Anti-siphon and reversible occlusion valves for shunting in hydrocephalus and preventing post-shunt subdural hematomas

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An anti-siphoning valve for use in hydrocephalic shunt systems is described. The addition of this valve to the system effectively reduces the hazard of negative intraventricular pressure when the patient is sitting or standing. The formation of post-shunt subdural hematomas was prevented by temporary post-operative occlusion of the shunt using a percutaneously reversible occlusion valve, which is also described.

KEY WORDS  • hydrocephalic shunts  • siphon effect  • ventricular hypotension  • subdural hematoma  • anti-siphon valve  • reversible occlusion valve

In 1892, Grashey on theoretical grounds suggested that the normal intracranial pressure in the erect position is subatmospheric, and in 1925, Ayer recorded the cerebrospinal fluid (CSF) pressure in the cisterna magna as being “below zero.” Masserman in 1934 observed that the mean CSF pressure in the sitting position is approximately equal to the distance to the cisterna magna. Pollock and Boshes confirmed experimentally the theoretical considerations of Grashey and concluded that “the cerebrospinal contents are exposed to atmospheric pressure only through the blood vessels,” presumably at the level of the foramen magnum since the major venous drainage emerges at the nearby jugular foramen.

Recently Bradley, Fox, et al., and McCullough, et al., demonstrated by direct measurement that pressure in the human ventricle is less than atmospheric pressure when the patient is erect. The normal intraventricular pressure (IVP) at the foramen of Monro is approximately −70 mm H$_2$O. 70 mm is the approximate vertical distance between the foramen magnum and foramen of Monro. Fox and McCullough also noted that ventriculoatrial (V-A) and ventriculoperitoneal (V-P)
shunts significantly lowered intraventricular pressure, the shunt acting as a siphon whose lower end vents to the atmosphere via the heart or peritoneal cavity at a significant distance below the foramen magnum.

Several problems have been noted as a consequence of lowered intracranial pressure secondary to shunting. In an infant, the erect posture can cause a sunken fontanel, overriding cranial bones, and irritability. Persistent close approximation of the cranial bones may be the cause of the cases of craniosynostosis reported as a complication of shunting. Similarly, persistent low IVP may convert a communicating hydrocephalus to a noncommunicating hydrocephalus with stenotic or occluded aqueduct. In the adult, low intracranial pressure may produce headaches. The most severe problem however, is the development of a subdural hematoma (SDH) or effusion.

To eliminate the deleterious "siphon effect," we (H.D.P., R.R.S.) have designed a valve that closes under the influence of a negative pressure. This report describes the "anti-siphon valve" and includes 13 cases in which the IVP was measured with and without the anti-siphon valve in the shunt system, demonstrating the effectiveness of the valve in limiting subnormal IVP in the erect position. Initially, we believed post-shunt subdural hematomas were caused solely by the siphon effect; however, six patients developed hematomas despite well-functioning anti-siphon valves. The data gleaned from these cases led to a re-evaluation of the hypothesis and a rationale and course of treatment of this complication.

Theory and Method

Theory

All present-day valves (Ames, Denver, Hakim, Holter, Pudenz) are differential pressure valves (DPV). The valve opens and closes whenever the hydrostatic opening or closing pressure of the valve is reached, whether this pressure is produced by a positive pressure at the inlet or a negative pressure at the outlet of the valve. As noted by Fox, et al., the perfusion pressure (P) through the shunt is equal to the IVP plus shunt hydrostatic pressure (HP) less the sum of the intra-atrial pressure (AP) (or intraperitoneal pressure) and the hydrostatic closing pressure of the shunt system (CP) (Fig. 1):

\[ P = IVP + HP - (AP + CP) \]

In the supine position the difference in height between the inlet of the shunt system in the cerebral ventricle and the outlet in the heart or peritoneum (HP) is nearly zero. This, however, is not the situation when the patient sits or stands; HP becomes a significant value and is proportional to the length of the distal catheter. Since a valve will remain open until P equals zero, when HP exceeds the sum of AP + CP, the intraventricular pressure will become negative.

In contrast to standard differential pressure valves, the anti-siphon valve closes whenever the pressure within becomes subatmospheric and thus eliminates the

\[ P = IVP + HP - (AP + CP) \]
Anti-siphon and reversible occlusion valves

![Diagram of anti-siphon valve in open and closed positions.](image)

Fig. 2. Diagram of anti-siphon valve in open and closed positions. Note a slight offset between diaphragm (D) and crown seat (CS). Also note that the area of diaphragm under the inlet is greater than that under the outlet (8:1).

siphon effect when the patient stands. Whenever the intraventricular pressure rises above atmospheric pressure, the valve opens, thus regulating ventricular pressure within a more normal range.

