PATHOLOGICAL ANATOMY AND ANGIOGRAPHY OF INTRACRANIAL VASCULAR ANOMALIES*

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Dorcus Hager Padget\(^1\) has pictured and described the changes in development of the cranial arteries of the human embryo in a beautiful monograph which is an amplification of the chapter written for Dandy's book\(^8\) on intracranial aneurysms. The information contained therein renders more comprehensible some of the anomalies of the adult cerebral vascular tree. Indeed, Dandy\(^4\) held that there could be but one explanation of aneurysms of the congenital type—that they have their origin in defects incident to the resolution of the early vascular bed. The alternative hypothesis is that of Forbus,\(^9\) which ascribes the formation of aneurysms to persistent pressure against the weak point of bifurcation of an artery—where the media may be defective. That this has not been a completely satisfactory explanation was pointed out by Richardson and Hyland.\(^16\) They felt it necessary to assume that although structural defects predispose and determine the sites of berry aneurysms, some additional, acquired lesion was required to weaken the elastica to cause the outpouching. Bremer\(^3\) has given explanations for the lack of development of arterial walls in outpouchings, based on the lack of pulsation which induces developing arteries to form walls to contain the pulsation, or to degeneration of the walls when they no longer function in this manner. He believes that rapid stretch at bifurcations of the middle cerebral from the carotid, and of the posterior communicating arteries at the basilar could contribute to aneurysmal formation since muscularis appears latest in the fork of such angles. Other aneurysms not at forks might be derangements of embryonic vessels (which develop before the muscular coats are laid down). It appears obvious that a more careful consideration of the embryologic features of the vessels of the brain may give better understanding of intracranial vascular anomalies. Mrs. Padget has graciously permitted me to reproduce some of the drawings from her monograph.

ANEURYSMS

Fig. 1 shows the mode of formation of the basilar artery from the two longitudinal neural arteries under the neural tube in the early embryo (Congdon,\(^6\) Padget\(^15\)). At the line of fusion one might expect medial defects to occur, and such opposing deficiencies have been described in the basilar

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artery of the child of 13 weeks whose case was reported by Forster and Alpers. The aneurysm in this case was at the bifurcation of the basilar into the posterior cerebral arteries. An exactly similar bulge was depicted in a human embryo of 42 weeks by Bremer—surely the earliest "berry aneurysm" known! The adult counterpart of this type of aneurysm is shown in Fig. 2. Since these long vessels fuse by growing toward 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 at the bifurcation (Figs. 3 and 4).

Another primitive vessel, the primitive vertebral-basilar anastomosis, is shown in Fig. 1. This disappears when the vertebral arteries form by fusion of cervical segmental arteries and come to join the basilar artery in the midline. Persistence of such an artery or its early branches might well account for the location of an aneurysm well to one side of the basilar artery, such as has been described by Schwartz in his case of successfully treated posterior fossa aneurysm. It is also probably some remnant such as this which accounted for a peculiar vessel which compressed the last four cranial arteries lateral to the medulla oblongata, in the man whose arteriogram is shown in Fig. 5. This should be compared with the normal axial vertebral angiogram shown in Fig. 6.

The primitive trigeminal artery (Fig. 1) is the earliest communication between the carotid and the basilar systems. With establishment of the posterior communicating artery (which in the embryo is one of the two basic termini of the internal carotid artery), this vessel dwindles and disappears. Occasionally it persists in the adult as a large artery passing between the internal carotid artery in the cavernous sinus and the basilar artery in the region of the trigeminal nerve roots. In this event, the vertebral artery on that side (or both vertebral arteries) may be considerably reduced in size (Sunderland, Altmann). When it does not persist, there may arise