Slit Ventricle Syndromes

Harold L. Rekate

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Abstract

The term "slit ventricle syndrome" (SVS) is widely used to describe a condition of severe, usually intermittent, headaches suffered by patients with long-standing ventricular shunting for hydrocephalus. It is important to differentiate this condition from the smaller than normal, even almost nonexistent, ventricles seen in some patients on routine imaging studies, since most of these patients are asymptomatic and require no intervention. Only when patients begin to suffer from a severe headache disorder that interferes with their normal lives does the presence of radiographic slit ventricles leads to the diagnosis of SVS.

Slit ventricle syndrome can be classified into five major subgroups according to the clinical presentation, results of ICP monitoring, and surgical findings: (1) extreme low-pressure headaches, (2) intermittent proximal obstruction, (3) shunt failure with ventricles that do not expand/"normal pressure hydrocephalus," (4) intracranial hypertension in patients with a working shunt, and (5) headaches in shunted patients not related to intracranial pressure. The pathophysiology, the clinical features, and the treatment algorithms of all these clinical forms are extensively reviewed.

Keywords

Hydrocephalus · Slit ventricle syndrome · Ventriculoperitoneal shunt · Overdrainage · Low-pressure headaches · Intracranial hypotension

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Introduction

It seems likely that ventricular shunts have saved more lives and more quality of life years than any other operation that neurosurgeons have ever performed. For the vast majority of patients, it has turned a death sentence into a chronic illness. This advance however has not been without unanticipated consequences. Early attempts to shunt CSF to the atrium or other termini often led to complete collapse of the brain and fatal subdural hematomas. Finally at the beginning of the second half of the twentieth century, several valves were developed that prevented the very rapid collapse of the ventricles and, in the case of atrial shunts, prevented reflux of blood into the ventricles (Pudenz 1981). Prior to the first reports of the use of the shunts with valves from the ventricles to the right atrium (Nulsen and Spitz 1951), very few infants with hydrocephalus in infancy survived, and those that did were likely to have severe developmental delays (Laurence and Coates 1962). There were treatments available prior to these early valve-regulated shunts such as choroid plexectomies, third ventriculostomies done open or using cystoscopes, shunts from the lumbar thecal sac to the ureter, and non-valved shunts from the ventricle to the spinal subarachnoid spaces (Torkildsen procedures). Unfortunately these were successful in a low percentage of patients and had very high complication rates (Pudenz 1981).

By the mid-1960s shunts had become a standard treatment for hydrocephalus when diagnosed in babies, children, and adults. There were several different valve designs including valves containing slit valves either within the valves itself or at the distal end of the catheter. This type of valve opened at a preset pressure, and the flow increased with increases in pressure differential from the ventricle to the distal terminus. Usually this was the right atrium or the peritoneum. These valves operated as a resistor in the system. Other valve designs were based on opening and closing diaphragms or springs opening and closing an orifice and thus acted as a switch that was open or closed (Pudenz 1981, 1986). By this time there was a recognition of a problem generally referred to as "overdrainage." This complication of working shunts led to subdural hematomas and ventricles that became smaller than normal leading to ventricular catheter failure. In those days there was a great fear of proximal shunt failure with no way of cannulation of the tiny ventricle in days before CT scans or neuronavigation.

Overdrainage also led to severe headache disorders particularly in adolescents. Over time the problem of overdrainage headaches became known as the "slit ventricle syndrome." The first reference of this nomenclature based on a PubMed search was from Holness and colleagues at The Hospital for Sick Children in Toronto (Holness et al. 1979). This group presented 22 children with severe headaches and ventricular shunting who were managed using bilateral subtemporal decompression with opening of the dura. The incidence of subsequent shunt failure and the quality of life improved substantially, and the treated condition became known as the "slit ventricle syndrome." It is of note that subtemporal decompression was the standard way to control high intracranial pressure prior to the development of shunts and was used for idiopathic intracranial hypertension and brain tumors causing hydrocephalus such as pineal tumors.

Three years later Hyde-Rowan and colleagues recognized the role being played by overdrainage or siphoning and managed six patients with intractable headaches and tiny ventricles with upgrade of the valve and the inclusion of an anti-siphon device (ASD) (Hyde-Rowan et al. 1982). The hydrostatic effect of upright positioning in shunted hydrocephalus had been recognized a decade earlier by Portnoy and colleagues who invented a device called the "anti-siphon device™" (Portnoy et al. 1973). This add-on device was to be placed under freely moving skin of the scalp. When the patient stood up and the pressure within the device became lower than atmospheric pressure, the diaphragm is closed until the upstream pressure rose sufficiently to open it again. Hyde-Rowan's study showed that by using this device and by raising the opening pressure of the primary valve, the ventricles could increase in size in a controlled fashion.