**Design**

The function of the anti-siphon valve can be understood by reference to Fig. 2. Whenever the pressure at the inlet rises above atmospheric, the diaphragm (D) is displaced away from the valve seat and fluid runs from inlet to outlet. If the pressure at the outlet drops below atmospheric, the diaphragm seats and closes the valve. With the areas beneath the diaphragms at the inlet and outlet the same size, negative pressure exerted at the outlet acting to close the valve requires a similar positive pressure at the inlet to open the valve. With this valve an adult hydrocephalic patient with a V-P shunt whose distal catheter is 800 mm long would require an IVP of 800 mm H₂O to overcome this siphon effect. To prevent such an increase in IVP, the area of the diaphragm under the inlet and outlet is adjusted to have a ratio of 8:1; thus, with 800 mm of distal catheter the valve opens at approximately 100 to 200 mm H₂O negative pressure is exerted at the outlet. The completed assembly is 9 mm in diameter and 3.3 mm in height. Since the anti-siphon valve has no mechanism for preventing reflux of blood into the shunt system, it must be used in conjunction with an appropriate differential pressure valve in V-A shunts.

**Testing**

Testing of the opening (OP) and closing (CP) pressures and flow through the valve was carried out as follows. A clear, water-filled, silicone rubber tube was suspended in front of a metric rule and attached to the inlet of the anti-siphon valve; by similar tubing the outlet was led beneath the water level of a glass reservoir. The distal reservoir was positioned so that its water level was between 0 and 800 mm below the anti-siphon valve in 100 mm intervals. The OP was determined for each negative outlet pressure by slowly raising the proximal tubing until flow began. The distance between the meniscus in the tubing and the ASV at the time of flow onset was considered the OP. For CP, flow was initiated in the proximal tube, and the tube lowered until flow stopped. For flow measurements a large bottle was substituted for the proximal tubing and a calibrated cylinder for the distal reservoir. Elevating the proximal water level above the anti-siphon valve produced positive pressures between 0 and 300 mm in 50 mm intervals, while lowering the distal catheter tip 0 to 800 mm in 100 mm intervals provided negative outlet pressures. Flow was then measured at a given inlet pressure for varying outlet pressure between 0 and −800 mm H₂O.

Figure 3 compares the OP and CP of an anti-siphon valve and the OP of a Hakim medium-pressure valve. Note that the OP of an anti-siphon valve is negative when there is a negative pressure of −100 to −200 mm H₂O at the outlet. This, however, is small (−50 mm H₂O) and not less than the normal negative pressure in the ventricle when the patient is standing (−70 mm H₂O). The inability of the Hakim valve to prevent the siphon effect is obvious. When the two valves are combined with the Hakim...
Fig. 3. Opening and closing inlet pressures of an anti-siphon valve (ASV) and the opening inlet pressure of a Hakim differential pressure valve under the influence of various negative outlet pressures. Note that the Hakim inlet pressure closely follows the negative outlet pressure while the ASV inlet pressure does not. When the Hakim is placed proximal to the ASV; the opening pressure of the Hakim valve becomes additive to the opening pressure of the ASV regardless of the negative outlet pressures.

Reversible Occlusion Valve

In all but Cases 2 and 3, a special valve assembly was used (Fig. 5). This consisted of a bubble reservoir (Foltz) connected to an anti-siphon valve (ASV) and a percutaneous reversible occlusion valve (PROV) which acted like an open-close switch (Fig. 6), the ASV and PROV being in parallel. When the PROV is closed, CSF must flow through ASV. When the PROV is open a direct pathway for CSF flow is established, and the ASV is bypassed. This latter condition is the same as that obtained with a standard V-P or V-A shunt. The open and closed position of the PROV could be determined both by palpitation and radiographically (Fig. 7). This combination al-
Anti-siphon and reversible occlusion valves

Fig. 5. Special valve combination used to evaluate intraventricular pressure in patients with anti-siphon valves. With percutaneously reversible occlusion valve (PROV) open (A), CSF flow bypasses ASV. With PROV closed (B), CSF is forced to flow through ASV.

Fig. 6. Percutaneously reversible occlusion valve. A. With valve in open position there is unobstructed flow from the ventricular to distal catheter. A differential pressure valve (DPV) can be incorporated into the base of the occlusion valve if desired. B. Finger pressure over dome (D) forces the radiopaque plug (P) into the outlet orifice (S), thus preventing the flow of CSF through the valve. C. Valve is reopened by occluding the inlet (arrow) of the reservoir and simultaneously depressing the reservoir dome. Trapped CSF is forced through orifice H and opens the valve when 1000 to 1500 mm Hg pressure is exceeded. Note also the radiopaque ring (M), which allows easy radiographic identification of the open or closed valve.

