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# The Corpus Callosum, a Unique White-Matter Tract: Anatomic Features That May Explain Sparing in Binswanger Disease and Resistance to Flow of Fluid Masses

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**The corpus callosum exhibits several unusual features: arteriole-venule pairs, perivascular fibrous alae, and recurrent companion arterioles. These and other anatomic aspects are compared and contrasted with those of the centrum semiovale. Vascular changes occurring with aging or hypertension, commonly seen in the centrum semiovale, rarely develop in the corpus callosum. The vascular supply to the central zone of the genu and body of the corpus callosum, via short penetrating arterioles, is similar to that of the cerebral cortex, whereas the vascular supply to the extreme lateral corpus callosum, centrum semiovale, and basal ganglia is substantially carried by long end-arteries.**

**When observed critically, the anatomic features of the corpus callosum may explain such clinically observed phenomena as its relative resistance to flow of fluid masses, radiation injury, Binswanger disease, lacunar infarction, hypoxia, and hypoperfusion.**

The corpus callosum, literally the "hard body," so-named because of its firmness in the fresh state, is the major white-matter tract crossing the interhemispheric fissure. It cannot strictly be termed a commissure. Although most axons cross directly to homologous areas of the opposite hemisphere, a significant number travel obliquely in the corpus callosum to reach contralateral association areas. Another group of axons pass downward from the indusium griseum to the fornix and from the cingulate gyrus to septal nuclei [1].

Experience with pathologic material and modern neuroimaging procedures leaves the impression that the corpus callosum has no inherent resistance to injury from vasospastic and thromboembolic strokes [2] or de(dys)myelinating diseases [3, 4]. On the other hand, the corpus callosum appears to be more resistant than central white matter to the flow of edema fluid [5], radiation injury [6, 7], hypoxemic injury [8], and Binswanger disease [9–11].

We report some interesting anatomic features in the corpus callosum observed as part of a continuing larger study of the vascular changes in hypertensive human subjects.

## Materials and Methods

The brains of 21 hypertensive and five normal subjects were obtained at autopsy. The hypertensives were 48–85 years old (average, 65 years) and the controls were 43–70 years old (average, 59 years). The brains were removed after a variable postmortem interval of 4–24 hr. They were placed in a refrigerator for 4 hr to attain a suitable consistency for sectioning. The cerebral hemispheres were sliced coronally, and large thick blocks of tissue (up to 5 × 5 × 1 cm) were cut from 10 areas. Two regions, the frontal lobe sections and thalamic sections, included the corpus callosum. From 12 of these brains a sagittal section of corpus callosum from rostrum to midbody was also obtained. In two other brains the corpora callosa were sectioned horizontally. This material serves as the basis of our report.

The tissue blocks were fixed in cold, weak formalin followed by progressively higher concentrations of alcohol prior to celloidin embedding and sectioning at 100, 500, and 1000

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μm on a base-sledge microtome. The sections were stained histochemically with the use of the activity of the native nonspecific alkaline phosphatase enzyme present in the endothelium of capillaries, arterioles, and the smallest arteries [12]. The final endothelial reaction product is brown-black lead sulfide, which makes the microvascular bed of the 100-μm sections suitable for light microscopic observation after first counterstaining with cresyl violet acetate and light green (or other counterstains such as Congo red and myelin), and then mounting and coverslipping. The presence of the lead precipitate also makes the endothelium relatively impervious to X-rays. High-resolution contact radiographs of the 1000- and 500-μm-thick sections were made at 10 kV with the copper anode tube of an ISBR-60 microradiographic unit* and Kodak SO-343 film.† The radiographs were mounted on glass slides with mounting medium and coverslips and examined with a light microscope.

Tissue blocks for routine paraffin sections and H and E stain were also obtained from adjacent slices of brain. These sections were studied for vessel-wall changes and used for overall correlation with the findings observed on the alkaline phosphatase preparations. When expedient, trichrome, Holzer, Bielschowsky, and gliofibrillary acidic protein stains were also used on 5-μm sections from the paraffin blocks.

MR images obtained with a 1.5-T unit were selected from patient material to illustrate certain features. Where stated in the figure legends, these patients are separate from those yielding the pathologic material.

## Results

The arteries‡ of the pial-arachnoidal plexus and their larger perforating branches to the cerebrum are negative for alkaline phosphatase. The enzyme first appears in a patchy or streaky fashion in penetrating and intraparenchymal vessels with diameters of approximately 200–50 μm; that is, in the smallest arteries and largest arterioles. In this size range exchange of nutrients begins to occur [13]. Smaller arterioles and the capillary bed are strongly positive for alkaline phosphatase. Usually, venules are alkaline phosphatase negative; most veins in the choroid plexus and a few in the subependymal region show moderate or patchy staining, but these are easily distinguished as veins by their wall structure.

