Neuroradiology (1995) 37: 212–218

E. Hofmann
T. Becker
M. Jackel
D. Metzner
M. Schneider
J. Meixensberger
H. Reichmann

The corpus callosum in communicating and noncommunicating hydrocephalus

Received: 29 November 1993
Accepted: 8 February 1994

Dedicated to Prof. M. Nadjmi on the occasion of his sixty-fifth birthday

E. Hofmann (✉) · M. Jackel · D. Metzner
Department of Neuroradiology, University of Würzburg Medical School, Josef-Schneider-Strasse 11, D-97080 Würzburg, Germany

T. Becker · M. Schneider
Department of Psychiatry, University of Würzburg Medical School, Würzburg, Germany

J. Meixensberger
Department of Neurosurgery, University of Würzburg Medical School, Würzburg, Germany

H. Reichmann
Department of Neurology, University of Würzburg Medical School, Würzburg, Germany

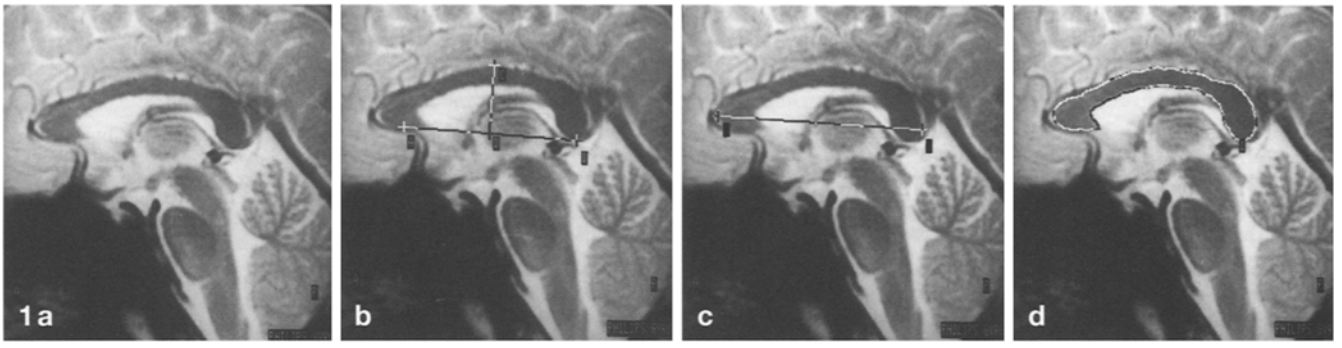
Abstract To investigate morphological changes in the corpus callosum in hydrocephalus and to correlate them with clinical findings we studied sagittal T2*-weighted cine MR images of 163 patients with hydrocephalus. The height, length and cross-sectional area of the corpus callosum were measured and related to the type of cerebrospinal fluid flow anomaly and to clinical features, especially dementia. With expansion of the lateral ventricles the corpus callosum showed mainly elevation of its body and, to a lesser degree, increase in length. Upward bowing was more pronounced in noncommunicating than in communicating hydrocephalus. Dorsal impingement on the corpus callosum by the free edge of the falx correlated with the height of the corpus callosum. Cross-sectional area did not correlate with either height, length or impingement; it was, however, the strongest anatomical discriminator between demented and nondemented patients. The area of the corpus callosum was significantly smaller in patients with white matter disease. Our findings suggest that, due to its plasticity, the corpus callosum can to some degree resist distortion in hydrocephalus. Dementia, although statistically related to atrophy of the corpus callosum, is possibly more directly related to white matter disease.

Key words Corpus callosum · Hydrocephalus · Dementia · Magnetic resonance imaging

Introduction

The corpus callosum is the major white matter tract crossing the interhemispheric fissure. It is a band of compact white matter composed of transversely oriented fibres, most of the axons crossing directly to homologous areas in the opposite hemisphere. A number of axons, however, travel obliquely to reach contralateral association areas [1, 2]. Important constituents of the limbic system run immediately adjacent to the corpus callosum, following its curve; the delicate supracallosal gyrus, containing both the grey matter of the indusium griseum and the related white matter of the lateral and medial longitudinal striae, is closely related to the superior surface of the corpus callosum. Another part of the limbic system, the cingulate gyrus, is located superior to the corpus callosum and can be traced posteriorly to the splenium. The crura of the fornix are major efferents of the limbic system and pass underneath the inferior surface of the corpus callosum [3–5].