Six years later Epstein and collaborators published a series of patients with severe overdrainage problems. Six of the 20 patients had proximal shunt failure without ventriculomegaly, and the other 14 were shown to have severely increased intracranial pressure with a shunt that was shown to be working and no ventriculomegaly. The first group was treated successfully with ventricular catheter replacement and upgrade of the valve with the incorporation of an ASD. The group with a working shunt was shown to have small heads and no CSF over the convexities. These patients were treated with a cranial expansion procedure again with satisfying results (Epstein et al. 1988). It was felt that these children suffered from secondary microcephaly as a result of overdrainage of CSF and therefore developed cephalo-cranial disproportion.

Subsequently Albright published a similar set of five patients shunted in infancy who had thick skulls and expansion of the now-closed sutures particularly the coronal sutures. His patients also had small heads that had stopped growing following the performance of the shunt. Albright referred to this as shunt-related secondary craniosynostosis (Albright and Tyler-Kabara 2001).

Are all patients with small ventricles and severe headaches the same? Is the problem overdrainage? Is the cranial vault too small or is the problem related to the shunt? Do patients with these issues share common underlying pathophysiologies?

Classification of Slit Ventricle Syndromes

Over time, the neurologic outcome in patients treated in infancy with shunts improved with a greater percentage of these patients with normal IQ and many with otherwise normal lives. As the children grew older and found themselves in school or later at work, it became obvious that there was a price to be paid for this miracle, and part of that price was the high instance of incapacitating headaches that interfered with normal life in adolescents and young adults. Why was this occurring? Headaches are among the most common burdens of mankind with 4% of the world's population having chronic daily headaches (CDH). The definition of CDH is severe headaches occurring at least 15 days per month. Two and a half more women than men are sufferers, and there is a massive literature regarding diagnosis and treatment of this condition. During training and as a junior faculty person in neurosurgery at Case Western Reserve University in Cleveland, Ohio, I had the opportunity to manage a large number of patients who had originally been treated there by one of pioneers in the treatment of hydrocephalus, Dr. Frank E. Nulsen, who began a hydrocephalus and spina bifida clinic there soon after arrival in 1953. All surviving shunted individuals who remained in northern Ohio were seen at least yearly, and immaculate handwritten records were maintained. The experiences at that institution in the early days of successful management of infantile hydrocephalus were carefully recorded and documented in the literature [Ref Becker, Young and Weiss]. Based on plane X-rays and pneumoventriculograms, the neurosurgeons and neuroradiologist became aware of the relationship between ventricles that had become smaller than normal and were associated with severe thickening of the skull with severe headaches that interfered with normal life (Kaufman et al. 1973). This question is still not completely answered. Should patients who have chronic daily headaches presumably related to the function of the shunt, specifically overdrainage, be treated surgically in an attempt to make the quality of their lives better? For most of these patients, we used a threshold of having headaches that required that the child had to leave school or not go more than twice a month as justification for intervention. At first this meant changing the valve opening pressure to a higher level and soon thereafter the addition of devices which prevented ICP which was negative. Preventing the ventricles from becoming too small became the goal of treatment (Hyde-Rowan et al. 1982).

Subsequently I moved to the Barrow Neurological Institute in Phoenix, AZ, where there existed a long-standing clinic for the care of children with birth defects and genetic diseases. This clinic was a carve-out clinic from Medicaid

(called ACCHS in Arizona). Through this clinic we followed about a thousand shunted individuals serving most of Arizona except Tucson that had its own such clinic. Between 1986 and 1991, we identified seven patients with incapacitating headaches that were resistant to medication. At that time there was enthusiasm for treating patients and in particular adolescents with shunt-related headaches as if they had migraine. These seven patients had all failed such trial using several different migraine medications. The headaches had continued and had also failed after a shunt revision with use of a device designed to retard siphoning. These patients underwent at least 48 h of invasive ICP monitoring using either a wire transducer in the parenchyma or a subdural bolt with a Statham pressure transducer. The results of this study led to a classification of shunt-related headaches (previously called slit ventricle syndrome). The classification resulted in treatment strategies related to each type of shunt headache (Rekate 1993).

