Trauma to the corpus callosum: MR features.

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Trauma to the Corpus Callosum:
MR Features

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The frequency, distribution, and appearance of corpus callosum injuries were evaluated with MR and CT in a prospective study of 78 patients with acute (n = 63) and chronic (n = 15) head injuries. Traumatic lesions of the corpus callosum were detected in 47% of patients. MR was significantly ($p < .001$) more sensitive than CT in the detection of callosal injuries. MR and CT visualized 100% and 27%, respectively, of the traumatic callosal lesions that were detected in the study population. The majority of lesions were located in the splenium but a few were also found in the body and genu. Patients with callosal injuries had a significantly higher incidence of primary brainstem injury ($p < .02$) as well as a greater number of subcortical gray-matter ($p < .05$) and diffuse axonal "shear" ($p < .001$) lesions. In addition, patients with callosal injuries had a significantly higher incidence of traumatic lesions of the septum pellucidum ($p < .007$) and fornix ($p < .001$). Intraventricular hemorrhage occurred significantly more often ($p < .002$) in patients with callosal injuries, especially if traumatic lesions of the fornix or septum pellucidum were also present. Patients with callosal injuries had significantly lower initial Glasgow Coma Scale scores (mean, 6.6) than those without injuries (mean, 10.7) ($p < .001$).

Injury to the corpus callosum occurs much more often with nonfatal head injuries than had been believed previously.

Traumatic lesions of the corpus callosum are commonly found at autopsy in patients who died of head injuries [1–19]. Despite being found in 16–100% of these specimens [8, 15, 18, 19], there has been little mention of corpus callosum injury in the radiographic literature. Although several authors have incidentally noted the presence of traumatic corpus callosum injury [20–29], no prior imaging study has dealt specifically with this condition. In a review of the CT scans of 286 patients with head injury, Zimmerman and Bilaniuk [23] reported that seven of eight patients with white-matter shearing injuries had hemorrhagic lesions of the corpus callosum. The frequency of callosal injuries in this and other series [23–31], however, is far less than that noted in the pathologic literature. We believe that this is primarily due to the low sensitivity of CT for detection of nonhemorrhagic lesions of the corpus callosum. MR has been shown to be considerably more sensitive than CT for detection of a wide variety of nonhemorrhagic traumatic cerebral lesions [32, 33]. Therefore, MR should be more helpful than CT for detection of callosal lesions. Although a few previous MR reports have incidentally noted callosal lesions in several instances [32–41], no prior study has dealt specifically with corpus callosum injury.

The purpose of this study was to prospectively characterize the appearance of traumatic corpus callosum injury in patients with acute (n = 63) and chronic (n = 15) head injuries. In this report we (1) describe the MR and CT appearances of corpus callosum injury; (2) evaluate its frequency, distribution, and anatomic features; (3) describe frequently associated traumatic intracerebral lesions; and (4) review the pathophysiologic mechanisms by which these lesions are produced.

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Subjects and Methods

The data from ongoing prospective MR studies of patients with acute ($n = 63$) and chronic ($n = 15$) head injuries were reviewed to assess the frequency of corpus callosum injury. From these 78 studies we identified 37 patients (47.4%) with CT or MR evidence of injury to the corpus callosum. Thirty-one patients with corpus callosum injuries were studied with MR in the acute phase of injury and six were scanned in the chronic phase. All studies were performed after informed consent was obtained and after prior approval of the study designs by our institutional review boards. A portion of the data obtained from 40 of these 78 patients was previously reported in an earlier series that investigated the overall sensitivities of CT and MR in the evaluation of closed head injury [32, 33].

In the subset of patients with acute corpus callosum injuries, MR images were obtained 1–19 days (mean, 5.4) after head trauma. Patients were included in the study if the trauma was of sufficient severity that some impairment of consciousness was present 12 hr after injury or if any residual neurologic deficit persisted beyond this time. Patients with contraindications to MR examination, those from whom informed consent could not be obtained, those with rapidly fatal head injuries, and those who remained medically or neurologically unstable for longer than 21 days were excluded from the study. Thus, patients who could not be safely studied with MR within the first 3 weeks were not entered into the study. With these inclusion and exclusion criteria, approximately 50% of all acute head injury patients who were seen in the emergency room were entered into the study.

The patients in this subset were 6–69 years old (mean, 26.5). Twenty-six were male and five were female. A detailed neurologic examination was performed in all patients within 6 hr of injury by an experienced neurosurgeon to determine the level of consciousness and to define the nature of any associated neurologic deficits. The severity of injury as measured by the admission Glasgow Coma Scale (GCS) ranged from 3 to 14 (mean, 6.6). Noncontrast CT scans were obtained in all patients within 24 hr of injury by using a late-generation CT scanner. Contiguous 8-mm sections were used in all instances. The MR images of this subset of patients were obtained with either a 0.5-T* or 1.5-T† cryogenic system with both T1- and T2-weighted pulse sequences. Contiguous, multiplanar, 5- to 10-mm-thick sections were obtained with an interleaved, multislice technique. T1-weighted images were obtained with an inversion recovery (IR), 2000–3050/500–600 (TR/TI ranges); partial saturation, 600/20–30 (TR/TE ranges); or spin-echo (SE), 300–1000/25–40; pulse sequence. T2-weighted images were obtained with an SE technique, 2000–2300/80–120.

The subset of patients with chronic corpus callosum injuries was evaluated with MR from 1 to 5 years after trauma, after informed consent was obtained. Inclusion criteria for this subgroup of patients were (1) prior trauma of sufficient severity that at least moderate posttraumatic neurologic disability (memory loss, intellectual decline, or focal neurologic deficit) was present, (2) no contraindications to MR evaluation, (3) head injury at least 1 year before the MR and CT studies, and (4) both CT and MR scans must have been requested by the referring clinician for evaluation of the patient's posttraumatic disability. Four men and two women 23–35 years old were in this subgroup of patients. CT scans were obtained in both the acute and chronic phases of injury by using a wide variety of late-generation CT scanners. Contiguous, 5- to 10-mm-thick MR images were obtained by using the 1.5-T cryogenic system† already described. T1-weighted images were obtained in the sagittal, axial, or coronal plane with a partial saturation, 600/20–30, technique. T2-weighted images were obtained in the axial or coronal plane with an SE sequence, 2000/90–120.

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† General Electric Co., Milwaukee, WI.

