Venous Sinus Pulsatility and the Potential Role of Dural Incompetence in Idiopathic Intracranial Hypertension

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BACKGROUND: Idiopathic intracranial hypertension (IIH) remains a poorly understood and therapeutically challenging disease. Enthusiasm has emerged for endovascular therapy with stent reconstruction of dural sinus narrowing; however, a complete understanding of the hydrodynamic dysequilibrium is lacking.

OBJECTIVE: To review and characterize catheter manometry findings including pulsatility changes within the venous sinuses in IIH.

METHODS: Cases of venous sinus stent implantation for IIH were retrospectively reviewed.

RESULTS: Three cases of venous sinus stent implantation for treatment of IIH are reported. All cases demonstrated severe narrowing (>70%) within the transverse sinus and a high pressure gradient across the lesion (>30 mm Hg). Stent implantation resulted in pulsatility attenuation, correction of pressure gradient, and improvement of flow.

CONCLUSION: We report the finding of high venous sinus pulsatility attenuation after stent implantation for dural sinus narrowing and propose the hypothesis that this finding is a marker of advanced dural sinus incompetence. This characteristic may be useful in identifying patients who would benefit from endovascular stent remodeling.

KEY WORDS: Angioplasty, Manometry, Pseudotumor cerebri, Stenting

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Idiopathic intracranial hypertension (IIH) is a common and easily diagnosed disorder, but many aspects of its pathophysiology remain poorly understood. Therapeutic management is not uniform and can be challenging in the 15% to 30% of patients presenting with debilitating headaches and visual impairment due to papilledema.[1,2] Recent investigation of the role of dural sinus narrowing (DSN) in IIH has generated interest in potential endovascular sinus stent reconstruction to restore venous outflow and cerebrospinal fluid (CSF) drainage. A considerable amount of information is available by catheter venogram evaluation; however, the role of each finding in the diagnosis and management of IIH is not well defined. Endovascular abnormalities primarily include focal DSN with an associated pressure gradient of greater than or equal to 10 mm Hg across the stenosis.[3] Venous sinus pulsatility characteristics have not been previously reported. We describe endovascular evaluation and treatment of 3 patients with IIH, highlight the finding of venous pressure pulsatility attenuation after stent implantation, discuss the relevance of these findings to the hypothesis that IIH is related to dural sinus incompetence, and suggest the possible important role of this waveform finding in diagnosis and management.

ABBREVIATIONS: DSN, dural sinus narrowing; **DSV,** digital subtraction venography; **ICP,** intracranial pressure; **IIH,** idiopathic intracranial hypertension; **MRV,** magnetic resonance venography

METHODS

Three patients were referred following the diagnosis of IIH by an experienced neuro-ophthalmologist. All fulfilled diagnostic criteria for IIH as outlined by the International Headache Society.[4] Patients either presented with progressive visual loss in the context of newly diagnosed IIH ("fulminant IIH"),[5] or due to chronic papilledema with failure of medical management. If indicated for prestent evaluation, diagnostic venography and manometry was performed under conscious sedation

for initial evaluation by the use of the manometry technique described below. For stent implantation, patients were prepared with a dual-antiplatelet regimen of aspirin 325 mg and clopidogrel 75 mg daily for 7 days or a loading dose of aspirin and clopidogrel if the procedure was urgently performed. Procedures were performed with patients under general anesthesia. A transfemoral venous approach was used. Patients were systemically heparinized to maintain an ACT level of 250 to 300 seconds, and a coaxial system including a 6F 070 Neuron guide catheter (Penumbra, Inc., San Leandro, California) was used to establish high cervical venous access in the internal jugular vein. Images were obtained with biplane digital subtraction venography. A Renegade Hi-Flo microcatheter (Stryker Neurovascular, Kalamazoo, Michigan) was advanced over a 0.014 microwire and positioned at the respective locations within the venous sinuses for venography and manometry. The external auditory canal was used for the manometry transducer zero point. Manometry measurements were obtained at the superior sagittal sinus, torcula, and bilaterally at the proximal transverse sinuses, distal transverse sinuses, sigmoid sinuses, and internal jugular veins. Stent deployment and balloon inflations were performed by using standard techniques. Poststent venography and manometry were obtained in all cases. Postoperatively, patients were treated with dual-antiplatelet therapy with aspirin 325 mg daily and clopidogrel 75 mg daily.

