neurocritical care society Neurocrit Care (2011) 15:461–468
DOI 10.1007/s12028-011-9543-6

ORIGINAL ARTICLE

The Genesis of Low Pressure Hydrocephalus

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Published online: 27 April 2011

Abstract
Background Low pressure hydrocephalus (LPH) is an uncommon entity. Recognition of this treatable condition is important when clinicians are faced with the paradox of symptomatic hydrocephalus despite low intracranial pressures (ICP). Its etiology remains enigmatic.
Methods We identified patients with LPH from the prospective, inpatient neuro-intensive care database over a 4-year period (2006–2010).
Results Nine patients with LPH were identified over a 4-year period. The time from diagnosis of the initial neurosurgical condition to development of LPH varied from 7 days to 5 years. The sub-zero drainage method of Pang and Altschuler was successful in all cases. LPH was accompanied by transependymal edema in five patients despite low ICP. Four patients developed LPH during their initial admission for intracranial bleeding. As patients entered the LPH state, the ICP remained in a normal range yet daily CSF output from the external ventricular drain was reduced. When LPH patients were drained at sub-zero levels, daily CSF output exceeded baseline values for several days and then receded to baseline. Long-term management was achieved with low pressure shunt systems: six programmable shunts; one valveless ventriculoperitoneal shunt; two ventriculopleural shunts. Conditions most commonly associated with LPH are: subarachnoid hemorrhage, chronic hydrocephalus, brain tumors, and chronic CNS infections.
Conclusions Low pressure hydrocephalus is a challenging diagnosis. The genesis of LPH was associated with a drop in EVD output, symptomatic ventriculomegaly, and a remarkable absence of intracranial hypertension. When LPH was treated with the sub-zero method, a 'diuresis' of CSF ensued. These observations support a Darcy's flux of brain interstitial fluid due to altered brain poroelastance; in simpler terms, a boggy brain state.

Keywords Hydrocephalus · Ventriculoperitoneal shunts · Chronic meningitis · Low pressure · Negative pressure · Elastance

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Introduction

Historically, hydrocephalus was classified as communicating or obstructive (Dandy; [1–3]). In another landmark article, Adams et al. [4] reported three hydrocephalus patients with intracranial pressure (ICP) in the normal range (less than 180 mm H_20), "disabling dementia with psychomotor retardation," and gratifying responses after placement of shunts. They named this condition "normal-pressure hydrocephalus (NPH)." During this historical period, there were additional reports of patients with "low pressure" hydrocephalus (LPH) (Ingram [5]; Bannister [6]; Singounas et al. [7]). These early articles used the terms, NPH and LPH, interchangeably. Efforts to treat LPH were disappointing, and modern neuroimaging was not available.

In 1994, Pang and Altschuler [8] described the low pressure hydrocephalic state in great detail (LPH; also called low pressure hydrocephalus and very low pressure hydrocephalus). Their diagnostic criteria for LPH were: (1) neurologic deterioration from the patient's baseline state in

the presence of medium pressure CSF diversion (either shunts or external ventricular drains (EVD)); (2) ventriculomegaly; (3) persistence of ventriculomegaly with ICP in the normal or low normal range; (4) clinical and radiographic response to sub-zero drainage; and (5) exclusion of other causes such as shunt or ventricular drain malfunction. A subset of their hydrocephalus patients treated with medium pressure shunting developed symptomatic hydrocephalus that was refractory to shunt revision with similar pressure or lower pressure valves. The authors describe a "sub-zero method" which employed sub-atmospheric ventricular drainage achieved by setting the EVD height below the level of the external auditory canal. After extended sub-zero drainage, the patients were successfully shunted using low pressure valves.

The article describes additional observations on sub-zero drainage of nine patients with LPH. We also present a review of LPH [8–14] and its treatment.

