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Postural Changes in Intracranial Pressure in Chronically Shunted Patients

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Key Words

Fluid shift · Intracranial pressure · Postural changes · Pressure-volume index · Shunted hydrocephalus

Abstract

A subset of hydrocephalic patients with indwelling shunts become symptomatic when they are upright and active. Intracranial pressure (ICP) measurements in these patients have shown a significant drop in pressure when the patient is upright with return to normal levels when the patient is supine. In 20 chronically shunted hydrocephalic patients who previously had no siphon protection devices, ICP changes in supine and upright position were studied at the time when the patient had external ventriculostomy for treatment of shunt infection. Our hypothesis was that these patients might display rapid changes in ICP from fluid shifts occurring in non-CSF compartments. To minimize the effects of hysteresis, drift and zero-point error, measurements were made using a fluid manometer rather than a strain gauge pressure transducer. The pressure-volume index was estimated using the standard technique of bolus injection. Intracranial CSF volume was estimated on CT scans. The fluid shift was calculated using a mathematical model of the CSF compartment that incorporates negative pressure and volume components that permits simulation of siphoning. Sixteen patients had small, slit ventricles; 3 patients had moderate-sized ventricles and in 1 patient the ventricular size was normal. The average intracranial CSF volume estimated on CT scan was 12 cm^3. There was a mean drop in ICP in the upright position of 159 mm H_2O. The mean PVI of 42 ml suggested a volume displacement out of proportion to the available intracranial CSF volume. Based on these findings, we conclude that even in the absence of drainage through the shunt, chronically shunted patients still display a fall in ICP when assuming the upright position. This raises the possibility of fluid shifts other than of CSF through nonshunt pathways. Possible mechanisms involving altered CSF-venous system interaction are discussed.

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Introduction

When a normal subject assumes the upright position, the intracranial pressure (ICP) falls to about negative 5 mm Hg and gradually approaches zero [1]. Previous studies have demonstrated a derangement of postural ICP regulation caused by placement of a shunt in hydrocephalic patients [1–3]. ICP falls dramatically in these patients with ventricular shunts. This has been explained by the 'siphoning phenomenon': the large pressure difference due to the hydrostatic effect of the vertical length of tubing from the ventricles to the point of termination, usually the peritoneum, atrium or pleura, causing overdrainage of CSF [1]. This phenomenon may be responsible for persistence of symptoms such as headaches, nausea, vomiting, lethargy and even diplopia, paralysis of upward gaze and development of subdural hematomas in these patients [4–7]. Many of these symptoms improve with the placement of siphon protection devices [7], yet our experience suggests that some of these patients remain symptomatic despite these devices.

In order to better understand the pathophysiology of the postural changes in chronically shunted patients, we measured the changes in ICP as well as the pressure-volume index (PVI) in 20 patients with chronic hydrocephalus whose shunts had been externalized. Intracranial CSF volume was quantitated on CT scans. The data generated allows us to speculate whether displacements in CSF volume account for the posture-related changes in ICP.

Clinical Material and Methods

Patient Population

Twenty patients with hydrocephalus ranging in age from 6 to 23 years were included in the study. There were 9 males and 11 females with the majority having congenital hydrocephalus or posthemorrhagic hydrocephalus secondary to prematurity. All patients had ventricular catheters that were externalized for treatment of shunt infection. All patients were shunted at an early age and none were preselected for any particular variable such as type of shunt system, etiology of hydrocephalus, gender or age. None of the patients were critically ill.

ICP Recording

During the first part of the study the patient was in a supine position and the ICP was recorded using a fluid-filled manometer. The external auditory meatus was used as the zero reference. Next the measurement was made in the upright position using the same technique again with the external auditory meatus as the zero reference. Subsequent measurements were made after 5 and 10 min of being upright. All measurements were done in triplicate.