The CT and MR scans of all 78 patients with head injuries were reviewed independently to identify patients with corpus callosum injuries. Because surgical confirmation was not possible in any of the patients, callosal lesions had to be seen on two different studies (CT or T1- or T2-weighted MR) or imaging planes to be considered true-positive lesions. The relative sensitivities of the CT and MR scans were compared. The size, shape, location, laterality, and extent of all traumatic callosal lesions were tabulated. These data were correlated with the clinical severity of injury as measured by the GCS, associated types of intracranial injuries, location of the primary traumatic force, and mechanism of injury. The MR images of 20 normal patients were evaluated to study the normal appearance of the corpus callosum, septum pellucidum, and fornix (Figs. 1 and 2). These normal age-matched cases also served as controls for determining the incidence and significance of traumatic lesions in our study population.

We used the following criteria to classify a lesion as hemorrhagic on MR. On T1-weighted images, we required that the shortening of T1 by the paramagnetic properties of methemoglobin be of sufficient degree that the hematoma was at least partially hyperintense relative to white matter. On strongly T2-weighted images, we required the presence of central hypointensity (compared with gray matter) within the hematoma. As recently described by Gomori et al. [42], central hypointensity in acute hematomas appears to be secondary to preferential T2 proton relaxation enhancement caused by deoxyhemoglobin or methemoglobin within intact RBCs. A lesion was classified as hemorrhagic if acute blood was visualized either on CT or on T1- or T2-weighted MR images.

Results

Callosal Injury

Injury to the corpus callosum was detected in 47.4% of head trauma patients in this series. Lesions were found in 31 (49.2%) of 63 patients examined in the acute phase of injury and in six (40%) of 15 patients studied several years after head trauma. Table 1 outlines the sensitivities of CT and MR for detection of traumatic corpus callosum injuries. All 37 traumatic callosal lesions (identified by either CT or MR) were visualized with MR (Figs. 3–8), while CT detected only 10 (27%) of 37 lesions so identified ($p < .001$) (Fig. 5). In addition, MR was judged to be superior to CT for the overall visualization of 36 (97.3%) of the 37 lesions. The only lesion that was visualized more clearly with CT was an acute callosal hematoma (Fig. 5). Seven of the 10 lesions that were seen with CT were hemorrhagic in nature.

T2-weighted MR images were judged to be superior to T1-weighted ones for detecting nonhemorrhagic lesions in 17 cases (63.0%), of equal value in five (18.5%), and inferior in five (18.5%). T1-weighted IR images, 2000–3050/500–600, were judged to be superior to T1-weighted SE images, 300–1000/20–40, in most cases because they usually provided superior contrast between lesions and adjacent white matter. T1- and T2-weighted pulse sequences were considered to be of equivalent value for evaluation of the hemorrhagic subset of lesions. Hemorrhagic lesions generally were seen more clearly in the first 4 days after injury with T2-weighted images, while those studied after 4 days were seen more clearly with T1-weighted images.

The majority (94.6%) of callosal lesions involved the splenium (Table 2) (Figs. 3–6). The lesions were confined to the splenium in 48.7% of cases; they also involved the body or

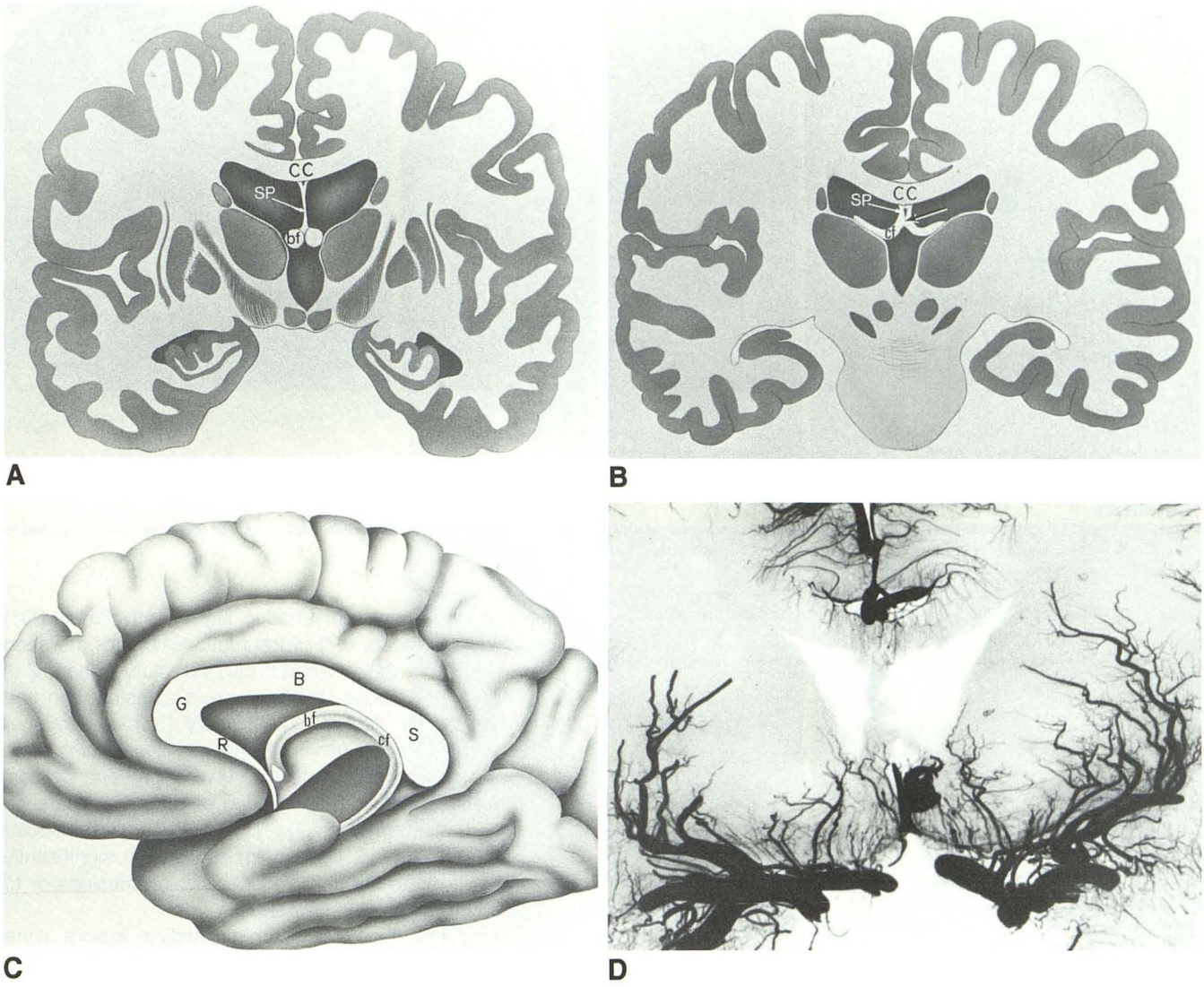

Fig. 1.—Coronal (*A* and *B*) and sagittal (*C*) diagrams reveal intimate relationships between septum pellucidum (SP), fornix, and corpus callosum (CC). Anteriorly, septum pellucidum connects corpus callosum to body of fornix (bf). Posteriorly, at level of fornix commissure (*arrow*), crus of fornix (cf) is more closely related to corpus callosum. Delicate connections of septum pellucidum and fornix to corpus callosum are easily severed by same shear strains that produce corpus callosum injury. G = genu; B = body; S = splenium; R = rostrum.

