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Original Article

Endoscopic third ventriculostomy as adjunctive therapy in the treatment of low-pressure hydrocephalus in adults

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Abstract

Background: Treatment of low-pressure hydrocephalus (LPH) may require prolonged external ventricular drainage (EVD) at sub-zero pressures to reverse ventriculomegaly. Endoscopic third ventriculostomy (ETV) has been used in the treatment of noncommunicating hydrocephalus; however, indications for ETV are expanding.

Methods: Patients with the diagnosis of LPH as defined by the Pang and Altschuler criteria who underwent sub-zero drainage treatment over an 8-year period were included. Patients were divided into two cohorts based on whether or not ETV was employed during their treatment. Time from EVD placement to internalization of shunt was recorded for both groups; time from ETV to placement of shunt was recorded for the patients undergoing ETV.

Results: Sixteen adult patients with LPH were managed with sub-zero drainage method. Ten (62.5%) patients did not undergo ETV and the average time from first ventriculostomy to shunting was 73 days (range 14–257 days). Six (37.5%) patients underwent ETV during the course of their treatment; average time from initial ventriculostomy to shunt was 114 days (range 0–236 days) ($P = 0.16$). Time from development of LPH to ETV ranged from 28 days to 6.5 months. In the ETV group, of the 4 patients who underwent shunting, the average time to shunting following ETV was 15.25 days.

Conclusions: ETV can be used successfully in the management of refractory LPH to decrease the duration of EVD.

Key Words: Adult hydrocephalus, endoscopic third ventriculostomy, intracranial pressure, low-pressure hydrocephalus, shunt, subarachnoid space

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INTRODUCTION

As initially described by Dandy, hydrocephalus (HCP) was defined as obstructive or communicating and typically associated with elevated intracranial pressure (ICP).[5,6] Later, Adams *et al*. reported on the condition of normal pressure HCP (NPH), describing adult patients with dementia and motor delay who responded well to ventricular shunting despite having normal ICP.[1] In 1994,

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Pang and Altschuler described in detail the rare entity of low-pressure HCP (LPH), distinguishing patients with LPH from "most shunt-dependent hydrocephalic patients (who) present with predictable symptoms of headache and mental status changes when their cerebrospinal fluid (CSF) shunts malfunction."[20] As the authors noted, patients with HCP typically manifest high ICP that generally responds well to shunt revision, which stands in sharp contrast to patients with LPH.

Pang and Altschuler reported 12 patients with a "full-blown" hydrocephalic syndrome who had all been previously managed by medium-pressure shunt systems; patients presented with headache, lethargy, obtundation, and cranial neuropathies but were found to have low or low-normal ICP. Nearly, all patients required prolonged external ventricular drainage (EVD) at negative pressure to reverse the ventriculomegaly and symptoms of HCP. Importantly, symptoms throughout the period of EVD drainage correlated with ventricular size only, not ICP. Their diagnostic criteria for LPH included: (1) Neurologic decline at normal EVD or shunt pressures; (2) ventriculomegaly; (3) persistence of ventriculomegaly with ICP in the normal to low-normal range; and (4) clinical and radiographic response to sub-zero drainage. This method consisted of prolonged subatmospheric pressure drainage of the ventricular system until the ventricular shrinkage was maintained at a positive pressure, and a new shunt system could be inserted.

Endoscopic third ventriculostomy (ETV) has been employed successfully in the treatment of HCP in specific populations, particularly those patients with what the literature commonly refers to as the "noncommunicating" form of the disease. Despite the traditional concept that ETV is best utilized in these patients, other uses for ETV have been suggested. We report on the successful application of ETV in the treatment of patients with LPH.