1. **Extreme low-pressure headaches**: ICPs normal after long periods of recumbency fell to −20 to −25 mmHg on sitting or standing up with severe headaches. The headaches resolved with returning to the supine position as the ICP rose into the positive range. Treatment using high-pressure valve containing or adding a device to prevent siphoning (DRC) was successful when the ICP did not fall below minus 5 mmHg. This condition is analogous to spinal headaches.
2. **Intermittent proximal obstruction**: ICPs were normal for the most part, but patients developed severe headaches lasting 10–90 min and associated with a valve that did not pump if the valve had a pumping chamber. ICPs went from normal to very high (30 mmHg) for the period of time the headaches lasted. The diagnosis of overdrainage was identified, and the strategy of upgrading the valve with the addition of a DRC manages these patients effectively.
3. **Shunt failure with ventricles that do not expand/"normal volume hydrocephalus."** Patients were identified that had very high intracranial pressures with small ventricles that do not change in size. I have recorded intracranial pressures of over 80 mmHg in two of these patients. This phenomenon relates to a failure of terminal absorption of CSF flow with no restriction of flow between the ventricles and the cortical subarachnoid spaces and, importantly, the brain which is stiffer than normal. This condition is the most challenging and most controversial. Effective treatment with a shunt system accesses the cortical subarachnoid space as well as the ventricle to prevent changes in the transmantle pressure and the collapse of the ventricle around the catheter. This condition may be caused by high pressures in the dural venous sinuses, and that is likely the cause in all such patients. This phenomenon has been confirmed in the case of achondroplasia where the hydrocephalus develops as a result of stenosis of the jugular foramina (Pierre-Kahn et al. 1980; Steinbok et al. 1989). Sainte-Rose actually prevented a dwarf from needing a shunt by doing a transverse sinus to jugular vein bypass (Sainte-Rose et al. 1984). Impeding intracranial venous drainage in children with closed cranial sutures leads to idiopathic intracranial hypertension (IIH), whereas in children whose skull can increase in size, the condition causes hydrocephalus and in some situations leads to secondary stenosis of the aqueduct of Sylvius (Nugent et al. 1979). Shunt failure in later life in these patients is the same as ventricular shunt failure in IIH patients. MRI studies in this condition will almost always show some distension of the cortical subarachnoid spaces. The term "normal volume hydrocephalus" (NVH) was originally coined by Engel, and it is a condition that occurs only in patients shunted for hydrocephalus originally early in life when the skull can expand. After suture closure the total intracranial volume cannot change, and the patients have non-responding ventricles throughout the remainder of their lives. NVH occurs in my series in 20% of such patients and 10% of patients in an unselected series (Engel et al. 1979; Baskin et al. 1998; Mcnatt et al. 2008). Excellent results

have been shown with the use of lumboperitoneal shunts and removing or disabling ventricular shunts (Le et al. 2002; Rekate and Wallace 2003). In patients such as those with spina bifida, Chiari I and II patients, and patients whose hydrocephalus is related to achondroplasia, it may not be possible to shunt the lumbar theca. In that case it may be necessary to utilize a shunt that accesses the cisterna magna (Nadkarni and Rekate 2005; Rekate et al. 2006).

4. **Intracranial hypertension in patients with a working shunt.** These patients have high intracranial pressure, but the shunts are shown (usually by shuntagrams) to be working. In these cases the key usually can be found in the CT scans or plain X-rays that show a beaten silver (kleeblattschädel) appearance. This occurs in patients with severe craniosynostosis such as Pfeiffer syndrome. ICPs may be rather normal when up and around and often go into the high 30 mmHg range when asleep. This condition requires the expansion of the skull. Note, there is often a significant degree of cerebellar tonsillar descent in these patients (Francis et al. 1992).
5. **Headaches in shunted patients not related to intracranial pressure**. As stated above chronic daily headaches are very common in patients who do not have shunts. The management here is extremely difficult because it is never completely safe to ignore severe headaches in shunted individuals. The management proposed here is very controversial among neurosurgeons so what follows has to be recognized to be my personal approach to this problem and is not widely accepted by the neurosurgical community. If the ventricles are "stable" meaning they are the same size with and without a headache, it is impossible to tell from a CT scan or MRI if the shunt is or is not working. If it is a severe headache such as a migraine, the emergency room staff is essentially obliged to get a CT scan or, more recently, a fast-sequence MRI looking just for ventricular size.