RESULTS

Case 1

A woman in her thirties with a body mass index (BMI) of 31.5 and a 4-year history of IIH presented with severe frequent headaches, tinnitus, and papilledema with progressive vision loss. Failed medical and surgical management included attempts at weight loss, acetazolamide, repeated lumbar punctures (most recent preadmission opening pressure $>$ 44 cm H_2O), and ventriculoperitoneal shunt placement. Magnetic resonance venography (MRV) suggested bilateral transverse sinus narrowing, and she was referred for digital subtraction venography (DSV). The DSV revealed severe narrowing at the distal left transverse sinus estimated at 80% to 90% and mild narrowing at the distal right transverse sinus estimated at 40%. A significant gradient (30 mm Hg) was present across the left transverse sinus stenosis. Prestenotic pressure recordings showed a pulsatile waveform (Figure 1). Under general anesthesia, angioplasty was performed with use of a Sterling 6 × 20 mm

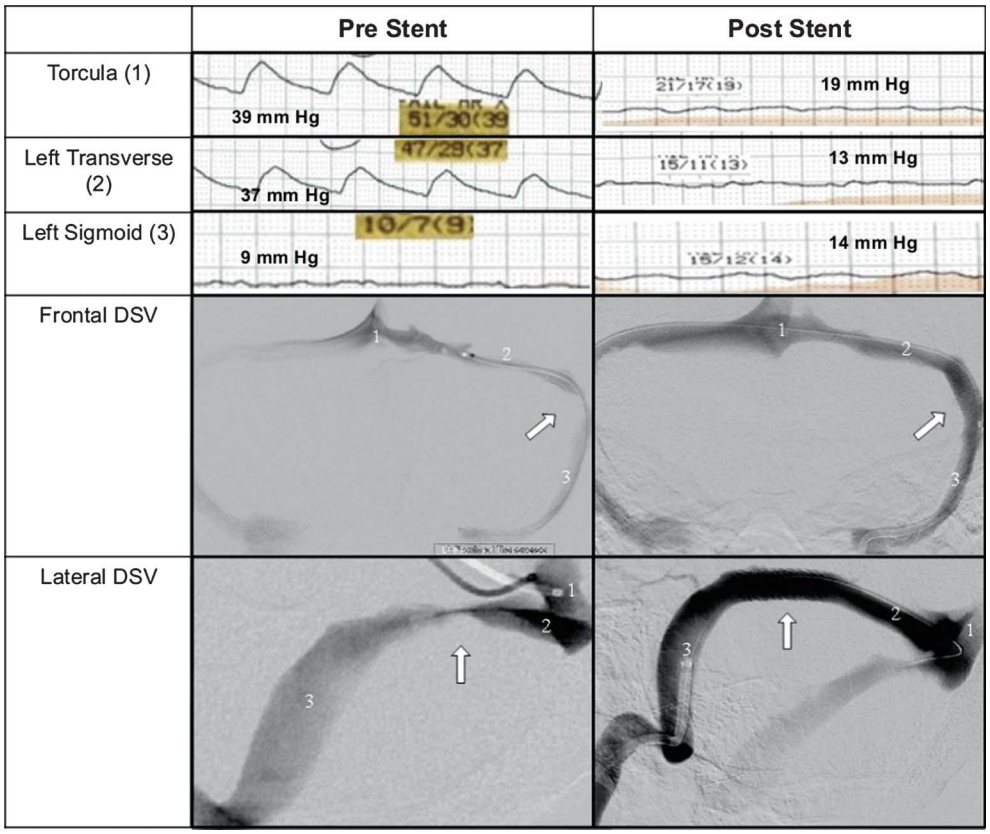

FIGURE 1. *Manometry tracings and reference venogram from case 1. Manometry tracings obtained at the torcula (1), left transverse sinus (2), and left midsigmoid sinus (3) demonstrate pulsatility proximal to the stenosis (prestent column), which resolved following stent implantation of the left transverse venous sinus narrowing (poststent column). Frontal and lateral digital subtraction venography (DSV) images are provided for reference of manometry locations and to illustrate the lesion (arrow) before and after treatment.*