PVI Measurements

During the second part of the study, with the patient in the supine position, the ICP was recorded first using the same technique mentioned. Next, based on the previously described method of bolus injection [8–10] a total of 4–6 cm^3 normal saline was injected intraventricularly and the ICP was recorded at 2-cm^3 intervals. None of the patients experienced any complications from the measurements.

Measuring Total Intracranial CSF

This was done by measuring the volume of both intraventricular and extraventricular CSF spaces, on the CT scans based on methods previously described [11, 12]. Briefly, the regions of interest (ROI), that is, CSF spaces, were outlined on the CT scanner screen via a manually operated computer cursor. The total number of CSF-dense pixels within the ROI was automatically calculated by the scanner's software and expressed in square millimeters. Lastly, all ROIs were summed and multiplied by their slice thickness to yield a final intracranial CSF volume. Estimations were done on a General Electric (Milwaukee, Wisc., USA) CT scanner.

Results

ICP Fall and PVI Measurements

On average the ICP dropped 159 mm H_2O ($\pm$14). PVI was calculated using the formula: PVI = log change in volume/Final pressure-initial pressure. The average PVI was calculated to be 42.8 ($\pm$4.5). There was no significant change in ICP noted over the 10-min measurement interval.

Volume Shift

As noted before, during all measurements, no CSF was allowed to escape through the closed system. The volume shift was calculated using formulas derived from a computer model of siphoning for CSF shunt design evaluation [13] referred to as 'the negatively symmetric nonlinear model'. This model is identical to the PVI model proposed by Marmarou et al. [14] for positive pressures with the pressure asymptomatically approaching zero as fluid is withdrawn. However, unlike the Marmarou model, the negatively symmetric model allowed us to calculate volume changes during negative pressures. The average volume shift was calculated as 21.7 cm^3 ($\pm$2.7). The formulas used were as follows:

For pressures >0: $\Delta V1 = \ln (10/Ps) \times PVI/2.3$
For changes <0: $\Delta V2 = \ln [2 - (Pu/10)] \times PVI/2.3$

where PVI = pressure volume index, Ps = pressure during supine position, Pu = pressure during upright position. The total volume change was calculated as $|\Delta V1| + |\Delta V2|$.

Table 1. List of patients in the study with corresponding ventricular size and etiology of hydrocephalus

Patient No.	Age years	Sex	Ventricular size[1]	Etiology of hydrocephalus	ICP change when up cm H_2O	PVI	CSF volume cm^3
1	10	M	moderate	congenital	16	50.5	38.6
2	14	F	small	postmeningitis	23	48	12.8
3	23	F	small	IVH-premature	11	61.9	5.2
4	14	M	moderate	myelomeningocele	15	85	26.6
5	17	M	small	congenital	11	75.8	15
6	11	M	small	congenital	10	21.7	3.6
7	17	F	small	IVH-premature	21	44.5	4.3
8	9	F	small	postmeningitic	22	45.1	5
9	21	M	moderate	tumor	30	34.1	31
10	12	M	small	congenital	6	19.6	2.5
11	9	M	small	myelomeningocele	7	27	4
12	17	M	small	congenital	18	47	7.9
13	20	F	small	posttraumatic	23	87.4	15.8
14	15	M	small	term-IVH	21	32	3.1
15	13	F	small	IVH-premature	11	25.6	19.2
16	17	F	small	congenital	11	38	15.4
17	13	F	small	IVH-premature	18	26.4	18.1
18	5	M	normal	IVH-term	18	37.2	11.9
19	8	F	small	postmeningitic	9	25.1	1.3
20	5	F	small	IVH-premature	12	22	14.7
					Avg = 15.9 ± 1.4	Avg = 42.8 ± 4.5	Avg = 12.8 ± 2.3

[1] Criteria for large ventricles: CSF volume >25 cm^3 based on CT scans or size of both temporal horns >2 mm in width or ratio of FH/ID >0.5, where FH is the largest diameter of the frontal horns and ID is the internal diameter from inner table to inner table. Criteria for small ventricles: ventricles appearing slit-like or collapsed. Ventricles between large and small categories were designated as normal.

