Periventricular Hyperintensity as Seen by Magnetic Resonance: Prevalence and Significance

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Periventricular Hyperintensity as Seen by Magnetic Resonance:
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Periventricular hyperintensity was identified using long repetition and echo times in spin-echo magnetic resonance imaging of patients with white-matter disease (e.g., multiple sclerosis) caused by local demyelination and in hydrocephalic patients caused by transependymal migration of spinal fluid. A review of 365 consecutives studies revealed that some degree of periventricular hyperintensity is present in most patients (93.5%) regardless of diagnosis. Mild periventricular hyperintensity was seen in patients with no other evidence of intracranial pathology. Periventricular hyperintensity is a normal finding that should not be considered indicative of either demyelinating disease or hydrocephalus. More extensive and severe periventricular hyperintensity is associated with intracerebral pathology, but the finding often is nonspecific. For example, mild periventricular edema from hydrocephalus is impossible to differentiate from the increased periventricular hyperintensity seen in patients with multiple white-matter lesions. Thus, the pattern of periventricular hyperintensity has proven to be of limited value in the clinical assessment of hydrocephalic patients.

One of the earliest and most impressive clinical applications of magnetic resonance imaging (MRI) has been in the evaluation of patients with multiple sclerosis [1–8] and other white-matter diseases [5] including subcortical atherosclerotic encephalopathy [5, 9], radiation necrosis [7], and systemic lupus erythematosus [10]. In these diseases focal and/or diffuse areas of supratentorial white-matter hyperintensity are commonly imaged adjacent to the ventricular lining. In patients with obstructive hydrocephalus MRI scans have demonstrated a smooth, hyperintense halo surrounding the ventricles believed to be indicative of edema from transependymal resorption of cerebrospinal fluid [2, 6, 7, 11]. Because focal and/or diffuse periventricular hyperintensity (PVH) seemed to be present in many patients with a variety of pathologic processes, and even in patients with otherwise normal examinations, a retrospective evaluation of the incidence and possible significance of PVH was undertaken.

Materials and Methods

We retrospectively evaluated MRI studies performed between October 1983 and April 1984 on a 0.5 T superconducting MR imager manufactured by the Technicare Corp. To be included in the study a transversely oriented spin-echo (SE) image with a repetition time (TR) of 1500 msec and echo time (TE) of 90 msec ("T2-weighted") of adequate quality had to be available for review. With this technique intraventricular spinal fluid was approximately isointense to white matter. All scans were obtained with a single-echo multislice technique. Clinical data were correlated with MR findings. A total of 365 cases were evaluated. The patients were 6–84 years of age (mean age, 42). Of the 365 cases, 17 were ≤ 10 years old, 27 were 11–20, 61 were 21–30, 72 were 31–40, 60 were 41–50, 65 were 51–60, 38 were 61–70, and 25 were ≥ 71. The gender distribution was roughly equal.

Five PVH patterns of increasing extent and intensity were delineated: (0) no PVH (fig. 1); (1) discontinuous PVH—rounded hyperintense foci seen at the angles of the frontal horns

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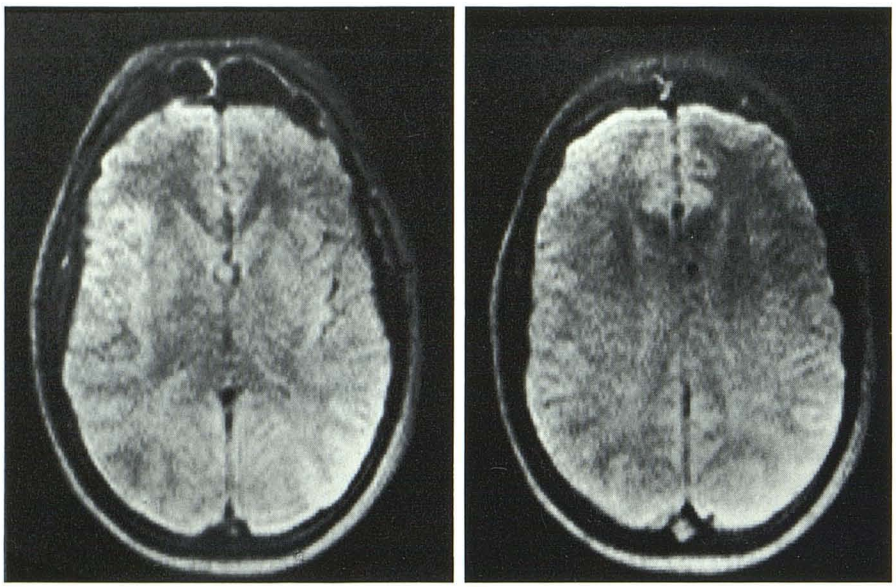


Fig. 1.—Pattern 0 in 17-year-old boy with history of seizures and negative CT scan. Scans to level of foramen of Monro (**A**) and bodies of lateral ventricles (**B**) show no PVH.