***D*, Coronal slice of injected specimen. Rich vascularity of corpus callosum, septum pellucidum, and fornix explains high frequency of intraventricular hemorrhage in patients with corpus callosum injury. (*D* courtesy of Drs. C. M. Strother and G. Salamon.)**

genu in 45.9% of cases (Figs. 4 and 5). Callosal injury was limited to the body in one case (2.7%) (Fig. 7) and to the genu in another (2.7%) (Fig. 8). Traumatic lesions were unilateral (sparing the midline) in nine (24.3%) patients, involved the midline with unilateral extension in 14 (37.8%), and involved the midline with bilateral extension in 14 (37.8%) (Figs. 3–6). We could find no significant associations between the eccentric location of the lesion (with respect to midline) and the direction of the blow, location of associated lesions, or presence of midline shift.

Acute callosal lesions usually were ovoid and were 0.5–7.0 cm in greatest dimension. Smaller lesions usually were confined to the splenium (Fig. 6), while larger lesions extended forward to involve the body and genu also (Figs. 3–5). Acute callosal lesions were most commonly (21 [67.7%] of 31 cases) nonhemorrhagic in nature. Foci of hemorrhage were seen in 10 (32.3%) of 31 cases, although in most instances the amount of blood was minimal. Usually, large nonhemorrhagic zones of injury surrounded the foci of blood. The vast majority (91.9%) of traumatic lesions involved the full thickness of the corpus callosum (Fig. 4). The three lesions that did not extend completely through the corpus callosum involved the superior aspects (Fig. 7).

The severity of injury, as manifested by the admission GCS scores, was greater in those patients with corpus callosum injury (mean score, 6.6) than in those without injury (mean score, 10.7) ($p < .001$). The severity of posttraumatic impairment of memory and overall clinical outcome were also significantly worse in the former group (J. C. Godersky et al., unpublished data). No consistent trends were detected in

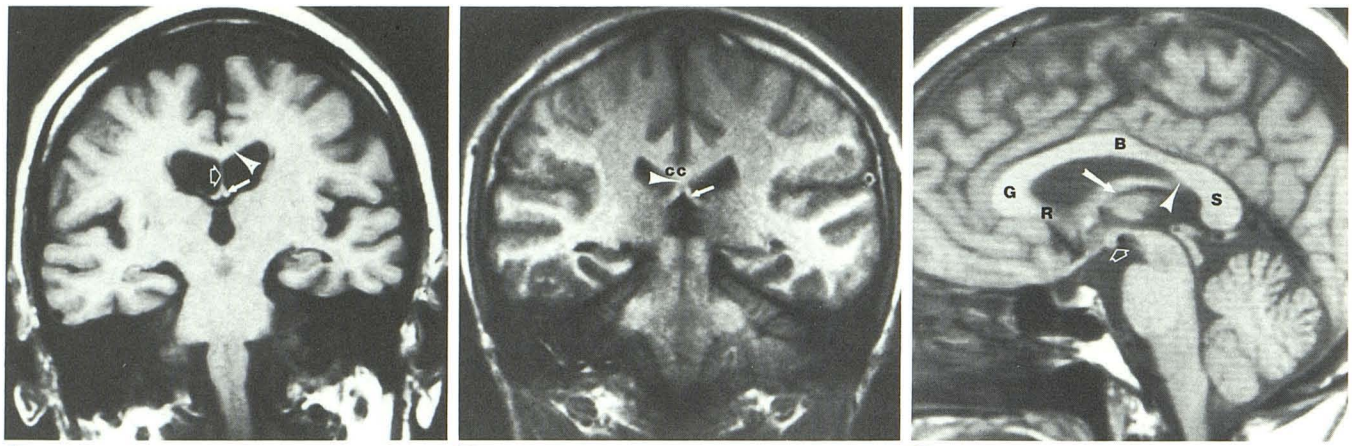

Fig. 2.—Normal coronal (*A* and *B*) and sagittal (*C*) T1-weighted MR images.
***A*, Anteriorly, septum pellucidum (*open arrow*) is very thin and long, stretching between body of fornix (*solid arrow*) and corpus callosum (*arrowhead*).**
***B*, More posteriorly, at level of commissure of fornix (*arrowhead*), septum is very short and crus of fornix (*arrow*) is more closely related to corpus callosum (cc).**
***C*, Fornix (*solid arrow*) is connected to mammillary body (*open arrow*) by way of its anterior column. Posterolaterally, crus of fornix is closely related to undersurface of corpus callosum (*arrowhead*). R = rostrum; G = genu; B = body; S = splenium.**

TABLE 1: Relative Visibility of Traumatic Lesions of the Corpus Callosum with CT and MR

Lesion Visibility	No. of Patients (%)		
	Acute CCI	Chronic CCI	Total
MR only	24 (77.4)	3 (50.0)	27 (73.0)
MR > CT	6 (19.4)	3 (50.0)	9 (24.3)
MR = CT	0	0	0
CT > MR	1 (3.2)	0	1 (2.7)
CT only	0	0	0
Total	31	6	37

Note.—The sensitivity of MR was 100%; the sensitivity of CT was 27%, $p < .001$ (chi-square with 2 df). CCI = corpus callosum injury.

patients with and without callosal injuries with regard to differences in the location and direction of the traumatic force. Blows to the orbitofrontotemporal region were most common in both groups of patients. However, a much higher percentage of patients with corpus callosum injury were victims of motorcycle and bicycle accidents. Falls were an uncommon cause of callosal injury in this series.

Chronic callosal injuries generally (five of six cases) showed no evidence of prior hemorrhage. In one instance, however, a peripheral rim of low signal intensity was present on the strongly T2-weighted images, indicating hemosiderin deposition from prior hemorrhage. All other lesions showed nonspecific hypointensity on T1-weighted images and hyperintensity on T2-weighted images. The primary feature of chronic callosal injury tended to be focal areas of callosal atrophy and encephalomalacia at the site of injury.

