HYDROCEPHALUS AND THE DURAL VENOUS SINUSES

MURL E. KINAL, M.D.

Department of Surgery, Division of Neurosurgery, Hamot Hospital, Erie, Pennsylvania

(Received for publication July 3, 1961)

In a classic monograph on the pathology of hydrocephalus, Russell\textsuperscript{21} recorded admirably all known causes of internal or obstructive hydrocephalus. At the same time she speculated about the relationship of thrombosis in the dural venous sinuses to hydrocephalus. The clinical and pathological material suggested that sinus thrombosis could be followed by a limited degree of hydrocephalus after a time and that the size of the ventricles could revert to normal with canalization of the thrombus. Further, roentgenographic evidence of antemortem hydrocephalus could not be corroborated at necropsy, because collapse of the ventricles was thought to be the cause of a normal ventricular pattern. Deliberate attempts to occlude the superior sagittal sinus in a series of animals produced only slight and equivocal degrees of hydrocephalus.\textsuperscript{3} The over-all results were attributed to compensation of circulation by collateral veins.

It is the purpose of this presentation to relate obstructive phenomena in the dural venous sinuses to states of intracranial hypertension and hydrocephalus. Aside from the customary diagnostic measures, the diagnostic value of measurements of intrasinus venous pressure and dural sinus venography will be emphasized.

The physiological studies of Becht\textsuperscript{2} indicated that pressure of the intracranial fluid was the result of the influence of venous and arterial pressure. Venous and fluid pressures varied in the same direction and to some degree proportionately in nearly every case. Raising the venous pressure raised the fluid pressure, while lowering the venous pressure lowered the fluid pressure. Increasing or decreasing the fluid pressure moderately did not alter the venous pressure unless the arterial pressure was affected. Weed and Flexner\textsuperscript{21} corroborated these studies.

In a series of dogs Bedford\textsuperscript{1,5} showed that with prolonged occlusion of the external jugular veins or the lateral sinuses, the venous pressure rose higher than the fluid pressure. Within minutes the fluid pressure gradually fell to its normal level; the venous pressure maintained itself above that of the spinal fluid until release of the occlusion. Bedford then ligated the external jugular veins and concluded that the procedure did not cause hydrocephalus after 6 weeks of observation. In this series the internal jugular veins, usually small accessory channels, were dilated considerably.

Stewart\textsuperscript{26} was able to produce a state of chronically increased intracranial pressure in cats by ligating both posterior sinuses of the vertebral column, both internal jugular veins and often one external jugular vein. The fluid pressure fell slowly as remaining venous channels grew larger and new venous channels developed. To maintain the raised pressure for 4 to 5 weeks required second and third operations, but no hydrocephalus resulted.

Bering and Salibi\textsuperscript{8} were able to produce increased cerebral venous pressure, cerebrospinal fluid pressure and hydrocephalus in 74 per cent of 21 dogs as a result of occlusion of the cephalic venous drainage. Ventricular enlargement reached its peak 2 or 3 weeks after the second dissection of the neck and then remained stabilized. This effect may have been the result of development and enlargement of remaining venous collateral drainage. They felt that the mechanisms involved in the ventricular enlargement were a combination of two factors: the possible failure of cerebrospinal fluid absorption in the face of increased venous pressure in the sagittal sinus and increased intraventricular pulse pressure from the choroid plexus.

In the human, deliberate ligation of the
jugular veins has been followed by varied effects. Rohrbach and Linser ligated the internal jugular vein unilaterally during dissection of the neck for cancer. In each case death resulted from acute passive congestion of the brain, because of the ligation of the major draining venous sinus. Guillain reported a case of ligation of the internal jugular vein followed by neurological symptoms and increased intracranial pressure relieved by repeated spinal punctures. Creyssel and Douillet reported 1 death and 2 cases in which neurologic complications followed ligation of a jugular vein. More recent reports of unilateral and bilateral ligation of the jugular vein indicate that the procedure may be innocuous or followed by mild neurological complications, and states of intracranial hypertension. The latter may be evanescent or prolonged and have not been evaluated with pneumoencephalography and dural sinus venography. It remains undetermined how many of these patients would show the ultimate effects of increased intracranial pressure, that is hydrocephalus.

An analysis of clinical entities that affect the dural venous sinuses, provides a correlation of obstructive phenomena in the dural venous sinuses with increased intracranial fluid pressure and communicating hydrocephalus. Symonds evolved the clinical picture of “otic hydrocephalus,” and demonstrated the relationship of otitis media to thrombophlebitis of the dural venous sinuses and the picture of benign intracranial hypertension. Gardner corroborated Symonds’ clinical observations and stated that encephalography showed no evidence of hydrocephalus. The roentgenograms reproduced in 3 cases indicated a mild but definite hydrocephalus. Foley and Bradshaw corroborated the clinical picture described by Symonds with their findings at operation, autopsy, ventriculography and arteriography. In a small per cent of cases the ventricles were larger than normal. Frenckner was the first to demonstrate by dural sinus venography the relation of otitis media to thrombosis in the lateral sinus. Since then Ray et al. and Frenckner have shown that thrombosis of a major dural venous sinus invariably leads to intracranial hypertension. Kinal and Jaeger were able to demonstrate hydrocephalus in 1 patient out of 3 with thrombosis of a major dural venous sinus.

Cranial trauma has afforded further case material for the study of obstructive phenomena in the dural venous sinuses and their relation to intracranial hypertension and communicating hydrocephalus. Martin first described cases of head injury in which signs of increased intracranial pressure developed, without signs of severe intracranial bleeding and in which ultimate recovery occurred without operation. One syndrome was attributed to obstruction in the sagittal sinus itself but not infrequently in the lateral sinus. Many of his cases had points of similarity to those of “otic hydrocephalus” and he used the term “traumatic hydrocephalus” in referring to them. In a discussion of benign forms of intracranial hypertension, Foley related 4 cases of mild head injury to inexplicable intracranial hypertension. By means of dural sinus venography, Kinal was able to demonstrate that in his cases the “traumatic hydrocephalus” of Martin was indeed caused by thrombosis in the dural venous sinuses. In these cases, the spinal fluid pressure and the intrasinal venous pressure were increased invariably, although they might be normal. In these and subsequent cases, fluid dynamic studies indicated that recanalization of the obstructed sinus was possible. The patent venous sinus system could dilate to accommodate the additional burden of blood. Collateral channels of drainage into the vertebral-azygos plexuses of Batson could drain excess quantities of blood thereby averting a state of intracranial hypertension. That a communicating hydrocephalus may result from traumatic thrombosis of a major draining dural venous sinus has been shown in a small per cent of unpublished cases.

It has been shown in the preceding discussion that deliberate or clinical interference of drainage through the major dural venous sinuses may induce states of intracranial hypertension. In the clinical group of throm-