It doesn't matter how many of these scans have been done in the past. This is quite expensive and utilizes a large number of healthcare dollars. With frequent visits to the emergency center, the repeat use of CT scans and the cumulative dose of radiation can be enormous. The overall cost of treatment of these patients can be enormous. Physicians are under great pressure to relieve the pain, and this leads to the prescription of narcotic medications and eventually to addiction that can be fatal. Also it is very likely that the largest majority of chronic daily headaches would be due to medication overuse.

What is the answer? In my opinion the most important thing is an accurate diagnosis. The simplest but least likely thing to do would be to measure the pressure. Almost all shunt systems have with them a tapping reservoir that allows the pressure to be measured manometrically at the time the shunt is assessed. Care must be taken to carefully prep the area around the reservoir and use sterile techniques to tap the shunt in order that an infection is not induced. In recurrent cases, I recommend early monitoring of intracranial pressure overnight in patients in whom there is a significant risk of analgesic dependence and who have had a significant number of emergency room visits. Any use of narcotics for headaches in patients with shunts should be avoided at all costs (Fig. 1).

Shunt-Independent Arrest on Hydrocephalus

After spending almost 40 years caring for patients with hydrocephalus, I have come to the conclusion that it is "Not a shunt, it's a sentence." Even in patients who have had very few problems with shunts, it is likely that the presence of a shunt represents a substantial burden. Is this headache a headache or is it a shunt failure? Can I really safely travel to Machu Picchu? Can I live alone? Must I tell my friends? The answer to these questions is not easy. If the patient also suffers from chronic daily headaches, the problem is multiplied many times. They are often operated over and over again to

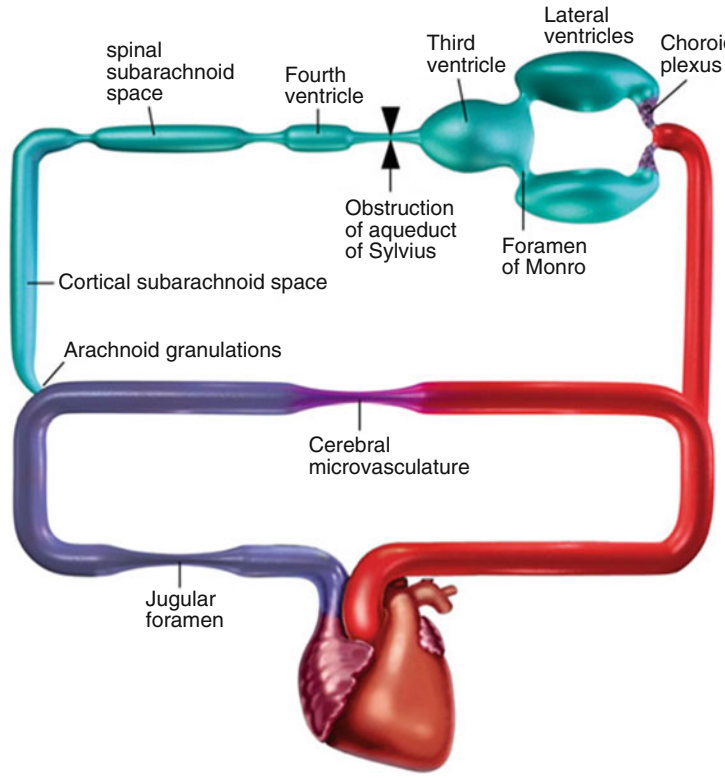


Fig. 1 Diagram of the flow of dynamics of Cerebrospinal fluid and Cerbral blood flow as a Circuit diagram showing the potential points of restriction of flow that can lead to hydrocephalus (Rekate 2008)

find the "right valve." Are they going to develop a malignancy due to the number of CT scans they have had? While there are some conditions in which it is very unlikely that they can never be shunt free, for many shunted patients there is a significant chance that the shunt can be removed safely. This result is very unlikely in patients whose hydrocephalus relates to myelomeningocele and who have a Chiari II malformation. This is because there are multiple sites that may be involved in the process, and bypassing one may not help at all (Fig. 2). The other condition in which it is very unlikely and probably not worth the risk of intervention is when the patient has suffered both a hemorrhagic and infectious cause for the hydrocephalus (Siomin et al. 2002). In patients with incapacitating headaches and a history of multiple shunt revisions, I will frequently spend time explaining how the hydrocephalus is affecting them related to the new consensus classification of hydrocephalus (Rekate 2011).