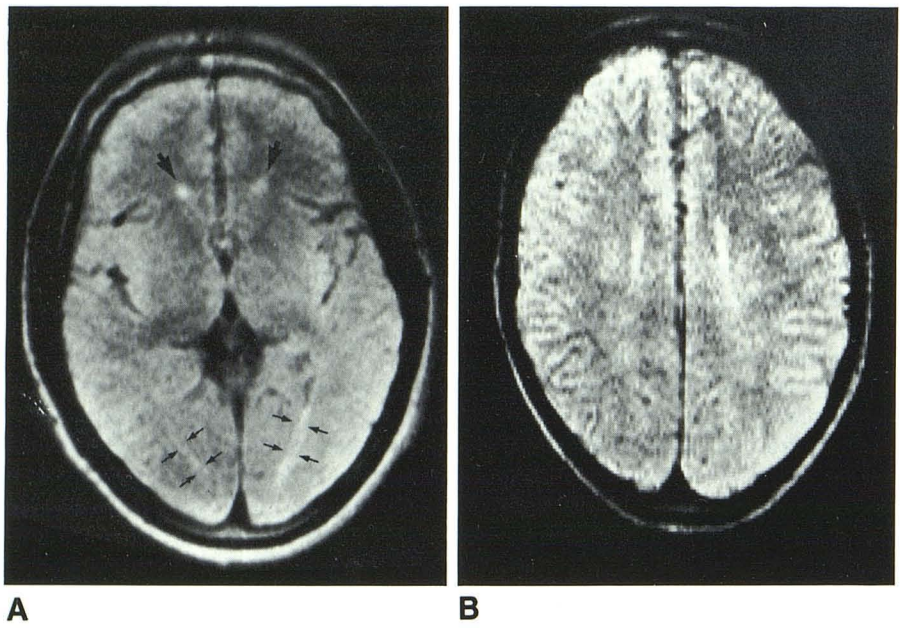
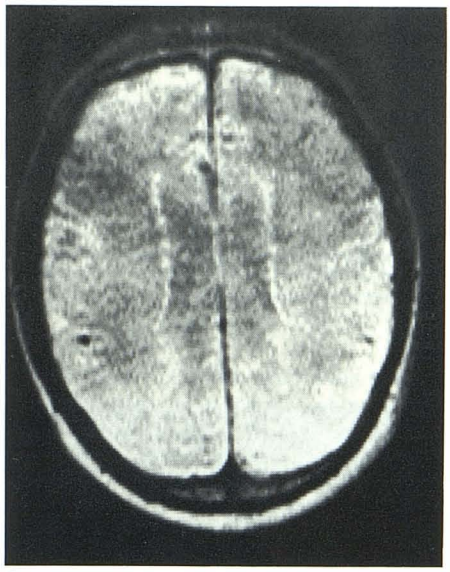


Fig. 2.—Pattern 1 in 32-year-old woman studied to rule out demyelinating disease. **A**, Level of foramen of Monro. Bilateral, symmetric, rounded foci of hyperintensity just anterior to frontal horns (*large arrows*). Thin caps of hyperintensity surround small occipital horns (*small arrows*). **B**, At level of body of lateral ventricles. Streaks of hyperintensity just lateral to atria.

bilaterally (fig. 2A), caps of hyperintensity surrounding the occipital horns medially and laterally (fig. 2A), and streaks of hyperintensity extending along the atria of the lateral ventricles (fig. 2B); (2) continuous PVH—a pencil-thin continuous line of hyperintensity surrounding the ventricles (fig. 3); (3) periventricular halo—a band of hyperintensity of variable thickness with smooth lateral margins surrounding the ventricles (fig. 4) (this pattern has been described as indicative of periventricular edema from hydrocephalus [2, 6, 7, 11]; and (4) diffuse white-matter abnormality—hyperintensity extending from the ventricular lining to the corticomedullary junction involving all or most of the white matter, the lateral margins of the hyperintensity being irregular (fig. 5). This pattern has been described in patients with diffuse demyelinating disease [1, 5, 8]. The studies were also evaluated for the presence and number of focal areas of white-matter hyperintensity independent of the PVH.

The MRI patterns were tabulated (table 1) and correlated with clinical data including CT scans when available. The studies were divided into those cases demonstrating no other evidence of abnormality on MRI ("otherwise normal group"—table 2, fig. 6A). And those cases in which pathology was present ("pathology group"— table 3, fig. 6B). The otherwise normal group was divided into patients under and over 30 years of age, and the pathologic cases were grouped into four broad diagnostic categories: (1) trauma, (2) supratentorial tumors, (3) white-matter diseases, and (4) hydrocephalus.