Magnetic resonance imaging (MRI) is ideally suited to study of the normal anatomy and pathology of the corpus callosum. The midsagittal section allows simultaneous visualisation of all four parts of the corpus callosum: the rostrum, located anteriorly and inferiorly; the genu, the bulbous anterior end; the splenium, its



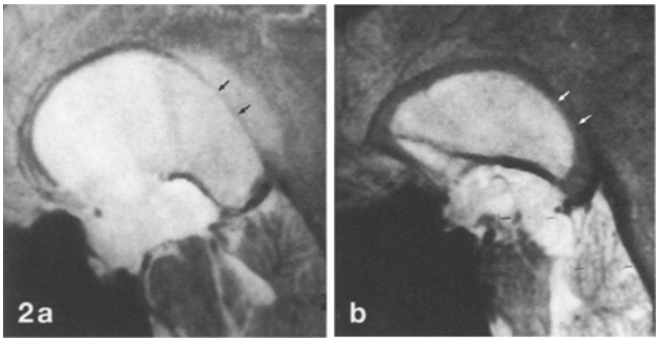


Fig. 1 **a** Normal corpus callosum on a midsagittal T2*-weighted gradient echo image. Height of the corpus callosum *(CD)* was measured above baseline *(AB)* (**b**), length was measured between genu and splenium (**c**), and area as shown in **d**

Fig. 2 Impingement by the falx cerebri *(arrows)* in patients with **a** communicating, **b** noncommunicating hydrocephalus

posterior extension, and the body or truncus between the genu and splenium [2]. Since the introduction of pneumencephalography and cerebral angiography, upward bowing of the corpus callosum and upward displacement of the pericallosal artery have been diagnostic features of marked hydrocephalus [6]. The corpus callosum is a good indicator of the size and location of parts of the lateral ventricles, since its body forms their roof and the genu curves down to form the anterior wall of the anterior horn [5]. It seems surprising that only a limited number of recent reports deal with the MRI appearances of the corpus callosum in hydrocephalus [7–10]. We performed a systematic analysis of corpus callosum pathology in communicating and noncommunicating hydrocephalus and correlated MRI and clinical findings.

Materials and methods

We reviewed the MRI studies of 163 patients (75 males, 88 females, aged 2–81 years, mean age 49 years) and compared them with a control group of 22 healthy individuals (8 men, 14 women, aged 22–92 years, mean age 45 years). All subjects were studied with a T2*-weighted, ECG-gated, multiphase fast-field-echo (FFE) sequence on a 1.5 Tesla imager. We recorded eight individual images ("heart phases") with different equidistant delays per cardiac cycle, using prospective cardiac gating. The flip angle was 10°, echo time (TE) 35 ms. Repetition time (TR) of the whole sequence varied due to variations in heart rate; a typical TR ("heart phase interval") was of the order of 80 ms. The slice was angulated and positioned in the midsagittal plane (Fig. 1 a). Reconstruction of magnitude images from the raw data allowed excellent demonstration of anatomy. Cranial cerebrospinal fluid (CSF) dynamics could be assessed by observing flow phenomena. CSF flowing in-plane showed a varying signal loss which was measured in the aqueduct of Sylvivs at the level of the intercollicular sulcus. The minimum value during the eight heart phases was used as a measure for maximum systolic craniocaudal CSF flow through the aqueduct and was used for further assessment. Pronounced pulsating aqueduct CSF flow has been associated with so-called normal-pressure hydrocephalus (NPH) [11–13]. Our patients were placed in the "hyperdynamic" aqueduct flow group if their systolic signal intensity value in the aqueduct was double standard deviations lower than the control group mean. This pronounced flow-related signal void could be the effect of increased flow velocity through the aqueduct or of dilatation of the aqueduct and consequent reduction in partial volume averaging. Irrespective of its cause, marked signal void in the aqueduct on the midsagittal image would always indicate pathologically increased pulsating flow rates.

The height of the corpus callosum above its baseline (Fig. 1 b), its length, between genu and splenium (Fig. 1 c), and its midsagittal area (Fig. 1 d) were measured manually at the operator's console using the image analysis software. Images in which the pericallosal sulcus was not clearly visible due to imperfect positioning were rejected as they carried the risk of spatial distortion and erroneous measurement.

Three of the authors (E. H., T. B., M. S.) independently rated the degree of dorsal impingement on the splenium of the corpus callosum by the free margin of the falx cerebri (Fig. 2), using a three-step scale (0 = no, 1 = questionable, 2 = definite impingement). Interobserver differences were eliminated at a consensus conference.

