Clinical manifestations of hydrocephalus caused by impingement of the corpus callosum on the falx: an MR study in 40 patients.

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Clinical Manifestations of Hydrocephalus Caused by Impingement of the Corpus Callosum on the Falx: An MR Study in 40 Patients

J. R. Jinkins

The clinical features of patients with hydrocephalus include generalized reductions in coordinated motor and cognitive functions. Although some group similarities have been noted, the outward manifestations of this dysfunction vary in degree and character, with some subjects revealing no overt signs of the underlying hydrocephalus. A retrospective review of subjects with MR criteria of hydrocephalus was undertaken to reevaluate the specific imaging correlates of the signs and symptoms associated with this pathologic process. Forty adults with hydrocephalus on MR evaluation were carefully scrutinized in an effort to elucidate specific clinicoradiologic patterns of abnormality. Spin-echo MR techniques were used with T1 and/or T2 weighting in three orthogonal planes. MR criteria of hydrocephalus encompassed dilated lateral ventricles to include the temporal horns, a pronounced upward elevation of the corpus callosum, and an outward expansion of the cerebral hemispheres at the expense of the subarachnoid space overlying the convexities. The significant related morphologic change on MR that has not been previously described in hydrocephalus was a localized dorsal flattening and thinning of the posterior body of the corpus callosum. Importantly, all but three of the 24 patients with this phenomenon manifested varying combinations of imbalance, gait disturbance, incontinence, short-term memory deficits, and global dementia. In the presence of hydrocephalus, but in the absence of this specific callosal configuration, only one of the remaining 16 subjects revealed symptoms that might suggest the presence of hydrocephalus (i.e., profound dementia). The structure responsible for this focal callosal flattening and thinning in hydrocephalus is the rigid free surface of the falx cerebri as it impinges on the caudal extent of the upwardly expanding corpus callosum and supracallosal hippocampal formation. This mechanical insult hypothetically causes variable axonal dysfunction, ranging from decreased to increased neurophysiologic activity.

In summary, it is postulated that callosal impingement represents a dynamic partial hemispheric disconnection and accounts, in part, for the complex clinical state associated with hydrocephalus.


A clinical evaluation of a group of patients with hydrocephalus as judged by MR has revealed that some individuals demonstrate gait disturbance, memory defects, and incontinence, while others do not. Perhaps in the former group, there exists an as yet undisclosed specific pathologic insult in addition to the global changes associated with ventricular dilatation with or without increased intracranial pressure. A retrospective review of subjects with MR criteria of hydrocephalus was undertaken to seek discrete abnormalities that might indicate clues to the pathophysiologic mechanisms underlying symptomatic hydrocephalus.

Materials and Methods

Forty adults with MR evidence of hydrocephalus were studied further in an effort to elucidate specific patterns of abnormality. Spin-echo MR techniques were performed with
T1-weighted, 500–600/20–40/2 (TR/TE/excitations), and/or T2-weighted double-echo (2000–3000/30–80/1–2) acquisitions in two or three orthogonal planes. MR criteria of hydrocephalus included dilated lateral ventricles, an upwardly elevated corpus callosum, and outwardly expanded cerebral hemispheres. These accepted radiologic standards of hydrocephalus are detailed elsewhere [1, 2]. Each case in the present study had to demonstrate all of these criteria in order to be included. These findings were believed to effectively discriminate between simple deep cerebral atrophy and ventricular dilatation on the basis of hydrocephalus. Two of the subjects in this series were given IV gadopentetate dimeglumine as an adjunct to the MR study for clinical reasons other than suspected hydrocephalus. A review of 10 normal enhanced studies was performed for purposes of comparison. All patient charts were subsequently reviewed in order to tabulate the major clinical signs and symptoms in each subject. The evaluation was limited to presenting symptomatology and to gross reductions in motor and cognitive function. While still undergoing a watchful period of conservative management, it is believed that none of the subjects in this series had undergone ventricular shunting at the time of publication.

**Results**

Among the 40 patients with ventricular enlargement, 10 subjects had varying degrees of imbalance, 12 had gait disturbance, 12 had mild memory deficits, three had profound dementia, three exhibited psychopathologic behavior, and one had urinary incontinence (Table 1).