The vessels of the corpus callosum have the same staining characteristics as vessels in other parts of the brain. The vascular supply to the genu and body of the corpus callosum is derived from the pericallosal artery and is provided by an irregular row of 80- to 100-μm paramedian arterioles, streaky with alkaline phosphatase stain, penetrating vertically or obliquely backward from the superior surface (Fig. 1). The earliest branching arterioles from the main callosal penetrators recur back up the same Virchow-Robin space for a distance before diverging to their ultimate destinations (Figs. 1 and 2). The sharply reversed course of these branches contrasts strongly with the gradual, "candelabra"-shaped curves of cortical arteriolar patterns (Fig. 2). Deeper in the corpus callosum the arteriolar branches are nearly perpendicular, and the deepest branches have a more conventional branch angle

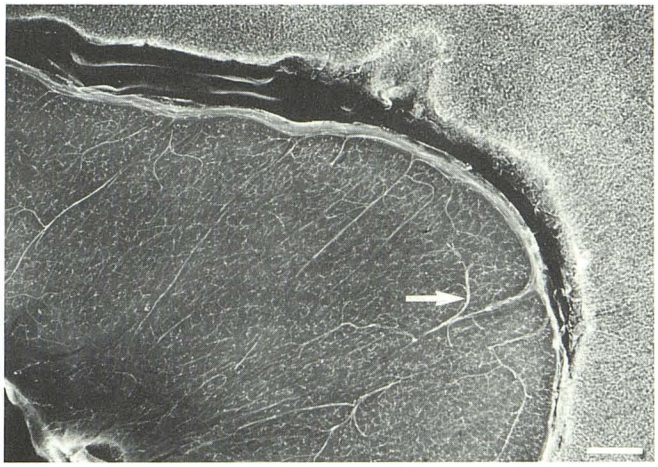


**Fig. 1.—Corpus callosum, genu. Contact microradiograph of parasagittal celloidin section, 500 μm thick. Lead sulfide reaction product of alkaline phosphatase stain in endothelial cells attenuates low-energy X-ray beam. Short (<8-mm) penetrating arterioles enter at irregular intervals from superior surface. Note recurrent companion branch (*arrow*). Others can be seen. This "row" of arterioles enters corpus callosum approximately 2–5 mm from midline. Bar = 1000 μm. (Reprinted from [14], with permission.)**

(i.e., the diverging blood flows make an acute angle) (Figs. 1 and 2). The distal branches reach to the subependymal region of the lateral ventricular roof and some branches enter and supply the septum pellucidum and fornix. These penetrating arterioles are usually 8 mm or less in length. An insignificant number of arterioles enter the undersurface of the corpus callosum from the fornix and septum pellucidum.

Laterally, a number of arterioles and small arteries (up to 175 μm) enter the brain in the depths of the callosal sulcus and supply both the corpus callosum and cingulate gyrus (Fig. 3), but the callosal branch is usually 100 μm or less. The largest of these arteries entering at the depths of the callosal sulcus may travel several millimeters anteriorly or posteriorly before giving branches to the cingulate medulla, the *lateral one-quarter* of the corpus callosum, and the territory where these structures blend. Such an artery, severely afflicted by lipohyalinosis and wall dissection, is seen in Figure 4 giving rise to a large lacunar infarct at the zone where corpus callosum and cingulum meet. These arteries, because of their extended anteroposterior course on penetration and their relatively large diameters, are comparable to the long medullary penetrating arteries of the centrum semiovale (deep hemispheric white matter at the level of the corpus callosum) and are subject to the same atherosclerotic and arteriosclerotic disease processes.

Still farther laterally, at a zone corresponding to the outer one-quarter of the lateral ventricular roof, blood supply is via long penetrating medullary arteries and arterioles entering the brain through the cortex of the cingulate gyrus (Fig. 5).

Arteriole-venule pairs were commonly found in the corpora callosa (Fig. 6). Not every arteriole had a companion venule, but at least one example was found in each corpus callosum studied on sagittal or axial sections.

While the majority of axons were cut in cross section on the sagittal sections, a significant minority of axon bundles

* Softex, Tokyo, Japan.

† Eastman Kodak, Rochester, NY.

‡ We have been careful to use *artery* when we mean a thick-walled afferent vessel greater than 100 μm in diameter. An *arteriole* is a thick-walled afferent vessel less than 100 μm in diameter ultimately supplying the capillary network.

