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Association of Deep White Matter Infarction with Chronic Communicating Hydrocephalus:
Implications Regarding the Possible Origin of Normal-Pressure Hydrocephalus

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The coexistence of cerebrovascular disease leading to deep white matter infarction and normal-pressure hydrocephalus has been noted previously in clinical studies, as both diseases can present with the triad of gait disturbance, dementia, and incontinence. The purpose of this MR study was to determine if the two diseases demonstrated a statistical association. Evidence of patchy periventricular hyperintensity representing presumed deep white matter infarction was sought in 20 patients shunted for normal-pressure hydrocephalus and in 35 additional consecutive patients with clinical symptoms and MR findings consistent with normal-pressure hydrocephalus. Deep white matter infarction was also sought in 62 consecutive age-matched control subjects. There was a statistically significant ($p < .001$) higher association (58%) of marked infarction in the 55 patients with normal-pressure hydrocephalus than in the age-matched controls (24%). MR findings of communicating hydrocephalus (ventriculomegaly and increased aqueductal CSF flow void) were sought in 78 consecutive patients with presumed deep white matter infarction, and the degree of severity of the two diseases was also found to be statistically significant ($p < .05$).

In view of this association, the possibility that the two diseases are related was considered. A potential mechanism is discussed whereby deep white matter infarction leading to decreased periventricular tensile strength could result in communicating hydrocephalus. It is plausible that normal-pressure hydrocephalus may result from a number of different insults to the brain.

Normal pressure hydrocephalus (NPH) is a form of chronic communicating hydrocephalus of unknown origin [1–4]. Although the mean CSF pressure is normal in NPH (hence the name), the CSF pulse pressure can be up to six times normal, producing the "water-hammer pulse" described by neurologists [4]. Tangential shearing forces on the paracentral fibers of the corona radiata are thought to produce the clinical triad of gait apraxia, dementia, and incontinence [5, 6]. These fibers are also involved in deep white matter infarction (DWMI) resulting from decreasing cerebral perfusion with advancing age [7, 8]. It is not surprising, therefore, that patients with severe DWMI should have the same clinical triad as those with NPH [9–12]. Both NPH and DWMI are more easily diagnosed by MR imaging [7, 8, 13, 14] than by CT [14, 15]. NPH and other forms of communicating hydrocephalus are diagnosed on the basis of ventricular dilatation out of proportion to sulcal enlargement (differentiating hydrocephalus from generalized atrophy) [14]. NPH is best distinguished from central atrophy on the basis of a marked CSF flow void resulting from the marked to-and-fro motion of CSF through the cerebral aqueduct and contiguous third and fourth ventricles [13]. The hyperdynamic CSF flow state that has been described in NPH [14] produces a flow void in these structures that is visible on MR (Fig. 1) but not on CT. The presence of a significant CSF flow void has recently been correlated with a favorable surgical response to ventriculoperitoneal shunting [15].
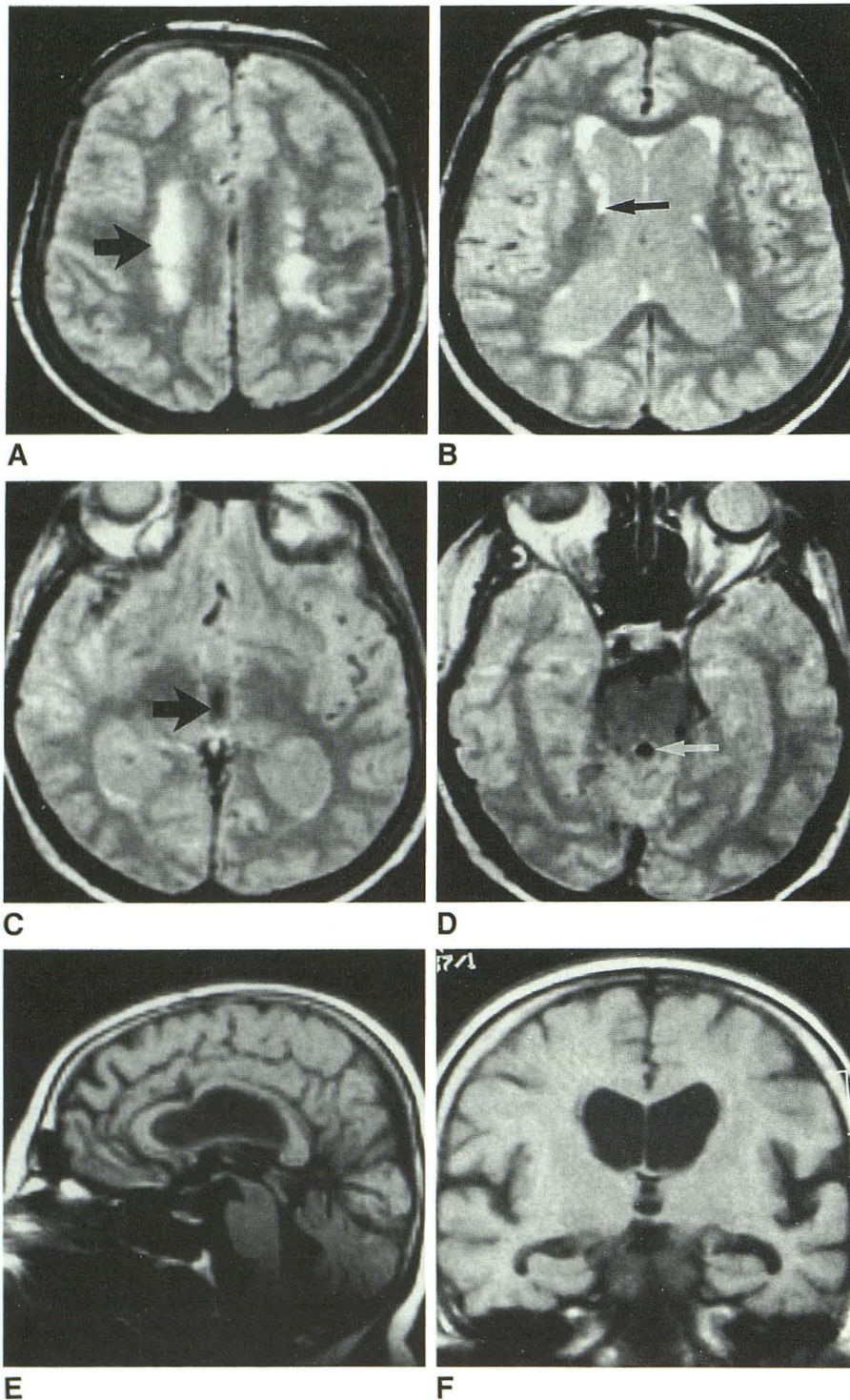
Fig. 1.—72-year-old patient from group 2 with marked deep white matter infarction (DWMI) and moderate communicating hydrocephalus.