Summary of Cases

All IVP measurements in these case reports are referred to the level of the foramen of Monro. Except when obtaining measurements or as indicated, the anti-siphon valve was included as part of the shunt pathway.

Table 1 summarizes 13 hydrocephalic patients in whom IVP measurements were obtained with and without an anti-siphon valve introduced into a V-P or V-A shunt system with a low opening pressure slit valve. In Cases 2 and 3, reoperation was necessary, and the initial shunt system was modified by the inclusion of the anti-siphon valve just distal to a Mishler valve-reservoir.

Discussion

The anti-siphon valve was developed to reduce subnormal IVP produced by present-day shunt systems. As clearly demonstrated by Cases 6 and 11, the ASV prevents depression of the fontanel and overriding of the cranial bones when an infant is held erect. When the cranial sutures are closed, the ASV prevents abnormally low IVP when the patient is sitting or standing, as in Cases 1-5 and 7-13. It will require an extensive series of patients to determine if the ASV eliminates low-pressure headaches, secondary aqueductal stenosis or occlusion, and post-shunt craniosynostosis; Cases 2, 6, 7, and 9 suggest that the ASV may alleviate low-pressure headaches.

The ASV alone will not decrease the incidence of post-shunt subdural hematomas. This was evident in Cases 1, 2, 5, 8, 10 and 13 despite the fact that in each instance the ASV was effective in preventing markedly low IVP when the patient was placed in the sitting position. It is probable that operative manipulation and the acute drop in ICP produced by removal of CSF at surgery cause a separation of arachnoid and dura as well as a rebound engorgement of the cerebral vasculature, particularly the veins. Both factors could cause sub-
TABLE 1

Summary of 13 cases of hydrocephalus treated by anti-siphon valve shunt system

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Etiology</th>
<th>Shunt*</th>
<th>Intraventricular Pressure (mm H₂O)†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Position</td>
<td>Without ASV</td>
</tr>
<tr>
<td>1</td>
<td>57 yrs, M</td>
<td>chronic arachnoiditis causing hydrocephalus as child (slowly progressive, untreated)</td>
<td>V-P</td>
<td>sitting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>45°</td>
<td>-180 (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>supine</td>
<td>-10 (10)</td>
</tr>
<tr>
<td>2</td>
<td>53 yrs, F</td>
<td>normal pressure hydrocephalus, disorientation, headache, abnormal gait</td>
<td>V-P</td>
<td>sitting</td>
</tr>
<tr>
<td>3</td>
<td>53 yrs, M</td>
<td>normal pressure hydrocephalus</td>
<td>V-A</td>
<td>sitting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>45°</td>
<td>-200 (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>supine</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>62 yrs, M</td>
<td>acute hemorrhagic necrosis of pituitary tumor, bitemporal hemianopsia</td>
<td>V-P</td>
<td>sitting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>45°</td>
<td>-85 (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>supine</td>
<td>+5 (10)</td>
</tr>
<tr>
<td>5</td>
<td>46 yrs, M</td>
<td>normal pressure hydrocephalus secondary to clipping of aneurysm of ant. comm. artery</td>
<td>V-P</td>
<td>sitting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>45°</td>
<td>-215 (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>supine</td>
<td>-60 (10)</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>Arnold-Chiari malformation with myelomeningocele, depressed fontanels, overriding cranial bones when sitting</td>
<td>V-P</td>
<td>sitting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>45°</td>
<td>-75 (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>supine</td>
<td>+50 (10)</td>
</tr>
<tr>
<td>7</td>
<td>15 mos, F</td>
<td>E. coli ventriculitis at 1 mo (before V-P shunt with Mishler reservoir, low pressure Pudenz peritoneal catheter), irritable when sitting</td>
<td>V-P</td>
<td>sitting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>45°</td>
<td>+5 (10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>supine</td>
<td>+70 (10)</td>
</tr>
</tbody>
</table>
### TABLE 1 Continued