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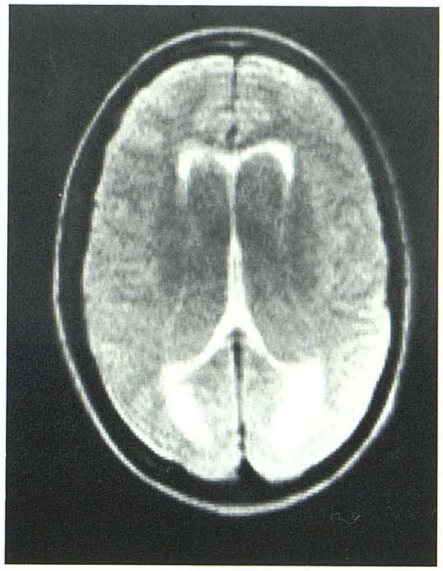

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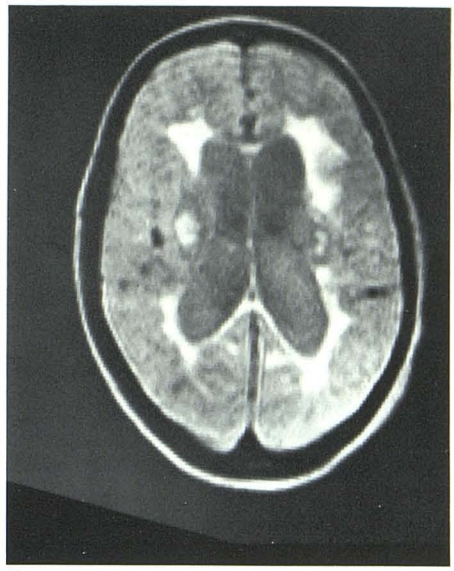

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Fig. 3.—Pattern 2 in 57-year-old man with seizures and/or transient ischemic attacks. Diffuse continuous pencil-thin line of hyperintensity surrounds ventricles.

Fig. 4.—Pattern 3 in 37-year-old woman with obstructive hydrocephalus secondary to pineal region tumor. Smooth halo of hyperintensity surrounds bodies of lateral ventricles. Findings reflect increase in periventricular water (edema) secondary to transependymal resorption of spinal fluid.

Fig. 5.—Pattern 4 in 47-year-old woman with long-standing history of multiple sclerosis. Diffuse periventricular hyperintensity with irregular lateral margins. In many areas hyperintensity extends to corticomedullary junction.

Results

PVH was seen in 93.4% of patients regardless of age, gender, clinical diagnosis, or other MRI findings (table 1). The PVH patterns most often encountered were 1 (43.3%) and 2 (29.9%). When the patients were divided into those with otherwise normal examinations and those with other evidence of pathology, several trends were noted both between the groups and within each group.

In patients with otherwise normal examinations (table 2, fig. 6A), patterns 0–2 (figs. 1–3) were encountered and pattern 1 (fig. 2) was seen most often (68.3% of cases). Within this group there was a definite increase in PVH with increasing age. Two-thirds of the patients in each subgroup had pattern 1, but in older patients (over 30 years of age), pattern 2 was twice as common as in younger patients (23.9% vs. 12.2%). Conversely, in younger patients (under 30 years of age), there was a higher incidence of pattern 0 (18.2% vs. 9.5%).

There was also a dramatic increase in PVH in patients with other evidence of pathology (table 3, fig. 6B). Thus, patterns 0 and 1 were seen in only 31% of these patients as opposed to 81% of the otherwise normal group. Patterns 3 and 4 were always associated with pathology, while patterns 0 and 1 were more typically seen in patients whose studies were otherwise normal. Pattern 2 was a nonspecific pattern frequently encountered in all groups.

Evaluation of the patients with other evidence of pathology revealed several findings. Patients with trauma (fig. 7) and supratentorial tumors had a greater degree of PVH than did patients with otherwise normal examinations. Fifty percent of trauma patients and 62.7% of supratentorial tumor patients had patterns 2–4 as compared with 19% of the otherwise normal group.

PVH was even more extensive in patients with white-matter diseases (fig. 8). Of these patients, 80% had patterns 2–4. When only several lesions were present, pattern 2 was usually encountered, and, as the number of lesions increased, so did the PVH, which became broader and more intense (pattern 3). When many lesions were present, these foci coalesced with each other and the PVH to produce an appearance that was intermediate between patterns 3 and 4 (fig. 8). In pattern 4 the white matter was diffusely hyperintense and there was no separation of the periventricular region from the rest of the white matter. This pattern was encountered in two patients with radiation change (diffuse necrotizing leukoencephalopathy) (fig. 9) and in several patients with long-standing multiple sclerosis (fig. 5).

The patients with hydrocephalus had the greatest degree of PVH (90.5% had patterns 2–4) (table 4). MRI was more sensitive than CT in detecting periventricular abnormalities in these cases. None of the patients with pattern 2 PVH on MRI had periventricular hypodensity on CT. Of 37 patients with patterns 3 and 4 PVH, 28 also had periventricular hypodensity on CT, but in all cases the degree of PVH on MRI was more extensive than the periventricular lucency on CT (fig. 10). Nine patients had pattern 3 PVH on MRI and no periventricular hypodensity on CT (fig. 11). PVH was more extensive in those patients with CT evidence of periventricular hypodensity than in those patients with no periventricular abnormalities on CT. The extent of the PVH was related to the severity and duration