METHODS

Patients

The authors performed a retrospective analysis of all adult inpatients admitted to the neurosurgical service at the University of Pittsburgh Medical Center between June 2005 and November 2013 to identify those patients who presented with LPH or developed this condition throughout their hospitalization. To meet inclusion criteria, patients with LPH were identified as those with (1) radiographic evidence of ventriculomegaly; (2) neurologic decline from baseline; (3) neurologic deficit despite conformation of normal or low ICP in the setting of a patent, functional EVD or ventricular shunt; and (4) clinical and radiographic response to the sub-zero drainage method. Within this group of patients, those who underwent ETV during the course of their treatment at the time an EVD was open and functional were identified. This review of patient data was approved by the Institutional Review Board.

Treatment of low-pressure hydrocephalus

All patients with LPH were treated with the sub-zero drainage method via external ventriculostomy. The EVD was initiated at a subatmospheric pressure, placed at a height ranging from 5 to 10 cmH_2O below midbrain, and incrementally raised 1–2 cmH_2O every 3–5 days. Prior to elevating the drain, a computed tomography (CT) scan was obtained to document stable or smaller ventricular caliber. If the patient demonstrated neurologic decline, the drain was again lowered to a more negative pressure and the cycle reinitiated. Patients with LPH underwent ETV while the EVD was still at a subatmospheric pressure. Patients underwent shunt placement once they demonstrated clinical and radiographic stability with an EVD leveled at or above midbrain.

Endoscopic third ventriculostomy

ETV was performed via a curvilinear skin incision in the right frontal region, with a single coronal burr hole placed at the mid-pupillary line. A small durotomy was created with electrocautery and scalpel, and a sheath was passed until CSF was obtained from the frontal horn of the lateral ventricle. Next, a rigid neuroendoscope was introduced and intraventricular anatomical landmarks were identified. A Bentson wire was used to create an ostomy in the floor of the third ventricle anterior to the mammillary bodies and a 2.0 French Fogarty balloon was inflated to increase the size of the ostomy. CSF flow was assessed and choroid plexus cauterization (CPC) was performed in some patients. Finally, the patient was then shunted at the time of ETV ± CPC or an EVD was replaced at the conclusion of the ETV, based on surgeon preference.

RESULTS

We identified 16 adult patients who met criteria necessary to obtain the diagnosis of LPH over the 8-year period in this study. All patients were managed with the sub-zero drainage method. Ten (62.5%) patients did not undergo ETV in the course of their treatment. All 10 (100%) patients in the non-ETV cohort required permanent shunting and the average time from placement of first ventriculostomy to shunting procedure in this group was 73 days (range 14–257 days).

Six (37.5%) patients underwent ETV during the course of their treatment; 4 were male and age ranged from 28 to 48 years. Two patients developed LPH following CSF diversion for HCP with elevated ICP secondary to tumor; one patient had a pineal germinoma causing obstructive HCP and the other patient developed communicating HCP secondary to a suprasellar cistern

epidermoid resected via the endoscopic endonasal approach complicated by intraoperative rupture and postoperative CSF leak. LPH occurred in 2 patients with prior aneurysmal SAH requiring permanent CSF diversion with shunting at the time of their initial admission. Two patients had a history of prior shunt placement many years before presenting with LPH. Features of the patients are shown in Table 1.

Clinical presentation was highly variable with signs and symptoms including headache, lethargy, seizure, upgaze restriction, and coma. No patients presented with autonomic instability or cardiac arrhythmias, as has been reported in other LPH series.[2] Time to development of LPH from initial neurologic insult (diagnosis of tumor, SAH, etc.,) ranged from 2 months to 26 years. All patients underwent neuroimaging with CT and/or magnetic resonance imaging (MRI). All patients had a significant ventriculomegaly, with universal involvement of the lateral, third, and fourth ventricles. All imaging demonstrated some degree of transependymal fluid absorption.

Five patients underwent ETV while the EVD was at a subatmospheric pressure (≤0 cmH_2O); 1 patient had a ventriculoperitoneal/pleural shunt in place at the time of ETV, which was removed temporarily for ETV and then replaced at the conclusion of the operation. Time from development of LPH to ETV ranged from 28 days to 6.5 months. Following ETV, the clinical course was highly variable. One patient underwent rapid EVD wean (in 8 days) and did not require shunting. Two patients underwent shunting at the time of ETV and have not demonstrated shunt failure to date. One patient was shunted 16 days after ETV. Two patients were deemed by the operative surgeon to have an unsuccessful ETV at the time of surgery; of these two, 1 patient died 2 months after ETV with an EVD still at a subatmospheric pressure, and the other proceeded to shunt internalization following another 1.5 months of sub-zero management post-ETV.

