The central canal of the filum terminale in communicating hydrocephalus

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Lumbar thecoperitoneal shunting was carried out in patients with communicating hydrocephalus due to long-standing tuberculous meningitis. At the time of this surgical procedure, the filum terminale was excised to achieve filum terminostomy. The central canal of the excised filum terminale in seven hydrocephalic children and an equal number from control cases was studied histologically. These observations indicate that the central canal of the filum terminale dilates in communicating hydrocephalus, and the dilatation is proportionate to the lateral ventricular enlargement.

Key Words • filum terminale • central canal • hydrocephalus • terminal ventricle

The concept that, in long-standing communicating hydrocephalus, the central canal may serve as an alternative route for resorption of the cerebrospinal fluid (CSF) through multiple communications with the spinal subarachnoid space has been disputed. Several studies based on experimental models have indicated an extensive dilatation of the central canal of the spinal cord and varying degrees of cleft formation in the posterior columns. This change extends to the filum terminale. Further communication is established with the spinal subarachnoid space through the clefts in the ependymal lining. In contrast, another experimental study has shown that the central canal of the spinal cord is of normal size and configuration. To the best of our knowledge, there is no similar study in humans to evaluate and verify these conflicting results. This paper records the results of our study of the central canal of the filum terminale in cases of communicating hydrocephalus.

Clinical Material and Methods

Patients with tuberculous meningitis accompanied by communicating hydrocephalus resemble an experimental hydrocephalic model. We have included in this study eight such patients under 10 years of age (Table 1). None of these patients had previously responded adequately to medical treatment, and all had experienced progressively rising intracranial tension. Contrast ventriculography established a diagnosis of communicating hydrocephalus in all of these patients. Lumbar thecoperitoneal shunting followed by filum terminostomy was carried out as a therapeutic measure. In one case (Case 1), the filum terminale could not be identified; in the other seven, however, it was definitely identified and the filum terminale excised for histological examination.

At operation, the filum terminale appeared grayish white, in contrast to the glistening white nerve roots. When flushed with saline, the nerve roots floated out more easily than the filum. In some cases, the identity of the filum was established by tracing its connection with the conus.

The excised filum was suspended in a bottle containing 10% formalin. After fixation, it was divided into four equal parts and processed for paraffin embedding. Serial sections 6 μ thick were cut and stained with hematoxylin and eosin for the microscopic study. Seven specimens of filum terminale from age-matched patients, who had died as a result of head injury, served as our normal controls.

Results

Examination of the filum terminale in the normal controls showed that the central canal was just a potential space lined by either a single columnar or pseudostratified columnar ependymal cells (Fig. 1). The canal extended through the filum for a variable distance, from one-third to two-thirds of its length.
Central canal of filum terminale

Based on contrast ventriculography, cases were grouped into three categories: 1) Minimal dilatation of the lateral ventricles; on brow-up lateral radiography, the anterior limit of the frontal horn was just at or within the level of the coronal suture. 2) Moderate dilatation of the lateral ventricles: the anterior limit of the frontal horn was less than 1 cm in front of the coronal suture. 3) Marked dilatation of the lateral ventricles: the anterior limit of the frontal horn was more than 1 cm in front of the coronal suture.

We found that the dilatation of the central canal was proportionate to the degree of lateral ventricular enlargement (Table 1). Since the dilated central canals in all the specimens of filum terminale were collapsed after fixation, it was not possible to measure the size of the canal under the microscope. We also found it difficult during surgery to differentiate the filum terminale from the nerve roots of the cauda equina in cases where the lateral ventricles were minimally dilated. In one of the patients who had minimal lateral ventricular dilatation, the filum had a normal-sized central canal (Fig. 2). In the two patients who had moderate ventricular dilatation, the central canal of the filum terminale showed minimal dilatation (Fig. 3), but the clefts were absent. In the four patients who

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Degree of Lateral Ventricle Enlargement</th>
<th>Degree of Central Canal Dilatation</th>
<th>Ependymal Clefts</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>minimal</td>
<td>filum terminale could not be identified</td>
<td>---</td>
</tr>
<tr>
<td>2</td>
<td>minimal</td>
<td>normal central canal</td>
<td>absent</td>
</tr>
<tr>
<td>3</td>
<td>moderate</td>
<td>minimal</td>
<td>absent</td>
</tr>
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<td>4</td>
<td>moderate</td>
<td>minimal</td>
<td>absent</td>
</tr>
<tr>
<td>5</td>
<td>marked</td>
<td>moderate</td>
<td>present</td>
</tr>
<tr>
<td>6</td>
<td>marked</td>
<td>moderate</td>
<td>present</td>
</tr>
<tr>
<td>7</td>
<td>marked</td>
<td>marked (tubercle visualized)</td>
<td>present</td>
</tr>
<tr>
<td>8</td>
<td>marked</td>
<td>marked</td>
<td>present</td>
</tr>
</tbody>
</table>

FIG. 1. Photomicrographs of the central canal of the filum terminale in normal control patients. The photographs are retouched to mark the boundary of the central canal. H & E, × 33. Left: Patient aged 12 years. A blood vessel is seen beside the central canal. Right: Patient aged 9 years. Note the proliferation of the ependymal cells within the lumen.

