The spinal cord central canal: response to experimental hydrocephalus and canal occlusion

DONALD P. BECKER, M.D., JIMMY A. WILSON, PH.D., AND G. WILLIAM WATSON, B.S.

Department of Surgery, Division of Neurosurgery, University of California at Los Angeles School of Medicine, and Harbor General Hospital, Torrance, California

The central canal of the spinal cord was studied with canal occlusion alone, and in experimental (kaolin) hydrocephalus without and with central canal occlusion. Massive dilatation of the canal occurred with kaolin hydrocephalus. Syrinxes extending into the gray and white matter of the cord and communicating with the central canal developed in both the upper and lower spinal cord. The completely isolated central canal (occlusion at the obex and ilium terminale) did not dilate, but remained patent. Canal occlusion at the obex and ilium terminale completely protected the spinal cord from central canal dilatation or syrinxes in kaolin hydrocephalus. These findings suggest that the choroid plexus is responsible for producing neural tube dilatation in hydrocephalus. It also supports the concept that syringomyelia results from inadequate drainage of cerebrospinal fluid and increased pressure (or pulse pressure) in the spinal cord central canal.

KEY WORDS - spinal cord - central canal - hydrocephalus - syringomyelia - hydromyelia - cerebrospinal fluid

The exact site of cerebrospinal fluid (CSF) formation, in particular the site of active secretion, has not been determined. With newer techniques available to study CSF production quantitatively, there has been a renewed interest in this topic. Recent studies have suggested that the choroid plexus contributes to CSF production in only a limited way, and that the ependyma and/or cerebral tissue may be responsible for the major component of CSF formation.

The spinal cord central canal offers a model that may improve understanding of this problem. It is lined with ependyma, surrounded by glia, axons, and neurons, but unlike the ventricle, is devoid of choroid plexus. In cats, as well as monkeys, infants, and children, it communicates with the fourth ventricle and is patent throughout to the filum terminale. In some species the filum canal may communicate with the subarachnoid space.

Our experiment was designed to answer the following questions: Will the central canal dilate in communicating hydrocephalus? Will cord syrinxes develop in communicating hydrocephalus? Will the central canal dilate when its orifices are occluded (obex and filum)? Will obex occlusion protect the central canal and spinal cord in communicating hydrocephalus? Answers to these questions can provide insight into mechanisms of CSF production as well as the etiology of sponta-
Response of the central canal of the spinal cord to hydrocephalus

Fro. 1. Gross cross sections of normal cat brain. The ventricles are very small.

neous syringomyelia and considerations of its treatment.

Methods

A total of 47 adult cats were used in this study, and all procedures were performed under sterile conditions with pentobarbital anesthesia. Hydrocephalus was induced by injecting 100 mg of kaolin percutaneously into the cisterna magna. Obex occlusion was performed via a suboccipital craniectomy. A cotton plug, 1.5 mm in diameter and 5 mm in length, tapered at its tip, was soaked in kaolin and coated with a thin layer of dental cement to give it resistance. This was placed into the central canal at the obex. A dural graft of dacron coated with silicone rubber was used to cover the exposed cerebellum. Filum terminale ligation was completed by low sacral laminectomy and extradural ligation of the filum and dural contents.

Animals were divided into the following groups:

Group 1. Control: normal canal (6 animals)

Group 2. Kaolin hydrocephalus: canal patent (6 animals)

Group 3. Central canal occlusion only (at the obex): (7 animals)

Group 4. Central canal isolation: obex occlusion and filum ligation (6 animals)

Group 5. Kaolin hydrocephalus with obex occlusion (11 animals)


In animals with hydrocephalus and canal occlusion, the kaolin injection and plug insertion were separated by 2 to 3 days. Our success rate in accomplishing both tasks was greater when the kaolin injection preceded open surgery than when the canal plug preceded the kaolin injection.

The cats were sacrificed at 2, 4, 6, and 8 weeks following the initial procedure. They were anesthetized with pentobarbital; total body perfusion via the left ventricle was then performed with 10% formalin. The brain, spinal cord, and filum down to the tail were
Fig. 2. *Left Column:* Photomicrographs from normal cat spinal cord. From top to bottom: cervical enlargement, ×8; thoracic, ×11; lumbar, ×8; sacral, ×15; filum, ×14. The central canal is patent throughout. *Right Column:* Photomicrographs of the spinal cord from a cat with kaolin hydrocephalus for 6 weeks. Magnifications and cord levels are comparable to the normal specimens on the left. The central canal is dilated up to 40 times its normal size.
removed and placed in 10% formalin. One week later the brain, spinal cord, and filum were cut and inspected grossly for hydrocephalus and canal dilatation. Serial sections of the spinal cord and filum were prepared and stained with hematoxylin and eosin for histological evaluation.