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Clinical Complex</th>
<th>Impingement</th>
<th>Other MR Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23 F</td>
<td></td>
<td>Headache, nausea &amp; vomiting</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>40 F</td>
<td></td>
<td>Imbalance, gait disturbance</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>52 F</td>
<td></td>
<td>Memory loss, seizure, headache, nausea &amp; vomiting</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>53 M</td>
<td></td>
<td>Headache, blurred vision</td>
<td>Not seen</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>55 M</td>
<td></td>
<td>Memory loss</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>58 F</td>
<td></td>
<td>Vertigo, headache, memory loss, gait apraxia</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>59 F</td>
<td></td>
<td>Acute weakness, R leg</td>
<td>Not seen</td>
<td>DWMI, cerebellar infarcts</td>
</tr>
<tr>
<td>8</td>
<td>62 F</td>
<td></td>
<td>Memory loss, gait disturbance</td>
<td>Mild</td>
<td>DWMI, R basal ganglia infarcts</td>
</tr>
<tr>
<td>9</td>
<td>64 M</td>
<td></td>
<td>Gait disturbance</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>64 M</td>
<td></td>
<td>Headache</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>65 F</td>
<td></td>
<td>Gait disturbance, vertigo, memory loss</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>65 F</td>
<td></td>
<td>Memory loss, anxiety</td>
<td>Moderate</td>
<td>DWMI, scattered lacunar infarcts</td>
</tr>
<tr>
<td>13</td>
<td>66 F</td>
<td></td>
<td>Vertigo</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>67 M</td>
<td></td>
<td>Headache</td>
<td>Severe</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>68 M</td>
<td></td>
<td>Gait disturbance, memory loss</td>
<td>Mild</td>
<td>DWMI, R pontine infarcts</td>
</tr>
<tr>
<td>16</td>
<td>69 F</td>
<td></td>
<td>Imbalance</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>69 F</td>
<td></td>
<td>Memory loss, gait apraxia, anxiety</td>
<td>Severe</td>
<td>Atrophy</td>
</tr>
<tr>
<td>18</td>
<td>69 F</td>
<td></td>
<td>Headache, memory loss</td>
<td>Moderate</td>
<td>Atrophy</td>
</tr>
<tr>
<td>19</td>
<td>70 F</td>
<td></td>
<td>Dementia, gait disturbance</td>
<td>Mild</td>
<td>DWMI</td>
</tr>
<tr>
<td>20</td>
<td>70 F</td>
<td></td>
<td>Syncopal episodes</td>
<td>Not seen</td>
<td>Atrophy, DWMI</td>
</tr>
<tr>
<td>21</td>
<td>71 M</td>
<td></td>
<td>Memory loss, headache</td>
<td>Mild</td>
<td>Atrophy</td>
</tr>
<tr>
<td>22</td>
<td>71 F</td>
<td></td>
<td>R-sided weakness</td>
<td>Not seen</td>
<td>DWMI, scattered lacunar infarcts</td>
</tr>
<tr>
<td>23</td>
<td>72 M</td>
<td></td>
<td>Parkinsonian gait</td>
<td>Mild</td>
<td>Atrophy</td>
</tr>
<tr>
<td>24</td>
<td>73 M</td>
<td></td>
<td>Seizure</td>
<td>Not seen</td>
<td>DWMI</td>
</tr>
<tr>
<td>25</td>
<td>73 F</td>
<td></td>
<td>Syncopal episodes</td>
<td>Not seen</td>
<td>Atrophy</td>
</tr>
<tr>
<td>26</td>
<td>73 M</td>
<td></td>
<td>Headache</td>
<td>Not seen</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>74 M</td>
<td></td>
<td>Visual disturbance</td>
<td>Not seen</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>74 M</td>
<td></td>
<td>Parkinsonian gait, ataxia, mild memory loss</td>
<td>Not seen</td>
<td>DWMI, R hemisphere infarcts</td>
</tr>
<tr>
<td>29</td>
<td>75 M</td>
<td></td>
<td>Memory loss, gait disturbance, confusion, imbalance</td>
<td>Mild</td>
<td>DWMI, atrophy</td>
</tr>
<tr>
<td>30</td>
<td>75 M</td>
<td></td>
<td>Dementia</td>
<td>Not seen</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>75 F</td>
<td></td>
<td>Headaches</td>
<td>Not seen</td>
<td></td>
</tr>
<tr>
<td>32</td>
<td>76 M</td>
<td></td>
<td>L-sided weakness</td>
<td>Not seen</td>
<td>DWMI, R hemisphere infarcts</td>
</tr>
<tr>
<td>33</td>
<td>76 F</td>
<td></td>
<td>L-sided weakness, progressive mild memory loss, gait disturbance</td>
<td>Mild</td>
<td>R hemisphere infarcts</td>
</tr>
<tr>
<td>34</td>
<td>79 M</td>
<td></td>
<td>Visual impairment, tremor</td>
<td>Not seen</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>81 M</td>
<td></td>
<td>Dizziness, memory loss, gait disturbance, incontinence</td>
<td>Moderate</td>
<td>DWMI, atrophy</td>
</tr>
<tr>
<td>36</td>
<td>82 F</td>
<td></td>
<td>Ataxia</td>
<td>Mild</td>
<td>DWMI</td>
</tr>
<tr>
<td>37</td>
<td>83 M</td>
<td></td>
<td>L-sided weakness</td>
<td>Not seen</td>
<td>R hemisphere infarcts, DWMI</td>
</tr>
<tr>
<td>38</td>
<td>85 M</td>
<td></td>
<td>R-sided weakness</td>
<td>Not seen</td>
<td>L hemisphere infarcts, atrophy</td>
</tr>
<tr>
<td>39</td>
<td>86 F</td>
<td></td>
<td>Dementia</td>
<td>Moderate</td>
<td>Atrophy</td>
</tr>
<tr>
<td>40</td>
<td>91 M</td>
<td></td>
<td>Anxiety, paranoia</td>
<td>Not seen</td>
<td>Atrophy, DWMI</td>
</tr>
</tbody>
</table>

