CIRCULATION OF THE CEREBROSPINAL FLUID

DEMONSTRATION OF THE CHOROID PLEXUSES AS THE GENERATOR OF THE FORCE FOR FLOW OF FLUID AND VENTRICAL ENLARGEMENT

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(Received for publication December 5, 1961)

This paper presents an experimental test of the assumption that the force of formation of cerebrospinal fluid causes the cerebrospinal fluid to circulate and the ventricles to enlarge when its pathways are blocked. These experiments show that this assumption is wrong and a new description of the circulation of the cerebrospinal fluid is given.

It has always been assumed that the cerebral ventricular enlargement of hydrocephalus was caused by the back pressure of blocked circulation of the cerebrospinal fluid, but this has never been put to experimental test. The clinical facts of hydrocephalus leave no doubt that if some cerebrospinal fluid escapes from the cerebral ventricles internal hydrocephalus will be prevented or cured, if established, and experimental work has generally confirmed these facts. Dandy, in his experiments on hydrocephalus and formation of cerebrospinal fluid in dogs, removed the choroid plexus from one lateral cerebral ventricle, plugged the foramen of Monro on the same side, and then occluded the aqueduct of Sylvius. In this animal there was an enlargement of the ventricle with the choroid plexus present and the foramen of Monro open, while the other lateral ventricle remained unchanged. This was interpreted as demonstrating that the choroid plexus produced the cerebrospinal fluid, and this has remained as the basic experimental work showing that the choroid plexuses were the major source of the cerebrospinal fluid. This conclusion was based upon the assumption that a symmetrical hydrocephalus would result from the pressure of accumulated cerebrospinal fluid if the choroid plexus of one lateral ventricle was removed, the foramen of Monro left open, and the aqueduct of Sylvius occluded. However, this assumption was never tested experimentally either by Dandy or others who repeated his work.

MATERIALS AND METHODS

Mongrel dogs weighing 15 to 20 kg. were the experimental animals. The procedures carried out were unilateral and bilateral ventriculotomy, unilateral choroid plexectomy with and without occluding the foramen of Monro on the same side, and bilateral choroid plexectomy. These procedures were tested for their effect, in the otherwise normal animal, on the development of hydrocephalus and on hydrocephalus induced previously.

The surgical procedures of ventriculotomy and choroid plexectomy were done aseptically in a manner similar to that of others. Hydrocephalus was produced by intracisternal injection of 0.25 to 0.5 gm. kaolin suspended in cerebrospinal fluid which produced a sterile reaction occluding the outlets to the 4th ventricle. Three to 4 weeks were allowed between any two procedures. Measurements of pressure were made with Statham strain gauges led to a Sanborn recorder. At the time of sacrifice the animals were given a lethal dose of pentobarbital, and the carotid arteries were cannulated, washed out with saline and then irrigated with 3 or more liters of 10 per cent formalin solution. The brain then was removed, and examined for ventricular size and completeness of the choroid plexectomies.

RESULTS

Ventriculotomy. Bilateral ventriculotomies were made in 2 normal dogs which were

* This work was supported by USPHS research grant 157 from the National Institute of Neurological Diseases and Blindness.
sacrificed 5 and 16 weeks later. The cortical scars had healed completely. The ventricles of these dogs were slightly larger than normal in size, but they were symmetrical without any distortion of the mid-line structures.

Unilateral ventriculotomies were done on 3 dogs and after intervals of 4, 6 and 8 weeks kaolin was injected intracisternally. They then were sacrificed 3, 4 and 12 weeks later. In all 3 animals hydrocephalus had developed and the cortical scars were healed and solid. In the 3-week animal there was some intra-ventricular scarring around the cortical incision in the body of the ventricle with slight asymmetry of the bodies of the ventricles, but there was no shift of mid-line structures and there was complete symmetry of the anterior and temporal horns. Difference in ventricular size was not apparent in the 4- or 12-week animals. The 4th ventricle of all animals had enlarged as expected.

Bilateral ventriculotomies were made in 3 normal dogs and then after periods from 2 to 6 weeks kaolin was injected intracisternally. In all these dogs symmetrical hydrocephalus developed including enlargement of the 4th ventricle and aqueduct of Sylvius. One animal had transventricular adhesions under the cortical scar, but in spite of this there was still a symmetrical enlargement of the ventricular system. The adhesions were stretched across the ventricle without distorting the mid-line structures. There were no adhesions on the opposite side which might have had a restraining influence on distortion (Fig. 1).

Unilateral ventriculotomy was carried out in 4 dogs previously made hydrocephalic. They died or were sacrificed at 1 day, 2 days, 4 weeks, and 12 weeks. The 2 dogs that died at 1 and 2 days postoperatively were not remarkable. The other 2 both showed well healed cortical scars and symmetrical hydrocephalus. In the 12-week animal the side with the ventriculotomy was larger than the other because of atrophy around the cortical incision, but there was no mid-line distortion.

Bilateral ventriculotomy was carried out in 5 hydrocephalic dogs which were sacrificed after another 3 weeks. They all showed well healed scars with symmetrical bilateral hydrocephalus.

In summary, experiments on 13 animals showed that ventriculotomy, unilateral or bilateral, will not affect the development of a symmetrical enlargement of all the cerebral ventricles following a cisternal injection of kaolin. Particularly important was the observation that the development of transventricular adhesions following surgical procedures did not prevent ventricular enlargement nor cause distortion or shift of mid-line structures.

Unilateral Choroid Plexectomy Without Blocking the Foramen of Monro. Experiments with unilateral choroid plexectomy without blocking the foramen of Monro were carried out in 19 animals, with 11 satisfactory survivors with complete choroid plexectomies. Five of these animals were subjected to unilateral plexectomy and then made hydrocephalic and the other 6 animals were hydrocephalic before plexectomy.

The 5 animals that had plexectomy before production of hydrocephalus were sacrificed 3 to 9 weeks after the injection of kaolin. The cortical incisions were all healed solidly. In all animals, there was enlargement of the lateral ventricle with the choroid plexus still present while the ventricle without the

Fig. 1. A coronal slice from brain of a dog which had in turn bilateral ventriculotomy, hydrocephalus produced by cisternal kaolin, and sacrifice 10 weeks later. Transventricular scarring did not prevent ventricular enlargement nor cause mid-line shift or distortion.