Sagittal Sinus Venous Pressure in Hydrocephalus*

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Previous workers have demonstrated a gradient of pressure between cerebrospinal-fluid pressure as measured either in the cisterna magna or ventricle and the venous pressure in the superior sagittal sinus. Data obtained in our laboratory have confirmed this difference in pressure in dogs with mean cerebrospinal-fluid pressure equal to 147 mm. H$_2$O and venous pressure in the superior sagittal sinus equal to 90 mm. H$_2$O. Acute elevations of cerebrospinal-fluid pressure in the normal dog did not cause a rise in the venous pressure in the superior sagittal sinus, but in the type of hydrocephalus induced by Kaolin, all rises of cerebrospinal-fluid pressure were accompanied by an increase in venous pressure in the superior sagittal sinus.

In an attempt to define the gradient of pressure available for shunting cerebrospinal fluid from the lateral ventricle to the superior sagittal sinus in infants with hydrocephalus, simultaneous recordings of pressure in the sagittal sinus and lateral ventricle have been accomplished in 15 infants. In 5 of these children simultaneous jugular venous pressure was measured. Three children had sinograms to visualize the dural venous system.

**Materials and Methods**

A. *Methods of Recording Pressure.* Hydrocephaic infants were studied at the time of ventriculojugular or ventriculopleural shunting. With the infants under general endotracheal anesthesia (Fluothane) and in the supine position with the head turned to the left, a burr hole was placed in the right occipital area. The dura mater was opened minimally in a cruciate fashion and after coagulation of the arachnoid and pia mater, a ventricular needle was passed into the lateral ventricle. This was withdrawn and a polyethylene catheter with an outside diameter of 1.7 mm. was introduced into the ventricle and held in place by a purse-string suture around the dural edges. This catheter was connected to a Statham P23 AA transducer and set at the level of the heart. A 1.5-cm. coronal incision was made over the posterior third of the anterior fontanelle straddling the midline, and the sagittal sinus was exposed. A purse-string 6-0 suture was placed in the wall of the sinus and a small opening was made, just large enough to admit a polyethylene catheter of similar size. The tip of the catheter was directed caudally for 2-3 cms. in the direction of the flow of blood, and sutured in place with the previously placed silk. A pledget of Gelfoam was fashioned around the catheter to control any bleeding. This venous catheter was continually irrigated with saline to maintain its patency, and its position was checked by aspirating blood. Recordings were done on an Electronics for Medicine PR-7 polygraph which allows full-screen deflection at low pressures (10 mm. Hg). In a number of infants after stable base-line pressures were recorded, fluid was allowed to escape from the ventricle to observe the effect of the lowered cerebrospinal-fluid pressure on the venous pressure in the superior sagittal sinus. In 5 infants undergoing shunting into the right atrium via the jugular vein, pressure in this structure at the level of the common facial vein was recorded by introducing a catheter via this side branch into the center of the internal jugular vein. This catheter was led off to a Statham P23 AA transducer.

B. *Sinography.* These studies were performed under local anesthesia usually prior to operation. A #20-gauge Cournard needle was introduced into the sinus in the direction of the flow of blood. Injection of 50 per cent Hypaque was done by hand and seriograms were taken. No complications occurred in the infants studied.

**Results**

Simultaneously obtained data on pressure in the equilibrium state are presented in
The mean pressure of the ventricular cerebrospinal fluid was $206 \pm 95$ mm. H$_2$O, while the venous pressure in the superior sagittal sinus was $222 \pm 94$ mm. H$_2$O. In 11 of 15 instances, the venous pressure in the superior sagittal sinus equalled or exceeded the ventricular cerebrospinal-fluid pressure. The ratio of mean venous pressure in the superior sagittal sinus to mean cerebrospinal-fluid pressure was 1.08. If the venous pressure in the superior sagittal sinus is plotted as a variable of the ventricular cerebrospinal-fluid pressure, the equation of the least squares straight line is

$$SSVP = (0.95) \times CSFP_{vent} + 27.5 \text{ mm. H}_2\text{O}.$$  

Such a plot is shown in Fig. 1. The regression coefficient, $b$, of SSVP as a function of CSFPvent is 0.95 with a standard error of 0.08. This regression is highly significant and shows that in different individuals high values of SSVP and CSFPvent go together, and low values of SSVP and CSFPvent go together. This also suggests that changes in SSVP are causally related to changes in CSFPvent. Indeed this observation is substantiated in Fig. 2, a graph obtained in patient J.S., where, by allowing the cerebrospinal fluid to escape, both the ventricular cerebrospinal-fluid pressure and the venous pressure in the superior sagittal sinus were lowered along a curve whose regression slope is 1. This line and that of the least squares obtained on all the children do not pass through the origin, but bisect the y axis at approximately 30 mm.
H₂O pressure. While not experimentally determined, this intercept might be thought of as the outflow pressure, i.e., jugular venous pressure. Indeed the mean pressure measured in the jugular vein of 5 children was 35 mm. H₂O (Table 1).