Results

Group 1

In each control animal the ventricles were quite small (Fig. 1), and the canal was noted to be patent throughout, to and including the filum terminale. Grossly, the normal canal could just barely be identified, but histologically it was clearly identified as a round or oval patent structure (Fig. 2 left column and Fig. 3). The ependymal cells in the cervical medullary canal (located just below the obex) were similar in appearance and orientation to ependymal cells lining the ventricular walls. There was a single continuous layer of cuboidal cells. In the upper cervical region the cells were longer with the nuclei appearing closer together. In the region of the cervical enlargement, they were columnar in their orientation to the lumen, and often there was a double layer of ependymal cells. In the thoracic region, the cells were sometimes lined up three deep from the canal lumen, but in most areas, a single layer of columnar cells predominated. As the canal became larger in the lumbar and sacral regions, the cells became progressively flatter, and in the filum, where the canal was largest, the cells again appeared cuboidal as in the ventricle and cervicomedullary regions. The canal cross-sectional area averaged 0.018 to 0.020 mm² in the cervical region, 0.010 mm² in the thoracic region, and up to 0.028 mm² in the lumbar area. The normal filum canal measured up to 0.10 mm² in diameter but was usually in the range of 0.050 mm².

Group 2

With kaolin injection alone, significant hydrocephalus was noted as early as 2 weeks. Minimal canal dilatation was evident at 2 weeks, became grossly apparent at 4 weeks, and became progressively more marked by 6 and 8 weeks (Fig. 4).

The canal enlarged up to 40 times its normal size (Fig. 2 right column). Ependymal cells became progressively more squamous in appearance and never more than one layer of cells was noted (Fig. 5).

In the 6- and 8-week animals with kaolin hydrocephalus, degenerative changes in the dorsal columns were present in the cervical region. Also, syrinxes or cord cavitations communicating with the central canal were present, usually in the cervical (Fig. 6) or upper thoracic regions, but in one animal in the lumbar region (Fig. 7). This lumbar cord lesion was histologically similar to the lesion of the syringomyelia, with cavitation extending into one ventral horn and surrounded by gliotic tissue. The involved half of the cord was expanded suggesting internal pressure from the syrinx.

Groups 3 and 4

We next sought to determine if the central canal would also dilate if its apertures were
Fig. 4. Sections of brain and spinal cord from a cat with kaolin hydrocephalus of 6 weeks' duration. The ventricles are very large. The flow of CSF from the ventricles occurred mainly from one foramen of Luschka (upper right). The central canal is markedly dilated. Spinal cord segments shown are the upper cervical (center) and (bottom row, left to right): the cervical enlargement, upper thoracic, lower thoracic, lumbar, and sacral. The central canal is clearly dilated.

occluded. However, even in animals followed up to 8 weeks, there was no canal dilatation either with occlusion at the obex alone, or with complete canal isolation performed by canal occlusion at the obex and filum terminale ligation (Fig. 8). It is important to note that the central canals in these animals maintained their patency and did not collapse. The canal sizes were the same as in the control group, and there was no evidence of cell desquamation or proteinaceous contents in the canal which would imply stagnation of CSF.

Group 5

We next set out to investigate what would happen to the spinal cord and central canal in kaolin hydrocephalus if the canal was occluded at the obex. Would canal occlusion prevent canal dilatation and/or syrinx formation? In this group of animals, no canal dilatation or syrinxes were noted in the cervical or upper thoracic region. In two animals, however, minimal dilatation of the lower thoracic, lumbar, and sacral canal was noted. Inspection of the filum terminale in these and several other animals revealed an opening into the subarachnoid space (Fig. 9).

In six other animals, the central canal was perfused at the obex with trypan blue at 20 mm H₂O pressure for 15 minutes. In all animals dye passed into the filum terminale, and in three it passed through the filum into the subarachnoid space, demonstrating either a canal connecting the filum directly to the subarachnoid space, or fluid transfer across the filum ependyma into the subarachnoid space. The lower filum canal in the cat is separated from the subarachnoid space by a single layer of cells and a very thin covering of glial tissue.
Response of the central canal of the spinal cord to hydrocephalus

**Group 6**

Our final group of animals had kaolin hydrocephalus with central canal occlusion at the obex and filum terminale ligation just below the conus medullaris. In no animal was there any evidence of canal dilatation or cord cavitation (Fig. 10). Animals in both this group and Group 5 demonstrated some minimal degenerative changes in the cervical dorsal columns, probably secondary to kaolin arachnoiditis in this region. But these dorsal column changes were much greater in animals with only kaolin hydrocephalus.