A, SE 3000/40 section through centrum semiovale shows patchy periventricular hyperintensity (*arrow*) characteristic of marked DWMI.

B, SE 3000/40 section through lateral ventricles shows ventriculomegaly as well as subependymal, deep white matter infarcts (*arrow*).

C, SE 3000/40 section through third ventricle shows mild enlargement and marked CSF flow void (*arrow*) in posterior portion.

D, SE 3000/40 section through midbrain shows aqueductal enlargement and CSF flow void (*arrow*). Extent of CSF flow void from posterior third ventricle through aqueduct into lower fourth ventricle (*not shown*) indicates presence of hyperdynamic flow state.

E, Midline sagittal section (SE 500/40) shows upward bowing of corpus callosum with flattening of cortical gyri against inner table of calvaria.

F, Coronal section (SE 600/30) through lateral ventricles shows ventricular enlargement out of proportion to cortical sulcal dilatation with flattening of cortical gyri against inner table of calvaria. There is also widening of callosal angle and narrowing of interhemispheric fissure.

Since chronic communicating hydrocephalus and DWMI may have such similar clinical presentations and are well visualized by MR imaging, the current study was undertaken to determine any statistical association between the two diseases and if they might represent different manifestations of the same disease process.

Materials and Methods

Four groups of patients were evaluated: 20 patients shunted for presumed NPH (group 1); 35 consecutive patients with MR findings and clinical symptoms consistent with the diagnosis of NPH (group 2); 78 consecutive patients with patchy periventricular hyperintensity, that is, presumed DWMI (group 3); and 62 consecutive patients over

age 60 without MR findings of communicating hydrocephalus (group 4), who served as a control for groups 1 and 2. Patients in groups 2–4 were gleaned from retrospective review of the MR log book, representing consecutive patients with presumed NPH (group 2), presumed DWMI (group 3), and presumed elderly normal (group 4). Patients in group 1 all presented with gait apraxia, dementia, and (in 17 of 20) incontinence, as determined by attending neurologists and neurosurgeons. These patients were studied in 1984 and early 1985 when ventriculoperitoneal shunting was being performed more frequently for presumed NPH. MR images reflect the typical technique used at that time: 128 × 128 acquisition matrix (1.7-mm spatial resolution); 7-mm slice thickness with a 3-mm gap; spin-echo (SE) sequence, 2000/28, 56 (TR/TE); and four excitations (17-min acquisition).

Group 2 represented the most recent 35 consecutive patients with gait disturbance, dementia, and MR findings consistent with NPH, including an increased CSF flow void. While many of these patients were shunted, most were not, reflecting the decreasing tendency nationwide to perform such CSF diversionary procedures in 1988–1989. The average age in group 2 was 75 years old (range, 61–83).

Group 3 comprised the 78 most recent, consecutive patients over age 60 with patchy, periventricular hyperintensity on MR, presumably DWMI. Their average age was 71 years old (range, 60–85). Their clinical presentations were diverse, some having dementia; some having difficulty walking; and some being evaluated for other diseases, for example, metastatic disease or primary tumor. No patient in group 3 was considered clinically to have NPH.

Group 4 comprised the 62 most recent, consecutive patients over age 60 without MR findings of communicating hydrocephalus. These patients served as an age-matched control group for groups 1 and 2. Their average age was 73 years old (range, 61–87).

Patients in groups 2–4 were studied with the technique typical for 1988 and early 1989: 0.9- to 1.0-mm in-plane spatial resolution, 5-mm contiguous slices, SE 3000/40, 80 sequences, two excitations, and 160 to 256 phase-encoded projections over a 16- to 24-cm lateral field of view (16- to 25-min acquisition). All studies were performed on a Diasonics MT/S MR imager at 0.35 T.

Grading of Patients in Groups 2 and 4

Communicating hydrocephalus was graded as either absent, mild, moderate, or marked on the basis of the size of the ventricles, the diameter of the aqueduct, the extent of the CSF flow void, and upward bowing of the corpus callosum [14, 15]. Communicating hydrocephalus was considered to be absent if the ventricles were normal in size or, if enlarged, increased in proportion to the enlarged cortical sulci. Some of these patients with prominent CSF spaces were considered to have atrophy. The corpus callosum was normally bowed and the CSF flow void was limited to the aqueduct (i.e., normal) without extension into the adjacent third or fourth ventricles (Fig. 2). It should be stressed that the aqueductal flow void depends on the gradient strength and the TE and thus is dependent on the machine and specific technique. There was no evidence of flattening of the cortical gyri against the inner table of the calvaria in these patients. All patients in group 4 (normal) also satisfied these criteria.

