Effect of the skull and dura on neural axis pressure-volume relationships and CSF hydrodynamics

KENNETH SHAPIRO, M.D., ARNO FRIED, M.D., FUTOSHI TAKEI, M.D., AND IRA KOHN, B.S.

Department of Neurosurgery, The Albert Einstein College of Medicine, Bronx, New York; and Tokai University School of Medicine, Kanagawa, Japan

The pressure-volume index (PVI) technique of bolus manipulation of cerebrospinal fluid (CSF) was used to measure changes of neural axis volume buffering-capacity and CSF dynamics produced by different conditions of the skull and dura. Twenty-eight cats were studied in the intact state, after bilateral craniectomy, and with the dura opened. At each stage of altering the container of the brain, the following parameters were obtained: steady-state intracranial pressure (ICP), sagittal sinus venous pressure, PVI, and the resistance to the absorption of CSF. The resistance to absorption of CSF was determined using both the bolus injection and the continuous infusion of fluid.

After craniectomy, PVI increased from 0.76 ± 0.04 to 1.3 ± 0.07 ml (± standard error of the mean) (p < 0.001) and increased further to 3.6 ± 0.17 ml (p < 0.001) after opening the dura. The resistance to absorption of CSF (Ro), determined by bolus injection, decreased after craniectomy from 91.3 ± 7.5 to 56.3 ± 6.2 mm Hg/ml/min (p < 0.001) and decreased further to 8.9 ± 0.66 mm Hg/ml/min (p < 0.001) after opening the dura. Although resistance determined by constant infusion was similar, results were dependent on the rate of infusion. Despite these changes of resistance and PVI, steady-state ICP and sagittal sinus venous pressure were similar in all three conditions of the skull and dura. These studies indicate that changes of the container of the brain affect pressure-volume relationships within the neural axis. However, the changes of resistance to absorption of CSF are in a direction that preserves a steady-state hydrodynamic equilibrium.

KEY WORDS - hydrocephalus - intracranial pressure - pressure-volume index - cerebrospinal fluid dynamics - craniectomy - durectomy

Traditional views of hydrocephalus unify the varying etiological processes as having in common a defect in the absorption of cerebrospinal fluid (CSF), creating an imbalance between the formation and absorption of CSF. This depiction of the hydrocephalic process implies that the ventricles will dilate inexorably at the expense of the brain as excess CSF is stored. However, this sequence rarely occurs in the majority of patients encountered in clinical practice. Based on studies of CSF absorption, investigators have suggested that a new steady-state balance between formation and absorption of CSF will occur at a higher CSF pressure resulting in stabilization of ventricular volume. However, many investigations have shown only modest elevations of CSF pressure in hydrocephalic patients who continue to exhibit progressive ventricular dilatation. From these empiric observations, many authors have concluded that this mechanism of compensation does not adequately describe the mechanical events in hydrocephalus.

Noting that the ventricular enlargement in hydrocephalic infants usually exceeds that found in older children and adults, Hochwald, et al., investigated the contribution of the container of the brain to the development of ventriculomegaly. These workers showed that the ventricular size in feline hydrocephalus increased dramatically after removing the calvaria and dura. Comparing ventricular size in hydrocephalic cats with intact skulls to that in animals subjected to alterations of the brain container, these authors concluded that the intact feline kaolin-induced hydrocephalus model leads to a stabilized or arrested form of ventricular enlargement and not to the progressive ventricular dilatation often seen in clinical practice.

Additional support for the role of the brain container in the progression of ventricular enlargement was contributed by clinical studies. By augmenting the mechanical properties of the container with compressive head wraps, these investigators stabilized ventricular size in a carefully selected group of hydrocephalic infants.
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Our own studies of hydrocephalic infants have demonstrated marked abnormalities in the biomechanical properties of the neural axis which result, in part, from the expansile skull. The alterations of the biomechanical properties of the infantile hydrocephalic brain are in a direction that facilitates ventricular enlargement.12

The evidence cited above supports the notion that container properties play a role in the hydrocephalic process. However, all studies were performed on subjects in whom the hydrocephalic process was advanced far enough to become clinically overt. In this and subsequent studies we have investigated the effect of biomechanical properties during the evolution of hydrocephalus. In order to understand the role played by the skull and dura in the steady state, we undertook the studies described here to document the effects of the calvaria and dura on CSF pressure, neural axis compliance (pressure-volume index (PVI)), and the resistance to the absorption of CSF.