The average time from placement of initial ventriculostomy to shunt in this group was 114 days (range 0–236 days). When comparing the ETV and non-ETV cohorts, the overall timing between first ventriculostomy and shunting did not reach statistical significance ($P = 0.16$). However, of the 4 patients in the ETV cohort who underwent shunting, the average time to shunting following ETV was 15.25 days (range 0–45 days). Of the entire study group, 1 patient avoided shunting altogether and this occurred following ETV. There were no procedure-related complications from ETV or shunt placement.

Illustrative case

A 31-year-old male with a medical history significant for Ehlers–Danlos type IV and smoking initially

Table 1: Features of patients with LPH managed with ETV as part of their treatment

Patient	Sex	Age (years)	Initial neurologic diagnosis	Time to LPH	Presenting symptoms	Lowest ICP (cmH_2O)	Transependymal absorption	Total duration of EVD	Time to ETV	ICP at ETV (cmH_2O)	Protein (mg/dL) at ETV	VPS placement	ICP at VPS (cmH_2O)	Protein (mg/dL) at VPS
1	Male	31	Aneurysmal SAH	7 years	Lethargy, headache	−12	Yes	48 days	32 days	−5	9	Yes	5	39
2	Male	46	Pineal region germinoma	26 years	Seizure, lethargy, headache	−5	Yes	0 days	28 days	VPS in place	<6	Yes	−5	13
3	Male	45	Adult HCP of unknown etiology	20 years	Upgaze palsy, lethargy	−5	Yes	3 months, 25 days	3 months, 17 days	−1	<10	No	N/A	23
4	Female	48	Aneurysmal SAH	1 year	Lethargy, headache	−8	Yes	8 months, never weaned	67 days	−1	NR	No	N/A	NR
5	Male	28	Suprasellar epidermoid	2 months	Coma	−10	Yes	7 months, 26 days	6 months, 14 days	0	<6	Yes	3	48
6	Female	34	Adult HCP of unknown etiology	17 years	Upgaze palsy	−3	Yes	2 months	2 months	−3	NR	Yes	1	NR

SAH: Subarachnoid hemorrhage, HCP: Hydrocephalus, LPH: Low-pressure hydrocephalus, ICP: Intracranial pressure, EVD: External ventricular drain, ETV: Endoscopic third ventriculostomy, VPS: Ventriculoperitoneal/pleural shunt, NR: Not recorded; N/A: Not applicable

presented at age 24 with Hunt Hess Grade 4 aneurysmal SAH and underwent craniotomy for evacuation of intraparenchymal clot and clipping of a carotid bifurcation aneurysm. He was managed with an EVD and subsequently underwent shunt internalization with a medium-pressure programmable valve. Initially with hemiplegia, he recovered well neurologically, regained all function and returned to his prior level of function. Three years later, he underwent elective coiling of a carotid wall aneurysm and at age 30 elected for an open craniotomy for clipping of ophthalmic and posterior communicating artery aneurysms. Throughout this time, he did not have evidence of shunt failure and required no revisions.

In the month prior to his diagnosis of LPH, he presented with headaches, and a shunt tap revealed elevated ICP, prompting shunt exploration and proximal catheter revision. A few weeks later, he presented with lethargy; CT revealed significant pan-ventriculomegaly with transependymal absorption and abdominal imaging showed an abdominal pseudocyst. He underwent removal of shunt and placement of EVD. CSF analysis at the time of shunt externalization revealed an *Enterobacter* infection and the patient completed a 3 weeks course of meropenem with resolution of his CSF infection. Of note, his course was also complicated by electrographic evidence of epileptiform discharges, managed by phenytoin.

