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Review Article

MR Imaging of the Corpus Callosum

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The corpus callosum is the major axonal commissure of the brain, connecting the two cerebral hemispheres and providing communication between the cortical and subcortical neurons. With MR imaging in the sagittal plane, the corpus callosum can be depicted in great detail. We review the normal anatomy, development, and process of myelination of the corpus callosum. The MR features of various pathologic conditions involving the corpus callosum are described. Finally, we discuss the evolving role of MR imaging in neuropsychiatric diseases with respect to the corpus callosum.

Normal Anatomy

The corpus callosum is a prominent band of compact white matter composed of transversely oriented nerve fibers by which every part of one hemisphere is connected with the corresponding part of the other hemisphere. It comprises four parts: (1) the reflected anterior portion, or the *rostrum*; (2) the *genu*, or the anterior bulbar end; (3) the *splenium*, or the posterior rounded end; and (4) the *body*, which lies between the genu and the splenium. Fibers from the inferior frontal lobes and anterior inferior parietal lobes cross in the genu, and those from the remaining part of the frontal area and from the parietal lobe cross in the body of the corpus callosum. Fibers from the temporal and occipital lobes cross in the splenium [1].

The corpus callosum has a rich blood supply of unique pattern, which is important for understanding the pathogenesis of certain lesions. The main arterial supplies are as follows [2]: The *pericallosal branch* of the anterior cerebral artery is the main vascular supply to the body; the *anterior artery* of the corpus callosum, a branch from the anterior communicating artery, supplies the rostrum and genu; and the *posterior pericallosal (splenial) artery*, a branch from the posterior cerebral artery, supplies the splenium.

Normal MR Features

The MR imaging characteristics of the corpus callosum are similar to those of white matter: high signal intensity on T1-weighted images and low signal intensity relative to gray matter on both T2- and proton density–weighted images. On midsagittal sections, the four parts of the corpus callosum are depicted well, with the cingulate gyrus and cingulate sulcus above and the lateral ventricles below. Measurements in the midsagittal plane are a subject of extensive investigation, especially in the field of neuropsychiatry. A larger main callosal area has been reported in men [3], and an 18–28% increase in the main callosal area has been reported in persons with right hemispheric cerebral speech dominance [4]. Other investigators [5], however, have found no sex or handedness differences related to callosal dimensions. Normal MR imaging measurements were reported by Okamoto et al. [6]. Focal thinning is seen superiorly at the junction of the body and splenium in 25% of persons, and is considered a normal variant [7].

On axial sections parallel to the canthomeatal line at the high ventricular level (5 cm above the external auditory meatus), the body of the corpus callosum is sectioned longitudinally, displaying a broad band of white matter bordered by the upper outer corners of the lateral ventricles. On lower cuts, the genu and splenium are sectioned transversely,

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anteriorly and posteriorly, respectively [8]. On midcoronal sections (3 cm in front of the external auditory meatus), perpendicular to Reid's base line, the body of the corpus callosum is seen above the frontal horns of the lateral ventricles. On more posterior cuts, the splenium lies at the bottom of the interhemispheric fissure, separated from the tectum of the midbrain by the contents of the quadrigeminal plate cistern, including the pineal gland and vein of Galen. At this level the lateral ventricles are diverging and lie lateral to the splenium.

Development and Myelination

Development begins anteriorly at the genu and proceeds posteriorly to the splenium, except for the rostrum, which is the last area to show crossed fibers [9]. In neonates, the corpus callosum appears flat, with no bulbous enlargement. After birth, segmental development and thickening progresses in a certain order in accordance with the development of the various cortical areas that send fibers into this commissure [10]. As in all other parts of the brain, myelination of the corpus callosum proceeds in a posterior to anterior direction. As myelination ensues, the corpus callosum, which is isointense with the rest of the brain on T1-weighted images at birth, shows an increase in signal intensity from posterior to anterior until it reaches its final MR signal characteristics at 9–12 months of age [11].