Methods

Under IRB approval, we identified LPH patients from a prospective inpatient database who received treatment for LPH at the Kaiser Sacramento Comprehensive Neurosurgery Center. Retrospective chart review was completed using a standardized data query form. We identified patients who met the following criteria: (1) neurologic decline from baseline; (2) radiographic evidence of ventriculomegaly; (3) elevated ICP present at the time of initial EVD placement; (4) clinical and radiographic response to EVD drainage at +5 to +10 cm height; (5) delayed neurologic decline accompanied by recurrent hydrocephalus despite confirmation of a patent EVD and normal ICP; (6) failure to respond to lowering EVD (0 to +5 cm); and (7) clinical and radiographic response to sub-zero drainage.

Following diagnosis of LPH, all patients were treated using the sub-zero method [8]. After clinical and radiographic improvement with sub-zero EVD drainage (typically, −3 to −10 cm), the EVD was raised in small increments (typically 3 cm steps) every 3–5 days. Prior to raising the EVD to a more positive height, clinical and radiographic stability was confirmed. If patients relapsed during EVD weaning, then the process was repeated. Shunt internalization was performed once clinical and radiographic stability was confirmed at neutral or positive EVD settings (typically 0 to +3 cm).

Results

We diagnosed LPH in nine patients over a 4-year period, representing 0.2% of the neuro-intensive care admissions. During this same period, we admitted 274 patients with subarachnoid hemorrhage (SAH), and EVD were placed in 102 on admission. LPH developed in four SAH patients treated initially with EVD for hydrocephalus and elevated ICP. Key features of the case series are summarized in Table 1. Four patients developed low pressure hydrocephalus during their initial admissions for cerebral hemorrhage (cases 4, 5, 7, and 9). We have included a detailed review of two of these patients (see Figs. 1, 2 and Illustrated Cases #4, #6 below). Pathology arising from the posterior fossa was present in five cases (case 1, 2, 5, 6, and 8). Clinical follow up is ongoing and ranges from 6 months to 4 years.

Clinical presentation of LPH varied. Two patients had dramatic and rapid deterioration to coma. Autonomic instability (brain storming) including hypertension, tachypnea, and fever was noted in two patients. Supraventricular tachyarrhythmias occurred in three patients with disproportionately dilated fourth ventricles. Symptoms resolved following LPH diagnosis and sub-zero treatment. Two patients (Cases 2 and 6) suffered relapse of LPH, but responded to sub-zero treatment. These patients restabilized with more aggressive CSF drainage using either a valveless ventriculoperitoneal shunt or a ventriculopleural shunt.

Neuroimaging was systematically reviewed (Table 1; Fig. 3). In five LPH patients, transependymal fluid was present and slowly resolved with sub-zero treatment. An example of transependymal edema in LPH and response to sub-zero treatment is shown in Fig. 2.

Shunt internalization was performed following LPH diagnosis and response to sub-zero drainage. Shunt treatment is summarized in Table 1. For most patients, we placed programmable shunts programmed to the lowest setting. Three patients had refractory LPH and were unable to tolerate positive pressure EVD drainage. A trial of cervical tourniquet in one patient was unsuccessful. For these three refractory LPH patients, two did well with ventriculopleural shunting and the third patient was managed with a valveless ventriculoperitoneal shunt. No long-term complications were observed with low pressure shunting such as shunt infection or subdural hematoma. LPH relapsed in 2/9 patients and ultimately stabilized with either a valveless ventriculoperitoneal shunt or a ventriculopleural shunt.

Illustrated Case #4

A 49-year-old male presented with Hunt Hess Grade 3 subarachnoid hemorrhage due to a ruptured anterior choroidal artery aneurysm (An). CT brain images are provided in Fig. 1a–d and graphical summary of his treatment and clinical course is provided in Fig. 1e. He clinically improved after EVD placement (post-bleed day 4, Fig. 1a) and then developed neurologic decline on post-bleed day 8. CT/CTA brain (Fig. 1b) imaging demonstrated hydrocephalus