FIG. 2. Case 2. Photomicrograph of the filum terminale in a 4-year-old child showing a normal-sized central canal. This patient had chronic tuberculous meningitis with minimal ventricular dilatation. H & E, × 30.
had marked ventricular enlargement, the central canal was moderately dilated in two patients (Fig. 4), and in the two others the canal had marked dilatation (Fig. 5). In all of these patients, focal areas of breach in the ependymal lining and cleft formation were observed (Fig. 6). Incidentally, a biopsy of the filum in Case 7 revealed a tubercle.

Discussion

This study supports the concept of the existence of an alternative pathway for resorption of CSF through the central canal, as has been observed in experimental hydrocephalic animals. The factors responsible for the changes in the central canal of the spinal cord in those experimental models are not well understood. McLaurin, et al., postulated two factors, namely, a severe inflammatory response of the spinal leptomeninges, and increased intraventricular pressure, which was caused in their study by kaolin injection into the cisterna magna. They believed that both these factors may be responsible for the dilatation of the central canal of the spinal cord. Becker, et al., confirmed these findings, and suggested that the central canal dilatation depends upon the intraventricular pressure. Where the pressure transmitted from the ventricles was not high, the central canal did not dilate. This may explain the fact that central canal dilatation has not been consistently observed in other studies.

Kasantikul, et al., studied the central canal of the spinal cord in man with and without hydrocephalus. They showed that, in the first two decades of life, the lumen of the central canal was patent in most of the patients. Of the three cases of severe hydrocephalus in patients below the age of 20 years, the canal was dilated in only one case. After the second decade, only 6% of the patients with various degrees of hydrocephalus had a patent central canal, and in others the canal was blocked. Hence, the authors concluded that the central canal of the spinal cord is not an important pathway for CSF absorption in most hydrocephalic patients, and especially not after the age of 20 years.

Bradbury and Latham have suggested that in rabbits there is a natural communication between the central canal and the spinal subarachnoid space, even under normal conditions. Many other species of animals, including higher mammals, have a patent central

Fig. 3. Case 3. Photomicrograph of the filum terminale showing minimal dilatation of the central canal. H & E, × 40.

Fig. 4. Photomicrographs of the filum terminale showing moderate dilatation of the central canal, a breach in the ependymal lining, and cleft formation (arrows). H & E, × 32. Left: Case 5. Right: Case 6.
Central canal of filum terminale

In man, the central canal is only a potential space. The canal can open if and when there is an obstruction to the CSF pathway beyond the fourth ventricular outlets. Through this newly opened central canal, CSF flows directly from the fourth ventricle, through the central canal of the spinal cord, and into the spinal subarachnoid space via the ependymal clefts of the central canal of the filum terminale.

The present study shows a consistent dilatation of the central canal of the filum terminale in cases of long-standing communicating hydrocephalus. Enlargement of the lumen is proportionate to the lateral ventricular dilatation. The changes in the ependymal lining of the canal probably reflect the amount of transmitted intraventricular pressure.

It should be emphasized that at an early stage of hydrocephalus, when the intraventricular pressure is not high, it is mainly the lateral ventricles that dilate, while the rest of the ventricular system is normal. Later, when the intraventricular pressure rises, the alternative resorption of CSF probably takes place through the clefts in the dilated central canal. At the same time, the CSF may reach the subarachnoid space through these newly formed breaches in the ependymal lining. An extreme example of this situation probably is the development of hydromyelia. Hydromyelia is also seen in every carefully studied case of syringomyelia. It is interesting that Gardner, et al., have demonstrated dilatation of the central canal of the filum terminale in all their 12 patients with syringomyelia who were subjected to terminal ventriculostomy. It is important to realize that this newly established pathway is inadequate for the total restoration of normal CSF resorption. Furthermore, it cannot reduce the size of the already dilated lateral ventricles. Hence, a more effective diversion procedure is essential.

Our own observations, although they are based on a
small number of cases, have led us to the conclusion that the central canal of the filum terminale in children with moderate and severe degrees of communicating hydrocephalus is consistently dilated. As a corollary to this, one may anticipate spinal central canal dilatation in all patients in whom the central canal of the filum is dilated. In adult cases of communicating hydrocephalus, however, the central canal of the spinal cord is not patent, so the filum terminale central canal may not be dilated.

Acknowledgments

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