At this time, it was found that he did not drain CSF at a positive pressure and so was managed with the sub-zero drainage method. His lowest EVD pressure was −12 cmH_2O. On EVD day 32, with EVD pressure of −5 cmH_2O, he underwent ETV with CPC (imaging 3 h prior to procedure shown in Figure 1a and b). An EVD was replaced at the time of ETV and was subsequently weaned to a nonzero pressure. Immediate post-ETV/CPC imaging is shown in Figure 1c and d. The patient clinically improved throughout this time with resolution of his headaches and motor/cognitive slowing. On post-ETV day 16, with the EVD at a pressure of +5 cmH_2O, he underwent successful placement of a ventriculopleural shunt (immediate postshunt imaging shown in Figure 1e and f). He was discharged to a rehabilitation facility (postshunt day 2 imaging shown in Figure 1g and h) and later returned to home, requiring minimal assistance from family. His most recent CT was obtained 3 months postshunting [Figure 1i and j]. Now, 2 years and 3 months since shunting, he is without neurologic deterioration or shunt-related complication.

DISCUSSION

LPH is a rare form of HCP consisting of neurologic decline and significant ventriculomegaly in the setting of low ICP. Review of literature since the landmark paper of

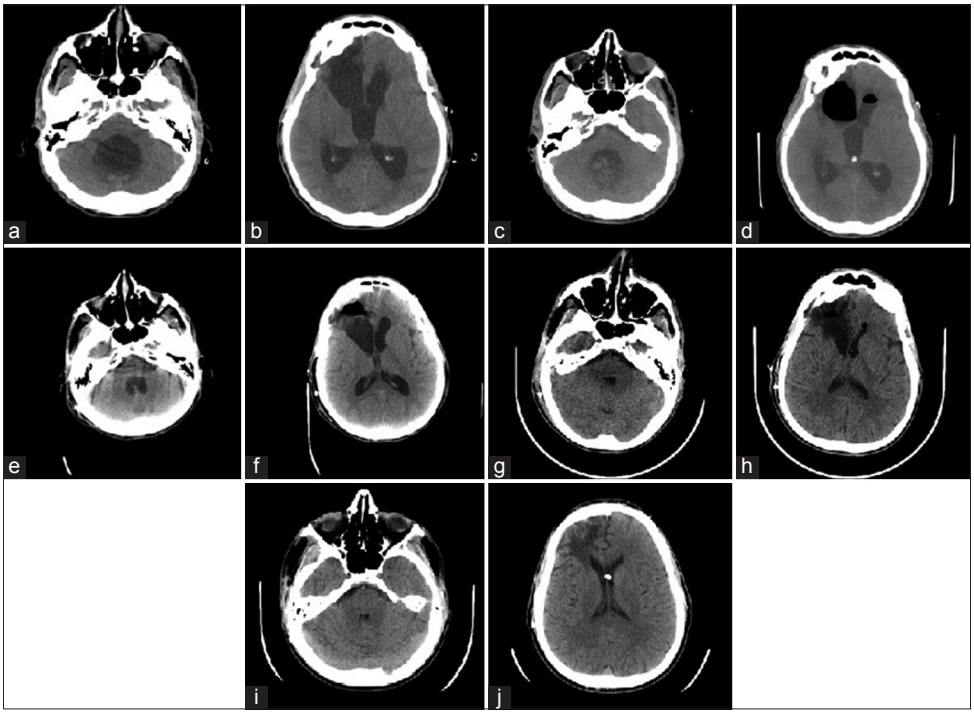

Figure 1: Axial computed tomography image showing ventriculomegaly of the fourth ventricle (a) and lateral ventricles (b) obtained 3 h prior to endoscopic third ventriculostomy/choroid plexus cauterization, with external ventricular drain positioned at 5 cmH_2O below midbrain. Computed tomography image obtained immediately following endoscopic third ventriculostomy/choroid plexus cauterization of the fourth ventricle (c) and lateral ventricles (d). Computed tomography image obtained 3 h prior to placement of ventriculopleural shunt, with external ventricular drain at 5 cmH_2O above midbrain, showing fourth (e) and lateral ventricles (f). Computed tomography imaging obtained on postoperative day 2 from shunting (g and h) and at 3 months neurosurgical follow-up (i and j)