Associated Traumatic Lesions

Several major types of traumatic intracranial injuries were significantly associated with corpus callosum injury (Table 3). Patients with callosal injury had a significantly ($p < .001$) greater number (mean, 6.7) of traumatic lesions of the deep white matter than those without callosal injury (mean, 2.0) (Table 3) (Fig. 7). These lesions were in a distribution and location typical for diffuse axonal injury [33]. In the age-matched control group ($n = 20$), however, only two deep white-matter abnormalities (mean, 0.1) were present. Callosal injury was also significantly associated with a higher incidence of primary brainstem injury ($p < .02$) and intraventricular hemorrhage ($p < .002$). Callosal injury was not significantly related, however, to the number of cortical contusions or to the presence of extraaxial hematomas.

Several specific types of traumatic midline lesions were also found to be present with a much greater frequency in patients with corpus callosum injury (Table 4). The strongest associations were between corpus callosum injury and traumatic lesions of the septum pellucidum ($p < .007$) (Figs. 3 and 4) and fornix ($p < .001$) (Figs. 4 and 6). Lesions of the anterior commissure also were found more often in patients with corpus callosum injury, although this relationship was not statistically significant ($p < .11$). Some traumatic midline lesions (contusions of the cerebellar vermis and cingulate gyrus and interhemispheric subdural hematomas) were not found to be significantly associated with corpus callosum injury. In those patients who had corpus callosum injury, we also found a strong relationship between the presence of intraventricular hemorrhage and traumatic lesions of the septum pellucidum ($p < .003$) and fornix (Table 5) (Figs. 3 and 4). When traumatic lesions of either of these two midline structures accompanied corpus callosum injury there was an extremely high frequency of intraventricular hemorrhage.

In general, MR was superior to CT in the evaluation of traumatic lesions of the anterior commissure, fornix, and septum pellucidum (Figs. 3 and 6). When there were hemorrhagic lesions, however, CT frequently was as helpful as MR (Figs. 4 and 6). Proton-density- or strongly T1-weighted MR

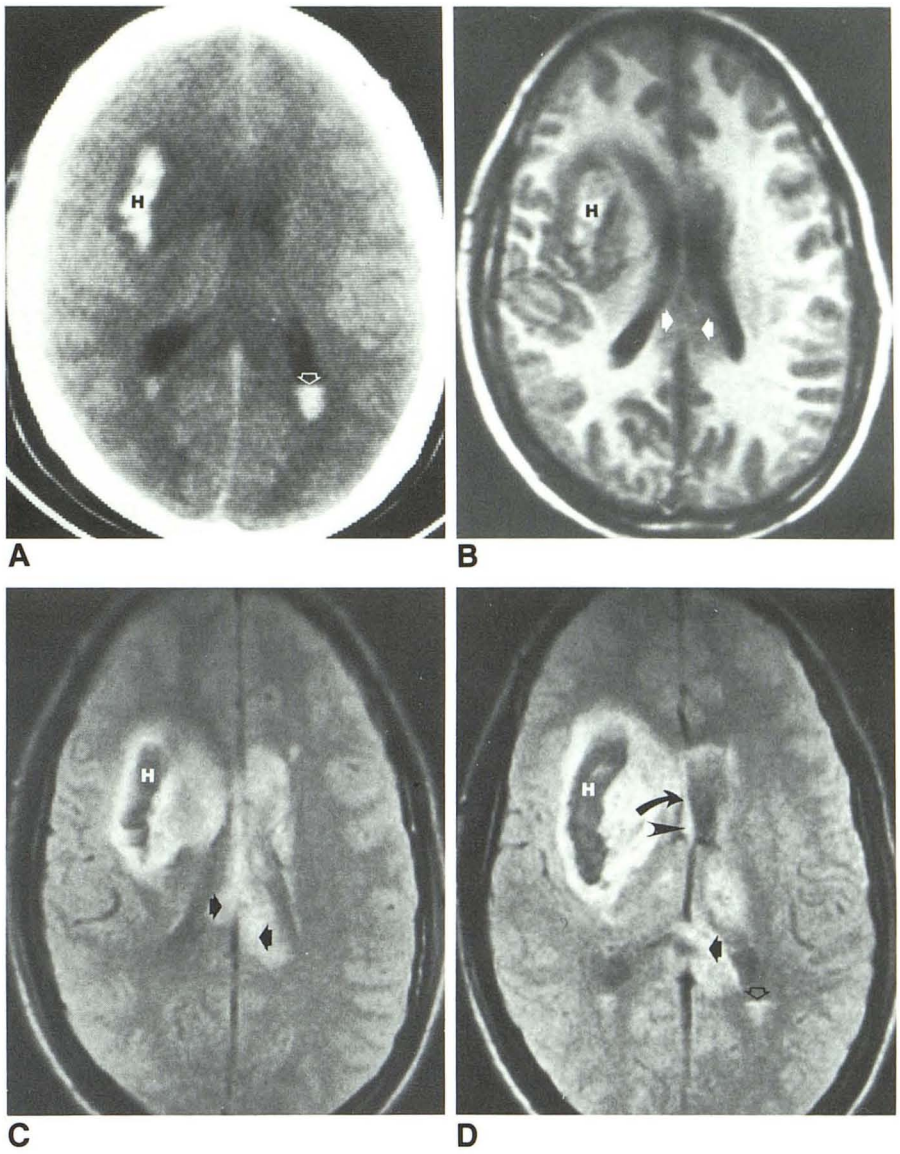

Fig. 3.—*A*, Axial CT scan in patient with severe head trauma reveals evidence of intraventricular hemorrhage (*arrow*) and large intracerebral hematoma (H) with surrounding edema. No definite abnormality of corpus callosum is seen.

***B–D*, T1-weighted IR, 2000/600 (*B*), and SE 2300/40 (*C* and *D*), images reveal extensive, primarily nonhemorrhagic, traumatic lesions of body and splenium of corpus callosum (*solid straight arrows*). Traumatic callosal lesion is also accompanied by intraventricular hemorrhage (*open arrow*). In *D*, the presence of marked edema and thickening of fornix (*arrowhead*) and septum pellucidum (*curved arrow*) indicates associated injury of these structures. Note that the 2-day-old basal ganglia hematoma (H) is slightly hypointense on spin-density and partially hyperintense on T1-weighted images. (*D* is reprinted from [32], with permission.)**

images were significantly better than strongly T2-weighted images for evaluating the fornix and septum pellucidum because the lesions often were obscured on the latter images by adjacent CSF.

Discussion

Frequency, Distribution, and Appearance of Callosal Injuries

Although traumatic corpus callosum injury has been extensively described by the neuropathologist [1–19], this topic has received little attention in the clinical and radiographic literature [20, 21, 23]. This is mainly because CT scanning is very insensitive for detecting these primarily nonhemorrhagic lesions [32, 33]. In addition, the absence of specific clinical manifestations of corpus callosum injury has provided little clinical impetus for specifically evaluating trauma patients for callosal injury. This does not mean, however, that recognition of callosal injury is of little importance [1–7, 11–13, 15].