In mild communicating hydrocephalus the lateral ventricles had begun to enlarge and were considered to be increased out of proportion to any enlargement of the cortical sulci. The aqueduct remained normal in diameter and the CSF flow void was confined to the aqueduct, that is, it was normal. There was mild upward bowing of the corpus callosum but no significant flattening of the gyri against the inner table of the calvaria (Fig. 3).

In moderate communicating hydrocephalus, there was somewhat greater ventricular dilatation relative to sulcal enlargement. The aqueduct was dilated and the CSF flow void was increased, extending from the posterior third to the mid fourth ventricle (Fig. 1). There was greater upward bowing of the corpus callosum and flattening of the cortical gyri against the inner table of the calvaria on parasagittal sections. On coronal views there was widening of the callosal angle.

In marked communicating hydrocephalus, there was additional ventricular and aqueductal enlargement. The CSF flow void extended from the anterior third ventricle through the obex of the fourth ventricle. Such a flow void is demonstrated in Figure 4 in a patient with chronic multiple sclerosis.

When the criteria for moderate or marked communicating hydrocephalus were met but the CSF flow void was not increased, central atrophy was considered to be present. Such patients have been found to not respond well to ventriculoperitoneal shunting [15].

Grading of Patients in Group 3

DWMI was graded as absent, mild, or marked. DWMI was considered to be absent when no patchy periventricular white matter disease was seen. This is illustrated in Figure 2. While smooth periventricular hyperintensity may be seen in the elderly, this has been reported to represent subependymal myelin pallor [16] without frank infarction. Mild DWMI was defined as focal, nonconfluent disease with no single lesion greater than 1 cm in diameter (Fig. 3). Marked DWMI was defined as confluent disease or individual lesions greater than 1 cm in diameter (Fig. 1). Infarction has been previously demonstrated pathologically in patients with focal periventricular hyperintensity [8].

In order to ascertain the association of NPH and presumed DWMI, groups 1 (shunted NPH) and 2 (consecutive patients with radiographic and clinical NPH) were evaluated for associated patchy periventricular hyperintensity and group 3 (presumed DWMI) was evaluated for evidence of coexisting communicating hydrocephalus.

Results

The data summarizing the prevalence and degree of communicating hydrocephalus and DWMI are given in Table 1. All patients in groups 1 and 2 (NPH) had moderate or marked communicating hydrocephalus. No patient in group 3 (DWMI) had marked communicating hydrocephalus and no patient in group 4 (controls) had any degree of communicating hydrocephalus (by definition).

Of the 20 patients shunted for presumed NPH (group 1), 19 (95%) had at least mild DWMI and 12 (60%) had marked DWMI. Of the 35 patients in group 2 with MR findings of chronic communicating hydrocephalus and clinical findings of NPH, 32 (91%) had at least mild DWMI and 20 (57%) had marked DWMI. This compares with the control patients in group 4, 41 (66%) of whom had at least mild DWMI and 15 (24%) of whom had marked DWMI. When the data from the 55 patients *with* NPH (groups 1 and 2) were compared with the data from the patients *without* NPH (group 4) using the chi-square test, there was a statistically significant association between DWMI and NPH ($p < .001$) (Table 2).

Of the 78 patients with DWMI (group 3), 63 (81%) had at the least mild communicating hydrocephalus and 24 (31%) had moderate communicating hydrocephalus. There was no marked communicating hydrocephalus in group 3. Of the 52 patients in group 3 with marked DWMI, 43 (83%) had at least
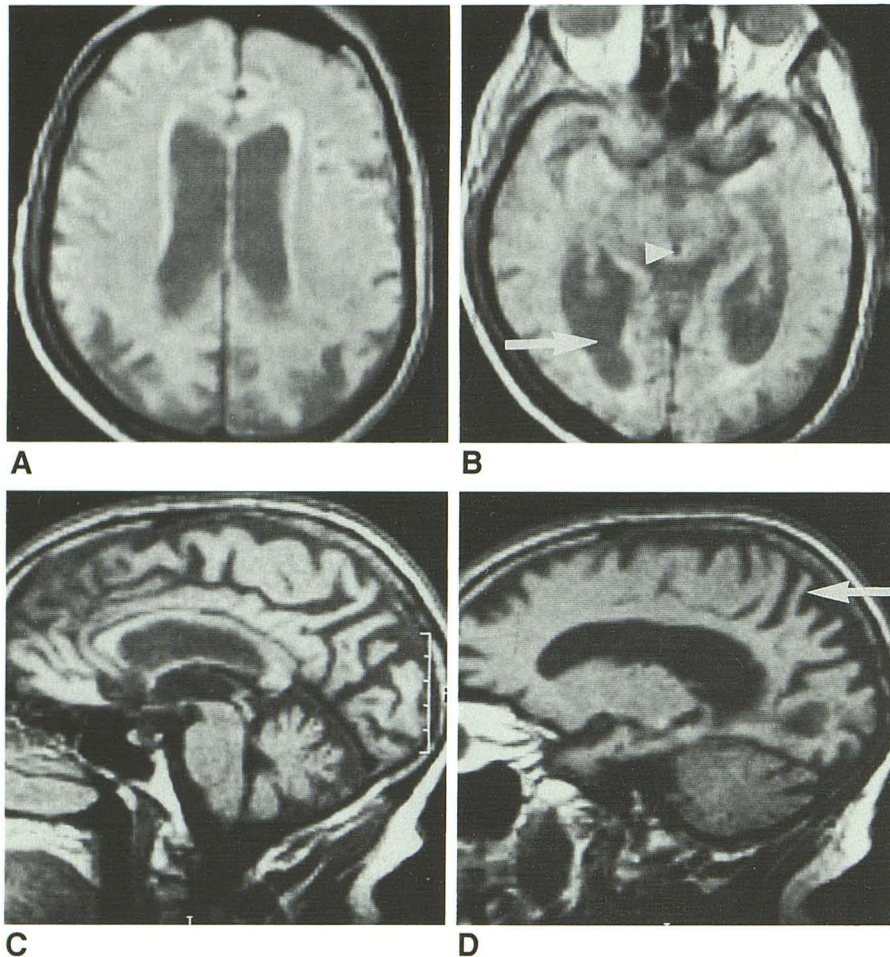
Fig. 2.—78-year-old man with atrophy.

