

# Lumbar CSF Shunting Preferentially Drains the Cerebral Subarachnoid over the Ventricular Spaces: Implications for the Treatment of Slit Ventricle Syndrome

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## Key Words

Lumboperitoneal shunt · Subarachnoid space · Slit ventricle syndrome

## Abstract

Based on a proposed pathophysiology of slit ventricle syndrome (SVS), we have hypothesized that lumboperitoneal shunting exerts effects in SVS patients by increasing the buffering capacity for raised intracranial pressure (ICP) via an increase in cerebrospinal fluid drainage from the cerebral subarachnoid space (SAS). We describe 3 SVS patients with patent lumbar subarachnoid drainage but under-functioning ventriculoperitoneal shunts (VPS) who presented with ventriculomegaly (not SVS), and persistence of shunt malfunction like symptoms. Revision of the VPS resulted in complete resolution of symptoms despite a finding of low pressure in the ventricular space. This supports the hypothesis that lumboperitoneal shunting preferentially drains the SAS over the intraventricular space and in these cases allows the 'SVS' ventricles to enlarge by creating a pressure gradient from ventricles to SAS through the cortical mantle.

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## Introduction

The modern era of shunting for hydrocephalic patients began in 1949 with Nulsen and Spitz inserting a unidirectional, pressure-responsive valve into a ventriculovenous shunt system [2, 3]. Since then, slit ventricle syndrome (SVS) has been well known as a complication in the treatment of hydrocephalus by shunting. In 1993, Rekate defined SVS as the triad of intermittent headaches lasting 10–30 min, smaller than normal ventricles on imaging, and slow refill of the palpable valve [4]. It is generally considered to be a chronic complication, occurring years after the initial shunt procedure with the average time from the first shunt operation to the development of SVS being approximately 6.5 years [5, 6]. The incidence of SVS is hard to ascertain from the literature with a range of 1–37% being reported [1, 6–10]. More specifically, of the 370 patients shunted by Walker et al. [11], 60–80% were reported as developing slit ventricles, whereas only 11.5% developed SVS, and only 6.5% of those patients required surgical intervention.

Over the past decade, the pathogenesis of SVS has resulted in a constellation of signs and symptoms in children with either a functional or a nonfunctional ventriculoperitoneal shunt (VPS) who concurrently have radiological findings of small ventricles on CT or MRI. SVS may be completely asymptomatic or have symptoms consistent with intracranial hypertension (nausea, vomiting,

**Table 1.** Treatment strategy for SVS

Symptoms	ICP on LP	VPS	Imaging	Drainage of CSF	Treatment
Asymptomatic	Normal	Functional	SVS	Overdrainage	Trial of externalization
Increased ICP (nausea, vomiting, decreased mental function, chronic migraines/headaches)	Increased	Non-functional	SVS	Underdrainage	Revision of VPS
Decreased ICP (postural headaches)	Decreased	Functional	SVS	Overdrainage	High-resistance valve, anti-siphon device, LPS
Increased ICP (nausea, vomiting, decreased mental function, chronic migraines/headaches)	Increased	Functional	SVS	Underdrainage	Cranial expansion, 3rd ventriculostomy, LPS placement

ICP = Intracranial pressure; LP = lumbar puncture; SVS = slit ventricle syndrome; LPS = lumboperitoneal shunt.

decreased mental function, chronic migraines/headaches) or intracranial hypotension (postural headaches) [10, 12].