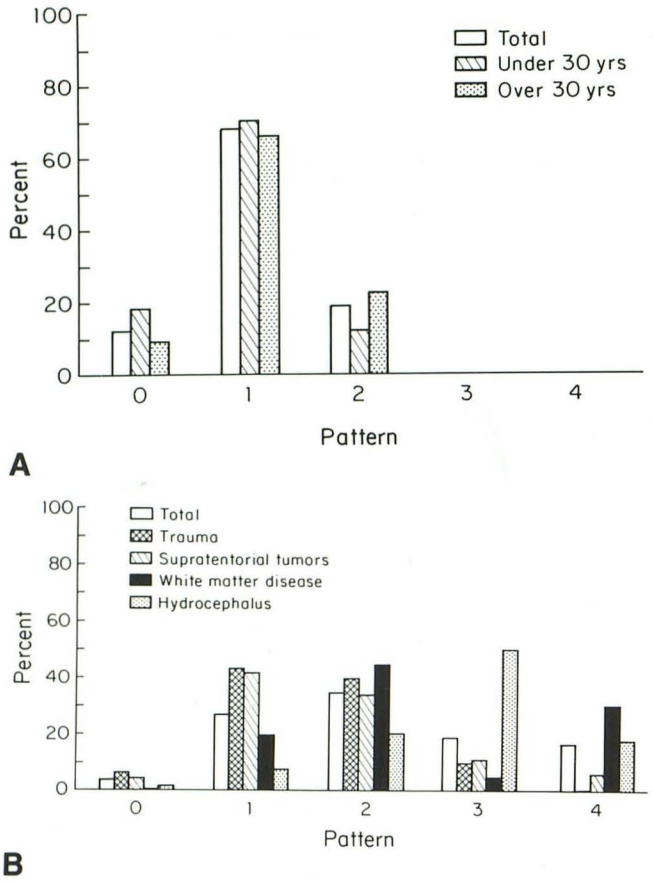


Fig. 6.—Incidence of PVH patterns. **A**, Otherwise normal group. All patients fall into patterns 0–2. Pattern 1 is most common. Pattern 2 is second most common in patients over 30 years of age, while pattern 0 is second most common pattern in patients under 30 years of age. **B**, In Patients with other evidence of pathology, all five patterns are present. Pattern 2 is seen in all groups. Pattern 3 is most typically seen in patients with hydrocephalus. Also, some hydrocephalic patients had patterns 2 and 4. Pattern 4 is commonly seen in patients with demyelinating disease.

TABLE 1: **Distribution of Patients by Pattern of Periventricular Hyperintensity**

Pattern	No. of Cases	(%)
0	24	(6.6)
1	158	(43.3)
2	109	(29.9)
3	41	(11.2)
4	33	(9.0)
Total	365	(100.0)

of the hydrocephalus (table 4). It was most extensive in patients with obstructive hydrocephalus secondary to mass lesions or basal meningitis. Thirty-one of these 34 patients had pattern 3 or 4 PVH. Two patients with early mild hydrocephalus had pattern 2. Of note was one patient with basal meningitis and 6 months of signs and symptoms of hydrocephalus with progressive ventricular dilatation identified on CT. Only pattern 1 PVH was identified on MRI (fig. 12). Patients with chronic and/or mild hydrocephalus had less severe PVH. Of six patients with normal-pressure hydrocephalus, two had mild, nonspecific PVH (pattern 2). Four had more severe PVH (patterns 3 and 4), but in all cases foci of hyperintensity were present in the white matter. The extent, configuration, and intensity of the PVH in these patients was indistinguishable from that seen in other elderly patients with white-matter infarctions who had no clinical or radiographic evidence of normal-pressure hydrocephalus (cf. figs. 8 and 13). Patients with arrested and adequately shunted hydrocephalus had absent (pattern 0) or mild (pattern 1 or 2) PVH. Two patients with shunt malfunction had more severe PVH (pattern 3).

TABLE 2: **Patterns of Periventricular Hyperintensity in Otherwise Normal Examinations**

Pattern	No. (%) Total		<30 Years		>30 Years	
0	13	(12.5)	7	(17.2)	6	(9.5)
1	71	(68.5)	29	(70.6)	42	(66.5)
2	20	(19.0)	5	(12.2)	15	(23.9)
Total	104		41		63	

Note.—There were no instances of pattern 3 or 4 in otherwise normal examinations.