Callosal Agenesis and Dysgenesis

A wide range of developmental malformations can affect the corpus callosum, ranging in severity from total absence (agenesis) to lesser degrees of deficiency (hypoplasia) involving only the splenium. As mentioned, embryonic development proceeds in an anterior to posterior direction, so if an injury occurs in a developing brain during the formation of the corpus callosum, the anterior part is usually present but the posterior part is deficient. Callosal dysgenesis usually occurs as a result of an injury during formation of its precursors rather than from an injury to the corpus itself [12]. However, a complete but atrophic corpus callosum most likely occurs as a result of a direct insult [13], such as hydrocephalus. Jinkins et al. [14] proposed a new classification of callosal abnormalities. Isolated agenesis tends to be asymptomatic; similarly, when dysgenesis is symptomatic, the associated anomalies usually cause the signs.

MR Imaging Features

In addition to demonstrating the absence of part or all of the corpus callosum, MR imaging can show other special features (Figs. 1 and 2).

Orientation of sulci and gyri.—The sulci and gyri along the medial surface of the cerebral hemispheres radiate directly toward the top of the third ventricle [10] owing to a lack of inversion of the cingulate gyrus and a failure to form the cingulate sulcus. Normally, crossing of the callosal fibers induces inversion of the cingulate gyrus.

Deformation of the lateral ventricle.—The MR imaging features vary, depending on the severity of the anomaly. In *complete agenesis*, bundles of white matter that normally would have crossed in the corpus callosum instead turn at the interhemispheric fissure and run parallel to it to form the longitudinal callosal bundles of Probst. Probst bundles indent the medial borders of the lateral ventricles, giving them a crescentic shape on coronal sections [12].

In *partial agenesis* of the corpus callosum [10], Probst bundles are present in regions where the corpus callosum is absent. In the absence of the splenium, the occipital horns migrate superiorly into the underdeveloped white matter. The resulting dilatation of the trigone, occipital horns, and posterior temporal horns is known as colpocephaly. The frontal horns become convex laterally instead of being concave when the genu is absent. When the body of the corpus callosum is absent, the bodies of the lateral ventricles are affected more, resulting in straight parallel ventricles on axial images. The lateral ventricles may expand superiorly into the frontal and parietal white matter [13].

Third ventricular enlargement.—The third ventricle is widened and extends superiorly into the interhemispheric fissure [10], forming an interhemispheric collection of CSF (interhemispheric cyst) that ultimately may or may not communicate with the third ventricle.

A wide variety of other anomalies has been described in association with dysgenesis of the corpus callosum: a primary defect in the development of the corpus callosum can

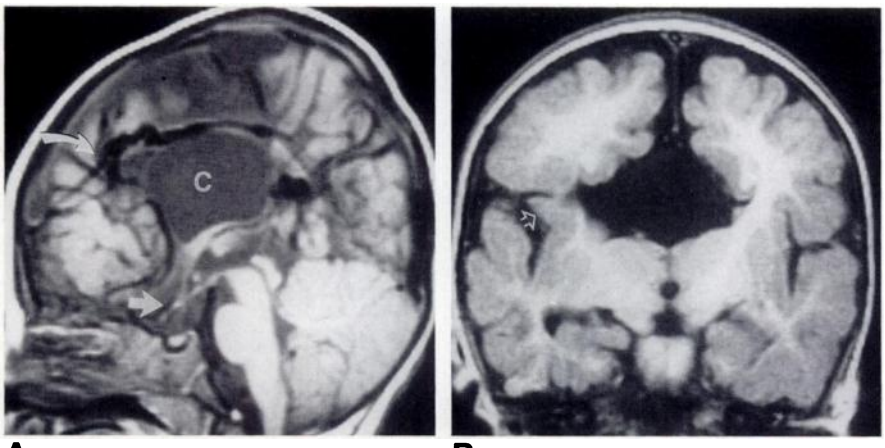

Fig. 1.—Complete agenesis of the corpus callosum in a 13-month-old boy.