A, Axial section (SE 2000/30) through lateral ventricles shows ventriculomegaly with concomitant enlargement of cortical sulci. No patchy periventricular hyperintensity is seen.

B, Axial section (SE 2000/30) through aqueduct shows normal CSF flow void (arrowhead) as well as enlarged occipital horns (arrow) and prominent cortical gyri.

C, Midline sagittal section (SE 500/40) shows thinning of corpus callosum without upward bowing. Cortical gyri are rounded and do not extend completely to inner table of calvaria.

D, Parasagittal section (SE 500/40) shows rounded or peglike cortical gyri (arrow), which are not compressed against inner table of calvaria.

mild and 19 (37%) had moderate communicating hydrocephalus. The results of comparing the degree of DWMI with the degree of communicating hydrocephalus (when present) in group 3 patients are shown in Table 3. (This represents the subset of group 3 patients with mild or moderate communicating hydrocephalus.) A chi-square test on this data demonstrated a statistically significant association between the two diseases ($p < .05$).

Of the 20 patients in group 1 who were shunted for presumed NPH, four were studied by MR both before and after shunting [15]. With MR techniques typical for 1984–1985, there was no discernible difference in the degree of deep white matter abnormality, the size of the ventricles, or the extent of the CSF flow void. Such comparisons are now underway using higher-resolution MR imaging and cardiac-gated CSF flow quantifying techniques.

There was no significant difference in the ages among the four groups.

Discussion

The coexistence of hypertensive cerebrovascular disease and NPH has been noted previously [9–12]. Earnest et al. [11] stated that "multiple deep cerebral infarctions may be

the initial pathologic process in some cases of NPH" through reduction of the periventricular tensile strength. Graff-Radford and Godersky [12] noted the association of hypertension and NPH, as did Koto et al. [10]. In a review of lacunar infarction, Fisher [9] noted that a number of patients considered to have état lacunaire by Marie [17] in his classic description in 1901 in fact had "enormously" dilated ventricles and probably had NPH instead. Fisher also commented that the compressive effects of NPH may predispose to deep white matter infarcts. Thus, the association of DWMI and NPH has been noted previously by both neuroclinicians and neuropathologists.

The diagnosis of NPH is facilitated by MR. The midline sagittal view (Fig. 1E) in MR demonstrates upward bowing of the corpus callosum and the coronal view demonstrates widening of the callosal angle (Fig. 1F). In addition, the parasagittal and coronal views demonstrate flattening of the cortical gyri against the inner table of the calvaria (Fig. 1E). This allows NPH to be distinguished from generalized atrophy [14], where the gyri do not extend all the way to the inner table and thus remain rounded or peglike (Fig. 2D).

It should be stressed that NPH is a clinical diagnosis while chronic communicating hydrocephalus is a radiologic diagnosis. To be rigorous, the two terms should be used only in the above contexts. On the other hand, the response to
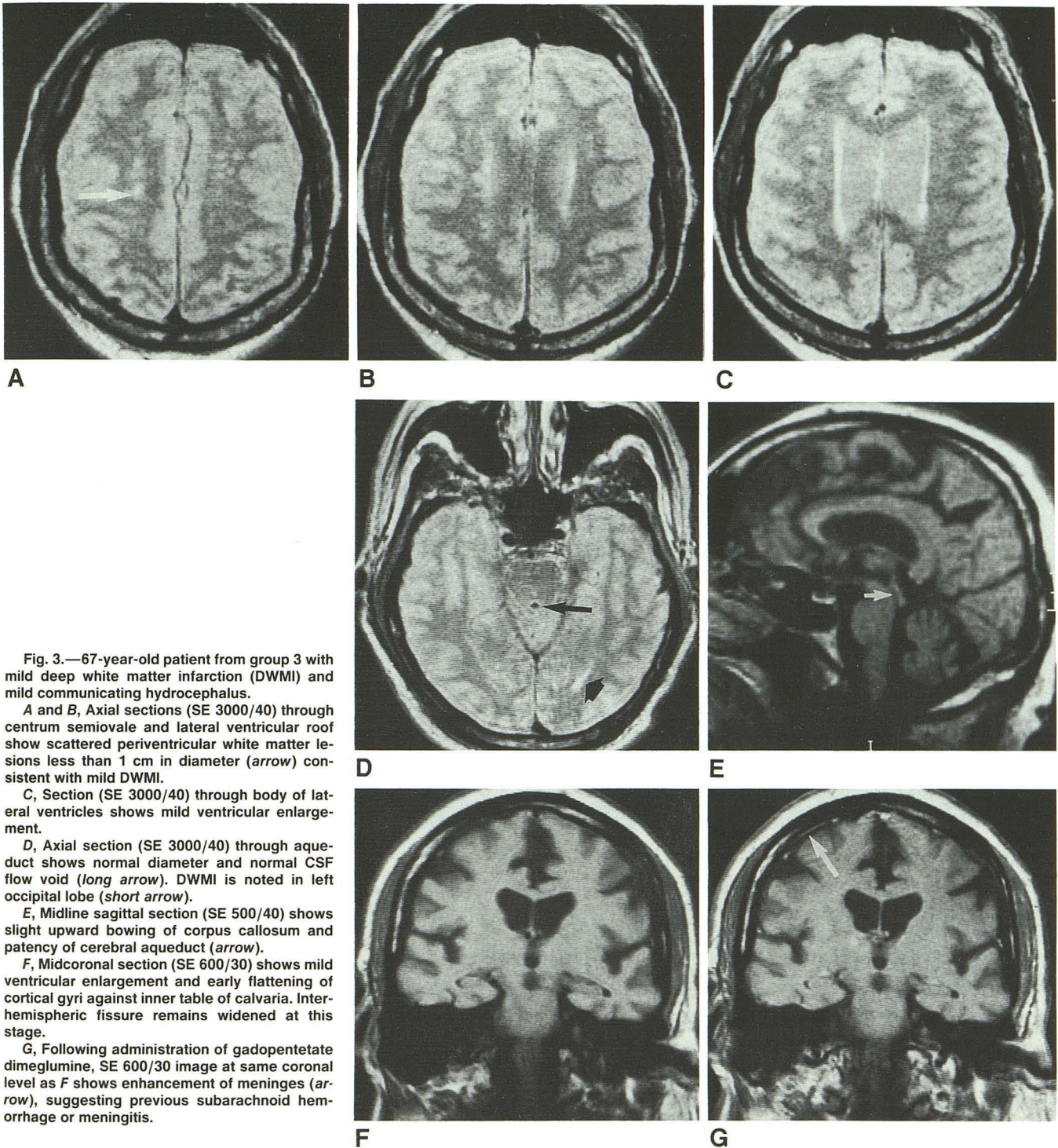
Fig. 3.—67-year-old patient from group 3 with mild deep white matter infarction (DWMI) and mild communicating hydrocephalus.