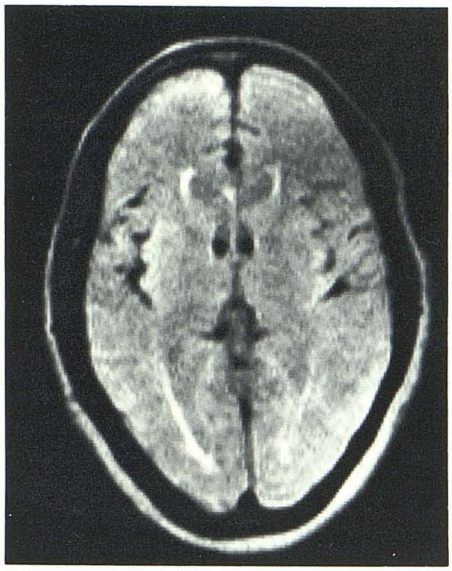
Discussion

PVH on long TR/long TE (T2-weighted) MRI has been reported in demyelinating diseases [1–10] and hydrocephalus [2, 6, 7, 11]. The hyperintensity is a reflection of a pathologic increase in tissue water resulting in prolongation of T2 relaxation [4–6, 8]. In demyelinating diseases the increased water and its resultant PVH is said to result from destruction of hydrophobic myelin [5] (figs. 5, 8, and 9), while in hydrocephalus periventricular edema from transependymal resorption of spinal fluid produces a halo of hyperintensity [7] (figs. 4 and 11). Our review of a large series of unselected cases confirms that PVH is most common and severe in patients with demyelinating disease and hydrocephalus, but the study also documents that some degree of PVH is present in most patients (93.4%) regardless of diagnosis (table 1).

The pathophysiologic mechanisms invoked to explain PVH in patients with demyelinating diseases and hydrocephalus do not explain the PVH seen in normal patients or in patients with abnormalities that do not directly involve the ependyma or adjacent white matter (tables 2 and 3, fig. 6). These phenomena require a physiologic rather than a pathophysiologic explanation for increased water in the periventricular region. The normal flow of interstitial water into the lateral ventricles from the extracellular space [12, 13] can be invoked to explain the higher concentration of water in the periventricular region than in the rest of the white matter. Water, which normally (or pathologically) escapes from the vascular system into the extracellular space, is reabsorbed across the epen-

TABLE 3: **Pathology Groups in Patients with Patterns of Periventricular Hyperintensity**

Pattern	No. (%)				
	Total	Trauma	Supratentorial Neoplasm	White-Matter Disease	Hydrocephalus
0	7 (3.2)	2 (6.7)	4 (5.6)	0	1 (1.9)
1	59 (27.5)	13 (43.3)	30 (41.7)	12 (20.0)	4 (7.5)
2	75 (34.9)	12 (40.0)	25 (34.7)	27 (45.0)	11 (20.8)
3	41 (19.1)	3 (10.0)	8 (11.1)	3 (5.0)	27 (50.9)
4	33 (15.3)	0	5 (6.9)	18 (30.0)	10 (18.9)
Total	215	30	72	60	53



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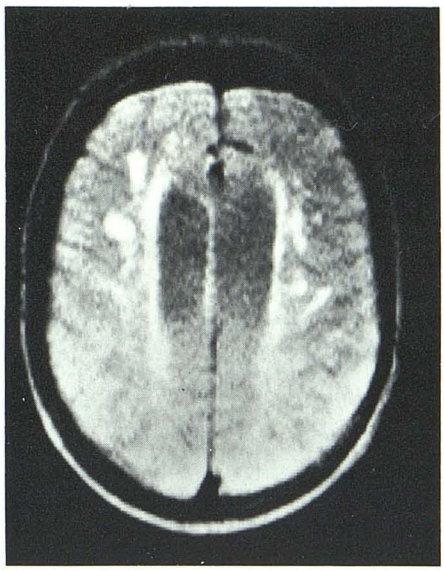

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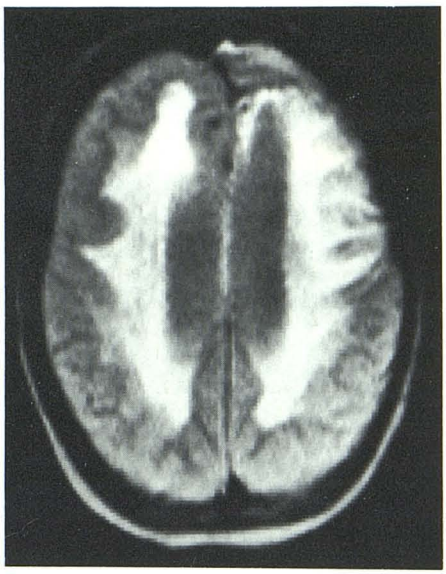

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Fig. 7.—47-year-old woman with history of head trauma several months after drainage of subdural hematoma. Thin, continuous line of PVH (pattern 2).

Fig. 8.—77-year-old hypertensive patient with dementia. CT scan (not shown) showed atrophy and subtle white-matter hypodensities, considered consistent with infarction. No clinical or CT evidence of hydrocephalus. MR image shows multiple foci of hyperintensity in supratentorial white matter. In addition, halo of PVH around ventricles (pattern 3) is indistinguishable from findings in patients with hydrocephalus. Several of foci in white matter are nearly confluent with each other and with band of PVH. Thus, findings are intermediate between patterns 3 and 4.