Intracranial CSF Volume

The average intracranial CSF volume based on CT scans was determined to be 12.8 cm^3 (± 2.3). The above data along with each patient's demographic and clinical information is presented in table 1.

Discussion

A number of investigators have estimated postural ICP changes by measuring lumbar CSF pressures [15–17]. The CSF pressure is equal to atmospheric pressure at the level of foramen magnum. Direct ventricular cannulation has confirmed that pressure above the foramen magnum is negative in upright posture [2, 18, 19]. Bradley [18] found an intraventricular pressure of negative 165 mm H_2O at the upper convexity of the head in a nonshunted patient with ventriculomegaly in the sitting position. Fox et al. [2] reported that, ICP varied from –140 to +280 mm H_2O in the sitting position in preshunted hydrocephalic patients. Following placement of shunt, upright ICP fell to –210 to –370 mm H_2O. In these studies, foramen of Monro (FOM) was used as the reference point. McCullough and Fox [6] in another larger series recorded pre- and post-shunt ICPs in the erect position referenced to the FOM and reported [2] that in the erect position, ventriculoatrial and ventriculoperitoneal shunt systems produced pressures averaging negative 238 and negative 251 mm H_2O, respectively. Chapman et al. [1] also investigated the relationship between ventricular fluid pressure and body position in normal subjects and chronically shunted patients using a telemetric device. They found that ventriculoatrial, ventriculoperitoneal and ventriculopleural shunts all caused similar severely abnormal ICP relation-

ships and that antisiphon devices were generally effective (with the exception of 1 out of 7 patients) in restoring normal pressures in patients in the upright position. They observed that ICP stabilized at markedly negative values within 15–30 s when subjects who had received shunts assumed an upright position. Our data suggest that in chronically shunted patients the almost instantaneous drop in ICP on upright position occurred even when the shunt was externalized and not allowed to drain.

Concept of the 'Starling Resistor' and 'Zero Point'

The exact mechanisms that result in changes in ICP in upright position are unclear. The two intracranial fluid compartments, which are coupled and can potentially change when a person goes from lying to a standing position, are the CSF and blood. The CSF is at a slightly higher pressure than the dural venous blood, which is why it moves from the subarachnoid space into the venous sinus [20–22]. As a consequence, it maintains a 'Starling resistor' relationship with the venous system [23]. This means that if the pressure in the venous sinuses drops below the ICP, the subarachnoid pressure will collapse the draining veins and the venous lacunae. This will shut off the communication between the veins and the dural sinus, thus preventing the siphoning of venous blood. In this way the venous drainage is independent of the drop in the dural sinus pressure and is autoregulated by the CSF pressure in upright position [24].

The compliance of the spinal compartment, however, allows movement of CSF from the intracranial compartment to the spinal compartment [25–27], resulting in the inferior displacement [15–17] of the zero point (ZP), the site at which the pressure in the craniospinal axis is atmospheric, which in the upright position, is at the level of the foramen magnum. As a consequence, the ICP, referenced to the FOM becomes negative. The subarachnoid pressure at the bridging veins is even more negative since it is situated above the ZP.

The 'ZP' of the venous blood column in upright position is at the right atrium, however, the jugular veins act as 'Starling resistors' since they are collapsible as soon as they exit the skull base and collapse shut if the intravenous pressure falls below atmospheric pressure. Since the jugular veins act as 'Starling resistors', the dural venous pressure is independent of the systemic venous pressure. Hence the pressure at the jugular bulb is close to atmospheric pressure and the sagittal sinus pressure is a reflection of the hydrostatic column from the jugular bulb to the vertex. Chapman et al. [1] also concluded from the data of Grady et al. [28] that the effective ZP for the cerebral venous system should be close to the jugular bulb and the external auditory meatus.