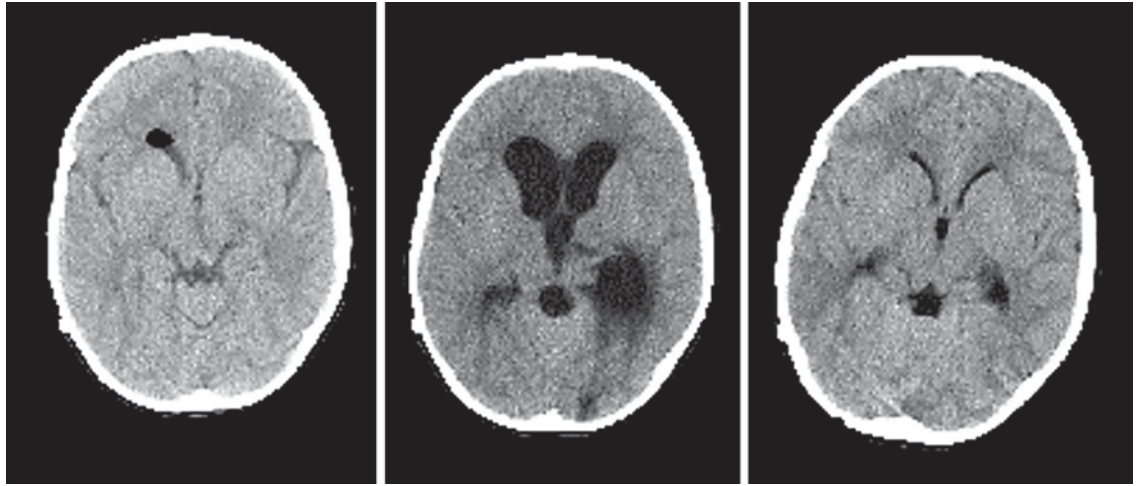
Patient management (table 1) is often determined depending on the patient's symptoms, the presence or absence of aberrant intracranial pressure (ICP), and the functional status of the VPS [4]. An asymptomatic and neurologically stable patient with radiological evidence of SVS may benefit from a trial of externalization with ICP monitoring for potential removal of the VPS [13]. A patient with symptoms of increased ICP, evidence of increased ICP on lumbar puncture (LP), small ventricles on imaging, and evidence of VPS malfunction will often see resolution of symptoms with a VPS revision [4, 11]. Moreover, a patient with symptoms of decreased ICP, evidence of decreased ICP on LP, small ventricles on imaging, and evidence of a functional VPS will often see resolution of symptoms with placement of a high-resistance valve, or antisiphon device [14]. However, a patient with symptoms of increased ICP, evidence of increased ICP on LP, small ventricles on imaging, and evidence of a functional VPS will often see resolution of symptoms with cranial expansion, subtemporal decompression, or third ventriculostomy [15–19]. More recently, Le et al. recommended the placement of a lumboperitoneal shunt (LPS) to preferentially drain the subarachnoid space (SAS) in this subgroup of patients [1, 20, 21].

The subgroup of patients with SVS who present with increased ICP, not related to shunt malfunction, represent a therapeutic problem. There exists no generally accepted treatment for this condition. Treatment strategies have ranged from subtemporal decompression and calvarian expansion surgeries to LPS placement all with the

intent of reducing the ICP [1, 11, 18, 22]. We describe 2 patients with a history of communicating hydrocephalus who were shunted at infancy and presented years later with symptoms of increased ICP, evidence of increased ICP on LP, small ventricles on imaging, and a functional VPS. They were diagnosed with SVS and a LPS was placed with resolution of their symptoms. A third patient presented with SVS and underwent a lumbar drain trial to resolve his symptoms. The first two patients, subsequently, presented with symptoms of increased ICP, but low ICP as evidenced by LP, large ventricles on imaging, a functional LPS, but underfunctioning VPS. At this time, they were diagnosed with VPS malfunction and underwent VPS revision with resolution of symptoms. Ventricular pressure at the time of revision was low. The third patient with lumbar drainage to a very low pressure also experienced symptomatic ventricular enlargement during the lumbar drain trial and was diagnosed with VPS malfunction but was found to have low pressure on VPS exploration. That VPS underdrainage in a patient with communicating hydrocephalus with a functional LPS or lumbar drain results in ventriculomegaly and that revision of the VPS to allow lower ventricular pressure resulted in resolution of symptoms, supports the hypothesis by Le et al. [1] that lumbar cerebrospinal fluid (CSF) catheters preferentially drain the convexity SAS over the ventricular system.