In patients for whom axial CT or MRI was available we calculated the volume of the lateral ventricles planimetrically. Periventricular high signal (PVH) on MRI was graded as suggested by Zimmerman et al. [14]: 0 = no PVH, 1 = discontinuous PVH, 2 = continuous PVH, 3 = periventricular halo and 4 = diffuse white

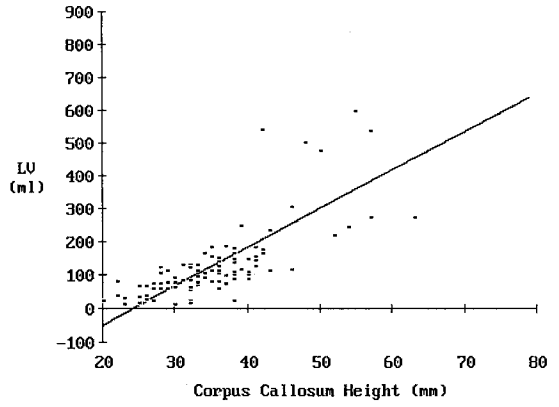


Fig. 3 Correlation between volume of left and right lateral ventricles and height of the corpus callosum (Y = 11.6611 * X-285.022, n = 103, r = 0.8097, $P < 0.0001$)

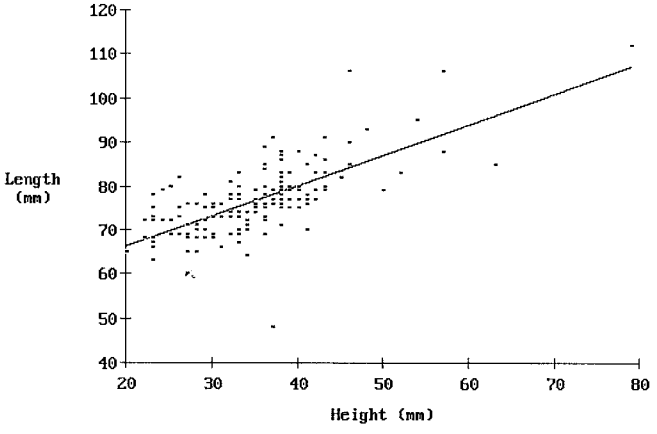


Fig. 4 Correlation between height and length of the corpus callosum (Y = 0.6951 * X + 52.32, n = 150, r = 0.7086, $P < 0.0001$)

matter high signal extending to the corticomedullary junction. PVH ratings of 0 and 1 were considered normal, ratings of 2–4 as pathological.

Clinical data were obtained retrospectively by analysing the casenotes. Dementia was assumed when there was evidence of permanent or transitory deficits in orientation or cognition. Disturbances of gait and continence were also looked for.

Results

When the combined volumes of both lateral ventricles were correlated with the height of the corpus callosum we found a fairly linear positive correlation ($r = 0.81$, Fig. 3). The height of the corpus callosum could thus be regarded as a reasonable measure of lateral ventricular volumes, which may be useful, as ventricular volumes were not available for all patients. Correlation between corpus callosum length – genu-splenium distance – and lateral ventricular volume was distinctly less good ($r = 0.64$). Figure 4 shows that in ventriculomegaly there is a stronger tendency for the body of the corpus callosum to bow upwards than for its length to increase. The corresponding length versus height graph, therefore, has a flat gradient.

Table 1 Dimensions of corpus callosum (mean ± standard deviation) in controls, hyperdynamic communicating hydrocephalus and noncommunicating hydrocephalus (aqueduct stenosis)

	Controls (22)	Communicating hydrocephalus (74)	Noncommunicating hydrocephalus (27)
Height (cm)	2.5 ± 0.4	3.6 ± 0.7	4.2 ± 1.1
Length (cm)	7.1 ± 0.5	7.7 ± 0.7	8.1 ± 1.3
Area (cm^2)	7.0 ± 1.2	6.7 ± 1.2	7.4 ± 1.1

Stretching and thinning of the corpus callosum were not reflected in terms of a negative correlation between cross-sectional area on the one hand and height, length and impingement on the other. However, there was a clear relation between height and impingement (χ^2 test, $P < 0.001$) and between length and impingement (χ^2 test, $P = 0.0029$). No significant sex difference in area was noted (6.9 ± 1.2 cm^2 in males vs 6.8 ± 1.2 cm^2 in females).

