Evaluation of the central canal of the spinal cord in experimentally induced hydrocephalus


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The central canal of the spinal cord has been proposed as a significant compensatory alternative pathway of cerebrospinal fluid (CSF) flow in hydrocephalus. Ten dogs were made hydrocephalic by a relatively atraumatic experimental model that simulates the human circumstance of chronic communicating hydrocephalus. The central canal was studied by histopathology and compared with 10 normal control dogs. In both groups the central canal of the spinal cord was normal in size, configuration, and histological appearance. In this experimental model dilatation of the canal and increased movement of CSF does not appear to be a compensatory alternative pathway.

Key Words • hydrocephalus • spinal cord • central canal • syringomyelia

The alternative pathways of absorption of cerebrospinal fluid (CSF) in patients and animals with hydrocephalus are not known. Some investigators have proposed that CSF is absorbed by the hydrocephalic brain. Others have produced experimental results suggesting that an important alternative pathway is through dilatation of the central canal of the spinal cord and establishment of a communication with the spinal subarachnoid space. The findings have only occurred in hydrocephalic animals following injection with kaolin, which occludes the ventricular outlets. Communicating hydrocephalus in humans usually results from obstruction of peripheral CSF pathways or at the level of the arachnoid villi. The animal model we used approximates the clinical circumstance, and does not cause obstruction of the fourth ventricular outlets. This study was undertaken to examine the central canal of the spinal cord in dogs with experimentally induced communicating hydrocephalus. The gross pathological and histological appearance of the central canals in experimental animals was compared with that in an equal number of normal dogs. We believed that determination of the status of the central canal in this animal model would provide information applicable to the situation encountered clinically in humans.

Materials and Methods

In 10 mongrel dogs, aged from 8 days old to adult, communicating hydrocephalus was
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induced by injection of a silicone rubber* in the subarachnoid space distal to the outlets of the fourth ventricle. This method was developed in our laboratory and has been described in other communications. A variable time was required for the development of hydrocephalus, ranging from 10 days in immature dogs to 45 days in adults. The development of hydrocephalus was demonstrated by cisternography or imaging with computerized tomography (CT).

After hydrocephalus had developed, the animals were sacrificed and examined pathologically. The animals were perfused by left cardiac ventricular cannulation at physiological pressures, with McEwen's saline followed by one-quarter strength and then full strength Karnovsky's fixative. The perfusion lasted 30 to 45 minutes, and required a volume of 2500 ml. The brain and cord were left in situ for an additional 18 hours, and then were removed en bloc and immersed in fixative for several hours. They were then rinsed in 0.05 N cacodylate buffer with 10% sucrose. The brains and spinal cords were weighed, cut in the coronal plane, photographed, processed for conventional histological studies, and stained with hematoxylin and eosin, and Luxol fast blue-cresyl violet. For ultrastructural study, tissue blocks from selected regions of interest were removed after fixation, postfixed in 1% osmium tetroxide in 0.05 N sodium cacodylate with 10% sucrose, and dehydrated and embedded in Epon 812 by standard techniques. Some sections were cut on an ultramicrotome and stained with toluidine blue. Thinner sections were stained with uranyl acetate and lead citrate, and examined by electron microscopy.

An equal number of normal animals were prepared in an identical manner and matched for age at time of sacrifice.

Results

In the control animals, the ventricles in the dogs were clearly identified as spaces. Normal cuboidal epithelium with microvilli and cilia at the ventricular surface were seen. The central canal was of normal size and morphology in all animals, and ranged in appearance from a small patent cylinder to a barely open slit (Fig. 1).

