autopsy that 52 per cent of the circles of Willis in a group of psychiatric patients were anomalous, concluded that anomalies of the circle of Willis might be a cause of psychiatric illness. Actually, if by normal we mean a completely symmetrical circle with thread-like communicating arteries illustrated in current texts (Fig. 1), then the incidence of anomalous circles is probably even greater than 50 per cent. In the treatment of intracranial aneurysms with subarachnoid hemorrhage, gross anatomical anomalies of the circle have been considered more as a factor in circulatory intolerance of carotid occlusion than as a cause of the aneurysm itself. Chase mentioned that gross congenital anomalies of the circle of Willis are very common and suggested that these anomalies may be expected to be accompanied by structural defects in the walls of the vessels. Wilson et al., however, reported that in 85 per cent of 40 aneurysms found on the anterior communicating artery were associated with hypoplasia of the first portion of one anterior cerebral artery. Stehbens stated that this was the only anatomical variation that he was able to correlate with the location of cerebral aneurysm in a study of 251 brains with 333 aneurysms.

Asymmetry of the circle of Willis seems to be primarily the result of a difference in size, and, in turn, a difference in the pattern of

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Fig. 1. Normal circle of Willis. It is complete, symmetrical and has thread-like communicating arteries.
branching of the internal carotid arteries. There are 2 relatively common, variations from the normal pattern of branching of this artery. One is the persistence of the fetal pattern of trifurcation in which all 3 of the ipsilateral major cerebral arteries arise from the internal carotid while the posterior cerebral segment of the circle continues, as in the early fetus, to be a small, thread-like, posterior communicating type of vessel (Fig. 2). In the other common variation the artery divides into the middle cerebral artery and an unusually large anterior cerebral branch. The latter in turn, divides at the anterior pole of the circle into both anterior cerebral arteries. This pattern of terminal branching of the internal carotid artery has been termed anterior trifurcation (Fig. 3). The proximal part of the branch to the opposite side is oriented in the manner of the usual anterior communicating artery but is several times larger. It usually is connected to the opposite internal carotid by a hair-like, communicating type of vessel. The opposite, anterior cerebral segment of the circle, however, may be missing, in which case the contralateral internal carotid simply continues as the middle cerebral artery (Fig. 4) or may bifurcate into the posterior cerebral artery and the middle cerebral artery (Fig. 3).

As would be expected, there are many gradations between the normal bifurcation of the internal carotid and each of these forms of trifurcation. A form representing a transition stage between early fetal trifurcation and normal adult bifurcation has been described as transitional trifurcation. In addition to the anterior trifurcation pattern of terminal branching, the internal carotid may have a transitional posterior communicating branch (Fig. 5). When combined with a contralateral internal carotid that fails to bifurcate (Fig. 5), this is the most dominant form of internal carotid. Of interest also in Fig. 5 is the congenitally small vertebral artery and the relatively large contralateral vertebral artery. A great discrepancy in the caliber of the vertebral arteries is not uncommon but we have noted no consistent relation between the asymmetry of these arteries and the asymmetry of the circle of Willis.

**Material and Methods**

The present study is an analysis of 1,000 circles of Willis obtained at autopsy at Ochsner Foundation Hospital. The circles, with the subarachnoid portion of each vertebral artery, the basilar artery, and each major cerebral artery to the point of its first major division distal to the circle, were fixed in 10 per cent formalin.

The incidence of aneurysms in this series may be higher than expected for 2 reasons. Our neurosurgical service represents a relatively large proportion of the hospital population and thus a comparatively large number of patients with subarachnoid hemorrhage are admitted. Moreover, the series represents a special professional interest in the dissections and probably few aneurysms were overlooked.