Patients with normal PVH ratings (0 and 1) were compared to patients with increased PVH (grades 2–4): mean cross-sectional area of the corpus callosum was significantly smaller in the latter group (6.3 ± 1.2 cm^2) than in the former (7.1 ± 1.2 cm^2) (Student's t-test: $t = 2.5$, $P = 0.007$; Wilcoxon test: $P = 0.017$).

All 22 healthy volunteers had rhythmic signal void in the aqueduct. Absence of flow phenomena therefore was regarded as pathological and was termed "noncommunicating hydrocephalus". In all patients without flow phenomena segmental or complete narrowing of the aqueduct was the reason. Of the 163 patients 27 had noncommunicating hydrocephalus ("aqueduct stenosis") and another 74 fulfilled the criteria of "hyperdynamic" communicating hydrocephalus with pronounced flow phenomena in the aqueduct. Table 1 gives the mean values and standard deviations of corpus callosum measurements for controls, patients with communicating "hyperdynamic" hydrocephalus and the "noncommunicating hydrocephalus" group. Communicating and noncommunicating hydrocephalus were associated with significantly higher and longer corpora callosa than in controls (Student's t-test and Wilcoxon test, $P < 0.01$). In noncommunicating hydrocephalus, the corpus callosum was higher than in the communicating hydrocephalus group (Student's t-test: $t = 2.51$, $P = 0.009$, Wilcoxon test: $P = 0.002$). Differences in corpus callosum length were less marked (Student's t-test: $t = 1.59$, $P = 0.06$; Wilcoxon test: $P = 0.02$). Corpus callosum area was slightly smaller in communicating than in noncommunicating hydrocephalus (Student's t-test: $t = 2.07$, $P = 0.02$; Wilcoxon test: $P = 0.02$), but patients in the noncommunicating group were, on average, al-

Table 2 Discriminative value of corpus callosum dimensions and impingement by falx cerebri between symptomatic and asymptomatic patients

	Gait problems	Dementia	Incontinence
Area[a]	$P < 0.01$	$P = 0.01$	$P = 0.38$
Height[a]	$P = 0.01$	$P = 0.11$	$P = 0.04$
Length[a]	$P < 0.01$	$P = 0.25$	$P = 0.06$
Impingement[b]	$P = 0.19$	$P = 0.15$	$P = 0.06$

[a] Student's *t*-test
[b] Chi-squared test

most a decade younger than patients with communicating hydrocephalus. Impingement ratings did not differ significantly between communicating and noncommunicating hydrocephalus. No healthy volunteer showed any impingement.

Among the clinical manifestations gait disturbance (93 patients, 57 %) was more frequent than dementia (82, 50 %). Incontinence, mainly urinary, occurred in a minority of patients (36, 22 %). The relative rates were similar in non-communicating hydrocephalus: 54 %, 50 % and 28 % compared with 67 %, 57 % and 24 % in patients with communicating hydrocephalus and increased aqueduct CSF flow.

Using parametric (Student's t) and non-parametric (χ^2) tests, we attempted to assess the power of the individual anatomical parameters in discriminating between patients with or without certain clinical deficits (Table 2); impingement was the weakest and cross-sectional area the strongest discriminator.

Discussion

To assess the validity of our anatomical measurements, our control results must be compared with those in the literature. The height, length and midsagittal area of the corpus callosum were studied at postmortem by Lang and Ederer [15]: in 100 individuals they found mean values of 2.2 ± 0.3 cm (height), 7.4 ± 0.4 cm (length) and 6.2 ± 0.4 cm^2 (midsagittal area). Laissy et al. [16] performed MRI measurements in 124 healthy subjects and found a mean length of 7.1 ± 0.5 cm, and mean area of 6.4 ± 1.2 cm^2. McLeod et al. [10] measured a "normal" length of 6.9 ± 0.5 cm, although they did not specify the number of individuals examined. The height and length measured in our controls (Table 1) fall well within these limits, although minor differences exist regarding cross-sectional area. This divergence may be attributed to differences in technique, materials and imaging sequences used.

Reports on anatomical distortion of the corpus callosum, seen on MRI in patients with hydrocephalus are scarce [7, 8, 10]. To our knowledge, no comparison between communicating and noncommunicating hydrocephalus has been performed. Our measurements confirm the visual impression that with increasing hydrocephalus the corpus callosum extends mainly superiorly and, to a lesser degree, in the anteroposterior direction [7]. In noncommunicating hydrocephalus (aqueduct stenosis) displacement of the corpus callosum was significantly more pronounced than in communicating hydrocephalus.