***A* and *B*, Sagittal T1-weighted (600/20, *A*) and coronal inversion-recovery (1500/25/708, *B*) MR images show complete absence of both corpus callosum and septum pellucidum. Large midline interhemispheric cyst (C) compresses roof of third ventricle. Large aberrant vascular channel (*curved arrow*) most likely represents a falcine sinus. Note mild pachygyria (*B*, *open arrow*) about right sylvian fissure, small optic nerve and chiasm (*A*, *straight arrow*), and normal myelination for age on coronal view.**

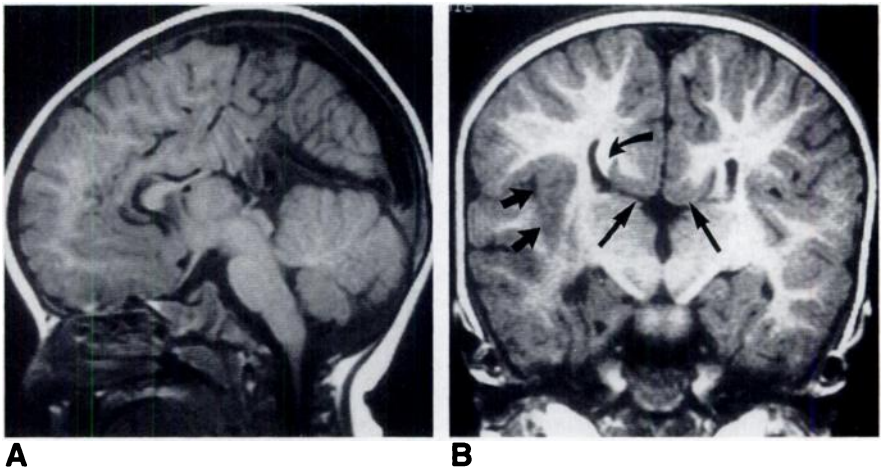

Fig. 2.—Partial agenesis of the corpus callosum in a 14-month-old boy.

***A* and *B*, On sagittal T1-weighted (600/20, *A*) and coronal inversion-recovery (1500/25/708, *B*) MR images, only the genu of the corpus callosum is formed; sulci and gyri along posterior part of medial surface of cerebral hemispheres radiate directly toward third ventricle. Coronal image (*B*) shows a crescentic right frontal horn, everted cingulate gyri (*long straight arrows*), prominent bundles of Probst (*curved arrow*), right pachygyria (*short straight arrows*), and normal myelination for age.**

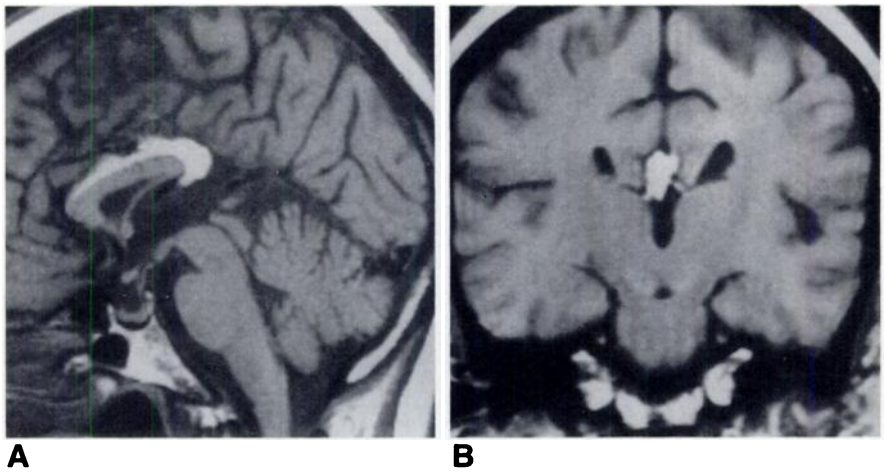

Fig. 3.—Lipoma of the corpus callosum in a 61-year-old woman with focal seizures.