Table 1 LPH case series

Age, sex	Diagnosis	Time to LPH	Symptoms	ICP	Transependymal fluid	Final shunt procedure	MRS	
22, M	Idiopathic hydrocephalus	1.5 years	HA, N/V, lethargy	3	Y	Programmable VPS	0	Dilated fourth ventricle
29, M	Ventral brainstem cavernoma	13 days	Coma, extensor posturing	3	Y	V-pleural shunt	3	Onset after cavernoma resection; three relapses
38, M	Aqueductal stenosis	14 years	Lethargy, Parinaud's syndrome	−2	Y	Programmable VPS	0	Nine prior shunt revisions
49, M	SAH, L ant choroidal an	7 days	HA, N/V	9	N	Programmable VPS	2	Fig. 1; EVD CSF output dropped first
52, M	SAH, L SCA an	24 days	HA, lethargy	3	N	Programmable VPS	1	EVD CSF output dropped first
53, M	CNS coccidiomycosis	5 years	Lethargy, Parinaud's syndrome	1	Y	Valveless VPS	0	Fig. 2; one relapse
60, M	SAH, Acomm an	15 days	Lethargy, mute	5	N	Programmable VPS	3	EVD CSF output dropped first
64, M	Cerebellar ICH, AVM resection, VP shunt	2 years	Lethargy, confusion	0	N	V-pleural shunt	3	
71, M	SAH, AcomAn	4 months	Confusion, mutism, gait apraxia	1	Y	Programmable VPS	2	

Key features of patients diagnosed and treated with LPH are listed. The time for initial presentation for neurosurgical management until the time of LPH diagnosis is provided. The Modified Rankin score (MRS) at long-term follow up is provided (range 6 months to 4 years)