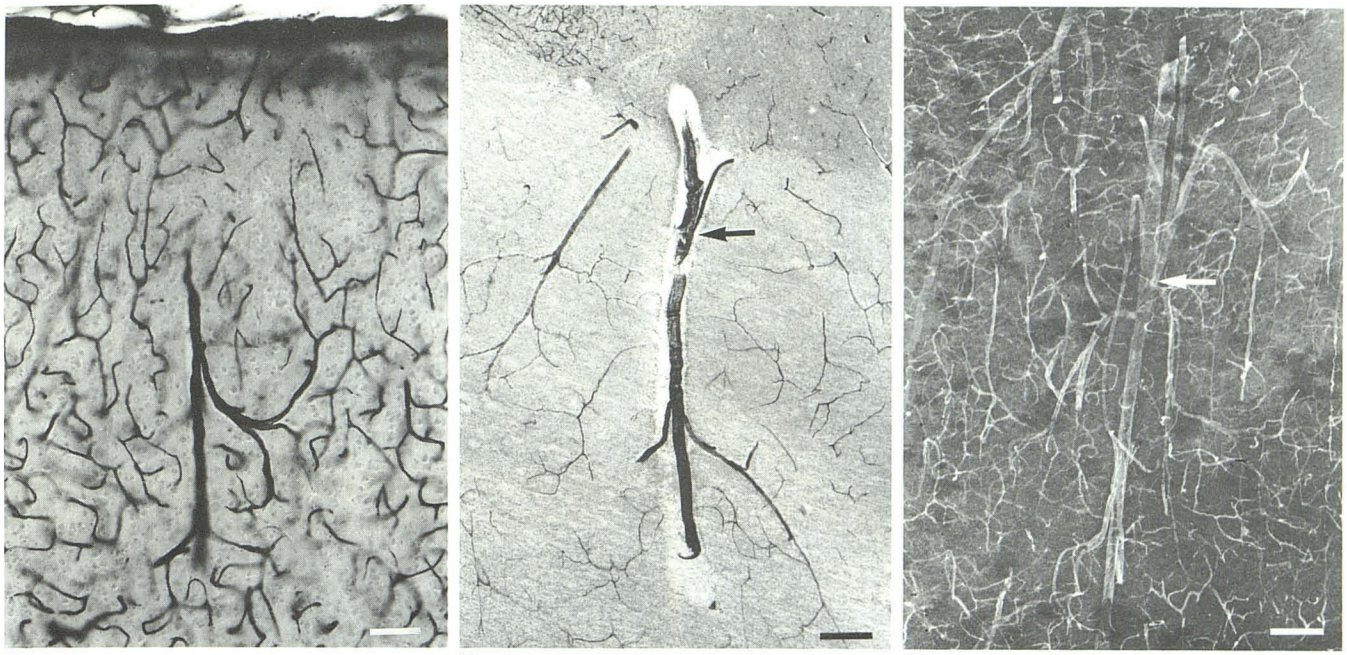



**Fig. 2.—Comparison of branching patterns in cortex and corpus callosum. Celloidin sections, alkaline phosphatase.**
***A*, Cortex, 100 μm thick. Arteriolar branches have candelabra shape. Bar = 100 μm.**
***B*, Corpus callosum, 100-μm-thick coronal section. Companion recurrent arteriolar branch (*arrow* at point of takeoff) is visible. Deeper in tissue, angle of branching becomes perpendicular and deeper still, gently curved like cortical branching pattern (see also Fig. 1). Bar = 250 μm.**
***C*, Microradiograph of 500-μm-thick section of corpus callosum shows several recurrent companion branches (*arrow*). Bar = 250 μm.**

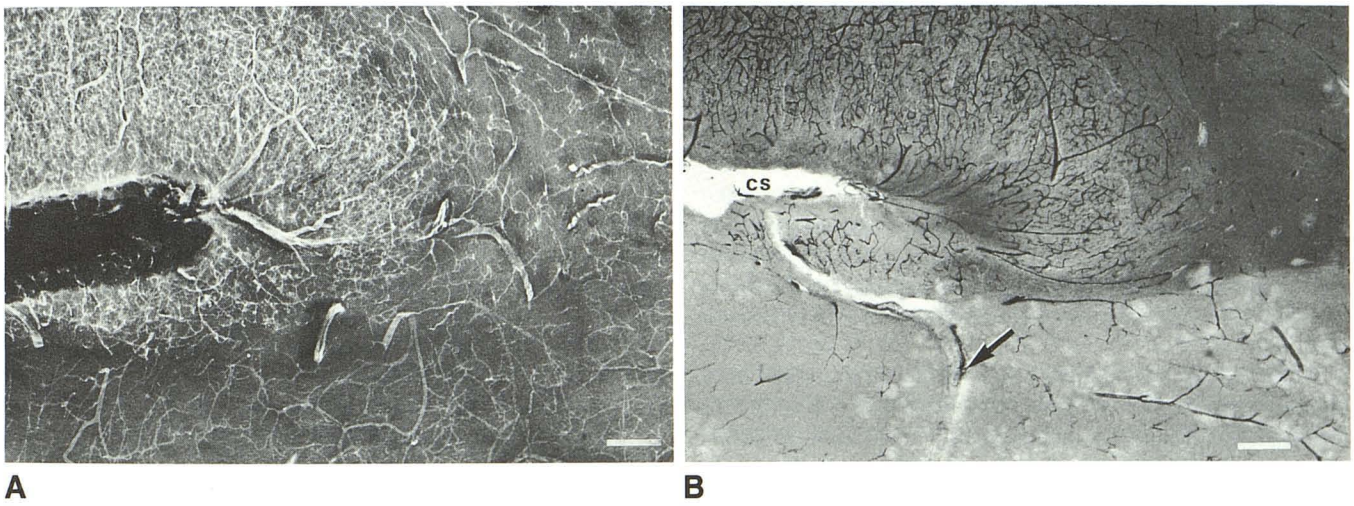
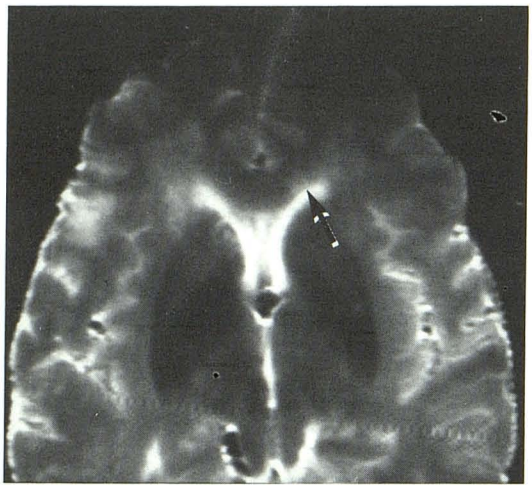