Note.—R = right; L = left; DWMI = deep white matter ischemic gliosis.
The significant related morphologic change observed in the majority of these patients on MR that has not been previously described in hydrocephalus was a dorsal flattening and/or thinning of the posterior body of the corpus callosum (Figs. 1 and 2). The severity of this configurational abnormality was graded as mild (slight dorsal flattening), moderate (associated mild thinning of the posterior callosum), or severe (marked thinning of the mid and posterior callosal body). All 24 subjects exhibiting this morphologic change were categorized as group 1. Importantly, all but three of the 24 individuals (87.5%) in group 1 demonstrated imbalance, gait disturbance, mild memory deficits or dementia, incontinence, and/or psychopathologic behavior.

Sixteen additional subjects with ventricular dilatation did not demonstrate this callosal abnormality and were categorized as group 2. Most of these patients presented with other nonspecific symptoms, some of which may be related to increased intracranial pressure, but not to hydrocephalus per se (e.g., headache, visual impairment, sudden paresis, syncope, seizure). Only one of the patients in group 2 revealed symptoms that might suggest hydrocephalus (i.e., dementia). All of the individuals in group 2 showed a single, uninterrupted curvilinear upward smooth expansion of the entire corpus callosum without focal thickening (Fig. 3).

One of the subjects with focal dorsal callosal thinning was imaged in the coronal plane with T2-weighted acquisitions for the purpose of evaluating clinically suspected temporal lobe seizures. Focal high intensity within the corpus callosum was identified at the point of thinning, but not elsewhere within the commissural white matter (Fig. 4).

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**Fig. 1.**—Moderate callosal impingement in hydrocephalus. Sagittal T1-weighted section shows mild curvilinear flattening of dorsal surface of posterior body of corpus callosum (arrow).

**Fig. 2.**—Marked callosal impingement in hydrocephalus. A, Sagittal T1-weighted section shows marked flattening and thinning of posterior body of corpus callosum (arrows). B, Coronal T1-weighted image shows stretching of fibers of corpus callosum over posterior falx with associated extreme thinning and focal loss of callosal signal (arrow).

**Fig. 3.**—Hydrocephalus without callosal impingement. Sagittal image shows smooth upward expansion of corpus callosum without focal caudal thinning or flattening of its dorsal surface.