Materials and Methods

Twenty-eight adult mongrel cats, each weighing 3 to 5 kg, were anesthetized with intraperitoneal pentobarbital (30 mg/kg) followed by endotracheal intubation. Arterial and venous catheters were implanted for measurement of systemic arterial blood pressure and administration of drugs. The animals were paralyzed with gallamine (4 mg/kg), mechanically ventilated using a conventional Starling respirator with a 2:1 mixture of nitrous oxide and oxygen, and secured in the sphinx position in a stereotaxic frame. Arterial blood gases were checked periodically: \(\text{PaCO}_2\) was maintained between 30 and 35 torr, and \(\text{PaO}_2\) at greater than 90 torr. Rectal temperature was monitored continuously and maintained within the physiological range.

A burr hole was made at the bregma. The sagittal sinus was cannulated using saline-filled PE 50 tubing coupled to a strain-gauge transducer in order to measure sagittal sinus venous pressure (SSVP). In the majority of experiments the sinus catheter was directed toward the torcula. A No. 19 scalp vein needle, coupled via saline-filled tubing to a strain-gauge transducer, was inserted into the cisterna magna to measure CSF pressure. The electrical output of all transducers was coupled to a strain-gauge transducer in order to measure the diastolic component of the cardiac and respiratory phases.

Manipulations of Cerebrospinal Fluid

After a steady-state baseline intracranial pressure (ICP) was established, bolus manipulation of CSF was performed by bolus injection of 0.1 to 0.4 ml mock CSF (\(\Delta V\)) at 0.1 ml/sec into the cisternal catheter while simultaneously recording the time course of CSF pressure. Strip chart recording was continued at 1 mm/sec until the cisterna magna pressure returned to baseline. At least three injection-recovery sequences were performed for each phase of these experiments. Pressure-volume index was calculated using the formula:

\[
PVI = \frac{\Delta V}{\log \left( \frac{P_p}{P_o} \right)}
\]

where \(P_o\) is the steady-state ICP prior to infusion, \(P_p\) is the steady-state ICP immediately following each injection, and \(\Delta V\) is the volume change due to bolus injection.

Resistance to the absorption of CSF (Ro) was determined using the continuous infusion technique.8 Rates for infusion varied from 0.06 to 0.18 ml/min. Resistance to the absorption of CSF (Ro) was extrapolated from the infusion sequence using the formula:

\[
Ro = \frac{P_o}{PVI} \times \log \left[ \frac{P_t}{P_p} \times \frac{P_t}{P_o} \right]
\]

where \(P_o\) and \(P_p\) are the CSF pressures at time 0 and immediately following each injection. In selected animals successive boluses were injected to establish the pressure-volume curve from 5 to 50 mm Hg. The resistance to the absorption of CSF (Ro) was calculated according to the equation:

\[
Ro = \frac{P_o}{PVI} \times \log \left[ \frac{P_t}{P_p} \times \frac{P_t}{P_o} \right]
\]

where \(P_o\) is the steady-state ICP achieved during continuous infusion, \(P_p\) is the steady-state ICP prior to infusion, and \(I_t\) is the rate of infusion.

These sequences were repeated after each change in condition of the brain container. Data were compiled for initial baseline ICP, SSVP, PVI, and resistance to the absorption of CSF (bolus and infusion) for 1) intact cats; 2) cats after craniectomy; and 3) cats after craniectomy and durectomy.

Alteration of Brain Container

After completing the PVI and resistance to CSF absorption determinations in the intact cat, bilateral craniectomies were performed. The calvaria was removed between the coronal and lambdoid sutures. The craniectomy extended medially to within 5 mm of the sagittal suture, and laterally to the floor of the middle fossa. Magnification was used during surgery to ensure integrity of the dura. Following the craniectomies, ICP was allowed to stabilize and a new steady-state baseline pressure was established. A second series of CSF manipulations was performed to determine the change in PVI and resistance to CSF absorption in each animal following the craniectomies.

After completion of CSF manipulation in craniec-tomized cats, cruciate incisions through the exposed dura were made under the operating microscope. The integrity of the arachnoid was confirmed by slowly infusing mock CSF through the cisternal catheter and visualizing the distended arachnoid under the operating microscope. The ICP was allowed to stabilize and the CSF manipulation sequence was repeated as described above.