Fig. 9.—54-year-old patient with reticulum cell sarcoma treated with radiotherapy about 2 years before. CT scan (not shown) showed no evidence of residual tumor but diffuse hypodensity of supratentorial white matter. MR image shows diffuse, irregular supratentorial white-matter PVH (pattern 4). Discrete periventricular halo of hyperintensity cannot be separated from rest of white-matter hyperintensity. Findings consistent with radiation damage (diffuse necrotizing leukoencephalopathy), but are indistinguishable from those seen in patients with other diffuse demyelinating processes, such as severe multiple sclerosis.

dymal lining into the lateral ventricles [13], accounting for 10%–60% of normal spinal fluid production [14–17]. The PVH seen in normal patients may, therefore, be a reflection of the higher concentration of interstitial water at the ventricular lining resulting from concentric bulk flow within the white matter from larger more peripheral sites toward the more confined periventricular region.

If this hypothesis is correct, it would also explain the trends identified in this study. The progressive increase in PVH seen in older patients with otherwise normal examinations might reflect a slight increase in interstitial water caused by vascular damage with leakage of fluid into the extracellular space even when obvious focal abnormalities are not seen. Any pathologic cause of increased interstitial water would produce a secondary increase in PVH as this water flows to the ependymal lining to be reabsorbed into the ventricles. The transependymal resorption of traumatic edema fluid has been documented in animal studies [18, 19], and it is likely that the increased PVH seen in patients with traumatic and neoplastic edema is a reflection of this phenomenon. There is also an increase in interstitial water in patients with demyelinating diseases caused by the destruction of hydrophobic myelin [5] and damage to the blood-brain barrier [20, 21]. Thus, at least some of the PVH in patients with demyelinating diseases may

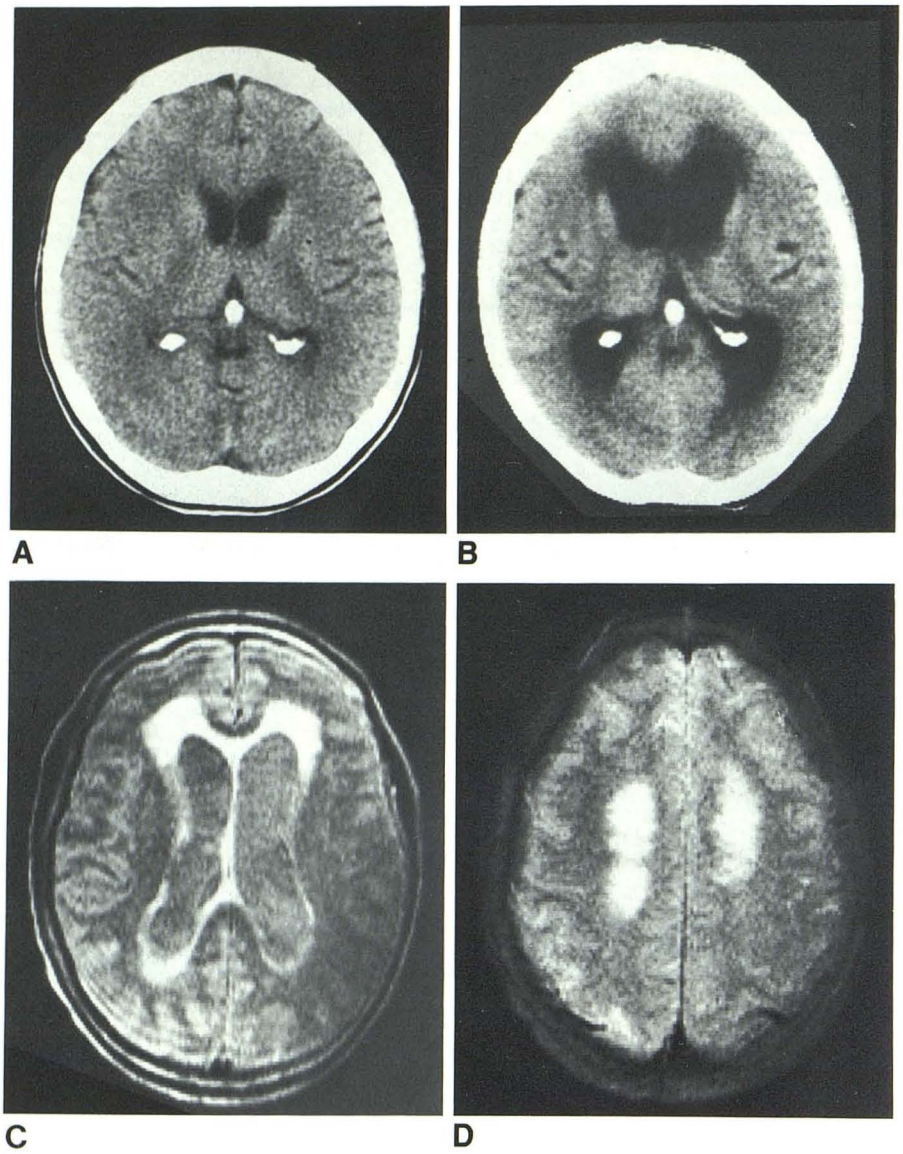


Fig. 10.—61-year-old patient with carcinomatous meningitis and secondary communicating hydrocephalus. **A**, Initial CT scan. Normal-sized ventricles. **B**, Follow-up CT scan 4 months later. Marked ventricular dilatation and bifrontal periventricular hypodensity, classic CT findings of periventricular edema due to transependymal resorption of spinal fluid. **C**, MR image at same time as **B**. Diffuse broad periventricular halo completely surrounds ventricles. Edema in **C** is more extensive than on CT. **D**, Higher level. Superior irregularities in periventricular edema cause superficial resemblance to diffuse demyelination. Irregularities are more lobulated than those seen in diffuse demyelinating disease. In addition, hyperintensity does not extend to corticomedullary junction.