## Methods

Charts were reviewed for patients with a diagnosis of SVS who had previously undergone LPS or lumbar drain placement per Le et al. [1] and subsequently presented with recurrence or persistence



**Fig. 1.** Left panel: patient 1 with SVS (functional VPS, but elevated lumbar CSF pressure); intraventricular air is from LP pressure measurement; middle panel: patient 1 with nonfunctional VPS and functional LPS; right panel: patient 1 with functional VPS and LPS.

of their hydrocephalus symptoms and ventriculomegaly on CT and low ICP on LP/drain. Determination of VPS system function was performed either via a shunt tap or by intraoperative VPS exploration. If a malfunctioning VPS system was discovered, the patient underwent VPS revision.

### Case Material

#### Case 1 (fig. 1)

A 9-year-old female was diagnosed with communicating hydrocephalus at 2 months of age at which time a VPS was placed. Since then she had undergone numerous revisions of her VPS. She presented with SVS and a LPS was placed at age 8 years when elevated CSF pressures were diagnosed and temporarily treated by LP with resolution of symptoms (fig. 1, left panel). One year later, she presented ventriculomegaly despite a working LPS and low lumbar-CSF pressures (<5 cm of water). Despite this, VPS exploration was undertaken and an occluded ventricular catheter was replaced. Upon placing the new catheter ventricular pressure was found to be 6.5 cm of water.

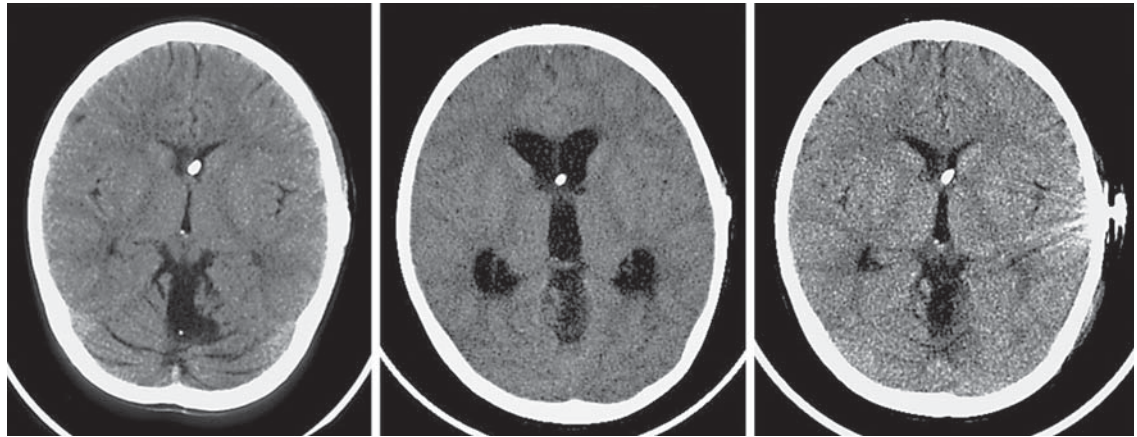
#### Case 2

A 6-year-old male was diagnosed with communicating hydrocephalus at 2 weeks of age at which time a VPS was placed. He had undergone numerous revisions of his VPS and eventually required revision of the VPS to a ventriculoatrial shunt. At 5 years of age he presented with SVS not treated by ventriculoatrial shunt revision and a LPS was placed. Nine months later, the patient presented with intermittent headache, irritability and fatigue. CT showed small ventricles and an intraoperative LP revealed an opening pressure of 45 cm of water. Because of this, the LPS was explored and an occluded distal catheter was replaced. The patient's preoperative symptoms did not improve postoperatively and another LP was performed and the patient was found to have an opening pressure

of 7 cm of water. CT scan performed at the time revealed gross ventriculomegaly observed for the first time in several years. The patient was returned to the operating room for investigation of his VPS and a partially occluded ventricular catheter was replaced. Intraventricular pressure at the time of catheter change was 8–10 cm of water. After VPS revision, the patient's neurological exam and his activity level returned to baseline. His CT showed small ventricles that were slightly larger than his SVS baseline (data not shown).