Results

Steady-State Intracranial Pressure

The mean steady-state ICP in the intact cats was 8.4 \(\pm 3.9\) mm Hg (± standard deviation (SD)) (Fig. 1). After the bilateral craniectomies had been performed, baseline ICP initially fell an average of 1 to 2 mm Hg.
FIG. 1. Bar graphs depicting steady-state intracranial pressure (ICP) and sagittal sinus venous pressure (SSVP) in normal cats with intact skulls, craniectomy, and durectomy. There was no statistical difference in ICP or sinus pressure in each state.

but with time became established at a new steady-state level of $8.4 \pm 3.9$ mm Hg. After the dura was opened, steady-state ICP fell still further, and found a new steady-state level of $8.1 \pm 1.7$ mm Hg. The pulse pressure diminished successively with each alteration in container property. These decrements in pulse pressure were consistent with the changes in the PVI and were not investigated further. There were no statistically significant differences among these steady-state pressures ($0.7 < p < 0.6$). With each alteration in container property the brain and its investing tissues appeared to expand slightly, but never bulged.

In the intact cat, sagittal sinus venous pressure (SSVP) was $7.9 \pm 0.5$ mm Hg (± SD) in the steady state (Fig. 1). Although this resulted in a CSF pressure-SSVP gradient of 0.5 mm Hg, the pulsatile excursions of the CSF pressure catheter were considerably in excess of those seen in the venous catheter, creating a higher net gradient between CSF pressure and SSVP. While there was some fluctuation in the SSVP after alterations of the container, these were not statistically significant ($0.3 < p < 0.2$) (Fig. 1). When CSF pressure was raised by bolus injection or by continuous infusion, the SSVP remained relatively stable so that the CSF-venous pressure gradient was maintained in these states. Early in the experimental series some of the sinus catheters were directed anteriorly and in several of these animals there appeared to be a linkage between the SSVP and CSF pressure. However, with the catheter directed toward the torcular, the apparent linkage disappeared.

Neural Axis Volume-Buffering Capacity

The mean PVI in the intact cats was $0.76 \pm 0.04$ ml (± standard error of the mean (SEM)) (Fig. 2). This value is within the normal range as previously determined for cats in this laboratory. Pressure-volume curves, established using stepwise increases in the volume of bolus injection, confirmed the validity of the pressure-volume curve projected from single-bolus injections. Following craniectomy, PVI increased to $1.3 \pm 0.07$ ml. This represented a 71% increase in volume-buffering capacity from the intact animal. The difference in PVI between the intact and craniectomy states was statistically significant ($p < 0.001$). After the dura was opened, the PVI increased to $3.6 \pm 0.17$ ml. This change in PVI was statistically significant ($p < 0.001$) and represented a further 177% increase in neural axis volume-buffering capacity. The exponential shape of the pressure-volume curve was not changed by the alterations of the container (Fig. 3). The PVI values determined at relatively low ICP were similar to those measured at high ICP. After the dura was opened, it was difficult to inject volumes large enough to raise ICP beyond 25 to 30 mm Hg due to the marked increase in neural axis volume-buffering capacity.

Resistance to Absorption of CSF

Resistance to the absorption of CSF, determined by the bolus injection technique, was $91.3 \pm 7.5$ mm Hg/ml/min (± SEM) in the intact cat (Fig. 2). It decreased to $56.3 \pm 6.2$ mm Hg/ml/min following craniectomy and still further to $8.9 \pm 0.66$ mm Hg/ml/min after the dura was opened. These changes with successive alterations of container properties were significant ($p < 0.0001$). The resistance was independent of the CSF pressure throughout the range of 5 to 30 mm Hg.

In contrast to the values determined by bolus testing, the values of resistance to the absorption of CSF, determined by continuous infusion, varied as a function...
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Fig. 3. Pressure-volume curves constructed after bolus manipulations of cerebrospinal fluid in the intact cat, after bilateral craniectomies and after opening the dura. The steady-state intracranial pressure (ICP) is indicated by an arrow at steady-state volume. The pressure-volume curves depict sequential increase in volume-buffering capacity after craniectomy and durectomy. However, the steady-state ICP and ventricular volume are preserved after each alteration of the container because of the reciprocal decrease in outflow resistance. PVI = pressure-volume index, expressed in milliliters.

Discussion

One way of analyzing steady-state CSF pressure is to relate the sagittal sinus venous pressure (SSVP), rate of CSF formation ($I_f$) and absorption of CSF ($R_o$) by the formula: CSF pressure = ($I_f \times R_o$) + SSVP. In the present series of experiments, we have shown that changes in the tissues enveloping the brain cause minimal alteration in SSVP and CSF pressure while markedly decreasing resistance to CSF absorption. Using this steady-state equation and assuming relatively constant CSF production, the contribution of the ($I_f \times R_o$) product to total CSF pressure decreases from 22% in the intact cat to 13% after craniectomy and 6% after durectomy. Although SSVP remains constant after each alteration of the container, the progressive decrease in resistance to CSF absorption demonstrated by both bolus and infusion techniques makes the relative contribution of SSVP to total CSF pressure greater with each alteration of the container. For the most part, SSVP determines CSF pressure in the steady state. In settings with large changes in CSF hydrodynamics, the apparent effect on CSF pressure is minimal so long as the sinus pressure remains stable.