**Summary of 13 cases of hydrocephalus treated by anti-siphon valve shunt system**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Etiology</th>
<th>Shunt*</th>
<th>Intraventricular Pressure (mm H₂O)†</th>
<th>Shunt Position</th>
<th>Without ASV</th>
<th>With ASV</th>
<th>Comments‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>77 yrs, M</td>
<td>normal pressure hydrocephalus secondary to traumatic subarachnoid hemorrhage and bilateral subdural hematomas</td>
<td>V-P</td>
<td>sitting</td>
<td>45°</td>
<td>-360 (60)</td>
<td>-90 (60)</td>
<td>No improvement; fell 6 wks post-shunt and underwent subtemporal craniectomies for bilateral SDH. Still no improvement.</td>
</tr>
<tr>
<td>9</td>
<td>43 yrs, M</td>
<td>normal pressure hydrocephalus secondary to subarachnoid hemorrhage from aneurysm of ant. comm. artery</td>
<td>V-P</td>
<td>sitting</td>
<td>45°</td>
<td>-340 (10)</td>
<td>0</td>
<td>Pre-shunt IVP: sitting = -80 mm H₂O; 45° = -80 mm H₂O; supine = +120 mm H₂O. No post-shunt improvement. Headache when PROV left open 5 days before IVP measurements, disappeared after PROV closed and CSF shunted via ASV.</td>
</tr>
<tr>
<td>10</td>
<td>5 yrs, F</td>
<td>optic chiasm glioma with partial obstruction of 3rd ventricle</td>
<td>V-P</td>
<td>sitting</td>
<td>45°</td>
<td>-130 (60)</td>
<td>-60 (60)</td>
<td>Bilateral SDH; subtemporal craniectomies for SDH and frontal craniotomy for tumor biopsy. Medium pressure valve substituted for low-pressure valve. SDH increased in size; subdural-peritoneal shunt followed by depression of craniectomies. Child steadily improved after initial shunt procedure despite SDH.</td>
</tr>
<tr>
<td>11</td>
<td>14 days, F</td>
<td>Arnold-Chiari malformation with myelomeningocele</td>
<td>V-P</td>
<td>sitting</td>
<td>45°</td>
<td>-55 (60)</td>
<td>+20 (60)</td>
<td>Normal head growth; fontanel flat but not depressed when child erect.</td>
</tr>
<tr>
<td>12</td>
<td>44 yrs, M</td>
<td>normal pressure hydrocephalus after clipping aneurysm of ant. comm. artery; stuporous, quadriaparetic, confused</td>
<td>V-A</td>
<td>sitting</td>
<td></td>
<td>-300 (60)</td>
<td>3 occasions</td>
<td>Rapid improvement after shunt, awake, feeds self, walks with help, still confused.</td>
</tr>
<tr>
<td>13</td>
<td>54 yrs, M</td>
<td>normal pressure hydrocephalus, confused, poor coordination, ataxic gait</td>
<td>V-A</td>
<td>sitting</td>
<td></td>
<td>-250 (60)</td>
<td>-20 (60)</td>
<td>After shunt, developed subdural effusion, with PROV left open (siphoning). Effusion drained and PROV closed; slow clinical improvement.</td>
</tr>
</tbody>
</table>

* V-P = ventriculoperitoneal shunt, V-A = ventriculoatrial shunt.
† IVP = Intraventricular pressure in mm H₂O with reference to foramen of Monro. Numbers in parenthesis following each pressure measurement indicate time in minutes patient was in indicated position before measurement obtained.
‡ SDH = subdural hematoma. PROV = percutaneously reversible occlusion valve.
subdural hematoma in animals. The situation is further enhanced by a continued reduction in ICP as a result of the shunt procedure. In fact, the continuation of a low-pressure shunt system with ASV still resulted in a subnormal IVP in these patients, particularly when measured with the patient supine. It is interesting that Bradley measured a particularly low IVP in the sitting position in a patient with cerebral atrophy. A low IVP in the sitting position was also found prior to shunting in our Case 8, a patient whose normal pressure hydrocephalus was secondary to bilateral subdural hematomas.

As Davidoff and Feiring noted, a subdural hematoma may produce symptoms after varying periods of time; in a hydrocephalic patient with an increased CSF compartment, this interval may be quite long. For example, in our Cases 1, 10 and 13, the subdural hematoma was asymptomatic and only found by routine postoperative angiography. The rapidity of development of a hematoma will depend on the degree of negative pressure that exists. The inclusion of an anti-siphon valve, which minimizes a negative IVP following a shunt, probably reduces the rate of formation of the hematoma when compared to that in a shunt system with only a differential pressure valve. These findings are confirmed by long-term measurements of intracranial pressure carried out by Symon on a patient who had a V-A shunt plus anti-siphon and reversible occlusion valves. Without the anti-siphon valve in the system, the IVP was 60 mm H₂O while the patient was supine and -425 mm H₂O after sitting 120 min. With the ASV in place, pressure in the supine position was 80 mm H₂O and after sitting 135 min, -70 mm H₂O. Prolonged epidural measurements with the patient in many positions revealed that there were long periods of time in which the ICP was equal to or below the atmospheric pressure with and without the ASV; the ASV, however, increased the length of positive pressure measurements. Symon's patient developed an asymptomatic subdural hematoma; the ASV, however, was functional only during the time ICP measurements were taken, and thus the system was primarily siphoning.