#### Case 3 (fig. 2)

An 8-year-old male had presented with cerebellar astrocytoma at an early age which required three attempts at resection before total removal. After the third resection, he developed four-ventricle communicating hydrocephalus and a VPS was placed. Shortly thereafter he developed SVS and underwent nearly 50 VPS revisions over a 2-year period at outside institutions for symptoms of VPS malfunction despite little or no change in his small ventricular size (fig. 2, left panel). He presented to our institution at age 8 years for a trial of lumbar drainage before LPS placement as per Le et al. [1] to provide him with adequate drainage. Several hours after lumbar drain placement, he presented with near coma and significant ventriculomegaly on CT (fig. 2, middle panel) despite a working lumbar drain set initially at –10 cm of water to his spine in the supine position. Urgent VPS exploration was undertaken and a possibly occluded ventricular catheter was replaced. The shunt was upgraded with a nonsiphoning programmable valve. Ventricular pressure measured at the time of exploration was 0 cm of water. The patient's symptoms resolved with the working VPS (set at a pressure of 7 cm water) and the lumbar drain also set at 7 cm of water. LPS was eventually placed with opening pressure of 7 cm of water with continued symptom resolution and ventricular size slightly larger than at presentation (fig. 2, right panel). Over the following 2 years he underwent only 2 LPS revisions for valve upgrades. His VPS remains functional.



**Fig. 2.** Left panel: patient 3 with SVS (VPS, but no LPS); middle panel: patient 3 with ventricular pressure found to be 0 cm water and lumbar drain set at -10 cm water; right panel: patient 3 with functional and pressure matched VPS and LPS.

**Table 2.** Patient profile

Gender, age (years)	Cause/type of hydrocephalus	Age of VPS placement	Age at diagnosis of SVS/LPS placement years	Ventricle size on CT at time of first VPS revision after lumbar drainage	VPS malfunction, component replaced	Ventricular pressure/lumbar pressure at revision of VPS cm of water
F, 9	congenital/communicating	2 months	8/8	Large (fig. 1)	Yes, proximal catheter	6.5/<5
M, 6	congenital/communicating	2 weeks	5/5	Large	Yes, proximal catheter	10/7
M, 8	Post tumor resection/communicating	5 years	6/8	Large (fig. 2)	?, valve	0/-10

VPS = Ventriculoperitoneal shunt; LPS = lumboperitoneal shunt; SVS = slit ventricle syndrome.

## Results

Patient demographics are displayed in table 2. The average time from initial shunt placement to the development of SVS was approximately 4.7 years (a range of 1–8 years). The patients were then treated with LPS or lumbar drain placement with resolution of signs and symptoms. Subsequently, all patients presented with symptomatic ventriculomegaly by CT and low lumbar CSF pressures (-10 to 7 cm of water). They were taken to the operating room for VPS exploration where a pressure gradient of between 1.5 and 10 cm of water was noted between ventricular and lumbar CSF (table 2). The VPS in each case was revised or upgraded to match the lumbar drainage. Postoperatively, in the presence of both a functioning VPS and LPS, all patients had improved neurological exam, resolved symptoms, and ventricles that had slightly enlarged from their SVS baseline.