Zimmerman and Bilaniuk [23] have shown that a normal or minimally abnormal CT scan will be present in as many as 41% of patients with significant head trauma. It is becoming increasingly clear that the neurologic findings in many of these patients are due to widespread disruption of axons, often at a microscopic level [1–7]. This type of injury has been termed "shear" injury, diffuse damage to the white matter of the immediate impact type, or diffuse axonal injury [1–15, 23]. Although it may vary in individual cases, the distribution of diffuse axonal injuries typically occurs in a predictable pattern. The most common sites of involvement (corpus callosum, brainstem, and cerebral lobar white matter) form a triad that has been described by Adams et al. [1–7]. The diffuse axonal lesions of the lobar white matter and brainstem usually are small to microscopic in size, while those of the corpus callosum typically are larger and more easily seen on CT or MR scans. The importance of observing diffuse axonal lesions of the corpus callosum lies in the fact that they serve as easily

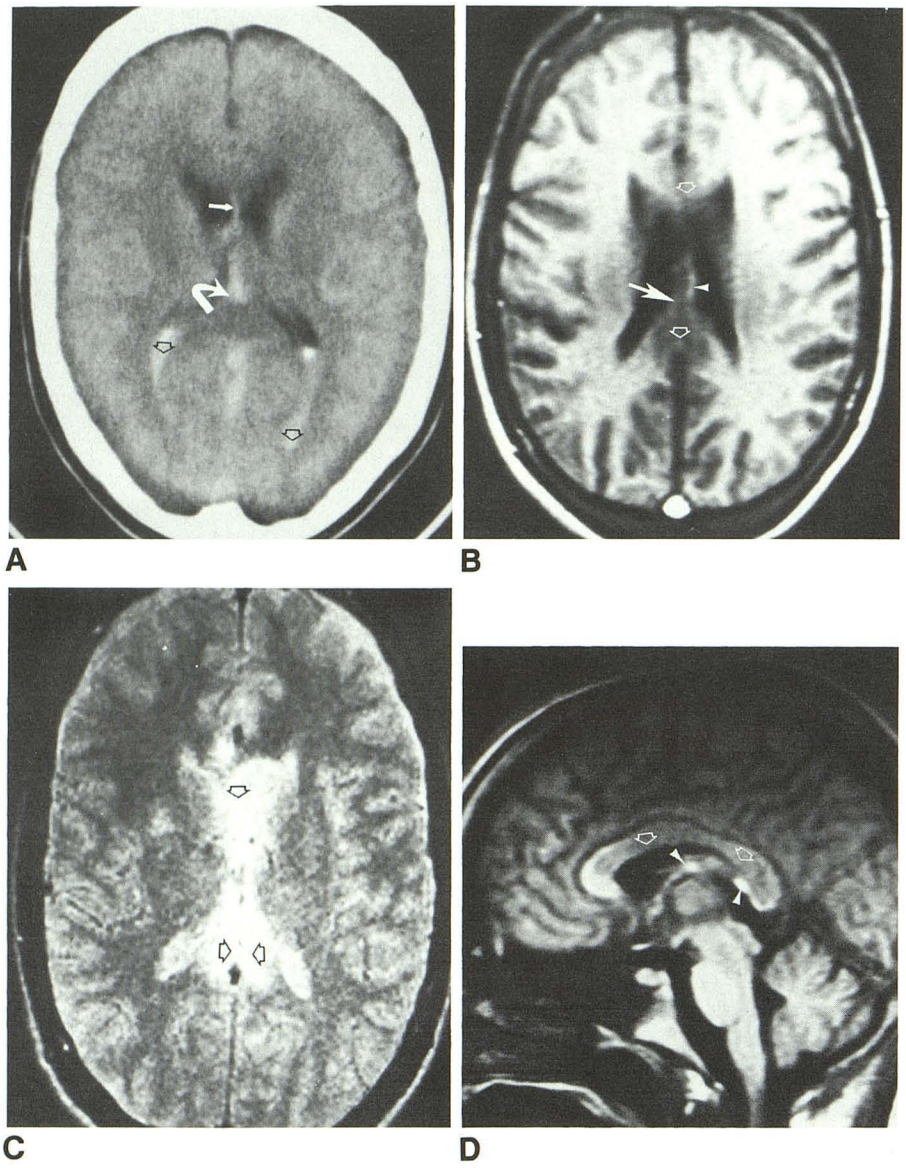

Fig. 4.—*A*, Axial CT scan reveals intraventricular hemorrhage (*open arrows*); small area of hemorrhage in septum pellucidum (*straight solid arrow*); and blood near junction of corpus callosum, fornix, and septum pellucidum (*curved arrow*). No definite lesions, however, are seen within corpus callosum.

***B–D*, Axial and sagittal T1-weighted IR, 2000/600 (*B* and *D*), and axial T2-weighted SE, 2300/80 (*C*), images, however, reveal severe callosal injury (*open arrows*) with involvement of genu, body, and splenium. In *B*, extensive edema and thickening of septum pellucidum is present near its junction with corpus callosum (*solid arrow*). Although lesion is predominantly nonhemorrhagic, small areas of hemorrhage (*arrowheads*) are present along periphery of septum pellucidum and fornix in *B* and *D*. Subacute hemorrhage is manifested by areas of signal hyperintensity (due to methemoglobin) on these T1-weighted images.**

visible "markers" of more widespread, but often less visible, diffuse axonal injury.

On the basis of evidence from pathologic studies, we know that microscopic diffuse axonal injury invariably will be present in the deep white matter if macroscopic lesions are present in the corpus callosum [1–7]. The visualization of corpus callosum injury on imaging studies indicates that a sufficient degree of the proper type of force (rotational acceleration) has been present such that widespread diffuse axonal injury has occurred [43, 44]. Careful evaluation of the brainstem, lobar white matter, and other vulnerable structures is then imperative. Conversely, in those patients with prolonged, unexplained impairment of consciousness or focal neurologic deficits, careful evaluation of the corpus callosum may reveal the presence of traumatic lesions. The presence of callosal lesions, in turn, suggests that the impairment of consciousness may be due to nonvisualized microscopic diffuse axonal injury of the brainstem and/or hemispheric white matter.

A few traumatic lesions of the corpus callosum have been previously described with CT [23–29, 32, 33]. The frequency of detection of corpus callosum injury with CT, however, has been far less than that reported in the pathologic literature [8, 15, 18, 19]. The primary reason for this discrepancy seems to be the relative insensitivity of CT in the detection of traumatic corpus callosum injury (Table 1) [32]. The frequency of callosal injuries detected by CT in our series (12.8%) is slightly higher than that reported in other CT head-injury articles [23–31]. This is probably because of interval improvements in CT scanning capabilities and because of the specific attention that we paid to the corpus callosum in this study. However, the frequency of corpus callosum injuries detected by MR (47.4%) in our series more closely corresponds to the 16–100% frequency reported in the pathologic literature [8, 15, 18, 19]. A greater sensitivity for MR in detecting callosal injury is not unexpected, since MR is known to be considerably more sensitive in the detection of many other types of nonhemorrhagic white-matter diseases.