With hydrocephalus, there is rounded upward bowing of the corpus callosum, as well as uniform, smooth thinning [7, 10]. We were unable to show a negative correlation between its height or length and the midsagittal cross-sectional area. This unexpected finding can be explained by a certain degree of physical plasticity of the brain. Ventricular dilatation appears to lead to remoulding of the corpus callosum with no evident loss of substance. Stretching and thinning of the corpus callosum does not necessarily mean atrophy; atrophy, with loss of fibres and a decrease in midsagittal area has to be attributed to a different mechanism, as discussed below.

As the corpus callosum is displaced upwards with increasing ventriculomegaly, it comes close to the rigid free edge of the falx cerebri. With increasing upward bowing the falx indents the posterior part of the corpus callosum, causing a kind of furrow. This indentation was found in communicating hydrocephalus and has been termed "callosal impingement" [8]. We observed callosal impingement with equal frequency in communicating and noncommunicating hydrocephalus. Although there was a significant correlation of impingement with the height of the corpus callosum, no such relationship could be established with its cross-sectional area. Again, plasticity of the corpus callosum and relative resistance to forces induced by changes in hydrodynamics may be the explanation [2].

As atrophy of the corpus callosum did not correlate with either the degree of upward bowing or impingement ratings, a different mechanism has to be postulated for atrophy in hydrocephalus. Relating cross-sectional area to periventricular signal on MRI gives important clues: in patients with pathological PVH the area was significantly smaller.

A close relationship between periventricular white matter pathology and atrophy of the corpus callosum has been found in a variety of diseases other than hydrocephalus, such as multiple sclerosis [17–21], HIV encephalopathy [22], subcortical vascular encephalopathy [23, 24], organic solvent intoxication [25] and complicated spastic paraplegia [26]. Therefore, damage to the periventricular white matter in hydrocephalus [27] is likely to lead to axonal damage in the commissural fibres of the corpus callosum, by some similar mechanism. Axonal loss then results in atrophy, clearly distinct from hydrocephalic distortion of anatomy. It is not then surprising that mere stretching of the corpus callosum in hydrocephalus is insufficient to distinguish

among patients with different clinical pictures [28]. The effect of additional loss of substance has to be taken into account.

Impingement on the corpus callosum has been associated with the clinical syndrome of NPH [8]. Reductions in cognitive and co-ordinated motor functions were attributed to a mechanical insult to the superiorly displaced corpus callosum. Our findings do not support this hypothesis with regard to gait disturbances, dementia or incontinence; impingement alone was no significant discriminator between symptomatic and asymptomatic patients. On the other hand, gait disturbance, the most frequent symptom, correlated with both anatomical distortion and atrophy of the corpus callosum. This is poorly understood as the literature yields very little information on gait and corpus callosum pathology. Mechanisms to be considered include disconnection of the vestibular system, damage to extrapyramidal projections via the corpus callosum, and interhemispheric disconnection of somatosensory and motor integration, postural adjustments and proprioceptive centres [8, 29, 30]. In Marchiava-Bignami disease, a toxic demyelination of the corpus callosum seen most frequently in heavy drinkers of red wine, walking difficulties are prominent [31–33]. However, potential damage to additional functional systems in this disease precludes a simple link between disease of the corpus callosum and clinical deficits.

In our patients atrophy of the corpus callosum was the best anatomical discriminator between demented and nondemented patients. Although morphological changes in the corpus callosum in schizophrenics are a matter of intense debate [1], associations between atrophy of the corpus callosum and dementia have attracted comparatively little interest. Several studies have reported it as the most salient finding in demented patients. For example, in multiple sclerosis a close association has been found between organic brain syndromes, cognitive deficits and atrophy of the corpus callosum [18, 19, 21]. In so-called lacunar dementia, demented patients had significantly more white matter high-signal lesions and more extensive atrophy of the corpus callosum on MRI than nondemented or borderline patients [23]. With electron microscopy, loss of nerve fibres in the anterior corpus callosum was found in subcortical vascular encephalopathy [24] and attributed to a loss of nerve fibres in the hemispheric white matter, mainly in the frontal lobe. The width of the anterior white matter bundle and the area of the corpus callosum were also said to be decreased in presumed Alzheimer-type and multi-infarct dementia [34, 35].