**Fig. 4.**—Edema and/or gliosis of corpus callosum in callosal impingement. A, Coronal intermediate-weighted image reveals transcallosal high intensity at point of impingement (arrow). B, T2-weighted coronal section further illustrates focal increase in intensity within posterior corpus callosum, indicating edema and/or gliosis (arrow).
In one of the two subjects with hydrocephalus and focal dorsal callosal thinning who was given IV gadopentetate dimeglumine as a part of a negative workup for possible neoplastic disease, focal enhancement of the corpus callosum at and near the point of callosal thinning was seen without evidence of abnormal cerebral enhancement elsewhere (Fig. 5). The ancillary review of 10 normal subjects with enhanced cranial MR studies demonstrated no such callosal enhancement on coronal imaging.

Discussion

The clinical signs and symptoms associated with uncomplicated hydrocephalus, regardless of cause or intracranial pressure considerations, are complex in their individual manifestations [3-6]. In addition, published reports indicate that the initial symptoms correlate poorly with ventricular size, and no consistent relationship between a reduction in ventricular volume following shunting and clinical improvement has been determined [7, 8]. Therefore, some factor other than absolute ventricular size must be responsible for the primary syndrome as well as for the observed positive effect of shunting in certain subjects [7, 8].

In many patients with hydrocephalus there is a generalized, temporally progressive reduction in coordinated motor and higher cognitive functions that correlates with forms of the acute cerebral hemispheric disconnection syndromes [3, 9-15]. This was seen in the present series in 87.5% of the 24 patients with dorsal flattening and/or thinning of the posterior body of the corpus callosum (group 1) and was manifested chiefly by imbalance, gait disturbance, and amnesic states. The structure physically responsible for this phenomenon is the rigid free surface of the falx cerebri in the sagittal midline. With upward expansion of the corpus callosum secondary to dilatation of the third and lateral ventricles in hydrocephalus, the falx first impinges on the outermost dorsal layer of the corpus callosum. As the falx is most complete posteriorly, and therefore extends farther ventrally in its posterior sector, the caudal extent of the corpus callosum naturally will be the most severely impinged on initially. Individual variations among patients in the embryologic completeness of the falx occasionally can be outlined in normal subjects owing to the flow-related void (or the paramagnetic contrast enhancement of blood) within the inferior sagittal sinus lying in or near the

Fig. 5.—Blood-brain barrier breakdown in callosal impingement.
A, Unenhanced T1-weighted coronal image immediately anterior to point of maximal callosal thinning.
B, Enhanced T1-weighted image shows vertical transcortical enhancement (straight arrow), indicating a focal disruption of blood-brain barrier within corpus callosum. Punctate enhancement within inferior sagittal sinus (curved arrow).

Fig. 6.—Normal appearances of inferior sagittal sinus running in edge of falx cerebri.
A, Normal enhancement within inferior sagittal sinus (arrow), illustrating a “high” or relatively developmentally incomplete falx.
B, More ventrally placed venous enhancement (arrow) in another normal subject indicates a “low” or relatively more developmentally complete falx.
The free edge of the falx cerebri (Fig. 6). The more completely ventrally developed falces obviously will induce callosal impingement earlier, and these callosi consequently may be more extensively affected relative to the degree of hydrocephalus.

The majority of the 16 subjects in group 2 did not have symptoms suggestive of hydrocephalus (i.e., no imbalance, gait disturbance, amnesia, dementia, or incontinence). It is important to appreciate that the corpus callosum in the group 2 individuals in this series demonstrated a smooth upward semicircular expansion without any degree of caudal flattening or focal thinning (i.e., no callosal impingement) (Fig. 3). These cases fall within a grouping that should be referred to as clinically occult hydrocephalus. This is not surprising as it is known that a certain rate of hydrocephalus is revealed in any review of normal age-matched controls [4, 10, 15-17].

However, the central question concerns the actual mechanism for the production of classical signs and symptoms of hydrocephalus in group 1 subjects with callosal impingement. The major objective and subjective complaints are best scrutinized separately. The most frequently encountered symptoms overall in group 1 were general imbalance (nine patients, 37.5%) and gait disturbance (12 patients, 50%). Some authors consider normal balance to be an integral part of posture and gait [4, 18]. If looked at this way, gait disturbance becomes the most common overall complaint. The fact that stance and gait are impaired yet motor strength is intact indicates that the focus of the abnormality is premotor rather than in the primary motor area [4].