balloon dilatation catheter (Stryker Neurovascular, Kalamazoo, Michigan); postplasty follow-up DSV revealed 70% residual narrowing. An Express 6 × 18 mm balloon mounted stent (Stryker Neurovascular, Kalamazoo, Michigan) was then centered across the narrowing and deployed by balloon inflation. Poststenting DSV confirmed accurate placement of the stent across the stenosis, with no evidence of residual narrowing. The poststent pressure gradient across the left transverse sinus was reduced to 5 mm Hg, and the waveform pulsatility attenuated (Figure 1). There were no complications. Upon discharge, the patient had only a mild residual headache and no tinnitus. Visual acuities and peripheral field limits had begun to improve. At 21-month follow-up, the patient had no recurrence of papilledema or IIH-related headaches. Follow-up noninvasive imaging at 6 months and 18 months demonstrated normal dural venous sinus patency with no significant stent restenosis.

Case 2

A woman in her twenties with obesity (BMI of 39.9) presented with a 3-week history of severe headaches and visual disturbances including advanced visual field constriction in both eyes, diminished visual clarity, and double vision. Following a negative magnetic resonance imaging (MRI), lumbar puncture was performed that yielded an opening pressure of 60 cm H_2O. Treatment with acetazolamide initiated by an outside physician several days earlier had provided no relief or improvement. She was diagnosed with fulminant IIH and underwent MRV, which showed severe bilateral transverse sinus stenosis. Subsequent DSV revealed 75% narrowing within the dominant left transverse sinus and an associated 36 mm Hg pressure gradient across the narrowed segment. A pulsatile waveform was noted in the superior sagittal sinus and the transverse sinus but not in the sigmoid sinus or the internal jugular vein (Figure 2). The patient underwent transverse sinus stenting with the use of a 7 × 40 mm Wallstent (Stryker Neurovascular, Kalamazoo, Michigan). Angioplasty was then performed with a 7 × 20 mm Sterling balloon dilatation catheter (Stryker Neurovascular, Kalamazoo, Michigan) to optimize wall apposition. Poststenting DSV confirmed accurate placement of the stent across the stenosis, with no evidence of residual narrowing. Postangioplasty, the pressure gradient had decreased to 5 mm Hg with significant attenuation in the pulsatile waveform. The patient reported immediate improvement in headaches and visual symptoms. One-month follow-up funduscopy demonstrated nearly