**Fig. 3.—Corpus callosum, body. Alkaline phosphatase–stained arterioles enter brain at callosal sulcus (cs). Some branches will supply cingulate gyrus (above) and others lateral corpus callosum (below).**
***A*, Contact microradiograph of 500-μm-thick coronal celloidin section. Bar = 250 μm.**
***B*, Photomicrograph of 100-μm coronal celloidin section. Large arteriole with patchy alkaline phosphatase stain and darkly stained recurrent companion arteriolar branch (origin at *arrow*) enters through indusium griseum to supply deeper portions of corpus callosum. Gray matter can be distinguished from white matter by greater density of gray-matter capillary bed. Bar = 250 μm.**

coursed obliquely backward or downward. This was apparent on thick (100-μm) sections counterstained with cresyl violet and light green, as well as on thin (10-μm) sections with Bielschowsky stain.

Not noticeable on routine H and E sections, but apparent on thick sagittal sections, were vertical columns of cells at irregular intervals in the corpus callosum. The character of these cell columns was elucidated on axial sections perpen-



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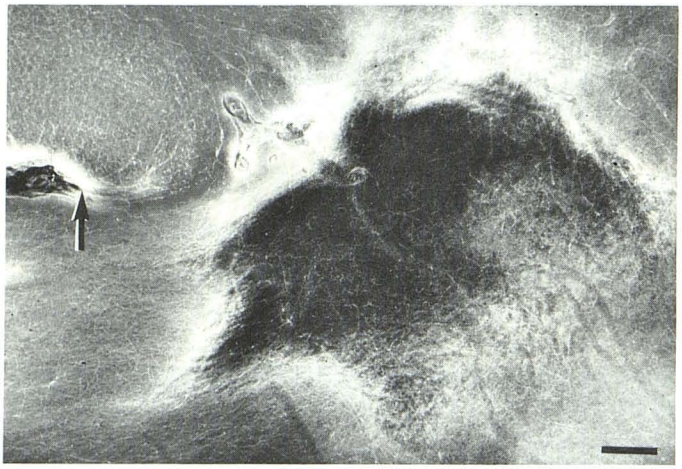

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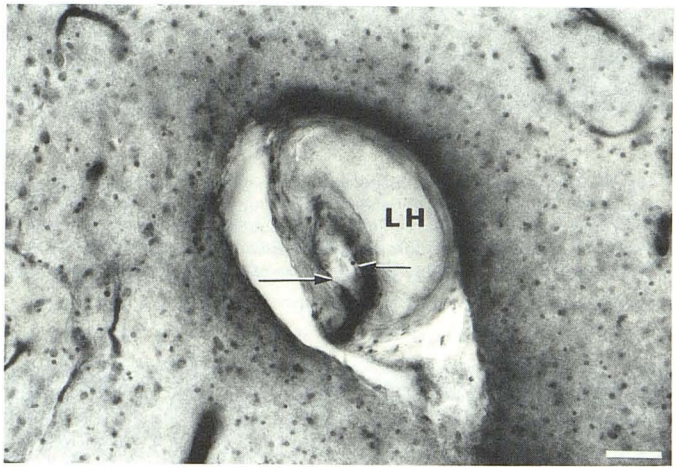



C

**Fig. 4.—Lacunar infarct, white matter, outer one-quarter of corpus callosum in hypertensive patient.**

***A*, Postmortem MR image, spin-echo pulse sequence, 1800/80/2 (TR/TE/excitations). Abnormal signal in extreme outer margin of corpus callosum on left (*arrow*).**

***B*, Microradiograph of 500-μm-thick alkaline phosphatase–stained section through same lesion. Depth of callosal sulcus is indicated by *arrow* at seam between cingulate gyrus (above) and corpus callosum (below). Radiolucent area with jagged edges represents lacuna loosely filled with cellular debris and vessels. Medullary artery supplying territory of lesion is seen with major branch passing into lacuna. Bar = 500 μm.**

***C*, Adjacent (proximal) 100-μm section; high-power view of artery supplying territory of lacuna. Narrowed true lumen is lightly outlined by alkaline phosphatase stain (*arrows*). Muscular wall is dissected by lipohyalinosis (LH). Note origin of darkly stained arteriolar branch below arrows. Bar = 50 μm.**

**Such arterial and lacunar lesions would not be anticipated in central portion of corpus callosum, an area supplied by short arterioles, and indeed were not observed there in any cases studied.**

dicular to the direction of the penetrating callosal arterioles. The cells were fibrocytes and were found in alae, composed of collagen but with a few glial fibers, which ran for a variable distance laterally after investing arterioles or arteriolar-venular pairs (Fig. 7).

When aging and hypertensive vascular changes were found, they occurred mainly in other regions of the cerebrum (basal ganglia, thalamus, and deep hemispheric white matter) and only rarely in the corpus callosum.