***A* and *B*, Sagittal T1-weighted (600/20, *A*) and coronal T1-weighted (800/20, *B*) MR images show a high-signal lipoma between cingulate gyrus and corpus callosum extending from the genu posteriorly, and wrapping around posterior margin of the body. The splenium is absent.**

occur, or the corpus callosum can become involved after it is formed [15–25].

Lipoma of the Corpus Callosum

Lipoma of the corpus callosum is a misnomer, because this tumor is most often pericallosal in location. The dorsal pericallosal location is particularly typical when the corpus callosum is completely formed [26]; however, the lipoma almost always occurs in association with callosal anomalies [10]. Pericallosal lipomas represent nearly 30–40% of intracranial lipomas. An intracranial lipoma is believed to be a kind of congenital malformation that results from abnormal persistence and aberrant differentiation of meninx primitiva, the mesenchymal anlage of the meninges [27].

On MR imaging studies [10, 26], a lipoma may have a globular shape at the site of agenesis, with a tongue of fatty tissue extending anteriorly over the dorsal surface of the corpus callosum (Fig. 3). A hyperintense midline mass superior and posterior to the corpus callosum is seen on T1-weighted and proton density–weighted images. The corpus callosum itself is always dysgenetic [10], and the normal part extends posteriorly from the genu to the lipoma; no callosal fibers are seen dorsal to the lipoma. Branches of the pericallosal artery frequently course through the lipoma, producing small round signal voids. Calcification also may be present in the lipoma, resulting in areas of low signal intensity.

Multiple Sclerosis

Three main changes can be seen in the corpus callosum in cases of multiple sclerosis: multiple sclerosis plaques, atrophy, and signal changes within the callososeptal interface (Fig. 4). Multiple sclerosis plaques appear as focal regions of high signal intensity on both proton density– and T2-weighted images [28]. They occur most often in the body of the corpus callosum, especially along the ependymal surface of the ventricles. Involvement of the corpus callosum is characteristic of multiple sclerosis [2], whereas the white matter changes of ischemia—e.g., deep white matter ischemia, arteritis, and microinfarcts—are uncommon in this location, thus providing distinguishing features of these entities.

Diffuse atrophy of the corpus callosum is seen in cases of multiple sclerosis and can be part of a generalized cerebral atrophy in long-standing cases [29], or it can be caused by wallerian degeneration and loss of axons within the corpus callosum as a consequence of demyelinating lesions in the periventricular areas of radiating white matter fibers [30]. Atrophy of the corpus callosum is seen more in advanced cases, with more severe clinical symptoms and advanced white matter changes.

The callososeptal interface is the inferior border of the corpus callosum and septum pellucidum and is seen well on coronal and midsagittal MR images [2]. In cases of multiple sclerosis, the callososeptal interface is seen as a nonuniform rim of hyperintensity along the inferior aspect of the

corpus on both midsagittal proton density–weighted and T2-weighted images [31].

Traumatic Lesions

Callosal injury usually occurs as a part of a triad of similar lesions in the upper brainstem and deep cerebral white matter. Callosal lesions, which are usually macroscopic, serve as an easily visible marker of more widespread but often less visible diffuse axonal injuries [32]. Patients with callosal lesions have a significantly low score on the Glasgow coma scale initially. The corpus callosum is second only to the lobar gray/white matter interface in frequency of diffuse axonal injuries [33]. Most injuries occur in the splenium (Fig. 5). Acute callosal injuries are commonly ovoid, 0.5–0.7 cm in size, and mostly nonhemorrhagic. They exhibit low signal intensity on T1-weighted images and high signal intensity on T2-weighted images [32]. Chronic lesions show regions of nonspecific hypointensity on T1-weighted images and hyperintensity on T2-weighted images, surrounded by hemosiderin rings from old blood.

Neoplastic Disorders

The corpus callosum is refractory to passage of edematous fluid from remote lesions, and it is unusual to see a bilateral pattern of hemispheric edema unless the lesion invades the corpus callosum primarily [34]. However, these statements are based on CT findings, and might not be accurate for MR imaging, which is more sensitive to edematous fluid.