Some of this symptomatic imbalance obviously may relate to a disturbed vestibular system. The vestibular cortex in humans is believed to be divided into multiple areas, some of which may be primary while others are associationals [19-22]. It is further known that input from the peripheral proprioceptive, visual, and auditory areas converges within the vestibular cortex [23-25]. In this way, the vestibular cortex is involved with much of the conscious perception of our position in space, posture, and righting reflexes. Importantly, both the posterior parietal and superior temporal regions containing these vestibular system related cortices transmit fibers through the posterior body of the corpus callosum, coincidently also the area of maximal callosal impingement (Fig. 7) [23].

Furthermore, the literature points toward the parkinsonian nature of the gait disturbance in hydrocephalus [26-28]. These observations become significant in callosal impingement because of the existence of a callosally mediated crossed corticostriate projection (motor cortex to basal ganglia) [29-32]. Owing to the compression of crossed corticostriate fibers in the impinged corpus callosum, varying degrees of axonal dysfunction may result in the positive and negative signs classically associated with extrapyramidal disease [33].

As a final consideration of the origin of the gait disorder, it is important to note that the rostral section of the corpus callosum causes the disruption of few tasks that require the interhemispheric integration of somatosensory information with motor control. However, the posterior one third of the corpus callosum, invariably involved in callosal impingement, contains commissural fibers that are essential to this integration in order to achieve united, controlled, coordinated bilateral motor performance in the normal individual [34].

Memory disturbances (short-term memory loss, profound dementia) constituted the second most common category of symptoms in group 1, occurring in 15 (62.5%) of 24 patients. As originally described, the dementia of hydrocephalus was in fact only a minor defect in short-term memory [4, 6, 35]. This condition may, however, progress over time to encompass a more severe form of global amnesia. It is known that the hippocampal formation is involved with the neural processing of memory. Lesions, particularly bilaterally, of the hippocampal formation produce a profound impairment of memory for recent events. More specifically, ongoing events in such circumstances cannot be retained for more than seconds or minutes, and cannot be converted into long-term memory storage. Memory for remote events prior to the insult usually is unaffected, and thus there seems to be no amnesia for these past engrams. Intellectual ability in general remains rather high, but the individual is incapable of assimilating new information.

Embryologically, the corpus callosum originates rostrally within the telencephalon and grows caudally. As it does so, it invaginates into the hippocampal primordium [36-38]. The result is that the dorsal surface of the corpus callosum is covered by the supracallosal component of the hippocampal formation, comprising the indusium griseum (gray matter extension of the dentate gyrus of the temporal lobe component of the hippocampal formation) and the medial and lateral striae lancelis (longitudinal association/projection white matter bundles of the hippocampal formation). These latter contain, in addition to other fibers, the supracallosal equivalents of the subcallosal fornices, which are the major efferent pathway of the hippocampal formation. Therefore, they are integral extensions of the hippocampal formation proper. Many of these supracallosal longitudinal fibers of the hippocampal formation eventually dive into the substance of the corpus callosum,
penetrate it, and subsequently join the main body of the subcallosal fornical fibers. Mingling diffusely with the pre- and postcommissural components of the subcallosal fornix, these fibers transmit to the medial and lateral preoptic areas, the medial mammillary nuclei, the superficial gyrus rectus, and the paraolfactory area [39]. It is plausible that impingement of the supracallosal fornix may be of significance in the genesis of the memory deficits associated with hydrocephalus [40–42]. In clinical situations, bilateral section of the hippocampal fornices causes acute disorientation and amnesia in the short term [43]. In addition, specific damage to the posterior fornices is especially prone to amnesic effects [44].