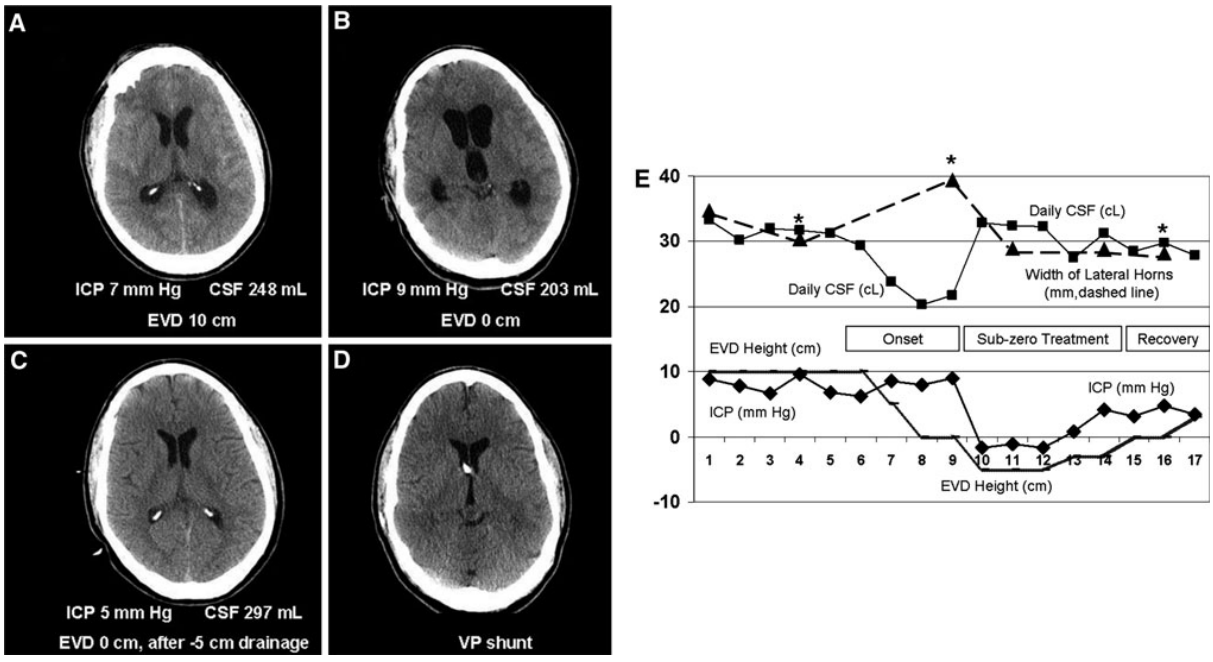

Fig. 1 The genesis of LPH. A 49-year-old male presented with Hunt Hess Grade 3 subarachnoid hemorrhage due to a ruptured anterior choroidal artery aneurysm. CT brain images are provided in **a**–**d** and graphical summary of his treatment and clinical course is provided in **e**. He clinically improved after EVD placement (post-bleed day 4, **a**) and then developed neurologic decline on post-bleed day 8. CT/CTA brain (**b**) imaging demonstrated hydrocephalus without angiographic vasospasm. EVD output declined with the genesis of LPH. EVD was lowered to +5 and then 0 cm without clinical or radiographic improvement (**e**). The sub-zero method leads to clinical and radiographic improvement (**c**) which was sustained following step-wise challenging of the EVD and subsequent placement of a low pressure ventriculoperitoneal shunt (**d**). The different ventricular configurations at similar ICP values (**a**–**c**) is an example of hysteresis

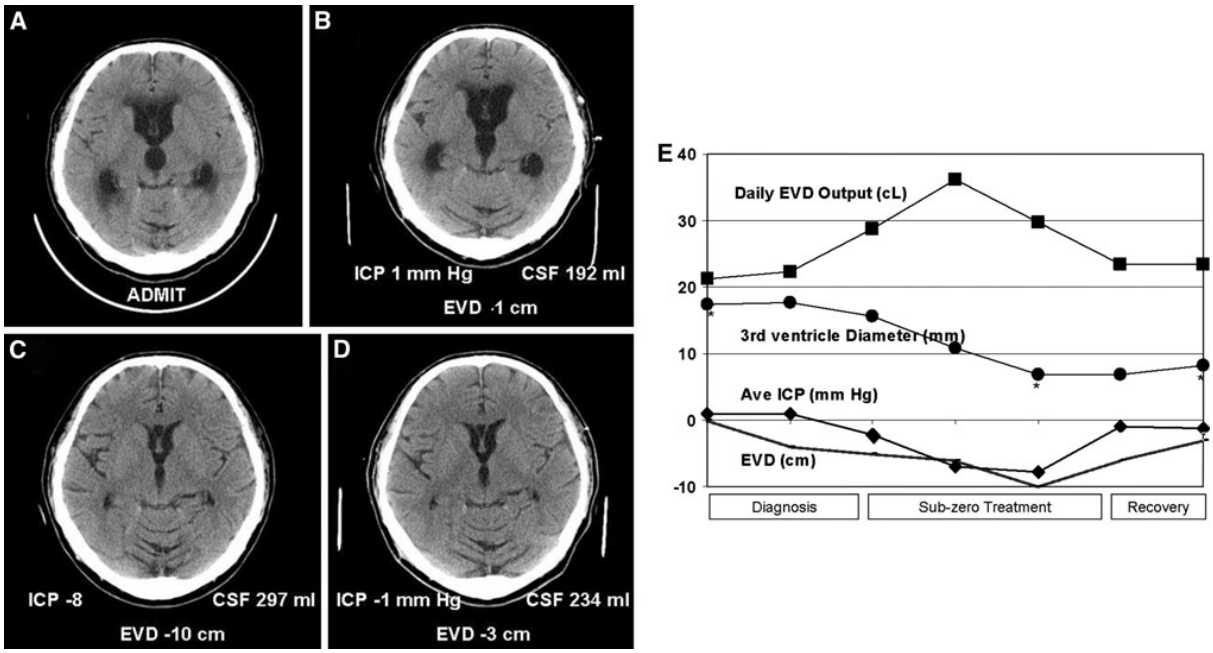

Fig. 2 A shunted patient with chronic coccidiomycosis and prior admission for LPH presented with symptoms of shunt failure (**a**). The shunt was removed and an EVD was placed. CSF routine and fungal cultures were sterile. HCP and transependymal fluid persisted despite EVD drainage at +1 cm and ICP at below normal values (**b**). The sub-zero method leads to a rise in EVD output, resolution of transependymal fluid, restoration of the subarachnoid space, clinical and radiographic improvement (**c**, **e**). After sustained improvement with EVD at −3 cm height (**d**), a valveless ventriculoperitoneal shunt was placed. The different ventricular configurations at similar ICP values (**b**, **d**) is an example of hysteresis