Glioblastoma has characteristic bihemispheric involvement with associated infiltration of the corpus callosum,

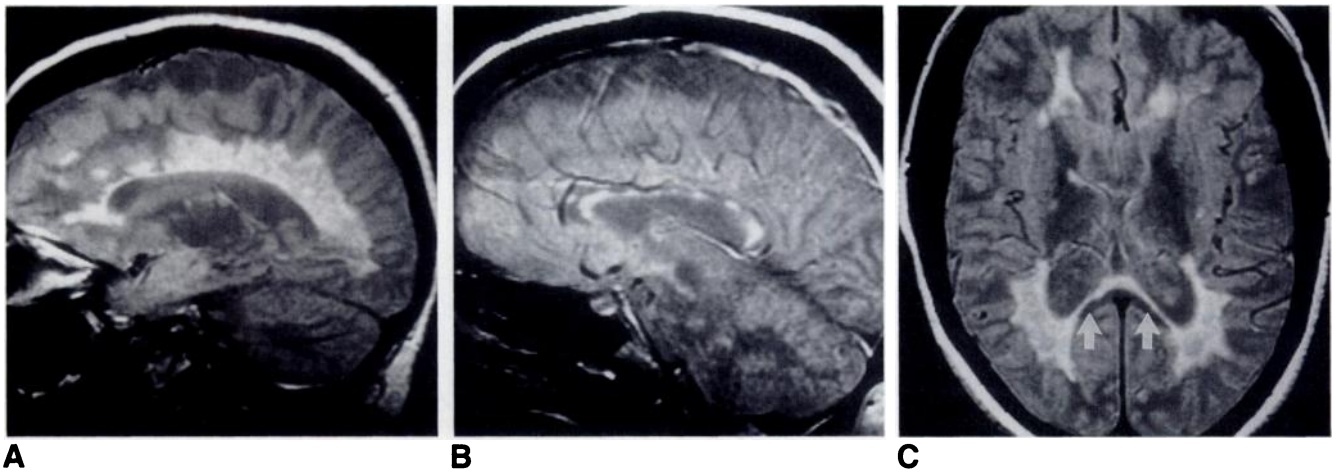

Fig. 4.—46-year-old woman with 18-year history of multiple sclerosis.
***A–C*, Sagittal (*A* and *B*) and axial (*C*) proton density–weighted (2500/30) MR images show multiple white matter plaques that have become confluent in periventricular area. Also note involvement of inner callosal fibers (*arrows*) and diffuse atrophy of corpus callosum.**