As a rule, any interference within a neural circuit causes a disruption of the proper functioning of that circuit. This has been experimentally and clinically demonstrated in regard to the laying down of memory traces [45–48]. Axonal dysfunction at the level of the supracallosal hippocampal formation might provide a sufficient degree of neurophysiologic interference within the complex network involved in memory processing and storage. In addition, homologous regions of the temporal lobes are also connected via true callosal commissural axons that pass through the presplenial posterior callosal body (Fig. 7). Therefore, impingement of axons directly linking the temporal lobes might substantially contribute to deficits in mnemonic function. Finally, the posterior parietal cortex has also been implicated in the generation of certain amnesic states [49]. Thus, the impingement of posterior parietal commissural fibers, which pass through the posterior body of the corpus callosum, may cause mnemonic deficits by similar axonal dysfunction.

That the mechanism of callosal impingement is an active injury in some individuals at the time of imaging seems to be confirmed in two of the subjects in the present study. Focal high intensity of the corpus callosum on T2-weighted images and enhancement with gadopentetate dimeglumine at and high intensity of the corpus callosum on T2-weighted images also have been experimentally and enhancement with gadopentetate dimeglumine at and high intensity of the corpus callosum on T2-weighted images been experimentally and enhancement with gadopentetate dimeglumine at and high intensity of the corpus callosum on T2-weighted images in patients with hydrocephalus [50]. Such focal of ectopic impulse generation have in fact been identified experimentally within chronically compressed axons [51].

Intermittent, sudden temporal pressure applied to axons of the corpus callosum would be the equivalent of periodic microtrauma, which is known to produce temporary paresthesias in the peripheral nervous system. This conceivably could be effected by the pulsatile expansion of the brain during arterial systole, causing the corpus callosum to periodically collide with or otherwise further impinge on the free edge of the falx cerebri. In addition, chronic pressure is known to induce extreme neuroelectrical mechanosensitivity in axonal structures, which would further enhance the effects of long-term callosal impingement but which might not be a major factor in acute cases of hydrocephalus with callosal impingement [51].

However, in order to be credited, the largely hypothetical evidence of a radiating callosal commissuropathy and its proposed effects requires electrophysiologic confirmation. Experimentally, direct electrical stimulation of the corpus callosum produces so-called "callosal potentials." These potentials reveal inhibitory and facilitory actions on the neuroelectric activity within the interconnected cortical neuronal field served by the stimulated callosal axons [52, 53]. The effect of such aberrant callosal activity on otherwise normal cortical function can thus be seen to be potentially significant.

Direct human evidence of abnormal neuroelectrical potentials is found in the literature that reviews the electroencephalographic (EEG) abnormalities in patients with hydrocephalus [15, 54–56]. Interestingly, these subjects exhibit discharges that are described as paroxysmal bursts of projected electrical activity [15, 54–56]. These EEG bursts are believed to be transmitted between bilateral homologous hemispheric regions, but up to the present time the source of these discharges has been unknown. Moreover, the EEG changes have been experimentally proved to be due to more than just simple increased intracranial pressure. Such EEG abnormalities are seen in approximately 50% of patients with hydrocephalus, which is consistent with the observed prevalence in the present series of callosal impingement in hydrocephalus [57]. These aberrant discharges are continuous or irregularly intermittent/periodic, diffuse or focal, and usually bilaterally asymmetric. In many individuals, the symptom complex associated with hydrocephalus fluctuates unduly from day to day and week to week [3, 4]. This fits with the electrophysiologic data of similar temporal fluctuations. These pathologic electrical potentials have been seen to largely disappear after days or weeks with resolution of the hydrocephalus. The improvement in the EEG pattern also parallels clinical improvement. It has even been suggested that the EEG be used as a monitor of when to shunt the cerebral ventricles and as a means of evaluating resolution of cerebral electrophysiologic abnormalities [15]. Nevertheless, there is frequently a time lag between shunting and resolution of symptoms, which would seem to indicate a slow healing of the primary neural insult following the removal of the noxious influence (i.e., callosal impingement). This EEG concept of irregular bursts of asymmetric bilateral discharges of aberrant electrical
activity fits well with a theory of a radiating callosal commissuropathy.

Experimentally, it has been observed that after a nonspecific reversible axonal insult, both prolonged and exaggerated, mechanically sensitive hyperexcitability is noted in both motor and sensory fibers of peripheral nerves [58–63]. It is quite likely that a similar response occurs in CNS axons in subjects with callosal impingement. In fact, experimental injury of CNS axons does result in spontaneous bursts of activity as well as amplitude-enhanced trains of action potentials in response to simple mechanical deformation [51, 59]. These impulses are bidirectional within axons, propagating away from the site of dynamic mechanical perturbation and/or frank injury.