Pang and Altschuler revealed nearly 70 reported cases of LPH. Many of these cases have been summarized in the work of Akins *et al*. and, in addition, the authors reported on 9 of their own patients.[2-4,7,9,16,18,20,26] Rekate *et al*. have contributed two reports including 5 total patients with the condition they refer to as negative pressure HCP[11,22] and Hamilton and Price report on 20 patients managed with the syndrome of inappropriately low-pressure acute hydrocephalus (SILPAH).[14]

LPH is associated with multiple conditions including SAH, tumor, and chronic HCP. Akins *et al*. noted a tendency for pathology isolated to the posterior fossa, including fourth ventricular tumors, hemorrhage, and basilar meningitis. The occurrence of LPH following lumbar puncture has been reported in a small case series of shunted patients who subsequently developed shunt failure due to LPH and an intraventricular pressure too low to open the shunt valve.[9] We found the underlying etiology of LPH in our patient population to be similar to those reported in literature.

Multiple theories attempt to explain the underlying pathophysiology of LPH. Early work to model HCP, including the contributions of Hakim *et al*., suggested that the brain should be viewed as a porous sponge that obeyed the principles of viscoelastic materials.[13] Based on this premise, Pang and Altschuler proposed the etiology of LPH to be related to an altered viscoelastic modulus of the brain; patients with inherently low brain elasticity were susceptible to development of LPH because water within the brain parenchyma would be forced out and into the ventricular system. The symptoms of LPH are related not to pressure changes but to cortical distortion and ischemia secondary to ventriculomegaly. The restoration of normal brain viscoelasticity could be achieved with prolonged sub-zero drainage, as water reenters the brain parenchyma.

Another model of LPH was based on the principle of hysteresis, as proposed by Lesniak *et al*.[16] The authors called the chaos theory of nonlinear dynamics, which explains how a system can exist in two different states (ventricular caliber) at a single parameter (ICP). Thus, LPH represents a biologic manifestation of hysteresis, and, in this theory, ventriculomegaly is initiated at a high ICP, but exhibits persistent ventriculomegaly at low ICP. Lesniak *et al*. incorporated changes in the viscoelastic properties of the brain as stated by Pang and Altschuler and proposed that the initial ventriculomegaly deranged normal brain physical properties (elasticity). Sub-zero drainage could restore brain elasticity and as ventriculomegaly resolved, so too would neurologic symptomatology.

The model of Akins *et al*. differs from the prior hypotheses and proposes the brain to be like a boggy sponge in LPH, in contrast to the "wrung-out" cortical mantle of other theories.[2] This poroelastic model describes the brain as a solid matrix with permeable pores filled with fluid (CSF). At the root of the problem, in the poroelastic theory, is a derangement in normal brain permeability. They noted that during the inception of LPH, EVD output declined (less output at a given height as LPH developed); fluid transitioned into the brain parenchyma because the inherent permeability of the brain had changed. Moreover, in support of their theory, they distinguish the periventricular white matter from the compact, cortical gray matter. As evidence for the poroelastic model, they noted the radiographic finding of transependymal fluid absorption (periventricular edema) in LPH and cite Darcy's law of fluid flux (see below) to explain why EVD output declines during development of LPH and rises during sub-zero treatment. We, too, noted decreased output during genesis of LPH with a significant increase in output during sub-zero drainage.