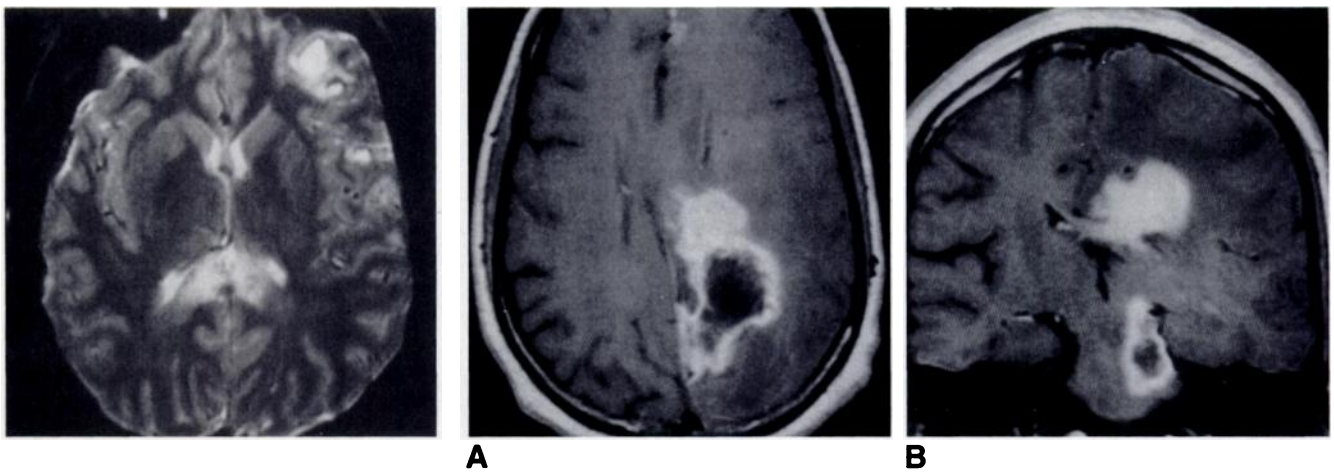

Fig. 5.—Traumatic shear injury of the corpus callosum in a 17-year-old boy. Axial T2-weighted (3000/80) MR image shows diffuse axonal injury involving the splenium of the corpus callosum. It was hyperintense on T1-weighted images, indicating a hemorrhagic shear injury. Another contusion is present in left frontal pole.

Fig. 6.—Multicentric glioma of the corpus callosum.
***A* and *B*, Contrast-enhanced axial (*A*) and coronal (*B*) T1-weighted (800/20) MR images in a 14-year-old boy show a large mass in left parietooccipital region that extends into posterior body of corpus callosum. Coronal view reveals a second lesion in left midbrain. Both lesions enhance brightly and exhibit central necrosis.**

resulting in a classic butterfly pattern (Fig. 6). Glioblastoma enhances heterogeneously after injection of a gadolinium-based contrast agent. Mass effect and edema are usually prominent. MR imaging has been reported to be more sensitive than CT for detecting communications between multicentric gliomas involving the corpus callosum [35].

Lymphomas frequently arise close to the corpus callosum and have a propensity to extend across the midline into the opposite hemisphere, a feature that may mimic glioblastoma [36]. Typically, lymphoma appears homogeneous and slightly hyperintense or isointense relative to brain on T2-weighted images. Usually, there is little mass effect or edema, features that help differentiate lymphoma from glioblastoma.

Vascular Problems

Infarcts of the corpus callosum have the same MR imaging features as infarcts elsewhere in the brain; they exhibit low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Areas of encephalomalacia and atrophy develop later [28]. Infarction of the corpus callosum is frequently associated with neuropsychiatric symptoms, mainly interhemispheric disconnection syndromes. Hemorrhage into the corpus callosum can occur in association with arteriovenous malformations (Fig. 7) and pericallosal aneurysms.

Toxic Disorders

In Marchiafava-Bignami disease, a rare complication of chronic alcoholism, the corpus callosum may show degeneration and necrosis [37]. Chronic sniffing of toluene has been reported to produce atrophy of the corpus callosum [38]. Agenesis of the corpus callosum has also been described after in utero exposure to vasoactive drugs such as cocaine and heroin [39]. In fetal alcohol syndrome, the corpus callosum may be hypoplastic or show complete agenesis (Marttson SN, Riley EP, Jernigan TL, et al., unpublished data).

The Corpus Callosum in Patients with AIDS

Nonenhancing areas of high signal have been reported in the splenium and crura of the fornix in patients with AIDS. These areas may be responsible for early memory dysfunction in patients with HIV-related cognitive impairment [40]. HIV encephalitis is characterized by brain damage and white matter atrophy and correlates clinically with the so-called AIDS dementia complex [41]. Progressive multifocal leukoencephalopathy is another white matter disease that can involve the corpus callosum. Generally, it does not exhibit mass effect or contrast enhancement [42]. Figure 8 shows a toxoplasmosis abscess in an AIDS patient.

Neuropsychiatric Diseases

The regional morphology of the corpus callosum has been the subject of extensive investigations. However, it is important to realize that the size or overt configuration of any brain structure does not necessarily determine its function [43]. Figure 9 shows a digital method for measuring the corpus callosum in neuropsychiatric disorders.

Changes in the corpus callosum in patients with schizophrenia are among the most interesting and controversial aspects of neuropsychiatric disorders. Previous reports have suggested that some schizophrenic patients may have dysfunction in the transfer of information between the two cerebral hemispheres via the corpus callosum. Thus, the presence of an abnormal corpus callosum in schizophrenic patients suggests a possible anatomic basis for abnormal cognition resulting from either an increase or a decrease in communications between the two cerebral hemispheres.