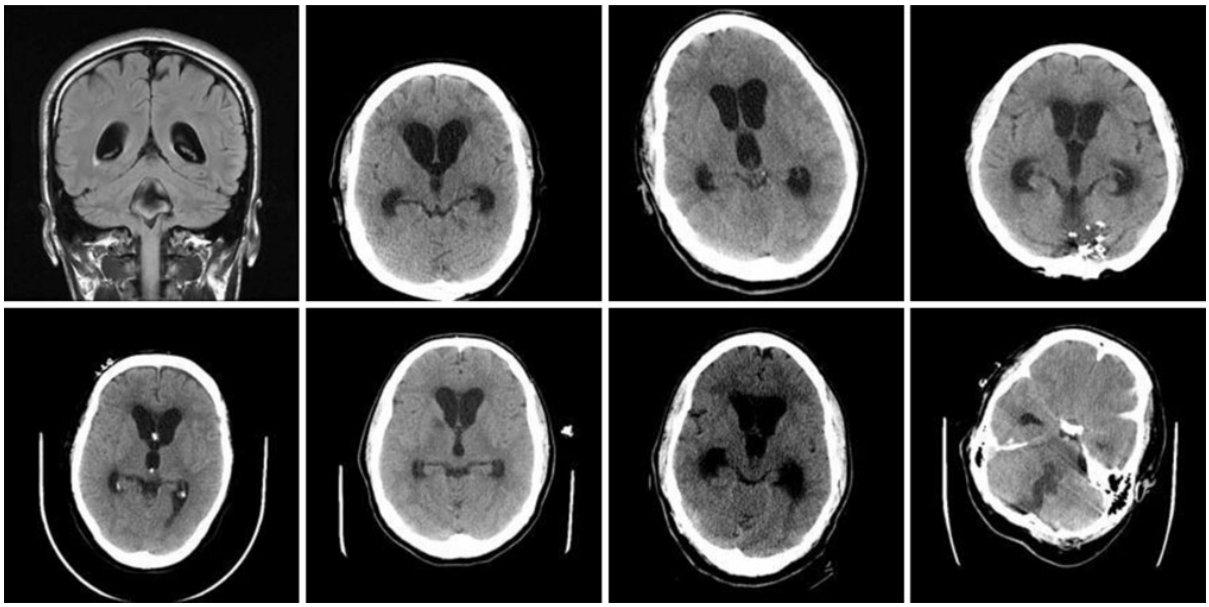

Fig. 3 Neuroimaging of LPH

without angiographic vasospasm. EVD output declined with the genesis of LPH. EVD was lowered to +5 and then 0 cm without clinical or radiographic improvement (Fig. 1e). The sub-zero method leads to clinical and radiographic improvement (Fig. 1c) which was sustained following stepwise challenging of the EVD and subsequent placement of a low pressure ventriculoperitoneal shunt (Fig. 1d). The different ventricular configurations at similar ICP values (Fig. 1a–c) provide an example of hysteresis [12].

Illustrated Case #6

A 53-year-old patient previously shunted for chronic coccidiomycosis presented with symptoms of shunt failure (Fig. 2a). The shunt was removed and an EVD was placed. Bacterial and fungal cultures of the CSF were sterile, and coccidiomycosis titers in the serum and CSF were stable compared to recent values. Ventriculomegaly and transependymal fluid persisted despite EVD drainage at +1 cm and ICP at below normal values (Fig. 2b). The sub-zero method leads to a rise in daily EVD output, resolution of transependymal fluid, restoration of the subarachnoid space, and clinical and radiographic improvement (Fig. 2c, e). After sustained improvement with EVD at −3 cm (Fig. 2d), a valveless ventriculoperitoneal shunt was placed. The different ventricular configurations at similar ICP values (Fig. 2b, d) again provide an example of hysteresis [12].