## Discussion

By introducing a shunt into a closed system, the system will adjust until a new equilibrium is reached. With an intraventricular catheter, a balance must be reached between forces dilating and contracting the ventricle. Forces contracting the ventricles include increased elastance over time due to maturation-associated changes such as myelination and glial proliferation. This is suggested by the peak prevalence of SVS being between 5 and 10 years of age in children who have been shunted at infancy [23, 24]. Another force resisting ventricular dilation in patients with VPS is the increase in elastance attributed to subependymal gliosis promoted by the long-term presence of ventricular drainage [19]. In addition to increased elastance of the brain parenchyma over time causing the ventricles to collapse, patients with a VPS often have depressed intraventricular pulse pressure, an important

**Table 3.** Summary of SVS etiology and possible treatment strategies

SVS causes	Treatment
<i>VPS mechanics</i>	
*Siphoning effect ↓ Intraventricular pressure	—————→ *High resistance or Antisiphon device
*↓ Intraventricular pulse pressure ↓ Brain compliance ↓ Force dilating ventricles	—————→ *Replace VPS for LPS
*Malfunction	—————→ *VPS revision
<i>Brain buffering capacity</i>	
*↑ Venous pressure ↑ Cerebral pressure d/t postshunt craniostylosis	} —————→ *LPS *Cranial expansion surgery
*↑ Extraparenchymal resistance d/t fused cranium	
*CSF absorptive capacity	
*Age-related ↑ resistance to cerebral deformity	

SVS = Slit ventricle syndrome; VPS = ventriculoperitoneal shunt; LPS = lumbo-peritoneal shunt; d/t = due to.

force in dilating the ventricles [25]. Hydraulic and mechanical mismatching of CSF shunts to individual patients may lead to overdrainage with resultant SVS or underdrainage with resultant ventriculomegaly [16].

*SVS Etiology and Treatment Strategies* (table 3)

In patients with a VPS, the siphoning effect of the distal catheter on the ventricular system results in overdrainage with symptoms which are postural in nature. In this setting of chronic overdrainage of CSF, a chronic decreased intraventricular volume results in decreased intraventricular pressure [7, 14, 21]. Both decreased intraventricular pressure and increased brain elastance of shunted patients help establish and maintain slit-like ventricles. Treatment options are therefore to increase brain compliance or increase intraventricular pressure. Normalizing the intraventricular pulse pressure, which can be achieved with the replacement of the VPS by an LPS, can increase compliance of the brain. LPS with its proximal catheter in the SAS – presumably preferentially draining the convexity of the SAS, not the intraventricular space, is considered not to affect intraventricular pulse pressure [26]. Decreasing the siphoning effect of the distal catheter with the use of a high resistance or antisiphon VPS can increase intraventricular pressure. A trial of externalization for potential VPS removal in this situation can also address the abnormal CSF shunting dynamics [13].

Ventricular underdrainage in the setting of SVS with increased ICP due to a malfunctioning VPS is treated with a VPS revision [4, 11]. If the VPS is functional, then the main pathophysiology for increased ICP with SVS is due to decreased buffering capacity of the brain and VPS mechanics.

Decreased buffering capacity can be due to increased venous pressures and hence reduced cerebral blood flow due to postshunting craniostylosis, increased extraparenchymal resistance due to a fused cranium, decreased CSF absorptive capacity in the SAS with communicating hydrocephalus, or age-related increased resistance to cerebral deformity [9, 15]. Chronic overdrainage of CSF via shunts during infancy dampens the normal cerebral pressure waves. Growth of the calvarium is thus understimulated leading to ossification of the sutures, which become unable to expand to allow normal brain growth [27]. By limiting the growth of the calvarium and hence the total calvarium volume, a new balance must be reached, often with resultant increased ICP. This is the rationale used by Eide et al. [17] for calvarial expansion surgery for treatment of SVS patients with small heads. In their study, they found that in children with SVS and intracranial hypertension, despite a functioning ventricular shunt, calvarial expansion may produce long-lasting relief from symptoms with an associated decrease in ICP and no increase in ventricular size.