A and *B*, Axial sections (SE 3000/40) through centrum semiovale and lateral ventricular roof show scattered periventricular white matter lesions less than 1 cm in diameter (*arrow*) consistent with mild DWMI.

C, Section (SE 3000/40) through body of lateral ventricles shows mild ventricular enlargement.

D, Axial section (SE 3000/40) through aqueduct shows normal diameter and normal CSF flow void (*long arrow*). DWMI is noted in left occipital lobe (*short arrow*).

E, Midline sagittal section (SE 500/40) shows slight upward bowing of corpus callosum and patency of cerebral aqueduct (*arrow*).

F, Midcoronal section (SE 600/30) shows mild ventricular enlargement and early flattening of cortical gyri against inner table of calvaria. Interhemispheric fissure remains widened at this stage.

G, Following administration of gadopentetate dimeglumine, SE 600/30 image at same coronal level as *F* shows enhancement of meninges (*arrow*), suggesting previous subarachnoid hemorrhage or meningitis.

ventriculoperitoneal shunting in patients who have both radiographic communicating hydrocephalus and a marked CSF flow void suggests that these patients in fact do have clinical NPH [15].

DWMI produces patchy periventricular hyperintensity that may extend from the ependymal surface of the lateral ventricle into the centrum semiovale (Fig. 1) [7, 8, 18–20]. The deep white matter infarcts occur in a watershed zone between the deep medullary and the superficial cortical circulation, and are due to decreased cerebral perfusion through the medium-sized lenticulostriate and thalamoperforator arteries [7, 18–21], which are also particularly prone to arteriolosclerosis. As these vessels arise from (or near) the circle of Willis, they have a particularly long intraparenchymal course [17–20].
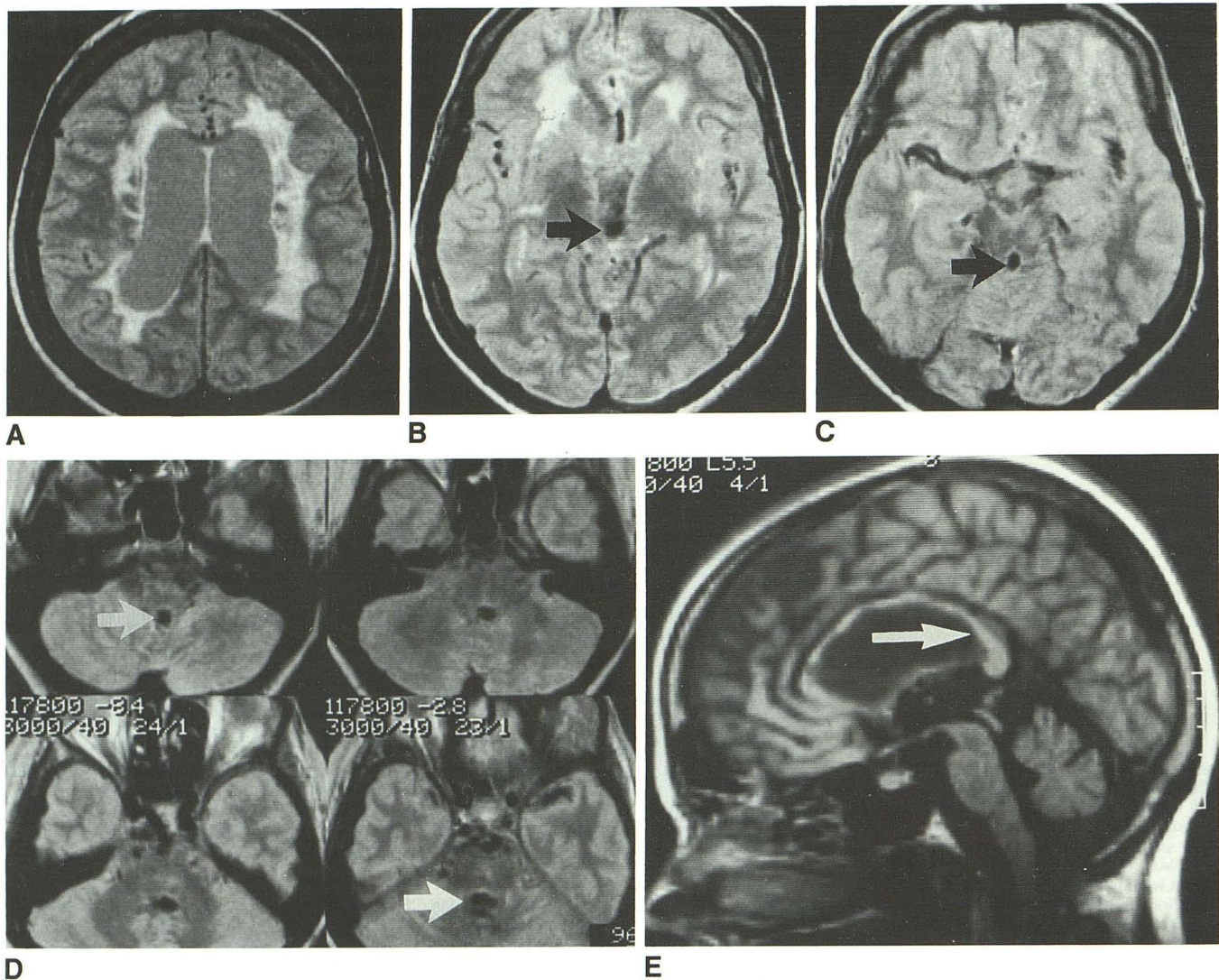
Fig. 4.—49-year-old woman with chronic multiple sclerosis leading to hyperdynamic CSF flow state.
A, Axial section (SE 3000/40) through lateral ventricles shows ventriculomegaly and periventricular hyperintensity secondary to demyelination.
B, Axial section (SE 3000/40) through third ventricle shows enlargement and marked CSF flow void (arrow).
C, Axial section (SE 3000/40) through midbrain shows marked CSF flow void (arrow).
D, Axial sections (SE 3000/40) through fourth ventricle show CSF flow void (arrows).
E, Midline sagittal section shows upward bowing of irregularly thinned corpus callosum (arrow), indicating concomitant communicating hydrocephalus. While cortical gyri are flattened against inner table of calvaria posteriorly, the plane is angled within interhemispheric fissure anteriorly.