Similar associations of atrophy of the corpus callosum with white matter disease and dementia have been described in HIV encephalopathy [22] and organic solvent sniffing [25]. In Marchiafava-Bignami disease the corpus callosum is selectively affected by demyelination, proceeding in some cases to frank necrosis [32, 36–38]; in patients with associated dementia disease of the white matter is also reported [31, 33, 38, 39].

In hydrocephalic children a relationship between the area of the corpus callosum, white matter loss and cognitive skills was reported [40]. However, in hydrocephalic children, delayed or altered myelin formation in the developing brain has to be considered as an additional pathophysiological mechanism for clinical deficits [41].

So far, there is no unifying theory to account for correlations between white matter disease, pathological change in the corpus callosum, cognition and memory. A relationship between the corpus callosum and cognition is conjectural [40]. Higher cortical functions in corpus callosum pathology are said to be impaired due to reduced interhemispheric communication [24]. However isolated surgical division of the corpus callosum appears unlikely to produce significant deficits of memory and cognition [42, 43]. Therefore, cognitive deficits with atrophy of the corpus callosum are more likely to reflect secondary changes due to hemisphere lesions rather than being a direct consequence of the atrophic changes [9, 18, 23]. Interstitial oedema in the periventricular white matter, as observed in hydrocephalus, has been suggested to constitute the initial lesion preceding white matter atrophy with axonal destruction, myelin disintegration and finally irreversible astrocytosis [44]. Extensive white matter disease, apart from involving the centrum semiovale and corpus callosum, also affects the subcortical area above the lateral ventricles. Tanaka et al. [3] drew attention to the fact that this zone corresponds to the white matter underlying the cingulate gyrus, part of the supracallosal limbic system [23]. Impairment of projection fibres to the cingulate gyrus seems a plausible explanation for dementia in hydrocephalus and other white matter disease [32, 45]. It has been speculated that impingement by the falx cerebri causes damage to the supracallosal component of the limbic system with subsequent deterioration of memory [8]. At a postmortem study, lesions on the surface of the corpus callosum attributable to impingement by the falx were incidental findings in about 5 % of autopsies [46]. Interestingly, the authors reported a 40 % prevalence of confusional and amnesic states in the elderly patients on whom they subsequently performed autopsies. The present study suggests that impingement alone is insufficient to produce dementia, unless additional atrophy of the corpus callosum and white matter disease are taken into account. Even after surgical division, as the most dramatic lesion of the corpus callosum, neurological deficits can often be detected only on subtle testing [42, 43]. Furthermore, the anterior part of the corpus callosum is most likely to be involved in interactions between cognitive systems [47], whereas impingement afflicts predominantly the posterior part. Amnesic syndromes

have been ascribed to disconnection of midbrain and temporal lobe structures from the frontal lobes [48]. Among the anatomical structures to be considered in this context, the fornix is prone to damage in hydrocephalus. In lesions of the corpus callosum, memory loss and amnesia syndromes have been attributed to involvement of adjacent anatomical structures such as the subjacent fornix and possibly the retrosplenial cortex [43]. In the Rhesus monkey hydrocephalus model, lesions of the corpus callosum were found adjacent to the junction between its body and the fornix [49], and in man, a similar case has been published exhibiting signal increase in the crura fornicis subjacent to the corpus callosum in a hydrocephalic individual [8]. However, we were unable to reliably assess this area due to inadequacy of midsagittal slices for demonstration of subtle forniceal abnormalities and partial volume averaging. Secondary damage to the fornix, however, may constitute a link between hydrocephalus and memory loss.