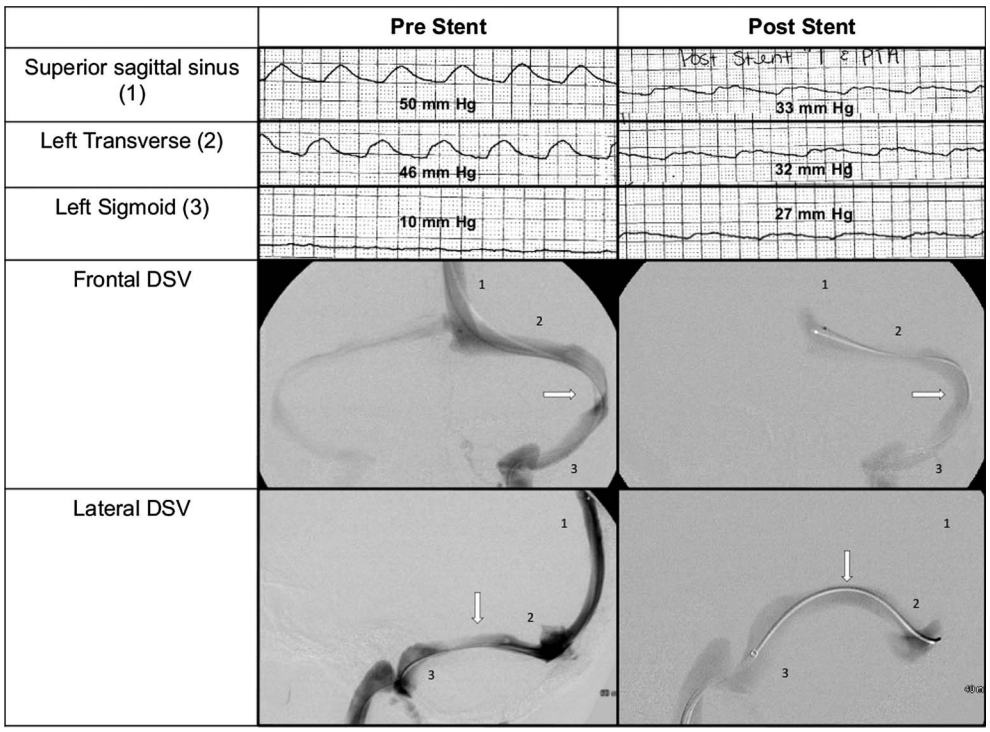

FIGURE 2. *Manometry tracings and reference venogram from case 2. Manometry tracings obtained at the superior sagittal sinus (1), left transverse sinus (2), and left midsigmoid sinus (3) demonstrate pulsatility proximal to the narrowing (prestent column), which resolved following stent implantation of the left transverse venous sinus narrowing (poststent column). Frontal and lateral digital subtraction venography (DSV) images are provided for reference of manometry locations and to illustrate the lesion (arrow) before and after treatment.*

complete resolution of disc swelling (Figure 3). At 3-month and 1-year follow-up visits, the patient reported continued improvement in headaches and complete recovery in her diplopia. There was residual optic atrophy, but visual functions had significantly recovered in comparison with her preadmission status. Noninvasive imaging demonstrated normal dural venous sinus patency with no significant stent restenosis.

Case 3

A woman in her twenties with severe obesity (BMI 47.7) reported worsening headaches over a period of approximately 1 year and bilateral peripheral visual field loss beginning 1 month before neurovascular referral. Her examination demonstrated bilateral papilledema and visual field restrictions. The MRV indicated bilateral transverse sinus narrowing. She was intolerant to acetazolamide and had been treated unsuccessfully with methazolamide. Lumbar puncture showed elevated opening pressure but offered little improvement in symptoms and findings. She underwent venous sinus manometry that showed severe narrowing (>70%) of the right transverse sinus with mean venous sinus pressures elevated to 45 mm Hg with an approximately 30 mm Hg gradient across the stenosed segment (Figure 4). Two Wallstents were successfully implanted with balloon angioplasty to optimize stent apposition. Poststent evaluation showed attenuation of venous sinus pulsatility, improvement of flow, increases in sinus caliber, and elimination of the pressure gradient. Poststent venous sinus pressures measured a mean value of 18 to 21 mm Hg on measurements throughout the superior sagittal sinus to the sigmoid