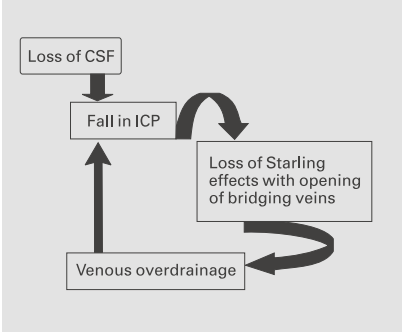

Fig. 1. 'Venous overdrainage' cascade.

Venous Relationships

During normal physiology, the subarachnoid pressure is higher (less negative) than the venous pressure and hence able to limit siphoning from the veins when a person stands up. The ICP changes are a result of movement of the CSF from the intracranial to the spinal compartment to the extent permitted by spinal compliance [27].

Our data suggests that in chronically shunted patients, for the PVI indices measured and the pressure changes recorded, the amount of fluid shift estimated to explain the degree of pressure drop exceeded the amount of CSF present intracranially. Based on this, we feel that the mechanism for ICP changes in upright position may be different in chronically shunted patients. If the subarachnoid pressure falls below the dural venous pressure, as a result of CSF drainage, the loss of the Starling effect will result in opening of the bridging veins, and siphoning of the venous blood, causing a rapid and marked drop in the ICP. This cascade of venous overdrainage (fig. 1) could explain the drop in ICP seen in this population of patients. The self-perpetuating cascade would terminate if, either, the dural venous pressure is made positive by lying down, resulting in a cessation of siphoning, or if the ICP is rapidly raised above the dural venous pressure. Lastly, significant vasodilation of cerebral arterioles, compensating for venous siphoning losses, could also terminate this cascade. To summarize, if the ZP is lowered below the dural venous sinus ZP, the uncontrolled venous overdrainage may result in the fall in ICP unless compensated by the mechanisms discussed above.

This study strongly suggests that the fall in ICP in chronically shunted patients is a result of uncontrolled 'venous overdrainage' since the CSF shifts are insufficient to explain the degree of the pressure drop. It confirms a previous observation by Chapman et al. [1] that a dramatic fall in ICP can be caused by a very small amount of CSF flow through the shunt. It reminds us that the shunted brain is in an abnormal state with altered physiology and that our understanding of the phenomenon known as siphoning is probably incomplete.

Pressure-Volume Index

We used the bolus injection method to measure the PVI. Previous studies have suggested that there is no substantial advantage of using continuous infusions that may be cumbersome and difficult to do in the population of patients in this study. Further bolus infusions may provide more stable and reliable estimates of PVI than the steady-state infusion [8]. We used the 'negatively symmetric nonlinear model' [13] to estimate the volume shifts. While conventional mathematical models of the pressure-volume relationship in the human central nervous system [29–32] were developed to understand the CSF dynamics in supine position, they cannot be extrapolated to allow calculations involving negative pressures in the upright position. We therefore used the 'negatively symmetric nonlinear model' [13] that is decidedly superior to using the conventional curves, which become mathematically void for negative pressure estimations (since there is no estimable log of a negative pressure). To estimate the volume shifts, we used the composite PVI of the cranial and the spinal axis. In reality, the volume shifts intracranially should be estimated using the cranial PVI only. Hence we may have overestimated the volume shifts. However, since the cranial PVI exceeds the spinal PVI, we feel this should not have significantly influenced our conclusions.

Conclusions

We conclude the following: (1) even in the absence of CSF drainage, chronically shunted patients display a fall in ICP when assuming the upright position, and (2) intracranial venous volume changes may have a greater role to play in the postural pressure changes than CSF fluid shifts in this population of patients. Together, these findings might explain why some of the chronically shunted patients may remain symptomatic despite addition of a siphon protection device. Moreover, this supports the concept that the intracranial venous system is of major importance in postural regulation of ICP in shunted patients.

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