Intuitively, one would expect that maneuvers that increase the neural axis volume-buffering capacity would lower CSF pressure. This study demonstrates that, despite dramatic changes in pressure-volume index (PVI), steady-state CSF pressure does not change significantly. Conceptually, the increase in PVI following craniectomy with or without durectomy should favor the accumulation of volume within the neural axis. The sequential decrease in resistance to CSF absorption following each of these maneuvers facilitates the absorption of CSF and maintains steady-state CSF pressure.

The depiction of steady-state CSF pressure cited above does not include a parameter of volume storage. This equation relates changes of CSF hydrodynamics to CSF pressure in a system described by a fixed neural axis pressure-volume curve. If volume is added to the system at steady state, CSF pressure will change transiently along the pressure-volume curve, but will return to a steady-state CSF pressure as volume is absorbed along outflow pathways described by the resistance to CSF absorption parameter. Thus, the steady state is reestablished over time.

In the present series of experiments, we have altered the container of the neural axis and caused progressive shifts to new hydrodynamic steady states on different pressure-volume curves. While these maneuvers seemingly depict the “resetting” of the system on a hydrodynamic basis, a closer look shows that manipulations of the steady state lead to markedly different responses after altering the brain’s container (Fig. 4). Although the steady-state CSF pressure does not change, the potential ability of the system to accommodate added volume is enhanced markedly by cranial or dural opening. However, we have shown that each condition is a stable one with respect to the interaction between CSF hydrodynamics and volume-buffering capacity. Regardless of the potential for accommodating volume added to the system, CSF pressure returns to steady state after volume manipulations in each of the three states (Fig. 4). Thus, the response of CSF pressure to volume manipulation can be predicted by a pressure-volume curve unique to each of the three container states which holds for a wide range of CSF pressures (Fig. 3).

While both the PVI and resistance to CSF absorption parameters are independent of one another, the changes in each after the container of the brain is altered are compatible with maintenance of a stable balance between volume buffering and the circulation of CSF. If the resistance to CSF absorption did not decrease with opening of the skull and dura, CSF volume would accumulate because of the ease of volume storage induced by the increase in PVI.
One reason for performing this series of experiments was to characterize pressure-volume relationships in a model that approximates the expansile skull associated with infantile hydrocephalus. The present studies, as well as other reports, show that removal of portions of the calvaria are associated with increases in neural axis volume-buffering capacity.\textsuperscript{19} In contrast, previous studies from this laboratory have shown that normal infants have PVI's that differ from adults only because the total size of the neural axis is smaller.\textsuperscript{13,14} In that study, we did not find evidence for enhanced volume-buffering capacity created by open fontanels or sutures. While this clinical study appears to conflict with the results reported in this series of laboratory experiments, several differences can be offered to account for these results. First, the area of calvaria removed in the laboratory experiments is far greater than the areas of unfused bone in the normal infants. Second, the tensile strength at the sutural lines of infants probably exceeds that of the feline dura. Finally, the intact scalp of the human subjects probably helped stabilize the container in contrast to the present series performed with the scalp opened.

These acute studies demonstrate that neural axis pressure-volume relationships depend on an interaction between hydrodynamic parameters and volume-buffering mechanisms. In nonpathological settings, changes in one parameter are offset by reciprocal changes in another, which act to preserve not only steady-state pressure but also steady-state volume. Further studies will show how these parameters respond to pathological conditions.

Acknowledgment

We would like to thank Mrs. Carole Alvino for the preparation of the manuscript.

References

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Manuscript received September 14, 1984.
This work was supported by Teacher-Investigator Award NS00769-02 from the National Institutes of Health, by March of Dimes Research Foundation Grant 6-238, and by Basil O’Connor Starter Research Grant 5-251.
Address for Dr. Takei: Department of Neurosurgery, Tokai University School of Medicine, Kanagawa, Japan.
Address for Dr. Fried and Mr. Kohn: Department of Neurosurgery, The Albert Einstein College of Medicine, Bronx, New York 10461.
Address reprint requests to: Kenneth Shapiro, M.D., Department of Neurosurgery, Montefiore Medical Center, Moses 3, 111 East 210th Street, Bronx, New York 10467.