The possibility must be briefly considered that the lesions

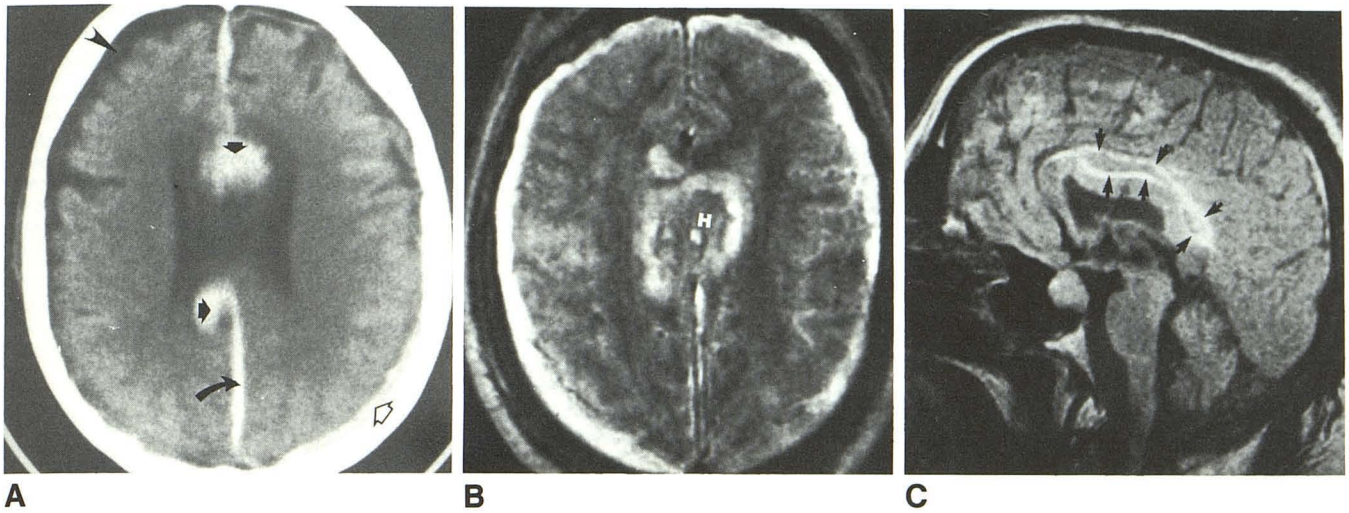

Fig. 5.—*A*, Axial CT scan reveals hemorrhagic injury of genu, body, and splenium of corpus callosum (*straight solid arrows*). Also present are acute interhemispheric (*curved arrow*) and convexity (*open arrow*) subdural hematomas as well as chronic right convexity (*arrowhead*) subdural.

***B*, Axial T2-weighted MR image, 2000/120, 5 days after injury. Marked hypointensity of acute callosal hematoma (H) probably is secondary to preferential T2 proton relaxation enhancement due to presence of intracellular methemoglobin/deoxyhemoglobin [42]. Other subdural hematomas are seen also.**

***C*, Sagittal T1-weighted image, 1000/30, more clearly reveals extent of hemorrhagic callosal lesion. Small, peripheral rim of signal hyperintensity, due to methemoglobin, is present around margin of 5-day-old hematoma (*arrows*).**

This was the only case in our series in which CT more precisely defined the extent and nature of corpus callosum injury.

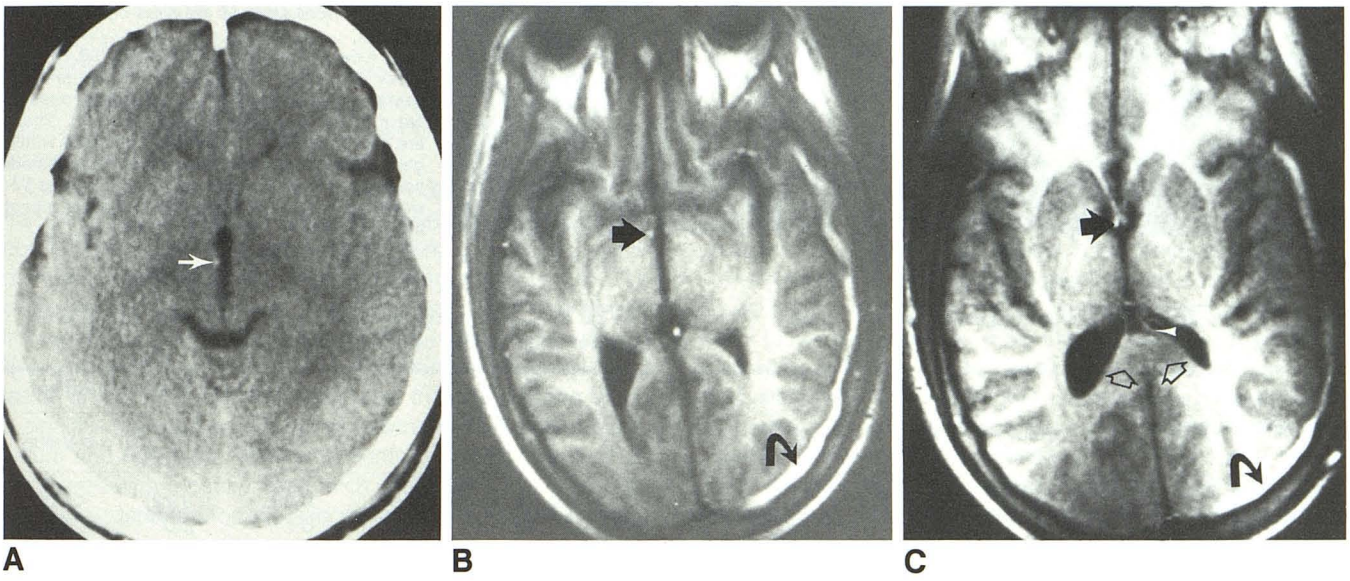

Fig. 6.—*A*, Axial CT scan appears normal except for small hemorrhagic lesion of anterior column of fornix (*arrow*).