A thickened corpus callosum has been described in association with both early onset of schizophrenia and with negative symptoms of the disease, and a thinner corpus callosum has been associated with late onset of schizophrenia and with positive symptoms of the disease [43]. However, others [44] have shown that an enlarged corpus callosum is connected with positive symptoms of the disease and with a good prognosis. The corpus callosum is described as longer in schizophrenic patients, with thickening of the anterior and middle parts [45]. Also, patients with schizophrenia were reported to have a smaller corpus callosum with an enlarged ventricular system compared with control subjects [46].

Interestingly, increased thickness of the corpus callosum was reported to be more specific to right-handed female

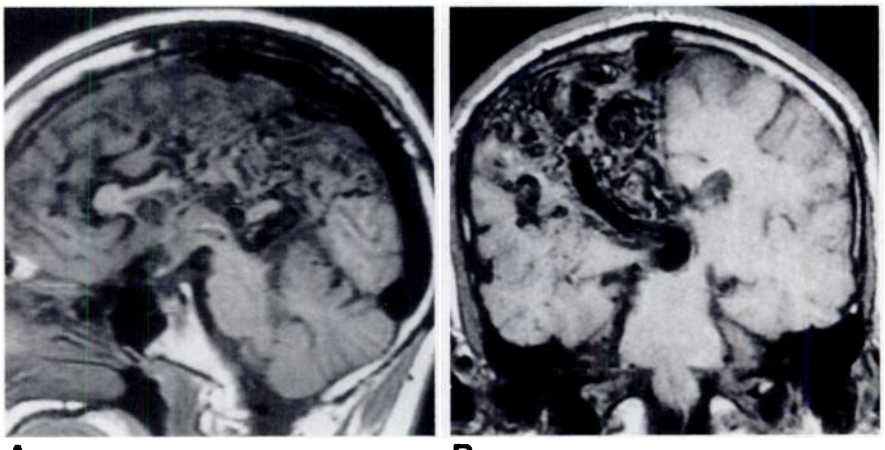

Fig. 7.—Arteriovenous malformation of the corpus callosum.

***A* and *B*, Sagittal (*A*) and coronal (*B*) T1-weighted (500/12) MR images show a large vascular mass in right frontoparietal region extending into body of corpus callosum. Multiple serpiginous areas of signal void represent the nidus, feeding arteries, and draining veins. Superior sagittal sinus is also dilated.**