Rekate *et al*. emphasized the importance of taking into account all compartments of CSF, which includes the ventricles, brain parenchyma, spinal subarachnoid space (SSAS), and the often neglected cortical subarachnoid space (CSAS).[22] Rekate *et al*. delineates three important principles important to HCP in general and applicable to LPH. First, all HCP is obstructive, whether or not it can be appreciated radiographically, and the point of obstruction may be at the level of the choroid plexus, ventricular system, SSAS, CSAS, arachnoid granulations, cerebral vasculature, or dural venous sinuses. Second, Rekate *et al*. reject the term compliance because it suggests a precise, fixed viscoelastic property of the brain; instead they support the property of brain turgor (Kb) which, similar to compliance, addresses the state of brain stiffness. Third, the CSAS plays a critical role in the etiology and maintenance of any type of HCP, including LPH. In short, when CSAS and the ventricles function in isolation (such as with a skull base CSF leak and postlumbar puncture), there is an obstruction in the ventricular system and the CSAS is selectively drained. As a result, the ventricles will expand volumetrically and push against a brain that is no longer being bolstered at the cortical (outer) surface by CSF in the CSAS. Alternatively, if the CSAS is distended or becomes "plump," it pushes the brain parenchyma inward, which displaces CSF from the ventricular system into the EVD or shunt. In principle, a mechanism by which the obstruction between the ventricular space and CSAS could be overcome, such as a communication between the cisternal space (CSAS) and the ventricles that could be achieved through ETV, would allow for the low-pressure ventriculomegaly to resolve.

As illustrated by Akins *et al*., movement of CSF in and out of the brain is critical in the development of LPH and can be explained by Darcy's law, which describes the movement of fluid in and out of a porous substance.[8] If we consider the brain a porous substance, then we can

apply Darcy's law: $q = -k\ (\Delta P)/\mu$ (q is discharge per unit area or, simply, movement of fluid; *k* is permeability of porous substance; ΔP is a pressure gradient; and μ is viscosity of the fluid). Movement of fluid into the porous substance is directly proportional to both the permeability of the brain and the pressure gradient; if there is a large pressure gradient between the CSAS and the ventricles, such as in the case of obstruction, there will be a significant movement of fluid across the brain parenchyma. When this pressure gradient is reduced, the movement is decreased (and when the pressure gradient is zero, movement of fluid into the brain halts completely). ETV functions to reduce this pressure gradient by equilibrating the pressure between the CSAS and the ventricles.

Permeability of the brain and viscosity of CSF also play an important role in LPH. If the permeability of the brain is increased, so will the movement of CSF. In LPH, it has been hypothesized that the permeability (*k*) of the brain itself is deranged, as evidenced by the presence of transependymal absorption seen commonly in imaging studies of patients with LPH.[2] We have also noted that CSF sampled during the course of LPH has very low protein content and cell count; we often refer to the CSF as "water" in these patients. Darcy's law states that movement (flux) of fluid is inversely proportional to viscosity of the fluid. In LPH, the CSF is "watery" with a very low viscosity, and movement of fluid into the parenchyma is increased. Following ETV, we noted an increase in CSF protein and cell count and, while we cannot assume causality, we highlight this interesting observation.

The role of endoscopic third ventriculostomy

ETV has generally been employed in the treatment of noncommunicating HCP. The ETV Success Score (ETVSS) is a recently designed, externally validated tool to guide the selection of patients for ETV.[15] The ETVSS was developed to predict the short-term success of ETV but has also been shown to correlate with longer term success of ETV.[10] As predicted by the ETVSS, the ideal patient for ETV has noncommunicating HCP (aqueductal stenosis) without a history of intraventricular hemorrhage or central nervous system (CNS) infection are ≥10 years of age and without a history of shunt.[15,23,27,28] Nonetheless, indications for ETV have been expanding and the successful treatment of patients with ETV in the setting of neural tube defects and CNS infection has been reported.[24,25,29,30] Other forms of HCP, such as NPH and postsubarachnoid hemorrhage HCP, have been successfully treated with ETV.[12,17,19,21]

To date, there is a little attention in literature given to the use of ETV in the treatment paradigm of LPH. Rekate *et al*. reports on 3 total patients who required sub-zero drainage for LPH and were treated with ETV; 1 patient still required placement of a shunt but no longer needed cervical wrapping following ETV, another managed with ETV alone[22] and a patient who died following ETV without resolution of LPH.[11] Hamilton and Price reported a cohort of 10 patients with the SILPAH managed with ETV and compared them to a matched cohort who did not undergo ETV.[14] Both groups of patients had similar clinical outcomes, and the ETV cohort included some patients who were shunted following ETV and some who did not require shunting.