***B* and *C*, Axial T1-weighted IR images, 2000/600, confirm hemorrhagic lesion in fornix (*straight solid arrows*). This small 9-day-old hematoma is hyperintense on T1-weighted images because of methemoglobin within lesion. An extensive, nonhemorrhagic lesion is also present bilaterally within splenium of corpus callosum (*open arrows*). Associated with callosal lesion is traumatic disruption of crus of fornix (*arrowhead*) near its junction with corpus callosum. A small subacute subdural hematoma is also noted (*curved arrows*).**

detected by MR in the corpus callosum and deep cerebral white matter of this study population were nontraumatic in nature. We addressed this question more comprehensively in our earlier publications [32, 33]. We believe that nontraumatic lesions are unlikely in view of the extremely young age of our patients (mean age, 26.5); the lack of prior history of demyelinating disease, hypertension, and vascular disease in our patients; and the lack of similar lesions in the age-matched controls.

The location of callosal injury in our series is similar, but not completely identical, to that reported in other studies. A preferential involvement of the more posterior portions of the corpus callosum has been noted by some [6, 10, 14, 18], but not all [8, 19, 21], authors. In our series, the frequency of callosal lesions progressively increased from anterior to posterior. A portion of the splenium was found to be involved in 94.6% of our cases. As has been noted in other series, the rostrum and inferior genu typically are spared [18, 19]. The

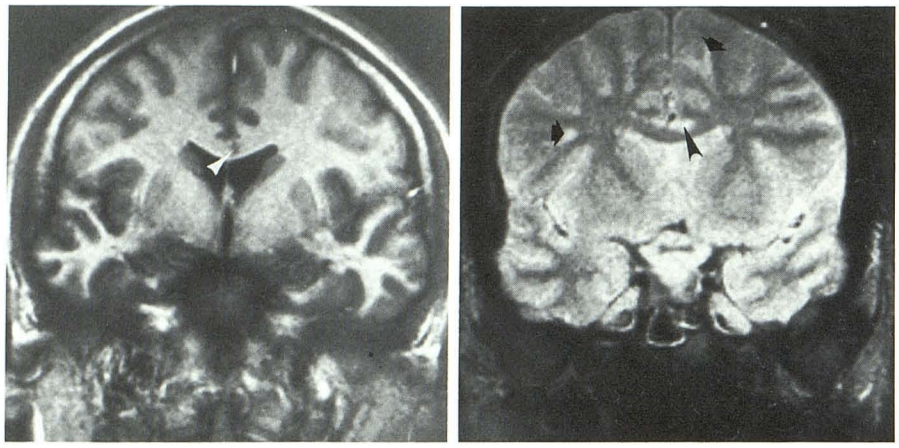

Fig. 7.—Coronal T1-weighted IR, 2000/500 (*A*), and T2-weighted SE, 2300/80 (*B*), images reveal minimal tear of superior aspect of body of corpus callosum near midline (*arrowheads*). Lesion does not extend through full thickness of corpus callosum. This nonhemorrhagic lesion (*arrowheads*) is hypointense on T1-weighted and hyperintense on T2-weighted images. Also evident are multiple other nonhemorrhagic diffuse axonal injury lesions (*arrows*) in cerebral white matter. Diffuse axonal injury lesions commonly occur in association with corpus callosum injuries.

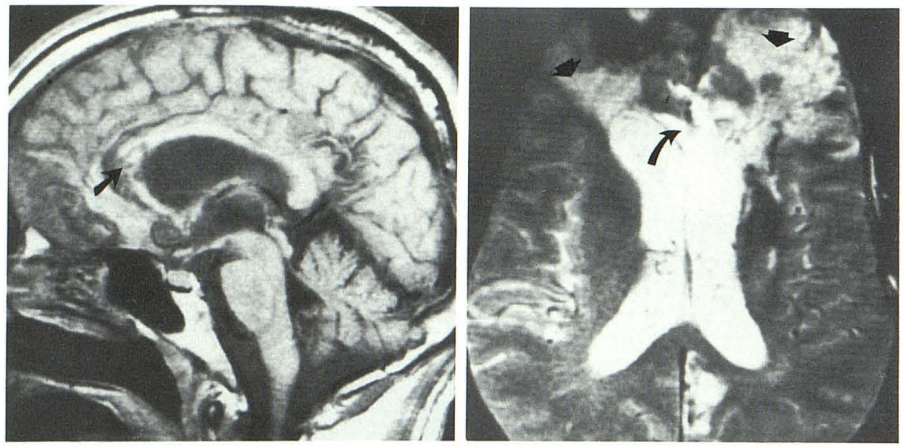

Fig. 8.—Patient with remote corpus callosum injury.

A, Sagittal T1-weighted image, 600/25, reveals posttraumatic atrophy in genu of corpus callosum. Areas of irregular signal hypointensity are present at sites of previous callosal injury (*arrow*).

B, Axial T2-weighted image, 2000/80, reveals areas of marked posttraumatic atrophy and encephalomalacia in both frontal lobes at sites of old cortical contusion (*straight arrows*). There are also extensive posttraumatic changes within genu of corpus callosum (*curved arrow*). Remote corpus callosum injury is manifested on this T2-weighted image by nonspecific areas of signal hyperintensity.

TABLE 2: Location of Traumatic Lesions of the Corpus Callosum

Lesion Location	No. of Patients (%)		
	Acute CCI	Chronic CCI	Total
Splenium	15 (48.4)	3 (50.0)	18 (48.7)
Splenium/body	11 (35.5)	2 (33.3)	13 (35.1)
Splenium/body/genu	4 (12.9)	0	4 (10.8)
Body	1 (3.2)	0	1 (2.7)
Genu	0	1 (16.7)	1 (2.7)
Rostrum	0	0	0
Total	31	6	37

Note.—CCI = corpus callosum injury.

TABLE 3: Relationship of Acute Corpus Callosum Injury (CCI) to Other Major Types of Intracranial Injury

Type of Injury	Average No. of Lesions/Patient		
	With CCI (n = 31)	Without CCI (n = 32)	p Value
Intraventricular hemorrhage	0.58	0.12	<.002[a]
Diffuse axonal injury	6.7	2.0	<.001[b]
Primary brainstem injury	0.48	0.12	<.02[a]
Subcortical gray-matter injury	0.55	0.22	<.05[a]
Cortical contusion	3.4	2.6	.40[b]
Extraaxial hematoma	1.0	0.8	.20[a]

[a] Chi-square, 2 df (based on absolute number of lesions).
[b] Student t test.

frequency of injury to the genu and body of the corpus callosum, with sparing of the splenium, was considerably less in our study (5.4%) than in others (45–67%) [18, 19]. This is possibly related to differences in the mechanisms of injury in our patients (principally automobile and motorcycle accidents) as compared with other studies (higher pecentages of falls). The lesser severity of injury in our patients may also contribute to the difference. We believe that the reason for a greater frequency of traumatic lesions in the splenium and posterior body of the corpus callosum lies in the greater vulnerability of this region to rotationally induced shear strains. The greater susceptibility of this region probably is due to specific anatomic features that will be discussed later.