Diseases that accelerate arteriolosclerosis, such as hypertension and diabetes, tend to accelerate DWMI as well. In advanced cases, a dementing, hypertensive white matter vasculopathy known as subcortical arteriosclerotic encephalopathy or Binswanger disease is present [7, 18–21].

DWMI is quite common, having been previously reported in 30% of all patients over age 60 [7]. Although deep white matter infarcts are seen easily on T2-weighted MR images, the central nonfunctional necrotic cavity is much smaller than the area of high signal on MR images [8]. Histopathologic studies have demonstrated that astrocytes surrounding a central infarct cavity become reactive, producing a pattern known as isomorphic gliosis [8]. Since the reactive astrocytes have both increased water and increased protein content, gliosis appears bright on T2-weighted MR images. The central

nonfunctional infarct cavity may be surrounded by reactive astrocytes to a diameter up to five times the size of the central lesion [8]. Since the isomorphic gliosis per se does not lead to a loss of function, there is very poor correlation between the degree of white matter abnormalities on MR [22] and the degree of psychometric impairment [22].

This study demonstrates a statistically significant association between DWMI and chronic communicating hydrocephalus. Given the recently noted [15] response to ventriculoperitoneal shunting in patients with communicating hydrocephalus and a prominent CSF flow void, this suggests that DWMI is related to NPH as well. The 24% prevalence of marked DWMI found in our elderly controls (group 4) compares favorably with that previously reported [7] and is significantly less than the 58% prevalence of marked DWMI found in

TABLE 1: Prevalence and Degree of Communicating Hydrocephalus and Deep White Matter Infarction in Four Groups of Patients

Condition/Degree	Group			
	1 (n = 20)	2 (n = 35)	3 (n = 78)	4 (n = 62)
Communicating hydrocephalus				
Absent	0	0	15	62
Mild	0	0	39	0
Moderate	10	19	24	0
Marked	10	16	0	0
Deep white matter infarction				
Absent	1	3	0	21
Mild	7	12	26	26
Marked	12	20	52	15

Note.—Group 1 comprised patients shunted for presumed normal-pressure hydrocephalus; group 2 patients had MR and clinical findings consistent with normal-pressure hydrocephalus; group 3 patients were presumed to have deep white matter infarction; and group 4 comprised normal control subjects.

TABLE 2: Prevalence of Deep White Matter Infarction (DWMI) in Patients with Normal-Pressure Hydrocephalus and Age-Matched Controls

Group(s)	Degree of DWMI (No.)			
	Marked	Mild	Absent	Total
1 and 2 (Normal-pressure hydrocephalus)	32	19	4	55
4 (Control subjects)	15	26	21	62

Note.—Chi-square test = 18.44 ($p < .001$).

TABLE 3: Correlation of Degree of Communicating Hydrocephalus and Deep White Matter Infarction (DWMI) in Group 3 Patients

Degree of Hydrocephalus	Degree of DWMI (No.)		
	Marked	Mild	Total
Moderate	19	5	24
Mild	24	15	39
Total	43	20	63

Note.—Chi-square test = 5.97 ($p < .05$). Group 3 patients were presumed to have DWMI.

patients with NPH (groups 1 and 2). In 78 consecutive patients with DWMI, 81% had at least mild associated communicating hydrocephalus and 31% had moderate communicating hydrocephalus. This group's statistically significant association of the two diseases suggests that they are not independent processes. This association has been noted previously by neuropathologists, neurologists, and neurosurgeons [9–12].