### *SVS and CSF Drainage Dynamics*

VPS mechanics include preferential drainage of the intraventricular space and decreased intraventricular pulse pressure and subsequent decreased ventricular compliance [7, 14, 21, 25]. The VPS's primary access is to the CSF in the lateral ventricle, with the foramen of Monro acting as a valve mechanism that usually closes in response to CSF withdrawal from the lateral ventricle [28]. With excess ventricular drainage, displacement of the septum pellucidum and functional occlusion of the foramen of Monro restricts flow from the remaining compartments to the lateral ventricle. With communicating hydrocephalus, when retrograde flow is impaired by occlusion of the foramen of Monro, the VPS no longer has access to CSF generated by choroid plexus in the third and fourth ventricles. There is redistribution of the CSF from the ventricular space to the SAS, distending the space, forcing the brain inward, and thereby causing temporary or permanent occlusion of the ventricular catheter [20]. The preferential drainage of the intraventricular space by a VPS causing decreased compliance, decreased dilating forces for the ventricles, and decreased pressure buffering capacity, allows for an increase in the extraventricular CSF volume with subsequent decrease in the intraventricular CSF volume. This situation seems likely to lead to a pressure gradient from SAS to ventricle and increased ICP with slit-like ventricles.

LPS placement and various cranial expansion surgeries can increase buffering capacity of the brain [15–19]. In addition to increasing the buffering capacity, LPS placement seems to circumvent VPS mechanics and directly drain the SAS [1]. LPS with access to the SAS prevents distention of the potential space, averting the inward force on the brain leading to slit-like ventricles and ventricular catheter occlusion. In a report comparing LPS to VPS, Aoki [26] found 2% of patients with VPS developed SVS, while none of their 16 patients with LPS developed SVS. Furthermore, since the lumbar catheter withdraws CSF from the SAS, it encourages forward flow through the foramen of Monro and may prevent closure of the foramen. Thus, an optimal balance between VPS and LPS systems can be used to treat the subgroup of SVS patients with high ICP and functional VPS.

Le et al. [1] demonstrated that in children with SVS and intracranial hypertension despite functioning ventricular shunts, LPS placement may produce long-lasting relief of symptoms. This effect was associated with a marked reduction of ICP, and was not related to significant changes in the size of the cerebral ventricles on the CT scans. An age-related resistance to cerebral deformity

and an increase in extraparenchymal resistance due to a fused cranium, and not ventricular adhesions, may explain the lack of ventricular dilation despite high ICP. As Del Bigio et al. [29] suggests, adhesions between chronically collapsed ventricles could retard, but not prevent enlargement of the ventricles with shunt obstruction. In their case report of an autopsy done on a patient with SVS, they found that the intraventricular adhesions were not dense and clearly did not seal the ventricles shut. In our series, 3 SVS patients who were treated with lumbar SAS drainage subsequently presented with ventriculomegaly and low ICP. Low-ICP related ventriculomegaly with a LPS functionally draining more than a VPS in a SVS patient likely indicates that lumboperitoneal shunting preferentially drains the SAS. This situation may decrease the extraparenchymal resistance and allow for ventricular enlargement by constructing a pressure gradient from ventricle to SAS – the opposite situation from one conceptualization of SVS etiology presented above. That a reduction in VPS drainage pressure to eliminate such a gradient in this setting (by catheter or valve revision) results in resolution of ventriculomegaly supports this hypothesis.

### **Conclusion**

LPS is considered to be an alternative method of treatment for SVS patients with communicating hydrocephalus. We described 3 SVS patients with low-ICP related ventriculomegaly and functional lumbar drainage – 2 with a functioning LPS and 1 with a functioning lumbar drain – and an underfunctioning VPS whose symptoms of shunt malfunction and ventriculomegaly resolved after VPS revision. That VPS underdrainage in a patient with communicating hydrocephalus with a functional LPS resulted in ventriculomegaly and revision of the VPS resulted in resolution of symptoms, likely indicates that lumboperitoneal shunting preferentially drains the SAS and allows the ventricles to enlarge by creating a transient pressure gradient from ventricles to SAS. Symptomatology in that situation, interestingly, may be a result of such a pressure gradient across the cortical mantle rather than absolute ICP.

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