Fig. 7. Radiographs of a valve assembly that includes a PROV as seen from the side and slightly oblique. The open position is readily determined by noting that the radiopaque plug is outside the radiopaque ring. In the closed position, the plug is inside the ring.
Anti-siphon and reversible occlusion valves

It is evident that prevention of a subdural hematoma depends on having a sealed subdural space and preventing an excessively low intraventricular pressure following a shunt procedure. Careful operative technique attempting to prevent opening of the subdural space and loss of ventricular fluid is important, but, in reality, difficult to achieve. Resealing of the subdural space can be accomplished if the shunt is temporarily rendered nonfunctional, thus maintaining the original pressure relationships. Temporary occlusion should not be difficult in patients with normal pressure hydrocephalus or craniocerebral disproportion due to an enlarged cranium, since their condition is chronic, not acute. These patients are also the individuals most likely to develop a post-shunt hematoma. This temporary occlusion, which would usually require reoperation to close and then re-establish shunt patency, can be accomplished without reoperation by using the reversible occlusion valve which can be turned on and off percutaneously. With such a unit, surgery is performed and the system left in the “off” position. After 1 week the patient is evaluated by angiography; if there is no hematoma, the shunt is opened. To prevent persistently low IVP, an anti-siphon valve should be used in conjunction with a standard differential pressure valve having a closing pressure of 70 to 100 mm H₂O and placed proximal to the ASV. With the use of this technique, a subdural hematoma has not formed in nine recently shunted patients with normal pressure hydrocephalus or craniocerebral disproportion.

Since there is a high incidence of post-shunt subdural hematomas in adults, anti-siphon valves depend on having a sealed subdural space and preventing an excessively low intraventricular pressure following a shunt procedure. Careful operative technique attempting to prevent opening of the subdural space and loss of ventricular fluid is important, but, in reality, difficult to achieve. Resealing of the subdural space can be accomplished if the shunt is temporarily rendered nonfunctional, thus maintaining the original pressure relationships. Temporary occlusion should not be difficult in patients with normal pressure hydrocephalus or craniocerebral disproportion due to an enlarged cranium, since their condition is chronic, not acute. These patients are also the individuals most likely to develop a post-shunt hematoma. This temporary occlusion, which would usually require reoperation to close and then re-establish shunt patency, can be accomplished without reoperation by using the reversible occlusion valve which can be turned on and off percutaneously. With such a unit, surgery is performed and the system left in the “off” position. After 1 week the patient is evaluated by angiography; if there is no hematoma, the shunt is opened. To prevent persistently low IVP, an anti-siphon valve should be used in conjunction with a standard differential pressure valve having a closing pressure of 70 to 100 mm H₂O and placed proximal to the ASV. With the use of this technique, a subdural hematoma has not formed in nine recently shunted patients with normal pressure hydrocephalus or craniocerebral disproportion.

Since there is a high incidence of post-shunt subdural hematomas in adults, anti-siphon valves should be performed as a precautionary measure 1 week after a shunt procedure. If a hematoma is present, initial drainage through a subtemporal craniectomy is recommended. As shown by Weed and Flexner, a craniectomy will serve to increase intracranial pressure toward atmospheric when the patient is upright and, in addition, act as an absorbing surface if the dura is left open. If simple drainage and subtemporal craniectomy fail, then the hematoma can be shunted, without an anti-siphon valve, into the peritoneal cavity as is done in infants. When the patient is erect, the anti-siphon valve, as part of the V-A or V-P shunt, prevents reduced pressure in the ventricles, while the subdural-peritoneal shunt without the anti-siphon valve tends to “suck” out the hematoma; thus a pressure gradient is developed that tends to collapse the subdural fluid collection.

Samuelson, et al., have reported five cases of normal pressure hydrocephalus that subsequently developed subdural hematomas. In two cases in which the subdural hematoma developed 10 and 11 months post-shunt, the onset of the hematoma could be related to antecedent head injury. The three other patients became symptomatic in the first month after surgery. We believe, contrary to the opinion of Samuelson, et al., that in these instances the hematomas were initiated at surgery and slowly progressed to become symptomatic over the ensuing month.

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