Given the statistical association and clinical similarity of the two diseases, it is tempting to speculate on a biologically possible relationship. The possibilities include: (1) communicating hydrocephalus causes DWMI, (2) a complication of communicating hydrocephalus causes DWMI, (3) DWMI causes communicating hydrocephalus, or (4) a complication of DWMI causes communicating hydrocephalus.

First consider possibilities 1 and 2 that communicating hydrocephalus or a complication of communicating hydrocephalus causes DWMI. Communicating hydrocephalus may follow subarachnoid hemorrhage or meningitis owing to decreased uptake of CSF by the arachnoid villi. This leads to an increase in the interstitial pressure in the brain. For the periventricular watershed regions, which may already be marginally supplied in the elderly, this may be a sufficient additional insult to decrease blood supply through the lenticulostriate and thalamoperforator arteries, leading to deep white matter ischemia and infarction. This combined insult to the paracentral fibers may lead to the clinical triad seen in both diseases.

While neither subarachnoid hemorrhage nor meningitis is likely to be causal in most cases of NPH, there is both pathologic [23–28] and MR evidence that meningeal irritation may be associated in some cases. Meningeal enhancement was noted after administration of gadopentetate dimeglumine in the patient with mild DWMI and communicating hydrocephalus illustrated in Figure 3. Since such benign meningeal enhancement is normally seen only after surgery [29] or
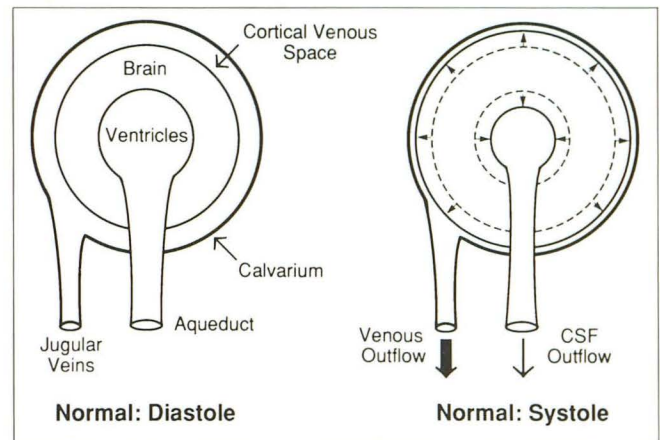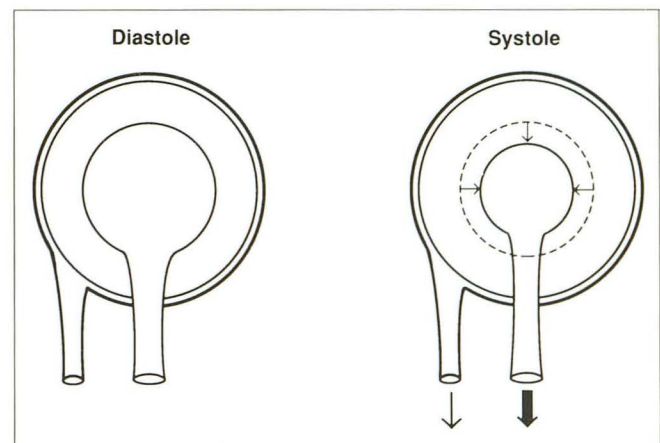
Fig. 5.—Schematic representation of systolic cerebral expansion in normal patients and those with communicating hydrocephalus.

A, In normal patients, systolic expansion of cerebral hemisphere occurs outward, compressing cortical veins and, to a lesser extent, inward, compressing lateral ventricles.

B, In communicating hydrocephalus, the brain has expanded against the inner table of the calvaria, compressing cortical veins. Since outward expansion of the brain is no longer possible, the entire increase in volume due to systolic expansion is directed inward, toward lateral ventricles. The combination of this greater inward displacement and mild aqueductal enlargement that accompanies communicating hydrocephalus leads to hyperdynamic CSF flow state.

following subarachnoid hemorrhage or meningitis, this indicates that meningeal irritation was present, possibly due to subclinical subarachnoid hemorrhage or meningitis.

Now consider possibilities 3 and 4. That DWMI might lead to hydrocephalus was first suggested by O'Connell [30] in 1943. Subsequently, a large body of experimental work has been performed to support the hypothesis that periventricular brain injury can lead to decreased periventricular tensile strength and subsequent ventricular dilatation [31–33]. While this was previously thought to be due to intraventricular CSF pulsations [31–33], in fact, it may result from inward expansion of the brain during cardiac systole [15].

In the normal brain, the intraventricular CSF pressure rise during systole reflects arterial inflow followed quickly by venting of blood from the cortical veins and venting of CSF from the convexity subarachnoid space and the lateral and third ventricles through the aqueduct [34, 35]. During systole, the brain normally expands outward (compressing cortical veins and convexity subarachnoid space) and, to a lesser extent, inward (compressing the lateral ventricles) (Fig. 5A). Inward motion of the lateral ventricles produces the CSF flow void in normal individuals. Should a significant percentage of the spin echoes be acquired during systole (i.e., systolic pseudogating), the flow void would be increased, even in normal individuals. Thus, an increased CSF flow void should not be used by itself as evidence of communicating hydrocephalus.