TABLE 4: **Types of Hydrocephalus in Patients with Patterns of Periventricular Hyperintensity**

Pattern	No. of Cases by Type of Hydrocephalus				
	Total	Obstructive	Normal-Pressure	Arrested	Shunted
0	1	0	0	1	0
1	4	1	0	2	1
2	11	2	2	3	4
3	27	22	3	0	2
4	10	9	1	0	0
Total	53	34	6	6	7

be secondary to increased reabsorption of interstitial water rather than a reflection of primary involvement of the periventricular white matter by the demyelinating process itself (fig. 8). This explanation would help explain the observation that as the number of white-matter lesions increased so did the extent of PVH.

In hydrocephalic patients the increased hydrostatic pressure causes a reversal of the direction of transependymal flow. Fluid is forced out of the ventricles into the periventricular white matter. Although the direction of flow is different, the distribution, and, in many cases, the amount of periventricular water is similar in patients with hydrocephalus and in those with increased interstitial water from parenchymal abnormalities; thus, the PVH patterns encountered on MRI in these conditions may be indistinguishable.

Elucidation of the causes (both physiologic and pathophysiologic) of PVH must await experimental verification, but there are several clinical implications of the phenomena observed in this study: (1) Mild PVH (pattern 1 or 2) is normal, and thus rounded hyperintense foci seen adjacent to the frontal horns (fig. 2A) should not be mistaken for areas of demyelination;

Fig. 11.—42-year-old woman with posterior fossa arachnoid cyst with secondary obstructive hydrocephalus. **A**, CT scan at level of lateral ventricles. Dilated ventricles but no obvious periventricular hypodensity. **B**, MR image at same level. Definite periventricular halo (pattern 3) indicative of edema from transependymal reabsorption of spinal fluid.

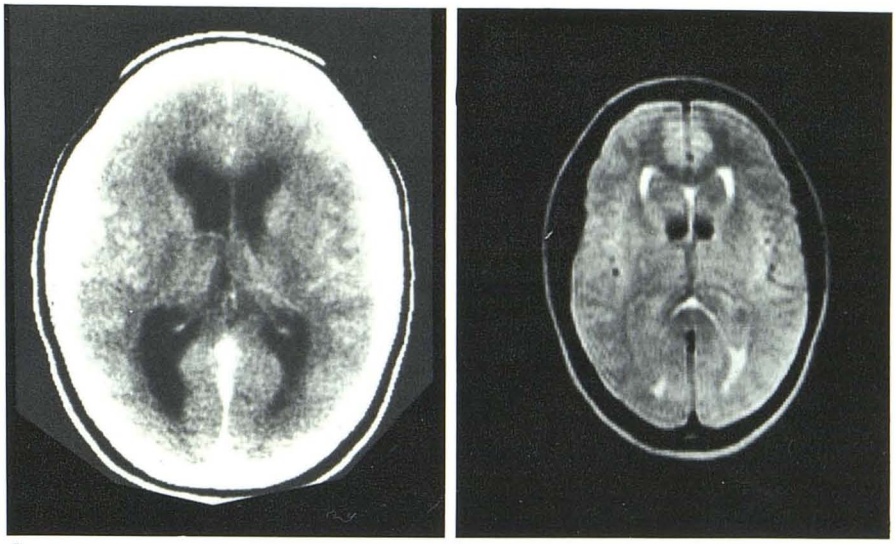
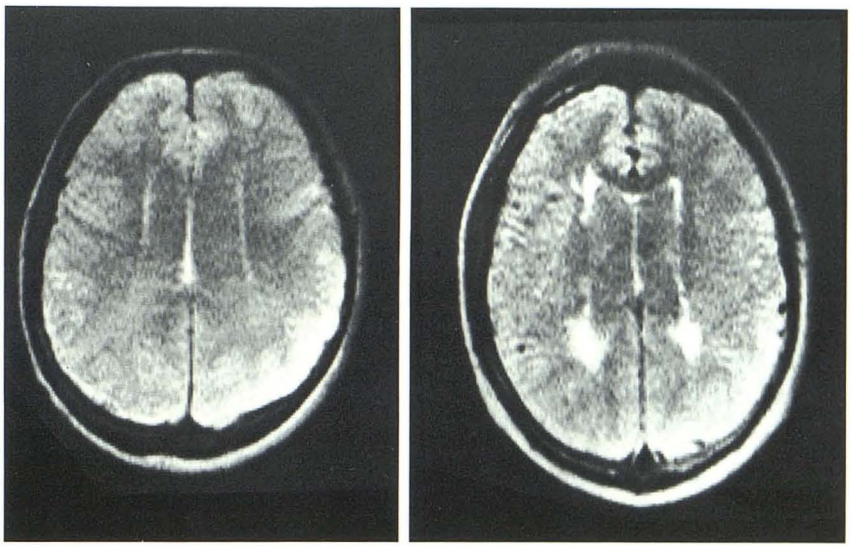


Fig. 12.—26-year-old man with communicating hydrocephalus secondary to basal meningitis of about 6 months duration. MR image shows dilated ventricles but only mild discontinuous PVH (pattern 1). Therefore, there is no evidence of transependymal resorption or spinal fluid or edema, despite good clinical evidence of active hydrocephalus.