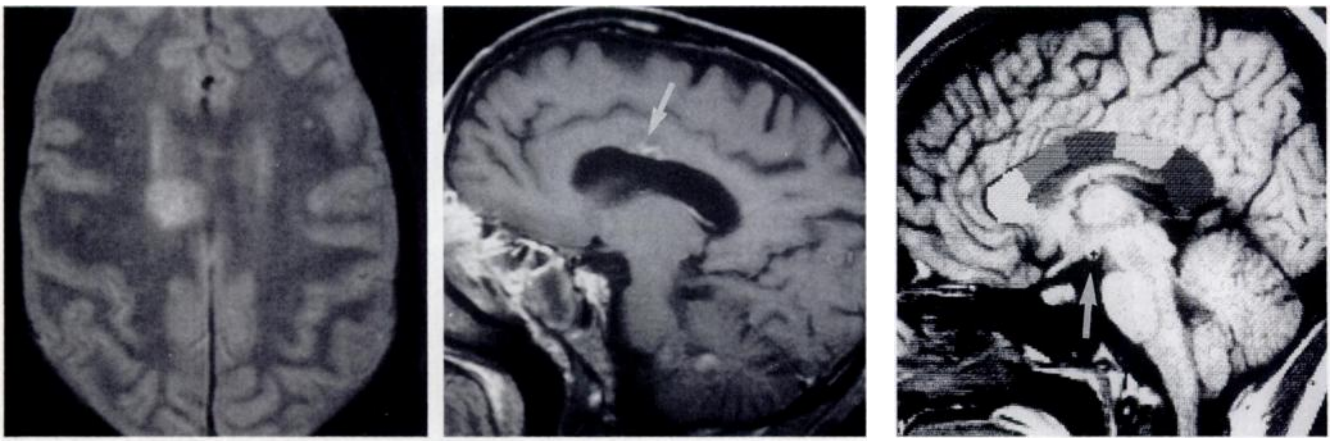

Fig. 8.—AIDS patient with cytomegalovirus encephalitis and toxoplasmosis.
***A*, Axial proton density–weighted (2500/30) MR image shows a high signal area in right side of the body of the corpus callosum.**
***B*, Enhanced sagittal T1-weighted (550/20) MR image shows partial enhancement of lesion (*arrow*). Additional similar brain lesions were seen on other sections, and the patient was treated empirically for toxoplasmosis.**

Fig. 9.—Digital method of measuring the corpus callosum in neuropsychiatric disorders. The edges of the corpus callosum are drawn on a midsagittal cut of a T1-weighted image, and the total volume is computed from the sum of all the pixels. With mammillary body as the center point (*arrow*), the angle subtended by lines drawn to the anterior and posterior extents of the corpus is divided into five equiangular sections. The size of the corpus callosum is also corrected for total cranial size.
(Method developed in the laboratory of T. L. Jernigan, University of California, San Diego).

schizophrenic patients [47], but sex differences in callosal thickness did not correlate with evidence of impaired interhemispheric transfer on neuropsychological tests [48]. Other researchers [49] found no significant differences in length or thickness of the corpus callosum in schizophrenic patients compared with control subjects. However, disease-related differences in the shape of the anterior and middle segments of the corpus callosum and a sex-related shape distribution suggest ventriculomegaly rather than an intrinsic abnormality of the corpus callosum itself.

Some authors [50] described an increased prevalence of partial agenesis of the corpus callosum in schizophrenic patients. Other reports indicate that there are no significant differences in the size of the corpus callosum between persons with and without schizophrenia [3]. To summarize, the reported morphologic data on the corpus callosum in patients with schizophrenia are inconsistent and conflicting. Interesting concepts have been raised, but the true role of the corpus callosum in schizophrenia has not been clearly defined.

A smaller genu and splenium are described in patients with attention deficit–hyperactivity disorders [51]. Also, a smaller mean callosal area has been described in bipolar disorders [52], but other investigators [3] have described no differences in the area of the corpus callosum, in the cerebral-callosal ratio, or in callosal width between patients with bipolar disorders and healthy subjects. A decrease in the area of the corpus callosum in the midsagittal plane in patients with Alzheimer's disease or multiinfarct dementia has also been reported [53].

Midcallosal thickening was described in epileptic patients with generalized seizures [54]. MR imaging is used for preoperative determination of the extent of callosotomy for epilepsy. Postoperative changes after callosotomy are seen well on MR images. T1-weighted images show the anatomic extent of the resection. T2-weighted images show areas of progressively higher signal intensity over time, representing ongoing demyelination, wallerian degeneration, and gliosis. MR imaging also shows signal changes consistent with surgically related edema and blood [55].

Conclusions

The corpus callosum is a prominent white matter structure connecting the two cerebral hemispheres of the brain. It has a very orderly development, and the timing of any in utero injury is reflected in the final morphologic alteration. It is affected by many of the white matter diseases, except those that are vascularly mediated. Structural differences in patients with various neuropsychiatric diseases raise interesting questions about specific functions of the corpus callosum. What is lacking to date are functional imaging data. Positron emission tomography has been used to elucidate the metabolism and function of cortical and deep gray matter structures in various disease states, but it has not yielded much information about the corpus callosum. Diffusion imaging can determine orientation of fiber tracts and structural integrity, but this information may not relate directly to function. Measures of neurotransmitter activity would provide such information, but imaging techniques are not yet sensitive enough to perform such tasks.

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