In animals with hydrocephalus, documented by either CT or cisternography, the average brain weight was not substantially different from that of the normal dogs, except in the puppies, in which the development of hydrocephalus was advanced (Fig. 2). The gyri were flattened and the entire ventricular system enlarged. The histological findings

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*Silicone rubber manufactured by Dow Corning Corp., Midland, Michigan.
FJG. 2. Gross specimen of a dog sacrificed at 16 weeks after Silastic implantation. There is generalized enlargement of the ventricular system. The cervical cord of this animal is shown in Fig. 1 right.

have been the subject of previous publications, and will only be summarized here. The ependymal lining of the lateral ventricles was stretched, flattened, and denuded in the area of the dorsolateral angles. Electron microscopic studies showed variations from attenuated ependymal cells with decreased numbers of cilia and microvilli to absence of ependymal cells in the most severely affected regions. In animals with advanced hydrocephalus, the ventricular ependyma had a “cobbled” appearance. The corpus callosum was thinned and the white matter of the corona radiata reduced in volume. In the periventricular white matter, cellular elements were separated by an enlarged extracellular space. This finding was confirmed by electron microscopic studies. Enlarged fibrous astrocytes showed abundant glial filaments, and macrophages were present. In the animals with severe ventricular enlargement, axons were swollen and myelin was

FIG. 3. Photomicrographs of spinal cord. Tissue perfusion fixed with Karnovsky’s paraformaldehyde-glutaraldehyde, embedded in Epon, sectioned at 1.5 μ, and stained with methylene blue. X 160. Upper Left: Cervical cord in a normal dog showing a patent central canal. The ciliary lining is complete and the surrounding parenchymal tissue is tightly arranged. Upper Right: Cervical cord in a dog with acute communicating hydrocephalus. The canal is slit-like in appearance and the surrounding tissue normal. Lower Right: Central canal of cervical cord in a dog with chronic communicating hydrocephalus 16 weeks after Silastic implantation. The canal is of normal size and appearance, and the surrounding gray matter is uniform, and tightly arranged. There is no histological evidence of edema, vacuolation, or tissue destruction.
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degenerated. Further caudally, changes within the midline ventricles were not as prominent but alterations in the surrounding parenchyma caused by ventricular enlargement were present.

In acutely hydrocephalic animals, aged from 10 days to 2 weeks, the central canal of the spinal cord was patent but not enlarged (Figs. 1 and 3 upper right). It ranged in size and profile from a mere slit to the dimensions found in the normal animal (Fig. 3 upper left). The surrounding tissue was normal. In animals with chronic communicating hydrocephalus, the canals were patent, with the orifice of the central canal approximately the same as in the normal dog (Fig. 3 lower right). The surrounding tissue was normal, although in the white matter degenerative changes were seen distal to the central canal.

In none of the animals made hydrocephalic by this method was there enlargement of the central canal of the spinal cord nor were there surrounding histological changes to suggest altered permeability of this structure.

Discussion

The purpose of this study was to evaluate the possible role of the central canal of the spinal cord as an alternative pathway of CSF flow and absorption in hydrocephalus. The experimental model reproduced many of the physiological circumstances encountered in human chronic communicating hydrocephalus. An obstruction to CSF flow outside the ventricular system is produced. The technique does not cause a significant inflammatory response that will in itself produce significant alterations in CSF movement. Injection techniques that produce severe inflammation will often cause an obstruction to CSF flow at the outlets of the ventricle or in the cerebral aqueduct between the lateral and midline ventricles. This model avoids that problem as evidenced by entry of the radio-pharmaceutical agent into the ventricles during cisternography and confirmed by pathological studies.

Conflicting results have been obtained using various animal models regarding the compensatory absorption of CSF in hydrocephalus. It is not firmly established that transependymal migration is the major alternative pathway for absorption in hydrocephalus, and dilation and absorption in the central canal have been suggested. The enlarged canal presumably functions as a conduit between the ventricular system and the spinal subarachnoid space. We have observed the same compensating changes in our experimental model and have related the CSF pressure return to normal with pathological alterations in the ventricular ependyma, enlargement of the extracellular space, and movement of labeled molecules into the brain by bulk flow.

In none of the animals made hydrocephalic by the Silastic model has dilatation of the central canal of the spinal cord been present, nor are we aware that this is a common pathological finding in human beings with hydrocephalus. Therefore, it is our consideration that the central canal of the spinal cord does not play an important role in the alternative pathways for CSF absorption in chronic communicating hydrocephalus.

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