In early communicating hydrocephalus, the ventricles eventually expand such that the convexity subarachnoid space

and the cortical veins are compressed. Since late systolic venting of venous blood is no longer possible, there is greater inward systolic motion of the lateral ventricles, the intraventricular CSF pulse pressure rises, and the CSF flow void is increased (Fig. 5B) [15]. As postulated by Hakim et al. [15], the resulting tangential periventricular shearing forces may then lead to further ventricular enlargement and the symptoms of NPH.

Whether DWMI causes NPH or vice versa, arteriolosclerosis of the long penetrating arteries is likely to be involved. This may explain why both NPH and DWMI are diseases of elderly patients [36]. In younger patients with severe multiple sclerosis, periventricular demyelination may also lead to decreased tensile strength and progressive ventricular enlargement in the pattern of communicating hydrocephalus. As long as cerebral blood flow is preserved, the hyperdynamic CSF flow state may result, as shown in Figure 4. Thus, ventricular enlargement in patients with multiple sclerosis may not be purely central atrophy.

Just as the neuroclinician's pressure transducer is sensitive to the bounding CSF pulse pressures, MR is sensitive to CSF flow [13–15]. Aqueductal CSF velocities six to eight times normal have been documented by using velocity-sensitive MR techniques in patients with NPH [14]. As long as the CSF flow void is present, therefore, these patients must have enough cerebral blood flow remaining to power the pulsatile motion of CSF [15, 37]. Shunting at this stage of the disease would be expected to lead to resolution (or partial resolution)
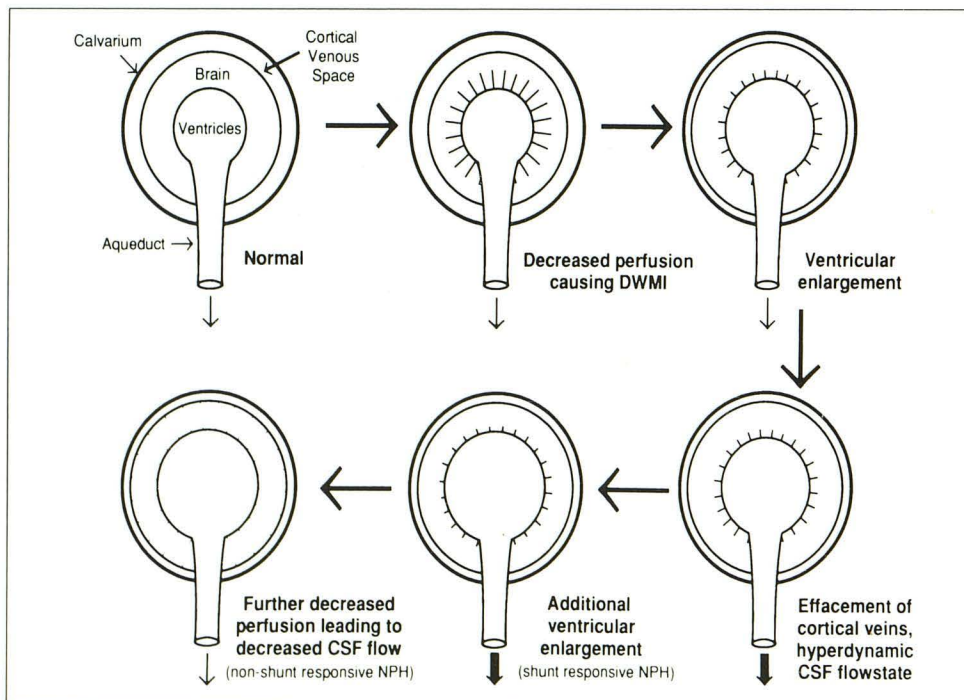

Fig. 6.—Proposed mechanism by which deep white matter infarction (DWMI) could lead to normal-pressure hydrocephalus (NPH). In the normal state, systolic expansion of cerebral hemispheres occurs outward and inward. Outward systolic expansion effaces cortical veins, leading to venous outflow; expansion inward compresses third and lateral ventricles with resultant outflow of CSF through the aqueduct. Initial decrease in deep cerebral perfusion leads to DWMI. The resulting decrease in periventricular tensile strength leads to ventricular enlargement from tangential shearing forces. As the ventricles expand, the cortical veins are effaced. Systolic expansion of the cerebral hemisphere is now directed inward, compressing third and lateral ventricles with secondary increased flow of CSF through aqueduct during systole. This produces the hyperdynamic CSF flow state that leads to additional periventricular motion, greater shearing, and additional ventricular enlargement. At this time, the patient is still shunt-responsive. Subsequent further decrease in blood supply to the brain leads to decrease in systolic expansion of cerebral hemisphere with secondary decrease in the outward propulsion of CSF during systole and, hence, a decrease in the CSF flow void. The decrease in total brain perfusion is indicative of atrophy. The resulting decreased CSF flow void indicates that the patient is not likely to respond to a ventriculoperitoneal shunt.

of symptoms. Subsequent decrease (or partial decrease) in cerebral blood flow [38] would result in decreasing inward systolic expansion of the cerebral hemispheres with resultant decreased pulsatile motion of CSF through the aqueduct and consequent decrease in the flow void (Fig. 6). Thus, an absent or "normal" CSF flow void in the setting of chronic communicating hydrocephalus probably indicates decreased cerebral blood flow and concomitant central atrophy [15]. Such patients are less likely to benefit from ventriculoperitoneal shunting. On the other hand, the presence of DWMI should not be considered a contraindication to ventriculoperitoneal shunting in patients with suspected NPH as it may be the cause in a certain number of cases. While DWMI may appear to be extensive, it should be emphasized that areas of hyperintensity do not represent nonfunctional brain [8], and, if a marked aqueductal flow void [15] is present, these patients should be considered for CSF diversionary procedures.

ACKNOWLEDGMENTS

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