THE PATHOLOGY OF SUPRATENTORIAL ANGIOMAS*

WALLACE B. HAMBY, M.D.

The University of Buffalo School of Medicine and the Buffalo General Hospital, Buffalo, New York

(Received for publication April 16, 1957)

The origin and anatomy of the cerebral angiomas has frustrated pathologists over the years as much as their treatment has baffled surgeons. An extensive literature has developed, replete with picturesque nomenclature based upon attempts to describe the appearance of lesions seen at the operating table or at necropsy. It is believed that no useful purpose would be served now in reviewing these and attempting to explain them in light of present information. The surgical descriptions are not entirely basic nor accurate because the bulk of the lesion is largely submerged under the cortex and hence invisible to the examiner. The pathologic descriptions have been faulty because of deflation of the lesions at the time of examination by lack of the expansile blood stream that characterizes them in life. Also confusing the picture of the dead lesion are the alterations produced in the component vessels by blood under arterial pressure, which dilates veins and "arterializes" them to withstand the added stress. Vascular resistance being lowered by the shunt, arteries dilate to carry more blood under less than usual pressure, and lose some of their usual characteristics. Histologically the lesions appear only as a tremendously convoluted mass of arteries, veins and intermediate types of vessels of extremely variable diameters traversing cerebral tissues altered in structure by the intervening and now deflated vessels.

CLASSIFICATION

Cushing and Bailey\(^2\) were able to separate the "intracranial blood-vessel tumors" into two principal groups, a) the hemangioblastomas, truly neoplastic masses confined chiefly to the posterior fossa, and b) the largely supratentorial group composed of blood vessels contained within cerebral tissue. We are concerned with the latter group. Cushing and Bailey divided these into a) telangiectases, b) venous angiomas and c) arterial (arteriovenous) angiomas. Again, it is with the latter two groups that we are concerned. Olivecrona and Riives\(^9\) agreed with Cushing and Bailey that these are histologically inseparable; the difference is physiologic and depends upon the degree of arterialization of the lesion. Upon the advent of angiography it became apparent that earlier expressed suspicions against the existence of purely venous angiomas were well grounded; in all reported cases the

---

lesions have proved to have arterial supply. The last persisting possibility remained in the very small intracerebral clusters of veins that occasionally are seen largely destroyed in the walls of intracerebral hematomas. Dorothy Russell was able to find a little better than usually preserved example of this type and histologic examination showed it to contain vessels having elastic laminas characteristic of arteries. It seems that there is little if any pathologic evidence to support the concept of purely venous angiomas. Physiologically there is reason to question why a cluster of veins deep within the cerebrum should rupture and produce a massive clot. It is believed reasonable then to ignore these time-honored concepts and to concentrate upon the pathology of the common arteriovenous malformation.

**GROSS APPEARANCE**

The malformation usually is cone-shaped, its base being on the surface and its apex near or intruding into a lateral ventricle. The apex may be demonstrated at times in cerebral air studies as a projection into the ventricle, and this may be lobulated, because of dilated veins on its tip. In most lesions large enough to produce symptoms leading to their disclosure, dilated cortical veins drain from the lesion and run to one or more of the dural venous sinuses. It is the appearance at operation of these huge veins, often bright with arterial blood, that has fathered so many of the colorfully descriptive titles given the lesions. Since the use of angiography, it has become recognized that many of them, especially behind the fissure of Rolando, also drain into the deep cerebral veins and thence into the Galenic system. Some, of course, drain exclusively by this route.

The arteries supplying the lesion usually are inconspicuous in the gross specimen, the large parent trunks being buried in the cerebral fissures and their branches being smaller than the more superficial dilated veins. The parent arterial trunk is usually that of the lobe of the brain harboring the mass, and commonly this means more than one vessel. The importance of supply from the posterior cerebral artery and the choroidal arteries has become properly appreciated only since the employment of angiography in diagnosis.

Inspection of the dead lesion shows it to be a spongy mass intruding into the brain substance. When it is cut across, the area containing the draining veins may appear cavernous. The complex of large veins usually lies within the subarachnoid spaces, which are expanded by the mass of vessels. Below this, and extending through the vascular cone of the lesion, the white matter is laced with numerous channels of various diameters. The identity of these vessels is impossible grossly, and very difficult under the microscope. Angiography provides an admirable method of delineating the gross vascular structure of the lesion and Potter considered this the most reliable facility at our command for the study. Unfortunately, it does not sharply reveal the vessels that usually intervene between the arterial and venous sides of the circulation. It was believed that dissection of such a lesion under the microscope might add information. Simmonds illustrated his Case 2 in this