Sinograms were performed in 3 instances. In 2 children, the sinus was elongated but no point of obstruction could be seen (Fig. 3). In the infant shown in Fig. 4, the lateral sinus on each side seems to taper to end proximal to the jugular foramen, and blood from the sinus is seen to be exiting via enlarged parietal and mastoid emissary channels. This suggests that considerable compression of the sinus has occurred near the point of exit from the skull, forcing blood to use unusual routes of exit from the sinus because of the induced increased resistance at the outflow of the lateral sinus.

Discussion

The normal superior sagittal sinus is an area of the venous system which is unique in that its walls are not thought to be collapsible. If this were entirely so, elevation of cerebrospinal fluid pressure would not affect the pressure within the sagittal sinus, and the only way that the venous pressure in the sagittal sinus could be elevated would be by a fixed obstructive lesion at its outflow or by increased blood flow through the sinus. Although obstructions have been occasionally demonstrated by sinograms, they are not a common finding in hydrocephalic material at autopsy. Our data show that there is an elevation of pressure within the sagittal sinus in the hydrocephalic state; the importance of this elevation is that it would be expected to negate the gradient of 30-40 mm. H₂O needed for absorption of cerebrospinal fluid across the arachnoid villi. To explain this elevation of pressure in the sagittal sinus, we postulate a partial collapse of the sinus possibly secondary to distortion in association with the enlargement of the ventricle and skull. In such a collapsible structure, the major determinant of pressure is the pressure in the surrounding tissue, and in the closed cranial cavity this pressure may be thought of as cerebrospinal-fluid pressure. That venous pressure in the superior sagittal sinus is determined by cerebrospinal-fluid pressure in hydrocephalus is suggested by the regression coefficient of venous pressure in the

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superior sagittal sinus as a function of cerebrospinal-fluid pressure of 0.95, and the data obtained in individual children by venting the cerebrospinal-fluid system and causing a concomitant drop in venous pressure in the superior sagittal sinus. In this collapsible system, the increased gradient of pressure from sagittal sinus to jugular vein need not be associated with an appreciable increase in blood flow. Flow does not vary directly with pressure gradient unless the conduit is rigid and there is no evidence demonstrating an increased cerebral blood flow in hydrocephalus. We need not postulate an anatomically obstructed sinus system as the cause of the hydrocephalus, but rather a secondarily partially collapsed sinus as the result of prolonged elevations of cerebrospinal-fluid pressure. As cerebrospinal-fluid pressure builds up further, the sinus is focally compressed at the jugular foramen, and in this state it becomes linked in a 1:1 fashion with pressure in the ventricular system. Certain consequences of this must occur. The pressure in the subarachnoid veins, which in the normal state is greater than the venous pressure in the superior sagittal sinus, would be expected to undergo a concomitant elevation. Any elevation of venous pressure at the outflow from the capillary bed must increase the pressure within that capillary bed, leading to venostasis and elevation of mean capillary pressure. It is conjectured that this increase in capillary venous pressure leads to a further decrease in absorption of cerebrospinal fluid at the capillary level, leading to a continuance of the hydrocephalic process.

Summary
Simultaneous recordings of pressure in the ventricle and superior sagittal sinus in 15 hydrocephalic children have shown an elevation of pressure in the sinus. The mechanism of this rise in pressure is thought to be a partial collapse of the sinus at or near the point of outflow from the skull. In this collapsible system, changes in cerebrospinal fluid pressure are accompanied by changes in the

FIG. 4a. Lateral sinus appears to end at double ended arrow (†). Large mastoid emissary vessel (▼▼) and dural venous channels in posterior fossa (▲) drain torcula.

FIG. 4b. Lateral sinuses are narrowed and partially collapsed bilaterally. Large mastoid emissary vein (↓) is draining laterally from torcula.
pressure in the sinus and the absorptive mechanism is further embarrassed.

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References