The pathologic manifestations of chronic callosal injuries primarily reflect focal loss of white matter at the sites of previous injury. Focal atrophy of the corpus callosum may be caused by direct callosal injury as well as from wallerian and antegrade axonal degeneration. Most authors have found shrunken, cystic scars at the sites of corpus callosum injury with occasional areas of hemosiderin deposition from prior

TABLE 4: Relationship of Acute Corpus Callosum Injury (CCI) to Other Traumatic Midline Intracranial Lesions

Location of Lesion	No. of Patients with Lesions (%)		
	With CCI ($n = 31$)	Without CCI ($n = 32$)	p Value[a]
Fornix	14 (45.2)	1 (3.1)	<.001
Septum pellucidum	12 (38.7)	3 (9.4)	<.007
Cingulate gyrus	7 (22.6)	4 (12.5)	.24
Cerebellar vermis	5 (16.1)	4 (12.5)	.48
Interhemispheric subdural	5 (16.1)	5 (15.6)	.61
Anterior commissure	3 (9.7)	0	.11

[a] Fisher exact test.

TABLE 5: Relationship of Traumatic Lesions of the Fornix and Septum Pellucidum to Intraventricular Hemorrhage (IVH)

Presence of Lesion	No. of Patients with Lesions	
	With IVH	Without IVH
Septum pellucidum:[a]		
Yes	11	1
No	7	12
Total	18	13
Fornix:[b]		
Yes	10	4
No	8	9
Total	18	13

[a] $p < .003$, Fisher exact test.
[b] $p = .16$, Fisher exact test.

hemorrhage [1, 2, 4, 6, 8, 10]. The MR appearance of the chronic callosal lesions in our series fits well with this pathologic description.

Associated Traumatic Lesions

Adams et al. [1–7] and others [11–15] have noted that corpus callosum injury occurs as part of a triad, with similar lesions being found in the upper brainstem and deep cerebral white matter. We also noticed a statistically significant association of callosal injury with primary brainstem injury and diffuse axonal injury of the lobar white matter. The association does not merely reflect a greater severity of injury to patients with callosal injury, since no such correlations were observed for cortical contusions or extraaxial hematomas; rather, the lesions are associated because they share similar mechanisms of injury.

Many pathologic reports have also noted the presence of traumatic lesions in structures that are adjacent to the injured corpus callosum (cerebellar vermis, fornix, septum pellucidum, cingulate gyrus, and anterior commissure) [1–6, 8–21]. Injury to these structures, however, has been mentioned infrequently in the imaging literature [23–42]. This is primarily because of the poor contrast discrimination between the lesion and normal tissue on CT scans. In our study, we found a significantly greater number of lesions in the septum pellucidum and fornix (Table 4) in patients with callosal injury as compared with those without such injury. We were unable to find an explanation for this association in the literature. However, we believe that it must be secondary to shear strains that develop at the junction of the corpus callosum with the septum pellucidum and fornix. The fornix and septum pellucidum are very delicate, tenuous structures that connect the relatively mobile corpus callosum with the relatively immobile diencephalon (Figs. 1 and 2). When the massive cerebral hemispheres and attached corpus callosum are displaced, shearing stresses are produced at the junction of the corpus callosum with the septum pellucidum and fornix. These delicate structures, therefore, are readily torn during abrupt callosal displacement. Injury to the fornix is of clinical concern in that this structure comprises projection and commissural fibers of the limbic system and hippocampal formation. Therefore, the fornix is related to memory and many other complex functions of the limbic system.

The same mechanism is also likely to be the explanation for the extremely high frequency of intraventricular hemorrhage in patients with corpus callosum injury. Figure 1D shows the extensive vascular plexus that is associated with the corpus callosum, septum pellucidum, and fornix. These minute vessels are susceptible to the same shear strains that disrupt the axons.

Pathophysiologic Mechanism of Corpus Callosum Injury

The mechanism of callosal injury has been debated for a long time. It was originally thought that callosal injury was caused by traumatic laceration by the free edge of the falx [10, 22]. It is now generally accepted that this mechanism is implausible except in a few rare instances [11, 17–19]. Sudden increases in intraventricular pressure, compression waves, and vibratory forces also have been discarded as unlikely means of injury [18]. Lindenberg et al. [16–18] proposed that shear-strain forces, acting in conjunction with direct blows to the vertex of the head, superior to the level of the corpus callosum, were responsible. It is becoming increasingly clear, however, that only shear-strain deformation is necessary [7]. Although Lindenberg et al. found that the vast majority of their patients received blows to the vertex of the head, we did not find such an association in our study. The patients in our study in whom the mechanism of injury could be determined received blows from a variety of directions, both above and below the level of the corpus callosum. There is also experimental evidence that callosal injury can occur even in the absence of direct blows to the head, provided that the force is of the proper type and of sufficient magnitude. Gennarelli et al. [7] have shown, in an important series of primate experiments, that nonimpact rotational acceleration of the head in the lateral or oblique-lateral direction will uniformly produce traumatic corpus callosum injury.

Although the falx does not play a direct role in corpus callosum injury, it may play an indirect one. Shear-strain forces do not develop between tissues of the same density if they are free to move together as a unit [43, 44]. When some portions of the accelerating and rotating brain lag behind adjacent faster moving areas, shear strains develop between tissues. This can easily occur in the callosal region because the bulky and independently mobile cerebral hemispheres are

connected by the less mobile corpus callosum. With lateral or oblique lateral movements of the head [7], the rigid falx prevents the cerebral hemispheres from moving across the midline. Shear strains, therefore, develop across the connecting point (corpus callosum) of the two hemispheres. Anteriorly, less strain develops in the corpus callosum since the falx is shorter and can allow transient displacement of portions of the brain across the midline [17, 18]. Posteriorly the falx is broader and effectively prevents this displacement, allowing greater shear and tensile strains to develop within the fibers of the corpus callosum. This would seem to be a possible explanation for the selective vulnerability of the posterior half of the corpus callosum that we observed in our study.

In conclusion, traumatic injuries of the corpus callosum occur much more often than previously believed in patients with nonfatal head injuries. Callosal injury commonly occurs in conjunction with diffuse axonal injury of the brainstem and lobar white matter. Because the callosal lesions are larger and more evident on imaging studies than brainstem and white-matter lesions, callosal injury often serves as a "marker" of injury to these vital areas. Callosal injury also is frequently associated with injury to other adjacent midline structures such as the fornix, septum pellucidum, and anterior commissure. Intraventricular hemorrhage is also a common companion to corpus callosum injury.

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