## Discussion

This method of anatomic investigation of the vascular tree with histochemical staining has advantages over injection techniques, which usually do not distinguish between arterial and venous systems and also may cause rupture and other artifacts (often mistaken for microaneurysms). Methods that stain intravascular contents are flawed because veins and arteries are hard to distinguish and the RBCs may leak out of the cut sections. Corrosion cast techniques destroy surrounding parenchyma and probably are unsuitable for study of the more sparsely vascularized deep white matter where the fragile cast will not support itself without fracture. Several of these techniques for vascular study preclude any examination of the neuropil; with alkaline phosphatase staining of the vessels, the surrounding neuropil can be concurrently examined with a choice of counterstains. Standard neuropathologic 5- to 10-μm paraffin sections can only show bits and fragments of vessels passing through the specimen. Our thick celloidin sections encompass an intact vascular pattern that can be examined by light microscopy (at 100 μm) and soft X-ray microscopy (at 500 and 1000 μm).

The active enzyme exploited in this histochemical stain (nonspecific alkaline phosphatase) is a phosphomonoesterase whose physiologic function in the endothelial cell in life is not clearly established [16]. (Interpretation is further complicated by the fact that the reaction used here, like most standard histochemical and biochemical tests for this enzyme, is carried out at nonphysiologic [alkaline] pH.) There is some circumstantial morphologic evidence that the presence of the endothelial alkaline phosphatase enzyme is related to exchanges across vessel wall membranes. When traced down the arterial

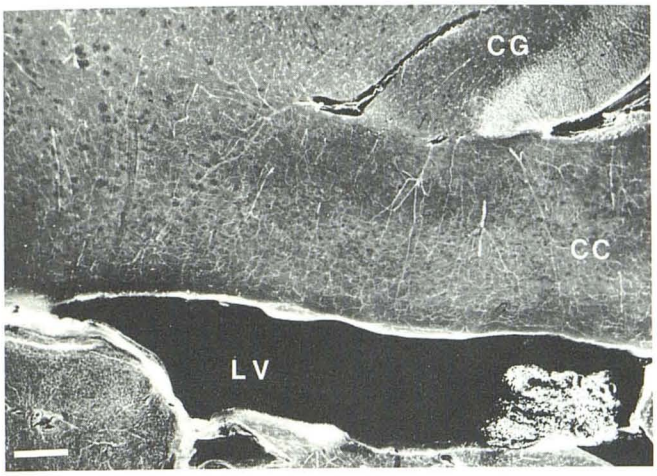



**Fig. 5.—Coronal section of frontoparietal area shows lateral ventricle (LV), corpus callosum (CC), and cingulate gyrus (CG). Microradiograph, 500-μm section, alkaline phosphatase. Note long medullary arteriole from cingulate gyrus supplying outer portion of lateral ventricular roof. This area is no longer exclusively corpus callosum and is subject to same vascular disease processes as centrum semiovale. Bar = 1000 μm.**

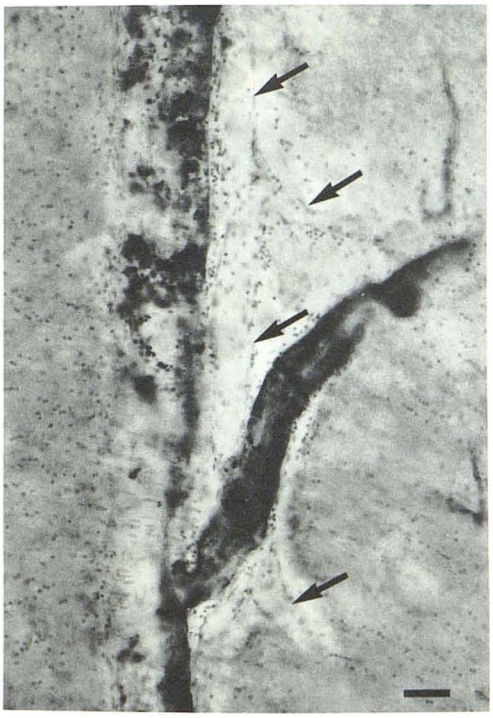


**Fig. 6.—Arteriole-venule pair in coronal 100-μm celloidin section of corpus callosum (up is superior). Unstained venule with its tributaries (*arrows*) parallels thick-walled arteriole with patchy alkaline phosphatase stain. Bar = 50 μm. (Reprinted from [14], with permission.)**

tree, the stain (indicating presence of alkaline phosphatase) is first seen in small arteries and large arterioles; these vessels are surrounded by a capillary-free zone of brain parenchyma [13, 17], implying that the vessel is itself nourishing the surrounding cylinder of brain parenchyma. By contrast, nourishing capillaries closely approach the walls of larger penetrating arteries that have no phosphatase stain.

The blood supply to the central corpus callosum is via short penetrating arterioles with a diameter not exceeding 100 μm and a length not greater than 8 mm. They enter the superior surface of the corpus callosum and run downward and backward. Blood flow in the earliest branches diverges from the flow in the parent vessel at an obtuse angle of nearly 180° (Figs. 2B and 2C). Except for rare examples elsewhere in white matter, this pattern is unique to the corpus callosum. The pattern of the earliest branches is probably imposed on the arterioles by the rapid differential growth pattern of the corpus callosum and the ingrowth of late-crossing axons [18] "shouldering" up these branches. The vascular pattern of the cortical gray matter and that of the corpus callosum are similar in that both areas are supplied by short arterioles, but the vessels supplying the cortical gray matter usually are less than 50 μm in diameter, and their branches have a gently curved candelabra configuration (Fig. 2).