References

1. Georgy BA, Hesselink JR, Jernigan TL (1993) MR imaging of the corpus callosum. AJR 160: 949–955
2. Moody DM, Bell MA, Challa VR (1988) The corpus callosum, a unique white-matter tract: anatomic features that may explain sparing in Binswanger disease and resistance to flow of fluid masses. AJNR 9: 1051–1059
3. Naidich TP, Daniels DL, Haughton VM, Williams A, Pojunas K, Palacios E (1987) Hippocampal formation and related structures of the limbic lobe: anatomic-MR correlation. I. Surface features and coronal sections. Radiology 162: 747–754
4. Naidich TP, Daniels DL, Haughton VM, Pech P, Williams A, Pojunas K, Palacios E (1987) Hippocampal formation and related structures of the limbic lobe: anatomic-MR correlation. II. Sagittal sections. Radiology 162: 755–761
5. Nolte J (1988) The human brain. An introduction to its functional anatomy. Mosby, St. Louis, pp 50, 358–359
6. Peterson HO, Kieffer SA (1972) Introduction to neuroradiology. Harper & Row, Hagerstown, pp 121, 157–163
7. El Gammal TE, Allen MB, Brooks BS, Mark EK (1987) MR evaluation of hydrocephalus. AJNR 8: 591–597
8. Jinkins JR (1991) Clinical manifestation of hydrocephalus caused by impingement of the corpus callosum on the falx: an MR study in 40 patients. AJNR 12: 331–340
9. Jinkins JR (1991) The MR equivalents of cerebral hemispheric disconnection: a telencephalic commissuropathy. Comput Med Imaging Graph 15: 323–331
10. McLeod NA, Williams JP, Machen B, Lum GB (1987) Normal and abnormal morphology of the corpus callosum. Neurology 37: 1240–1242
11. Bradley WG (1992) Magnetic resonance imaging in the evaluation of cerebrospinal fluid flow abnormalities. Magn Reson Q 8: 169–196
12. Bradley WG, Whittemore AR, Kortman KE, Watanabe AS, Homyak M, Teresi LM, Davis SJ (1991) Cerebrospinal fluid void: indicator of successful shunt in patients with suspected normal-pressure hydrocephalus. Radiology 178: 459–466
13. Kunz U, Heintz P, Ehrenheim C, Stolke D, Dietz H, Hundeshagen H (1989) MRI as the primary diagnostic instrument in normal pressure hydrocephalus. Psychiatry Res 29: 287–288
14. Zimmerman RD, Fleming CA, Lee BCP, Saint-Louis LA, Deck MDF (1986) Periventricular hyperintensity as seen by magnetic resonance: prevalence and significance. AJNR 7: 13–20
15. Lang J, Ederer M (1980) Über Form und Größe des Corpus callosum und das Septum pellucidum. Gegenbaurs Morphol Jahrb 126: 949–958
16. Laissy JP, Patrux B, Duchateau C, Hannequin D, Hugonet P, Ait-Yahia H, Thiebot J (1993) Midsagittal MR measurements of the corpus callosum in healthy subjects and diseased patients: a prospective survey. AJNR 14: 145–154
17. Dieteman JL, Beigelman C, Rumbach L, Vogue M, Tajahmady T, Faubert C, Jeung MY, Wackenheim A (1988) Multiple sclerosis and corpus callosum atrophy: relationship of MRI findings to clinical data. Neuroradiology 30: 478–480
18. Huber SJ, Bornstein RA, Rammohan KW, Christy JA, Chakeres DW, McGee RB (1992) Magnetic resonance imaging correlates of neuropsychological impairment in multiple sclerosis. J Neuropsychiatr 4: 152–158
19. Huber SJ, Paulson GW, Shuttleworth EC, Chakeres D, Clapp LE, Pakalnis A, Weiss K, Rammohan K (1987) Magnetic resonance imaging correlates of dementia in multiple sclerosis. Arch Neurol 44: 732–736
20. Pelletier J, Habib M, Lyon-Caen O, Salomon G, Poncet M, Khalil R (1993) Functional and magnetic resonance imaging correlates of callosal involvement in multiple sclerosis. Arch Neurol 50: 1077–1082
21. Weihe W, Loew M, Schulze-Siedschlag J, Horstmann A, Welter FL, Mariß G (1989) Multiple Sklerose: Balkenatrophie und Psychosyndrom. Nervenarzt 60: 414–419
22. Schlote W (1991) HIV-Enzephalopathie. Verh Dtsch Ges Pathol 75: 51–60
23. Tanaka Y, Tanaka O, Mizuno Y, Mitsuo Y (1989) A radiologic study of dynamic process in lacunar dementia. Stroke 20: 1488–1493
24. Yamanouchi H, Sugiura S, Shimada H (1990) Loss of nerve fibres in the corpus callosum of progressive subcortical vascular encephalopathy. J Neurol 237: 39–41
25. Ikeda M, Tsukagoshi H (1990) Encephalopathy due to toluene sniffing. Report of a case with magnetic resonance imaging. Eur Neurol 30: 347–349
26. Iwabuchi K, Yagishita S, Amano M, Yokoi S, Honda H, Tanabe T, Kinoshita J, Kosaka K (1990) An autopsy case of complicated form of spastic paraplegia with amyotrophy, mental deficiency, sensory impairment, and parkinsonism. No To Shinkei 42: 1075–1083
27. George AE (1991) Chronic communicating hydrocephalus and periventricular white matter disease: a debate with regard to cause and effect. AJNR 12: 42–44
28. Jack CR, Mokri B, Laws ER, Houser OW, Baker HL, Petersen RC (1987) MR findings in normal-pressure hydrocephalus: significance and comparison with other forms of dementia. J Comput Assist Tomogr 11: 923–931
29. Benes V (1982) Sequelae of transcallosal surgery. Child's Brain 9: 69–72