Fig. 13.—76-year-old man with progressive dementia and gait disturbances. CT scans (not shown) showed progressive ventricular dilatation over a 2 year period. Clinical and CT findings were consistent with normal-pressure hydrocephalus. Patient responded well to shunting. MR image shows narrow halo of PVH that could represent transependymal resorption of spinal fluid. In addition to halo, there are multiple foci of hyperintensity in white matter confluent with periventricular halo, presumably representing coexistent white-matter infarcts. MRI features in this patient are indistinguishable from those in patient with multiple infarcts alone (cf. fig. 8).



(2) Pattern 2 is nonspecific and may be seen in a wide range of circumstances. Its presence in older patients without other abnormalities may be considered normal (fig. 3), just as atrophy may be normal in elderly patients but in younger patients it may be a nonspecific indicator of cerebral insult (e.g., trauma [fig. 7] or early mild hydrocephalus), even in the absence of other MRI abnormalities; (3) Patterns 3 and 4 are always pathologic and are most typically seen in hydrocephalus (figs. 4 and 10) and extensive demyelination (figs. 5 and 9), respectively. There is, however, overlap between the two patterns; (4) In severe hydrocephalus, the periventricular halo becomes irregular, especially at its superior margins (fig. 10D), thus mimicking the hyperintensity seen in diffuse demyelination (pattern 4). The hyperintensity seen in severely hydrocephalic patients (nine cases) could, however, be distinguished from the hyperintensity seen in patients with diffuse demyelination (18 cases) on the basis of subtle differences in the configuration of the hyperintensity. In severe hydrocephalus, the peripheral margins of the PVH are irregular but blunted, and they do not extend to the corticomedullary junction (fig. 10D). In demyelinating diseases, the PVH is more sharply angled and usually does extend to the corticomedullary junction (figs. 5 and 9); (5) In patients with extensive multifocal demyelination there is a general increase in PVH (pattern 3) indistinguishable from mild to moderate periventricular edema secondary to hydrocephalus (cf. figs. 8 and 13).

These phenomena have a significant impact on the use of MRI in the evaluations of patients for hydrocephalus. The

greater sensitivity of MRI relative to CT in the detection of edema [22] had led to the hope that MRI would be more sensitive than CT in detecting mild periventricular edema and that this finding could be used as a criterion for shunting patients in whom a diagnosis of hydrocephalus could not be made by other criteria [23] (e.g., normal-pressure hydrocephalus suspects). This study confirms that MRI is more sensitive than CT in detecting periventricular edema (cf. figs. 10 and 11), but it also indicates that this finding is of limited clinical value. Those hydrocephalic patients with severe PVH on MRI with or without CT evidence of edema were easily diagnosed using standard clinical and radiographic criteria. The diagnoses of mild hydrocephalus and normal-pressure hydrocephalus remain difficult with MRI because its increased sensitivity is accompanied by a decrease in specificity. In mild or early active hydrocephalus only a thin band of PVH (pattern 2) may be seen, and this nonspecific finding cannot be used as a criterion for shunting. Our experience in patients with normal-pressure hydrocephalus, although limited to six cases, has been particularly disappointing. Two patients had mild nonspecific PVH (pattern 2). Four patients had prominent PVH (three patients, pattern 3; one patient, pattern 4), but in all cases multiple white-matter hyperintense foci (presumably representing infarction) were also present (fig. 13). The degree of PVH was similar to that seen in nonhydrocephalic elderly patients with white-matter infarcts alone, and, therefore, the PVH pattern could not be used as a criterion for shunting. Since these infarcts are commonly seen on MRI in elderly patients [24], their concurrent occurrence in patients with normal-pressure hydrocephalus limits the usefulness of the PVH pattern to those few cases where PVH is seen in the absence of focal white-matter lesions.

While most patients with hydrocephalus show relatively extensive PVH, absent or mild PVH (pattern 0 or 1) was also encountered occasionally. In four cases this reflected either arrested hydrocephalus or adequate shunt function, but one patient with active hydrocephalus who subsequently responded to shunting showed only mild PVH (fig. 12). It is unclear why periventricular edema did not develop, and one might speculate that spinal fluid absorption occurred via alternate routes (e.g., perineural or perivascular lymphatics [14]), but it is clear that simple reliance on the degree of PVH cannot be used as a criterion for shunting.

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