Discussion

LPH is a striking and paradoxical example of symptomatic hydrocephalus with low ICP. In addition to LPH, other examples of neurological syndromes occur in the setting of intracranial hypotension including: spontaneous intracranial hypotension [15]; sinking skin flap syndrome and paradoxical herniation in craniectomy patients [16]; and subdural hematomas in NPH patients with shunt overdrainage.

Review of the literature has identified 34 reported LPH cases that met Pang/Altschuler criteria (Table 2; [8–14]). Conditions associated with LPH include: chronically shunted hydrocephalus patients, subarachnoid hemorrhage, and tumor. Two articles observed the onset of LPH after lumbar puncture in shunted patients (Ingram [5]; Dias et al. [10]) and one article [9] described the onset of negative pressure hydrocephalus following cystopleural shunting in patients with pre-existing VP shunts for Arnold-Chiari malformations with myelodysplasia. We noted a predilection for LPH patients to have pathology arising from the posterior fossa such as: medulloblastoma, ependymoma, cerebellar hemorrhage, pontine hemorrhage, and basilar meningitis. One center placed valveless shunts as an alternative to the sub-zero treatment method (Owler et al. [11]). We did not find any reported complications of treated LPH patients, such as slit ventricle syndrome or SDH formation.

The pathophysiology of LPH is complex. Viscoelastic principles have been employed to explain LPH. In the classic model of elasticity, linear changes in shape (strain) are observed in response to external forces (stress) as described by Hooke's law (strain is directly proportional to stress). For non-Newtonian fluids, stresses applied over a prolonged period will also cause the material to flow like a viscous liquid. Hakim et al. [17] proposed that hydrocephalus could be modeled by treating the brain as a porous sponge with viscoelastic properties. Pang and Altschuler [8] postulated that LPH symptoms were due to the severe ventricular distortion and associated elevated radial forces on the ventricles rather than elevated ICP; susceptible patients had brains with low elasticity; water within the brain tissue was forced out (reduced); and that sub-zero treatment would restore brain viscoelasticity and allow water entry back into the brain parenchyma.

Table 2 Summary of LPH literature

Reference	Cases	SAH	Tumor	Chronic HCP	Chiari, myelomeningocele	Other conditions
Modern series						
Pang and Altschuler [8]	12	4	6	2	0	Multiple shunt systems present
Vassilyadi et al. [9]	2	0	0	0	2	
Dias et al. [10]	2	0	1	0	1	Posterior fossa cyst; cryptococcal meningitis
Owler et al. [11]	5	0	1	2	0	
Lesniak et al. [12], ten cases	10	5	0	3	0	IVH; meningitis
Daniel et al. [13]	1	0	0	0	0	Hemispherectomy
Clarke and Meyer [20]	2	1	0	1	0	
Akins and Guppy [16]	9	4	0	2	0	Coccidiomycosis; cerebellar AVM; pontine cavernoma
Total	43	14	8	10	3	8