30. Viallet F, Massion J, Massarino R, Khalil R (1992) Coordination between posture and movement in a bimanual lifting task: putative role of a medial frontal region including the supplementary motor area. Exp Brain Res 88: 674–684
31. Baron R, Heuser K, Marioth G (1989) Marchiafava-Bignami disease with recovery diagnosed by CT and MRI: demyelination affects several CNS structures. J Neurol 236: 364–366
32. Bracard S, Claude D, Vespignani H, Almeras M, Carsin M, Lambert H, Picard L (1986) Scanographie et IRM de la maladie de Marchiafava-Bignami. J Neuroradiol 13: 87–94
33. Chang KH, Cha SH, Han MH, Park SH, Nah DL, Hong JH (1992) Marchiafava-Bignami disease: serial changes in corpus callosum on MRI. Neuroradiology 34: 480–482
34. Yoshii F, Shinohara Y, Duara R (1990) Cerebral white matter bundle alterations in patients with dementia of Alzheimer type and patients with multi-infarct dementia – magnetic resonance imaging study. Rinso Shink 30: 110–112
35. Yamanouchi H, Sugiura S, Shimada H (1989) Decrease of nerve fibres in the anterior corpus callosum of senile dementia of Alzheimer type (letter). J Neurol 236: 491–492
36. Delangre T, Hannequin D, Clavier E, Denis P, Mihout B, Samson M (1986) Maladie de Marchiafava-Bignami d'evolution favorable. Rev Neurol 142: 933–936
37. Namba Y, Bando M, Takeda K, Iwata M, Mannen T (1991) Marchiafava-Bignami disease with symptoms of the motor impersistence and unilateral hemispatial neglect. Rinso Shink 31: 632–635
38. Rosa A, Demiati M, Cartz L, Mizon JP (1991) Marchiafava-Bignami disease, syndrome of interhemispheric disconnection and right-handed agraphia in a left-hander. Arch Neurol 48: 986–988
39. Canaple S, Rosa A, Mizon JP (1992) Maladie de Marchiafava-Bignami: disconnexion interhémispherique, evolution favorable. Aspect neuroradiologique. Rev Neurol 148: 638–640
40. Fletcher JM, Bohan TP, Brandt ME, Brookshire BL, Beaver SR, Francis DJ, Davidson KC, Thompson NM, Miner ME (1992) Cerebral white matter and cognition in hydrocephalic children. Arch Neurol 49: 818–824
41. Gadsdon DR, Variend S, Emery JL (1978) The effect of hydrocephalus upon the myelination of the corpus callosum. Z Kinderchir 25: 311–318
42. Castro-Caldas A, Poppe P, Antunes JL, Campos J (1989) Partial section of the corpus callosum: focal signs and their recovery. Neurosurgery 25: 442–447
43. Rudge P, Warrington EK (1991) Selective impairment of memory and visual perception in splenial tumours. Brain 114: 349–360
44. Rubin RC, Hochwaldt GM, Tiell M, Mizutani H, Ghatak N (1976) Hydrocephalus. I. Histological and ultrastructural changes in the pre-shunted cortical mantle. Surg Neurol 5: 109–114
45. Kameyama M (1973) Vascular lesions in the frontal association field and dementia. Clin Psychiatr 15: 357–366
46. Roessmann U, Friede RL (1968) Surface lesions of corpus callosum. Acta Neuropathol 10: 151–158
47. Sidtis JJ, Volpe BT, Holtzman JD, Wilson DH, Gazzaniga MS (1981) Cognitive interaction after staged callosal section. Evidence for transfer of semantic activation. Science 212: 344–346
48. Warrington EK, Weiskrantz L (1982) Amnesia: a disconnection syndrome? Neuropsychologia 20: 233–248
49. Clark RC, Milhorat TH (1976) Experimental hydrocephalus, part 3. Light microscopic findings in acute and subacute obstructive hydrocephalus in the monkey. J Neurosurg 32: 400–413