Until recently the "shunt removal protocol" was used to attempt safe shunt removal (Table 1). First the shunt was either removed and replaced with an external ventricular drain or the distal end externalized. I prefer the former option of an EVD so ICP monitoring can be done. In the ICU the drain is clamped and the patient is observed carefully. The following morning there are three options:

1. The patient is well, the ventricles are mildly enlarged or the same, and the pressure is low. In this case the shunt or EVD can be removed safely. This occurs most commonly in patients with posthemorrhagic hydrocephalus.
2. The patient is symptomatic and the ventricles have enlarged significantly. In this case the patient is an excellent candidate for endoscopic third ventriculostomy, and 80% of such patients will respond to ETV with no need for a reimplanted shunt. No patient undergoing this protocol has had late return of symptoms,

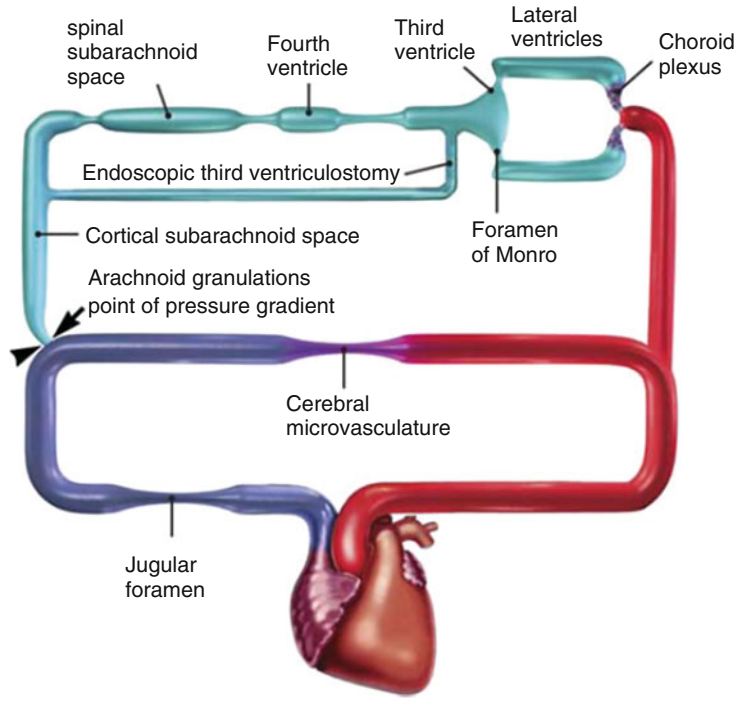


Fig. 2 Diagram of CSF and cerebral blood flow showing points of potential sites of flow restriction and what is actually done by an ETV. Note the effect not only treats aqueductal stenosis but also completes failure of flow out of the fourth ventricle and restriction of flow between the spinal and cortical subarachnoid spaces (Rekate 2008, 206)

but it is important that bad headaches should not be ignored as possible failure of the ETV as others have reported late deaths and sudden deaths from reclosure of the stoma (Lipina et al. 2007).