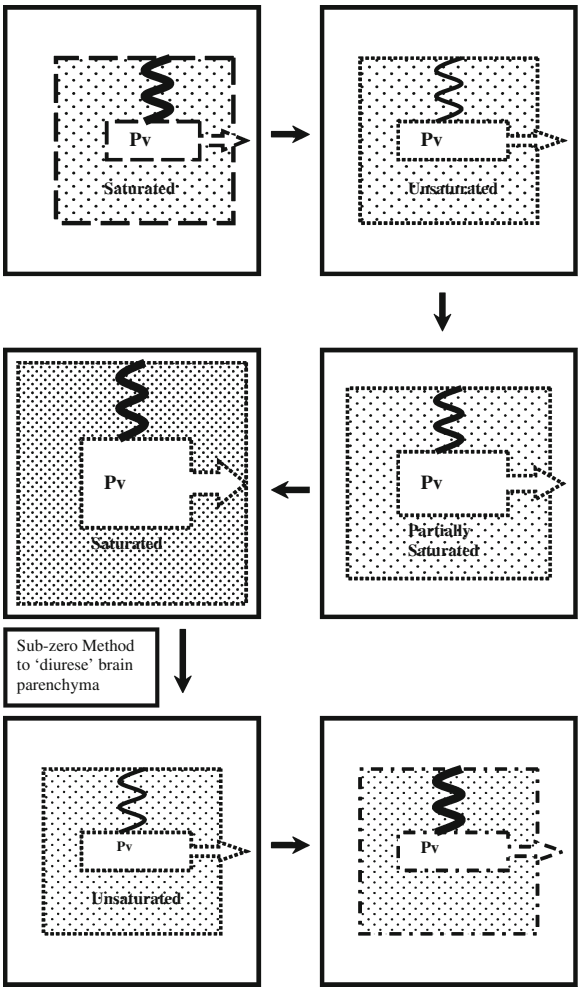

Fig. 4 A theoretical diagram for LPH using the brain poroelastic model. The poroelastic model treats the brain like a porous sponge with elastic properties (Young's modulus). The *outer box* (*solid line*) represents the inflexible and non-porous skull and dura. The *inner box* (*dash line*) represents the transcortical mantle. The permeability of the cortical mantle is illustrated by the integrity of the *dash line*. This is a simplification since permeability is different in white and gray matter. The *dots* within the cortical mantle reflect the interstitial brain water content. The *inner rectangle* and *arrow* represent the ventricular system and the intraventricular pressure is denoted Pv. For simplification, the pressure in the subarachnoid space has been held constant. The *wavy line* represents the elastic modulus (Young's modulus) of the transcortical mantle. *Top/Left* Baseline configuration where the force exerted on the cortical mantle by intraventricular pressure is matched by the elastic potential energy (heavy spring) of the cortical mantle. At steady state, intracranial pressure, intraventricular pressure (Pv) and intraparenchymal pressure are the same. When brain permeability or elasticity is altered, differences in intraventricular and intraparenchymal pressures arise and lead to either expansion or contraction of the ventricular system (with corresponding reciprocal changes in the cortical mantle). *Top/right* as brain permeability increases, the brain "sponge" will initially be unsaturated. This state is associated with a time-dependent increase in brain elasticity illustrated by a thinner spring. *Middle/right* with the increased brain permeability, the brain sponge will absorb more water and water will flow from the ventricles into the parenchyma. This flow constitutes a Darcy flux (see "Discussion" section). In the partially saturated state, the brain elasticity will increase (thicker spring). *Middle/left* at the new steady state, the force exerted on the cortical mantle by the same intraventricular pressure will lead to ventricular dilation and reduction in the subarachnoid space. When the elastic potential energy (heavy spring) matches the force exerted across the cortical mantle, the ventricular configuration will reach equilibrium. Brain water content is increased, due to altered brain permeability. *Bottom/left* a reduction in the force exerted on the cortical mantle (sub-zero treatment) will "wring out" the sponge-like cortical mantle leading to an unsaturated state. Water will flow from the parenchyma into the ventricular system. Again, this constitutes a Darcy flux. The unsaturated state will have greater elasticity. Since the force exerted across the cortical mantle is less, the strain on the cortical mantle is less, the ventricular size is reduced, and the subarachnoid space is restored. *Bottom/right* long-term maintenance of the desired ventricular configuration in LPH relies on low pressure shunt systems

The LPH model proposed by Lesniak et al. [12] melded features of Adam's "hydraulic press" model for NPH [4] and the viscoelastic model of Pang and Altschuler [8]. In response to changes in ICP, the authors observed how LPH brains demonstrate hysteresis, which is a lagging of an effect behind its cause. In essence, the ventricular system in LPH patients behaves in a similar manner to the lung during inspiration and expiration, where the intrapleural pressure is less during deflation and greater during inflation for a given lung volume. The authors postulated that ICP increases would lead to ventricular dilation. The ventricular dilation would have secondary effects on brain elastance. Therefore, the ventricular system would remain dilated even after ICP was lowered through CSF drainage.