**Discussion**

In 1954, McLaurin, et al., described central canal dilatation and progressive necrosis of the dorsal columns in dogs with kaolin hydrocephalus. Progressive cavitation of the cord was noted and when India ink was injected into the fourth ventricle before death, the material was visible in the central canal and cavitations. They attributed the cord changes to arachnoiditis and vascular occlusion. We have confirmed and extended their findings, but suggest that internal canal pressure is responsible for the canal dilatation and extension of the syrinx.

We would like to emphasize that the histological picture of the syrinx in our animals is not unlike that seen in human syringomyelia. There is cavitation with surrounding gliosis, and ependymal cells are noted only in part of the syrinx wall, if at all. Our find-
FIG. 9. Photomicrograph of cat filum terminale. Notice the opening of the ependyma toward the subarachnoid space. The rounded appearance of the ependyma at this orifice eliminates the possibility of artifact. H & E, ×156.

ings support Gardner's concepts regarding a hydrodynamic mechanism in the development of syringomyelia. Briefly stated, he believes the etiology to be secondary to impaired CSF circulation at the fourth ventricle outlets, beginning prenatally but extending into postnatal life.

We have demonstrated that the cat central canal will dilate when there is a block to CSF flow in the subarachnoid space. Although it will dilate behind the pressure head of active CSF formation, it will not dilate when its orifices are occluded. This provides indirect evidence that ependyma and/or surrounding central nervous system tissue may not be responsible for active CSF formation. It further suggests that choroid plexus must be present to cause ventricular or canal dilatation. Whether the enlargement of the ventricles or canal is the result of active CSF formation by the choroid plexus or the pulse pressure transmitted to the CSF by the choroid plexus as suggested by Bering remains to be determined.

Fig. 8. Photomicrographs of the spinal cord from an animal with filum terminale ligation and canal occlusion at the obex for 8 weeks. Levels and magnification are the same as those in the left column (top to bottom) in Fig. 2. The central canal is patent but not dilated.
Response of the central canal of the spinal cord to hydrocephalus

In hydrocephalus secondary to subarachnoid fibrosis, occlusion of the canal at the obex and filum completely protected the central canal from dilatation and the cord from the formation of syrinxes. This finding, in light of the above results, may justify Gardner’s recommendation to plug the obex with muscle or cotton in human syringomyelia associated with Arnold Chiari malformation. It is, however, a technically difficult task to completely occlude the canal at the obex without causing neurological damage. Furthermore, in cats, obex occlusion alone did not always totally protect the central canal. In these animals canal dilatation occurred in the lower cord, and it is postulated that increased canal pressure was transmitted via the filum terminale.

The ideal treatment for tension syringomyelia remains to be determined. If hydrocephalus accompanies the syrinx state, perhaps a ventricular shunting procedure should be considered. In the absence of gross hydrocephalus, plugging of the obex, shunting the syrinx to the subarachnoid space, and transcutaneous needle puncture have all been recommended. Further experience with these procedures needs to be documented.

Summary

Massive dilatation of the central canal was observed in cats with kaolin hydrocephalus when the central canal was in communication with the fourth ventricle. Syrinxes in communication with the central canal occurred in both the upper and lower spinal cord. The completely isolated but patent central canal did not dilate. Central canal occlusion at the obex partially protected the central canal and spinal cord in kaolin hydrocephalus. Filum ligation and central canal occlusion at the obex completely protected the central canal and spinal cord in kaolin hydrocephalus.

The above findings suggest that, in hydrocephalus, neural tube dilatation occurs as a result of choroid plexus activity and that fluid formation by or across the ependyma alone is not sufficient to cause neural tube enlargement.

References

2. Bradbury MWB, Lathem W: A flow of cerebrospinal fluid along the central canal of the spinal cord of the rabbit and communications between this canal and the sacral subarachnoid space. J Physiol 181:785–800, 1965
8. Milhorat TH: Choroid plexus and cerebrospinal fluid


Presented at the annual meeting of the Society of Neurological Surgeons, Phoenix, Arizona, February 14–17, 1971. This work was supported by a grant from the National Easter Seal Research Foundation and Grant RR-05551-09 from the U.S. Public Health Service.

*Address reprint requests to:* Donald P. Becker, M.D., Division of Neurosurgery, Medical College of Virginia, 1200 East Broad Street, Richmond, Virginia 23219.