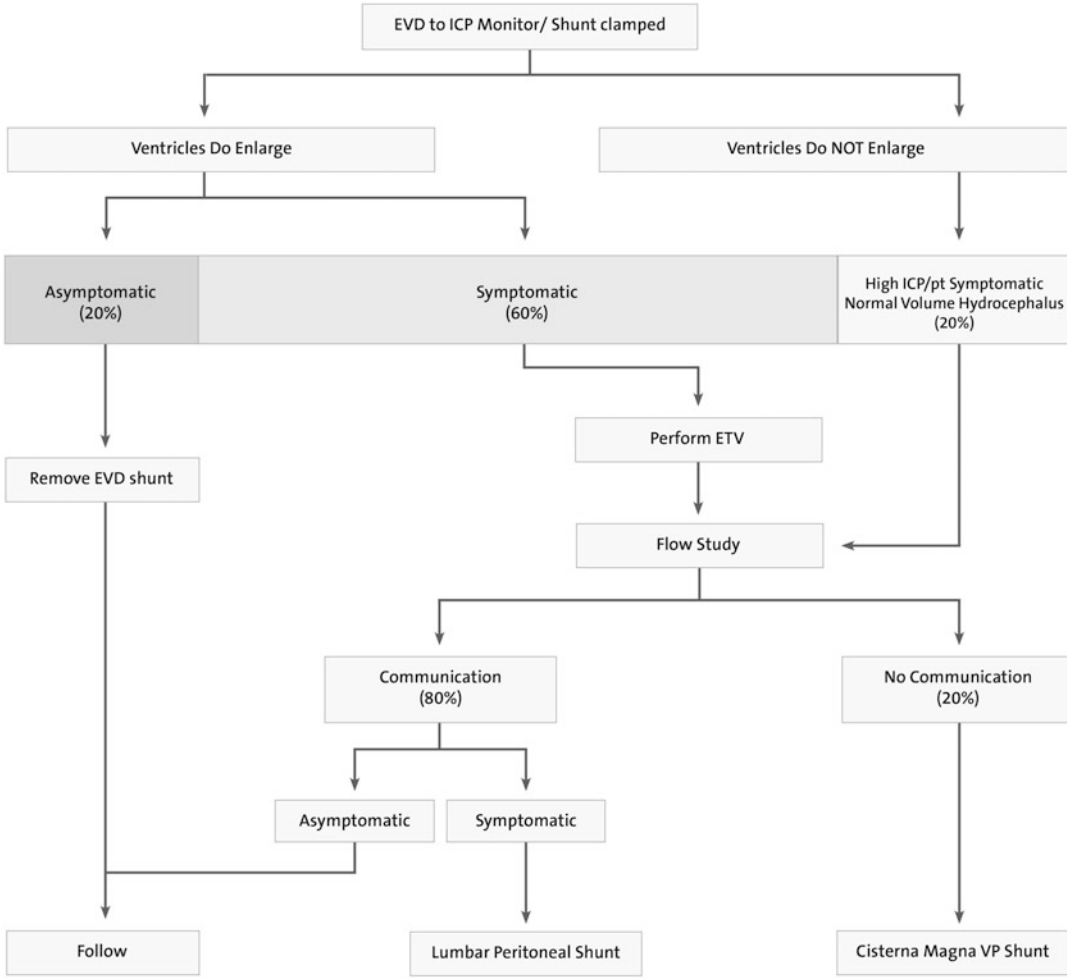
3. The patient is sick and the pressure is high, but the ventricles have not expanded. You now have a diagnosis of "normal volume hydrocephalus." These patients should have a shunt that accesses the cortical subarachnoid space either from the lumbar theca or the cisterna magna. I would recommend continuing to monitor ICP for a while after that to make certain that the pressures are normal. In this case I recommend a MedicAlert bracelet that states that the ventricles do not enlarge and scans cannot predict ICP (Baskin et al. 1998, 133; Rekate et al. 2006, 1498).

Conclusion

Hydrocephalus that begins in infancy must be seen as a chronic disease, and for most of the infants who need a shunt, they will probably remain shunt dependent for life and face many trials as a result. As a result of the Management of Myelomeningocele (MOMS) trial, a lower percentage of patients with spina bifida cystica are being shunted with an outcome at least as good and arguably better than had been expected previously. While the treatment group showed better results related to the hydrocephalus than those babies brought to full term, a significantly smaller number of the standard treatment babies were not shunted either leading to a better quality of life in those babies as well (Adzick et al. 2011).

We don't know how to gauge how much hydrocephalus it takes to lead to neurologic damage in children or young adults. We do know that there are many adults seen in neurosurgical offices with scans done for other reasons in which moderate hydrocephalus is diagnosed. These adults have had the hydrocephalus for years without symptoms. They do have a potential for later deterioration or the development of normal pressure hydrocephalus when they reach old age but have been and are living normal lives without surgery or surgical complications (Cowan et al. 2005). Deterioration in this group

Table 1 Algorithm for "shunt removal protocol"



Notice: For the patients with non-responding ventricles, the final treatment pathway requires drainage of both the ventricles and the cortical subarachnoid space. For most cases that means a single shunt from the lumbar theca to peritoneum, but for some it could mean that there must be a communication between the ventricular system and the cortical subarachnoid spacek

of patients is almost always related to balance, gait, and bladder control (NPH-type symptoms) and rarely present with headaches even though that might be why they were scanned. I would say that if ventriculomegaly is found unexpectedly on a scan, there should be an assessment of balance and bladder function as well as cognitive function and treatment of hydrocephalus only be considered if abnormalities are found. Headaches are rarely if ever signs of deterioration in these patients, nor do they often improve when treatment is given (Rekate 2007).

The most important conclusion to take home from this discussion is that approximately 10% of all patients who are shunted early in life will end up with ventricles that do not expand at the time of shunt failure. They can sicken and die if their "normal" MRIs and CTs, read as normal, do not lead to further investigation. They may really need help.

Appendix: Members of the Study Group for Definition and Classification of Hydrocephalus

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