The blood supply to the centrum semiovale is primarily via long (20- to 50-mm) arterioles and *arteries*, which may enter the apical area of a gyrus or the depths of a sulcus. The terminal ramifications of several lenticulostriate arteries also supply the inferior zone of the centrum semiovale. Unlike arterioles, these arteries (>100 μm, by definition) are vulnerable to atherosclerotic disease (Fig. 8) and dissection (Fig. 9). Furthermore, arteries penetrating into the brain are, with rare exceptions, end-arteries [19], and a luminal narrowing in these vessels would be expected to have a much greater clinical impact than a similar narrowing in the subarachnoid surface arteries, which are interconnected by a rich network of anastomoses.

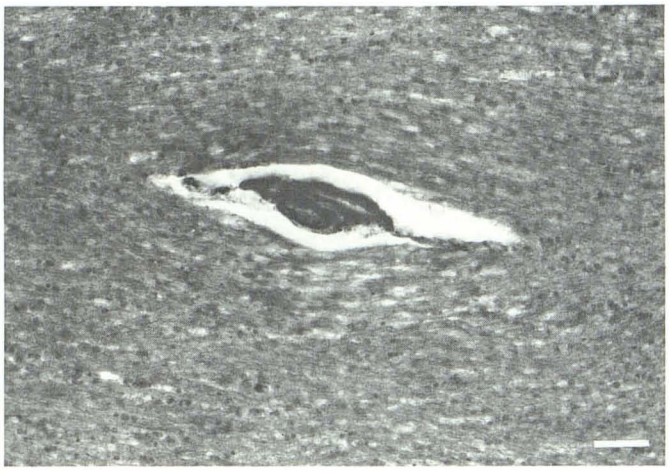
Where the axons and vessels of the cingulate gyrus begin to intermingle with those of the corpus callosum (Figs. 4 and 5), an area begins that is subject to the same vascular disease processes as the centrum semiovale. This area corresponds roughly to the outer one-quarter of the lateral ventricular roof as seen on coronal sections by modern clinical imaging techniques (Figs. 4 and 5).

Encephalic arteriole-venule pairs are thought to be rare in lower animals; when found they are in the phylogenetically oldest areas of the brain [19]. Such pairs were commonly seen in the corpus callosum in our human material (Figs. 6 and 7B). This variation is also found in the lateral geniculate body, basal ganglia, and thalamus, but not in the cortex. It is unclear what function, if any, this arrangement might serve. We hypothesize that the venule might be able to signal the arteriole to adjust its diameter and flow rate. Alternatively, there may be a "countercurrent" passive exchange of oxygen from a venule supplied by arteriole 1 to a portion of arteriole 2 (and hence its territory) distal to a damaged segment (Fig. 10).

The perivascular fibrous alae, which are thin sheets of collagen fibers and fibrocytes mixed with a few glial fibrils, extend for a variable distance lateral to the penetrating arterioles or arteriolar-venular pairs. The alae appear to be an extension of the adventitial investment of these vessels (Fig. 7). They are not found exclusively in the corpus callosum. We

A

B



C

**Fig. 7.—Collagenous perivascular alae, 100-μm alkaline phosphatase–stained sections.**

***A*, Sagittal view. Superior surface of corpus callosum can be seen at top. Several dark vertical lines occur at irregular intervals, contrasted against background of glial nuclei; these represent perivascular alae seen on edge. Occasionally, alkaline phosphatase–stained arterioles can be seen emerging from these lines. Bar = 500 μm.**

***B*, Axial view. Paired arteriole and unstained venule (*arrow*) are penetrating perpendicular to section and axons are running across. Alae invest such vessels, running laterally for variable distances; in this example, one ala is clearly seen as a dark band on the right. Bar = 100 μm.**

***C*, Microtome cut in this axial section was oriented so as to separate axon bundles, which run across illustration. With Masson trichrome technique, alae (seen here investing an arteriole) stained green, confirming their collagenous nature. This appearance may have been mistakenly identified as thrombosed capillary branches in previous reports that used routine neuropathologic techniques [15]. Bar = 50 μm.**

have also seen them in dense homogeneous axon bundles like the cingulum. Their sheetlike nature is revealed by the thick serial sections used here; although small vascular branches and tributaries often travel within them, they should not be mistaken for fibrosed vessels when seen on thin sections. The presence of fibrocytes and collagen deep within the brain may explain why meningiomas are sometimes found in an intraaxial location without dural attachment.

Cowley [5] described the preferential flow patterns of cerebral fluid masses. There is relative resistance to the transverse flow of fluid across the corpus callosum. However, clinical experience reveals that when fluid masses (edema, tumor, hemorrhage) are present focally in the corpus callosum, they tend to spread laterally rather than in an anteroposterior direction. This is due in large part to the predominant direction of the myelinated fiber tract scaffolding (notwithstanding a significant number that are obliquely oriented; see Results), but the orientation of the perivascular fibrous alae would also direct the fluid mass across the corpus callosum and retard anteroposterior migration.