A commissuropathy and perhaps an associated hippocampal fornixopathy thus seems to represent an electrophysiologic response to chronic mechanical compression of commissural and fornical axons by the falx cerebri and the attendant pathophysiologic sequelae of this insult. Physical factors believed to be responsible for this remarkable degree and quality of proposed abnormal ectopic electrical activity include: (1) the excellent contact of the insulting factor (falx cerebri) directly with commissural and supracommissural fibers of the hippocampal formation; (2) the compact segregation of commissural/fornical fibers, which should produce an enhanced, synchronized impulse volley with little temporal dispersion; and (3) the reduced distance of aberrant conduction, and therefore reduced conduction time, owing to midaxonal injury (as opposed to the greater conduction time over the normal complete interhemispheric axon length) [52]. On a basic pathophysiologic level, chronic axon compression leads to an increase in the sodium channel density within the axonal membrane, which results in spontaneous neuroelectric impulse generation [51]. The specific clinical manifestation of a projected commissuroopathy should depend on the individual callosal axons involved in the impingement, the qualitative and quantitative aspects of the induced electrophysiologic dysfunction, and the distribution of the respective callosal neurons within the corresponding hemispheric cortical and subcortical gray matter.

The relative infrequency of incontinence in this series and in others reported in the literature likely indicates the acute, mild, or perhaps arrested nature of the hydrocephalus in the majority of these patients. In many published cases, incontinence seems to be an end stage or at least a very late event in the natural history of the hydrocephalus [6]. In this regard it should be noted that even decerebrate animals have automatic urination, which seems to be driven under such circumstances by centers in the brainstem and spinal cord [64]. Alternatively, a type of global apraxia may occur whereby, because of the extreme binehemispheric neuroelectrical interference, the patient simply forgets how and when to urinate. In any case, the result would be a loss of control over urine and occasionally bowel contents.

The psychiatric aspects, peripheral sensory complaints, and seizure activity observed in some individuals with hydrocephalus may be related to the pathophysiologic mechanisms of callosal impingement, but will not be further elaborated here as they are largely speculative [29, 65–70]. Other symptoms common to hydrocephalus such as headache and visual impairment, on the other hand, are more probably caused by the direct effects of raised intracranial pressure and not by callosal impingement.

To a point, these processes are reversible. However, following irreversible callosal axonal injury, altered cortical neuronal projection fields secondary to the chronic radiating axonopathy, and/or further pathologic change of the cerebrum due to the supplemental direct damaging effects of prolonged hydrocephalus on the cerebral parenchyma, the clinical syndrome may not be correctable. The predictability of this reversibility remains obscure. On average, 40–60% of subjects with cerebral ventricular shunts do not improve substantially [71].

In this regard, the other effects of hydrocephalus on the cerebrum that likely have a clinical counterpart must not be overlooked in any evaluation of hydrocephalus and its related symptomatology. In addition to the factors outlined above, long-standing globally reduced cerebral blood flow, prolonged alteration of neuronal metabolism, and reactive periventricular gliosis sometimes associated with hydrocephalus may partially explain the clinical status as well as the failure of clinical success following ventricular shunting in certain individuals [72–75]. Actual destruction of cerebral neurons of the cortical mantle, however, does not by contrast seem to be a major factor in the pathologic process [74].

The superimposition of other disease processes, such as Alzheimer or chronic hypertensive/multinfarct dementia, must also be considered as a potential source of symptoms [17, 76]. All three major signs of hydrocephalus (gait disturbance, dementia, and incontinence) are seen in the older population but can be unrelated to hydrocephalus [77]. Therefore, there is bound to be some overlap of preexistent symptoms with similar symptoms attributable directly to the acquired hydrocephalus.

It seems, then, that the treatment of hydrocephalus fails because of preexisting primary or acquired degenerative brain disease and/or concomitant irreversible parenchymal brain injury induced by long-standing hydrocephalus [78]. Failures may thus result from attempting treatment too late in the disease process. In fact, patients who do not respond to measures aimed at treating the hydrocephalus may already be maximally compensated at the time therapy is initiated [78].