We favor a poroelastic model for LPH. We will briefly introduce readers to poroelastic principles [17–22]. Porous materials have a solid matrix and permeable pores filled with fluid (liquid or gas). The poroelastic theory has been used to model brain biomechanics as well as hydrocephalus (Hakim et al. [17]; Nagashima et al. [18]; Pena et al. [19]; Clarke and Meyer [20]; Momjian and Bichsel [21]; Linninger et al. [22]). Modeling of hydrocephalus using a poroelastic model revealed ventricular dilation at lower ICP values compared to the elastic model [18, 19, 21] and the presence of interstitial fluid capping the horns of the ventricles [19, 21].

We observed a decline in EVD output during the genesis of LPH (Fig. 1; Table 1) which we attribute to an increase in both brain extracellular fluid and ventricular volume. We conclude that the increase in brain extracellular fluid was a result of increased brain permeability in the patients who developed LPH despite a functioning EVD and normal ICP. This finding is consistent with the Nagashima et al. [18] and Pena et al. [19] hydrocephalus model which predicts greater edema in the periventricular white matter and more compaction of gray matter. The findings are in

conflict with the model of Hakim et al. [17] for NPH, Pang and Alschuler for LPH [8], and Linninger et al. [22] for communicating hydrocephalus which predicts that the cortical mantle will be "wrung out" as the ventricles expand. During sub-zero treatment, we observed a rise in EVD output which we attribute to reduction in ventricular volume and reduction in brain extracellular fluid (Fig. 2; Table 1). Owler et al. [11] was the first to draw attention to periventricular edema in LPH, and they also concluded that some LPH patients could demonstrate radiographic signs of increased brain extracellular fluid in the face of low ICP. Other LPH patients do not have radiographic signs of transependymal fluid increases and there may be other contributing factors. Ventricular configuration may play a role, since two of the patients had surgically altered fourth ventricles and disproportionate enlargement of the fourth ventricle was observed in two other patients. Also, lowering the pressure in the subarachnoid space may precipitate LPH in susceptible patients [5, 9, 10], presumably due to a pressure gradient across the cortical mantle in patients with highly compliant brains.

The development of HCP at low pressures rather than elevated pressures is an important observation. In most clinical settings, the finding of periventricular transependymal fluid is interpreted as a sign of elevated ICP. Paradoxically, we observed transependymal fluid in 5 of 9 LPH patients. The movement (q) of fluid in and out of a porous substance is dependent on pressure gradients (ΔP) rather than absolute pressure, as illustrated by Darcy's law: $q = -k(\Delta P)/\mu$), where q is the flux of fluid (q, discharge per unit area), k is permeability, and μ is fluid viscosity. The finding of increased periventricular fluid at normal or low ICP suggests that LPH patients have enhanced brain permeability in these zones. We hypothesize that underlying inflammatory or degenerative changes alter periventricular brain permeability in LPH patients, for example, chronic CNS infection or chemical meningitis from subarachnoid hemorrhage. In addition, as demonstrated by the pressure–volume index measurements [8], LPH patients have unusually compliant brains. Given the diversity of conditions associated with LPH (see Table 2), we predict that there may be multiple pathophysiologic paths that lead to LHP. We have not treated patients with tumor-related LPH, and our observations may not apply to this subtype.

The decline in EVD output during the genesis of LPH and a rise in EVD output during sub-zero treatment are consistent with a Darcy flux of fluid into and out of the sponge-like brain parenchyma due to increased brain permeability (Fig. 4; [18, 19]) in additional to expanded ventricular volume and displacement of the subarachnoid space. Conceptually, as brain permeability increases, brain water increases, and the brain becomes boggy. The change in brain permeability is likely heterogeneous, with greater permeability involving white matter and periventricular zones [18, 19]. Brain elasticity would be affected by the degree of saturation of brain water. For example, a sponge in a "wrung out" desaturated state offers less resistance to an external force that a saturated sponge. Hysteresis can arise from such time-dependent process (Figs. 1, 2).

In conclusion, this case series is confirmatory regarding the safety and effectiveness of the sub-zero method in patients with LPH. These observations support the poro-elastic model for LPH. The case series and literature review provide examples of LPH presenting in a fulminant manner along with the more typical chronic decline.

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