Vascular changes attributed to aging, hypertension, or both will be the subject of a later article when more control brains have been examined, but preliminary evidence suggests that the centrum semiovale and basal ganglia are much more likely to exhibit these findings than are the corpus callosum or cortex.

Table 1 catalogs our semiquantitative observations of the anatomic and morphologic differences between the corpus

Fig. 8.—Intimal atherosclerotic plaque in lenticulostriate artery. H and E stain, 5-μm-thick, paraffin section. This type of vessel also supplies inferior zone of centrum semiovale. Note slits formed by cholesterol crystals. Bar = 100 μm. (Reprinted from [14], with permission.)

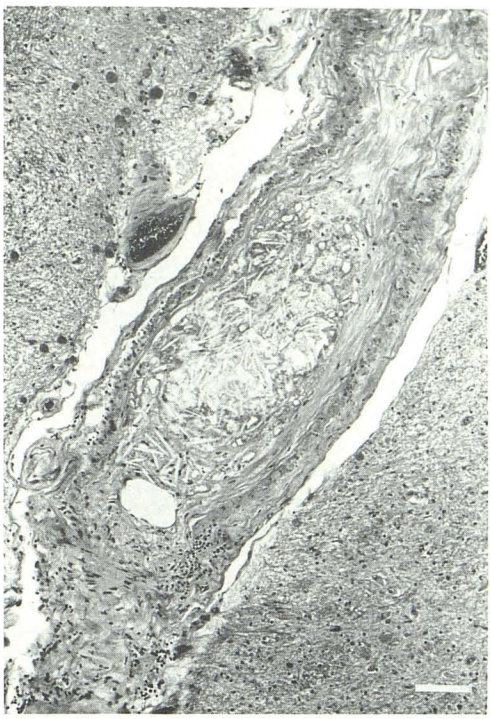


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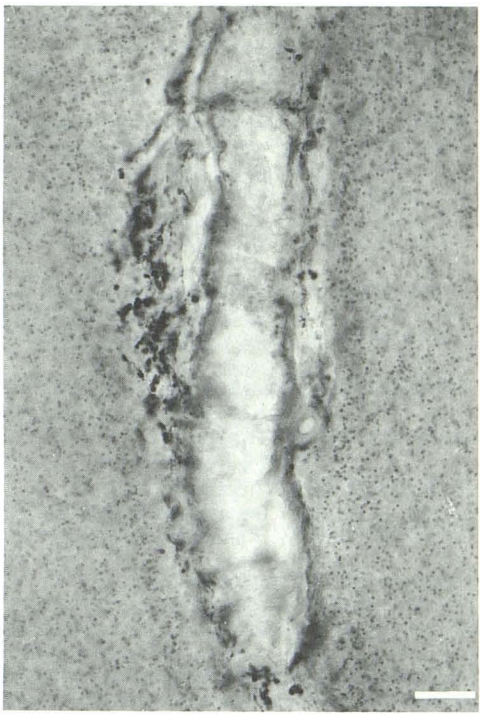
Fig. 9.—Medullary artery dissection, old, white matter of frontal gyrus, 100-μm celloidin section. Muscular coat of artery is split apart with hemosiderin-laden macrophages in separated area. This vessel passes through cortex without branches and supplies subcortical U fibers and deeper white matter. No vessels as large as this penetrate corpus callosum. Bar = 100 μm. (Reprinted from [14], with permission.)



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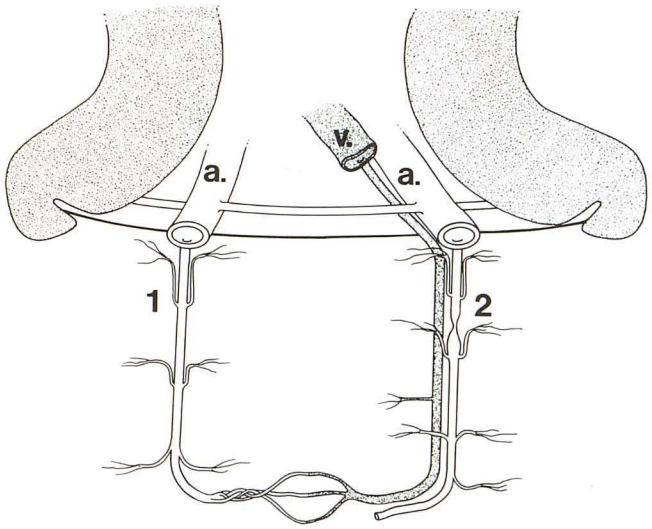



Fig. 10.—Schematic of arteriole-venule pairs observed in corpus callosum. Whether this is developmental curiosity or has functional significance is speculative and is discussed in text. Arteriole 2 is drawn with "focal pathologic narrowing." a = artery; v = vein. (Reprinted from [14], with permission.)

callosum and the centrum semiovale. Also included are obvious anatomic features not discussed in this article but tabulated for completeness; that is, proximity of CSF space and direction of arterioles relative to the direction of the majority of axons. The relevance of these differences to the modification of fluid movement and to injury from vascular disease has not been emphasized previously.