One final theory concerning failure of therapy must be further elaborated. In some individuals, irreversible surrounding parenchymal brain damage may occur owing to the direct effects of progressive, chronic ventricular dilatation. This will thereby result in continued ventricular enlargement, even subsequent to effective shunting, because the dilatation may be secondary to the residual atrophy and not necessarily to persistently elevated pressure [79]. Therefore, without the resolution of ventricular dilatation, callosal impingement must also still obtain in these subjects attended by all of the proposed clinical effects, even in the face of normal CSF pressure measurements. Shunting in such cases may thus be ineffective, because the pathologic focus of the origin of much of the signs and symptoms (i.e., callosal impingement) has not been remedied.

Degrees of partial hemispheric disconnection are seen to
exist in hydrocephalus clinically. Although not performed in this series, careful patient examination with specialized techniques designed to reveal this active, acute disconnection may be helpful in the future to more precisely assess the neurologic status and prognosis of subjects with hydrocephalus and suspected callosal impingement. New approaches to therapy taking the combined clinical, EEG, and MR features of callosal impingement into account may warrant consideration.

It is of interest that none of the subjects in this study were shunted at the time of publication. At this institution a conservative attitude has been adopted owing to the current unpredictability of ventricular shunting in terms of the probable resolution of signs and symptoms and because of the relative risk of shunt malfunction. Therefore, watchful supportive therapy has been used, and shunting is considered only for rapidly progressive deterioration in motor or cognitive functions.

A careful, critical evaluation of signs and symptoms, which can only be adequately provided by a prospective clinical study, was not feasible in this retrospective MR study of hydrocephalus. In the future, such a corroborative effort will be essential to further elucidate the significance of callosal impingement. In addition, subsequent neurophysiologic study of axonal dysfunction in a laboratory model of callosal impingement will be mandatory for confirmation of the postulated but as yet unproved cerebral commissuropathy.

Summary

The wide range and severity of symptoms associated with hydrocephalus have never been fully accounted for with reference to a specific cause. The linking of many of the significant elements of the clinical syndrome of hydrocephalus with dorsal flattening and thinning of the posterior corpus callosum would seem to implicate this observation to its genesis. The structure responsible for this pathologic change in hydrocephalus is the rigid free surface of the falx cerebri as it impinges on the caudal extent of the upwardly expanding corpus callosum and supracallosal hippocampal formation (Fig. 8). Hypothetically, this mechanical insult results in neuroelectrically active axonal dysfunction. It is postulated that in part this radiating callosal/hippocampal commissuropathy and fornicothalamus in turn yields the nebulous clinical state allied with hydrocephalus owing to a dynamic associational, commissural, and projectional cerebral disconnection. Future clinical and laboratory studies should be directed toward the corroboration of the relationship of callosal impingement to axonal dysfunction and to the confirmation of the role of this dysfunction in the generation of symptoms in patients with hydrocephalus.

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Fig. 8.—Schematic representation of callosal impingement. In callosal impingement of hydrocephalus, there is an upward expansion of corpus callosum (CC) (small arrows) and an associated curvilinear flattening and thinning of caudal aspect of dorsal surface of corpus callosum (large arrows) caused by impinging falx cerebri (F). I = inferior sagittal sinus.

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CALLOSAL IMPINGEMENT IN HYDROCEPHALUS


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**Editor’s Note.**—In the process of categorizing the preceding paper for the Table of Contents, I realized that it did not clearly fall under the heading of a specific anatomic location, disease, or imaging technique. Therefore, I have chosen to list it in the Table of Contents under the heading “Plausible Hypotheses.” According to the *Oxford English Dictionary*, something that is plausible has “an appearance or show of truth, reasonableness, or worth.” By the same authority, a hypothesis is “a supposition or conjecture put forth to account for a known fact; especially in the sciences, a provisional supposition from which to draw conclusions that shall be in accordance with known facts, and which serves as a starting point for further investigation by which it may be proved or disproved and the true theory arrived at.” I believe it is the duty of a scientific journal to occasionally bring articles of this nature to its readers for the purpose of stimulating debate and suggesting new avenues of investigation. This category seems to be an appropriate heading under which to list such articles.