In our LPH population, patients required prolonged hospitalizations for protracted sub-zero EVD, often on the order of months, before an atmospheric pressure could be obtained and a shunt could be placed. Our goal was to employ ETV to decrease the amount of time an EVD was required and allow for earlier shunting in adult patients who had developed LPH. There was no statistically significant difference in the overall time from ventriculostomy to shunt placement (or wean) between those patients undergoing ETV and those who did not. However, this is due to an inherent bias to perform ETV on patients who were refractory to sub-zero drainage and, importantly, all but 1 patient undergoing ETV was at sub-zero (one at zero) pressures at the time of ETV. Conversely, patients who demonstrated response to drainage were not readily considered for ETV. The difference in the clinical courses of the two cohorts should be highlighted post-ETV, as the rapid return to shunting occurred in this time period for those patients undergoing ETV (mean time to shunt was approximately 2 weeks). Moreover, only in the ETV cohort did any patient avoid a permanent shunt. ETV did not allow rapid shunting in all patients and, admittedly, we need to refine our ability to predict which patients in our LPH cohort would benefit from ETV.

This study has multiple limitations. It is a retrospective review and suffers from the inherent biases as such; a prospective, randomized evaluation of patients with LPH managed with and without ETV would definitively address the utility of ETV, if any, in this population. The most important bias in our study is selection bias. Throughout the time of this study, patients with LPH were managed by multiple different physicians and surgeons. The neurosurgical subspecialist treating the original neurosurgical problem (tumor, aneurysm, etc.,) may or may not have cared for the patient throughout their LPH state and, given the very long clinical course of most patients, many were managed by different physicians and care teams. Thus, the decision to perform ETV was based on attending physician preference and, admittedly at our institution, the comfort with performing ETV and the acceptance of ETV indications are variable and surgeon-dependent. Selection bias also explains why some patients remained in the non-ETV cohort (these patients were not considered for ETV), despite very long

hospitalizations. Moreover, we do not know what may have happened to those patients in the ETV cohort had they never undergone ETV.

In the patient who remained shunt-free after ETV, our imaging modalities (cranial CT and MRI) demonstrated that the patient had radiographic communicating HCP (all ventricles demonstrated the same extent of ventriculomegaly). While ETV is a well-accepted modality in noncommunicating (obstructive) HCP, we are suggesting it has value in the communicating form of the disease we defined our LPH patients as communicating. The definitions of communicating and noncommunicating HCP are debated, and a deeper understanding of anatomical site of abnormality is needed (as discussed earlier, all HCP may be obstructive), and a full discussion is beyond the scope of this manuscript.

CONCLUSIONS

ETV can be used successfully in the management of patients with refractory LPH. The benefit of ETV in the setting of LPH may be based on the concept of a pressure gradient, not an absolute intraventricular pressure, which exists across the cortical mantle in patients with altered brain viscoelastic properties. Ultimately, ETV could normalize (decrease) this pressure gradient and bring the important CSAS into communication with the ventricular CSF space. While ETV alone may not be sufficient in the treatment of LPH in all patients, it may allow for earlier shunting, or even a decrease in the number of shunt-dependent patients. Future investigations will need to clarify the appropriate timing of ETV and subsequent shunting, as well as identify those patients who will benefit most from ETV in the treatment of LPH.

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Conflicts of interest
There are no conflicts of interest.

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Commentaries

Hydrocephalus is very problematic and very difficult problem to treat and still to know many things for a simple, comprehensive solution of it. This statement is truer in cases of low-pressure hydrocephalus (LPH).