Table 1 also contrasts the relative susceptibility of the corpus callosum and centrum semiovale to categories of different disease processes. Both are heavily myelinated white-matter areas, but their vascular patterns are quite different, and so are their responses to vascular diseases.

A full discussion of Binswanger disease is beyond the scope of this article, but we were greatly influenced by the animal experiments of Abramowicz [22] and Symon et al. [23], who concluded: "With reduction of perfusion pressure, . . . it appeared that zero blood flow could be more readily induced in the white matter than in gray matter." (The "white-matter" probe of Symon et al. was recording from the centrum semiovale.)

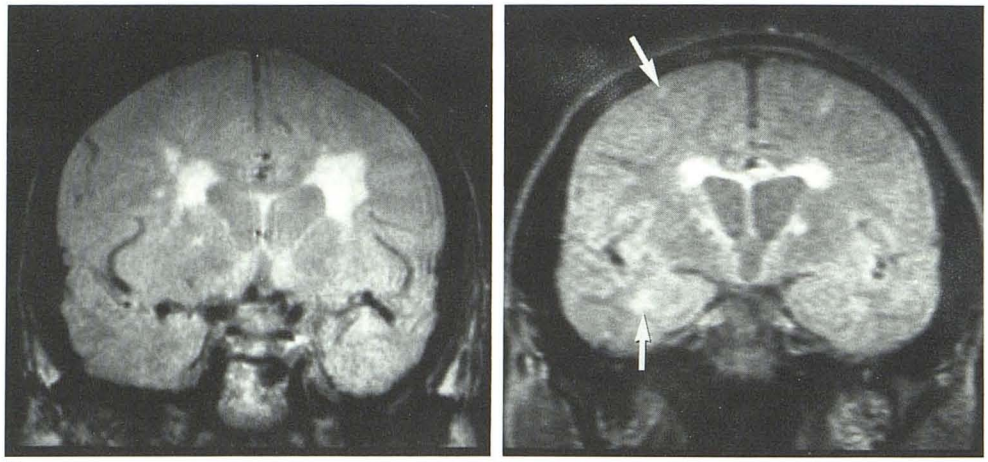
We speculate that there is an anatomic reason for this; that is, there is a greater pressure drop along a long penetrating arteriole or artery (supplying the centrum semiovale) than there is in a short arteriole (to the cortex and corpus callosum), and this pressure drop is exacerbated by tortuosity and other age-related changes. The intrinsic ability of the brain vasculature to autoregulate is then overwhelmed in conditions of hypoperfusion such as one might see in cardiac arrhythmia, in overzealous antihypertensive therapy, or distal to a high-grade carotid artery stenosis.

Accordingly, we believe that an MR image such as Figure 11B favors a diagnosis of primary demyelinating condition, whereas Figure 11A would favor the diagnosis of a vascular hypoperfusion state, because, in the latter, no abnormal signals are seen in those regions, including the corpus callosum, supplied by short arterioles.

**TABLE 1: Anatomic and Clinical Comparisons of Corpus Callosum and Centrum Semiovale**

| | Corpus Callosum | Centrum Semiovale |
|---|---|---|
| Anatomic: | | |
| Proximity of CSF, including Virchow-Robin spaces | ++++ | 0/+ |
| Blood supply | Short arterioles <8 mm long | Medullary arteries and arterioles >20 mm long |
| Arteriole-venule pairs | ++ | 0/+ |
| Direction of arterioles | Perpendicular to axons | Parallel to axons |
| Companion recurrent arterioles | +++ | 0/+ |
| Vascular aging changes | + | ++++ |
| Vascular hypertensive changes | + | ++++ |
| Periarteriolar fibrous alae | +++ | +/++ |
| Clinical: | | |
| Flow of fluid masses [5] | + | ++++ |
| De(dys)myelinating conditions [3, 4] | ++++ | ++++ |
| Vasospastic or thromboembolic stroke [2, 20] | +++ | +++ |
| Generalized small artery and arteriolar disease [6, 7, 9–11] | + | ++++ |
| Hypoxemia [8, 21] | + | + |
| Hypotension [8, 22, 23] | + | ++++ |

Note.—Scores were assigned on an arbitrary scale of 0 to ++++. The clinical scores of parenchymal susceptibility to damage from certain disease processes were based on a combination of observations on the material in our study, clinical experience in our institution, and reports in the literature.



**A** **B**

**Fig. 11.—Coronal MR images show difference between demyelinating condition and brain vascular hypoperfusion state (Binswanger disease).**

***A*, Binswanger disease (demented, elderly, hypertensive patient), coronal spin-echo image, 1800/40 (TR/TE). There are confluent areas of abnormal signal in centrum semiovale with focal abnormality in basal ganglia/internal capsule region. High signal where septum pellucidum attaches to corpus callosum is normal. Areas supplied by short arterioles (corpus callosum, cortex, subcortical U bundles) are spared. Areas supplied by long penetrating medullary arteries and arterioles are involved.**

***B*, Multiple sclerosis, coronal spin-echo image, 1800/40. There are abnormal signals in many areas of white matter, including corpus callosum and subcortical U fibers (*arrows* indicate two less obvious lesions).**

**These cases were not included in pathologic study.**

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