Though obstructive hydrocephalus is the main indication of endoscopic third ventriculostomy (ETV), it is now using in different types of selective communicating hydrocephalus.[3] This is an interesting paper where the authors efficiently managed 16 cases of the "Difficult" LPH. These patients needs prolong external ventricular drainage (EVD) in subatmospheric pressure to reduce the size of ventricles followed by shunting. Management of such EVD for a long time is very difficult, expansive and of course associated with complications. Though idea of use of ETV in the management of LPH is not new,[1,2] the authors set a background to look at it in different way/s. The authors managed their series in two groups with ETV and without ETV. When comparing the ETV and non-ETV group, the overall time interval between first ventriculostomy and shunting did not reach statistical significance. However, 4 patients in the ETV group who underwent shunting, the average time to shunting following ETV was 15.25 days (range 0–45 days). So after the diagnosis of LPH, early ETV (with EVD) can reduce the time interval from EVD to shunt. In 1 patient of ETV group did not require shunt; this indicates there are some patients (with LPH) who can be treated with ETV only (without shunt)!!! We should look for these patients. In pre-ETV images [Figure 1a and b] it is seemed to me that all basal cisternal spaces and cortical cerebrospinal fluid spaces are compressed and did not open up even with subatmospheric EVD. This patient responded with ETV and post-ETV images showed opening of cisterns and cortical subarachnoid spaces. So impotence of cortical subarachnoid spaces cannot be ignored[4] (though not relevant: From my experience, In a case of high pressure ? communicating hydrocephalus with such images, I found ETV is usually successful). So there may be a scope to look at this point in LPH. During the long standing management of EVD, infection is common and serious problem. In this series, it is not clear whether EVD-related infection occurred or not. The authors mentioned many limitations of the study and I do agree with them but at the same time I feel, it can be an important paper in the management of patients with LPH.

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In this well written article, the authors described their experiences with the surgical management of low-pressure hydrocephalus, which is a very rare, complicated, and difficult condition. Their surgical management is mainly based on endoscopic third ventriculostomy (ETV), after sub-zero drainage. I do agree that in the hard clinical situation with LPH, when simple shunt is highly possible to be ineffective, ETV can be a good alternative for the management of hydrocephalus, either for short or longer period of time. However, the result of this article is not persuasive enough. In this retrospective, small sample size study from experiences of one single center, totally 16 patients were enrolled. Among them, 10 cases had sub-zero external ventricular drainage (EVD), followed by shunt, while the rest 6 cases had EVD, ETV and shunt (4 cases). In both groups (with or without ETV), the LPH was eventually successfully managed. For those 4 cases who had ETV and shunt, the time between ETV and shunt is significantly shorter than those 10 cases with only EVD and shunt. However, if we compare the time between initial EVD and final shunt of two groups (with/without ETV), the period of time is slightly longer in the ETV group, but the *P* value is 0.16. This result is very difficult to be interpreted. The small sample size may be a possible reason. But other reasons should be further analyzed with more accumulated cases and data.

LPH is a very complicated condition. Its etiology is still controversial. The authors proposed that low brain elasticity may probably play an important role, because water within the brain parenchyma would be forced out and into the ventricular system. This mechanism is still worth further exploration. But to me, another possible mechanism, which was already proved by animal model in normal pressure hydrocephalus (NPH), appears more convincing. Di Rocco *et al.* reported experimental hydrocephalus induced by mechanical increment of intraventricular pulse pressure.[1] This mechanism was used to explain the etiology of NPH when the brain "compliance" or "elasticity" is significantly decreased. This elasticity change is very similar to the condition of LPH. Hence, it is possible to explain the etiology of LPH in similar way. In recent years, with the advancement of magnetic resonance (MR) imaging technique, the

MR elastography (MRE) became a promising method to evaluate the compliance or elasticity of brain tissue. I expect that future imaging research will enable us to use this new MRE method to quantitatively evaluate the brain elasticity and give us more evidence.

After all, in this small sample size study, ETV appears to be a possible alternative to manage LPH. The potential mechanism is still not clear and needs further research.

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