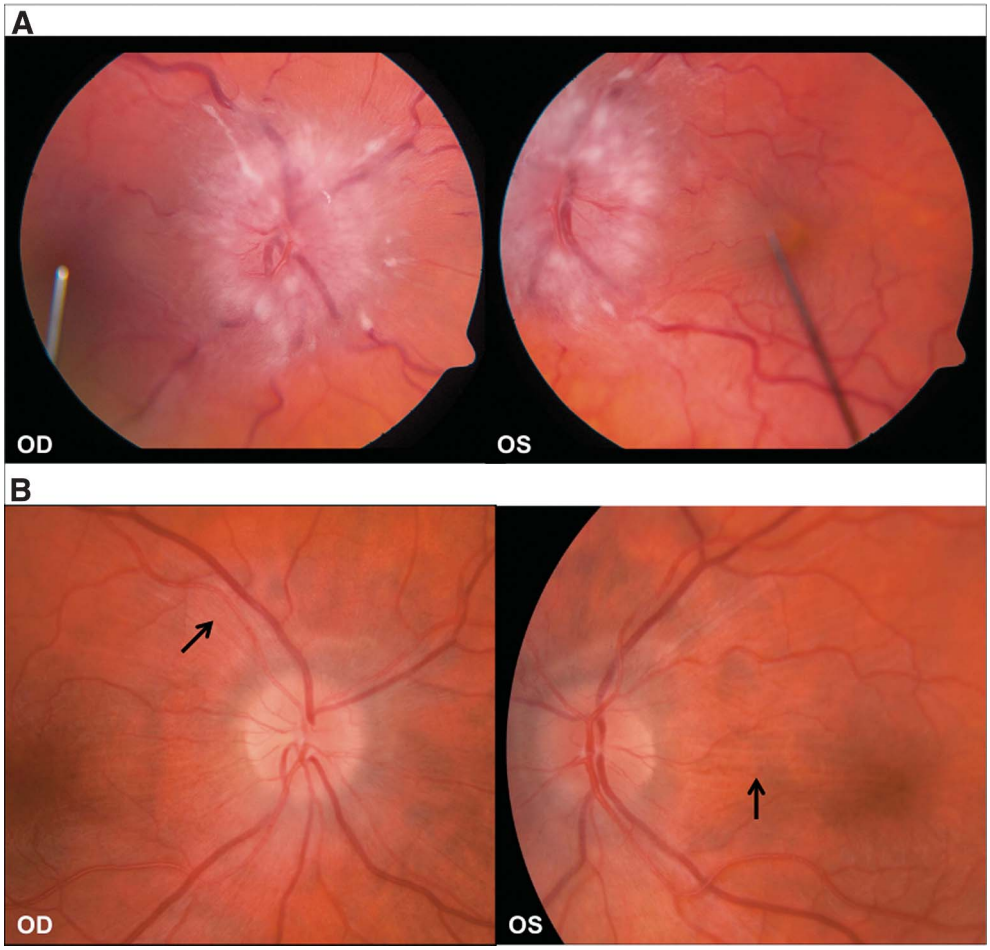

FIGURE 3. *Fundus photographs, case 2.* **A**, *fundus photographs from 1 day before stent implantation demonstrate severe papilledema in both eyes with extensive microvascular ischemic changes ("cotton wool spots") of the proximal nerve fiber layer. There is mild edema in the papillomacular bundle and the macula (OS).* **B**, *subsequent photographs 1 month after stent implantation show nearly resolved papilledema in the right eye (OD) and mild residual nasal disc swelling in the left eye (OS). Subtle striations in the nerve fiber layer of both eyes indicate resolving edema and loss of nerve fibers (arrows). OD, right eye; OS, left eye.*

sinus and the gradient was reduced to 1 mm Hg. One month after stent placement, she had persistent headaches, but marked improvement of papilledema and visual functions. A repeat venogram with manometry was performed followed by a lumbar drain. Venography demonstrated widely patent stents with no evidence of residual stenosis or in-stent thrombosis. Mean venous sinus pressures were 21 to 22 mm Hg. Lumbar drain showed no evidence of elevated CSF pressure. She was continued on dual-antiplatelet therapy with aspirin and clopidogrel.

Lesion characteristics, manometry values, and follow-up are summarized in the Table.

DISCUSSION

We describe high-amplitude venous pulsatility in the transverse sinus of 3 IIH patients that attenuated after stent reconstruction. This finding may contribute to the understanding of the pathophysiology, venous sinus manometry interpretation, and treatment planning of IIH. The presence of high venous pulsatility in IIH is likely due to dural sinus incompetence that leads to intracranial pressure (ICP) waveform transmission. Therefore, high pulsatility may be a marker of an advanced disease state in which chronically raised ICP leads to dural weakening, venous outflow obstruction, and a subsequent self-perpetuating cycle. The common location of stenosis within the transverse sinuses suggests a susceptibility of this region to weakening from chronic increased ICP. Improvement in pulsatility after stent implantation, which in our patients was not sustained with angioplasty alone, supports the theory of dural sinus incompetence. The manometry finding of high pulsatility may serve as an important marker for identifying patients who have developed dural sinus incompetence and thus may benefit from endoluminal wall stabilization with stent implantation.

Over a century has passed since Quincke first described the syndrome of what is currently known as IIH, and still the pathogenesis remains uncertain.[6] The syndrome is puzzling because increases in ICP are seen without any organic space-occupying lesion. The possible pathophysiological role of cerebral DSN in IIH has recently sparked considerable interest and debate.[7-9] It has been proposed that increased venous pressure may be a final common pathway for IIH.[10] Increased venous pressure can be theoretically caused by a number of mechanisms,

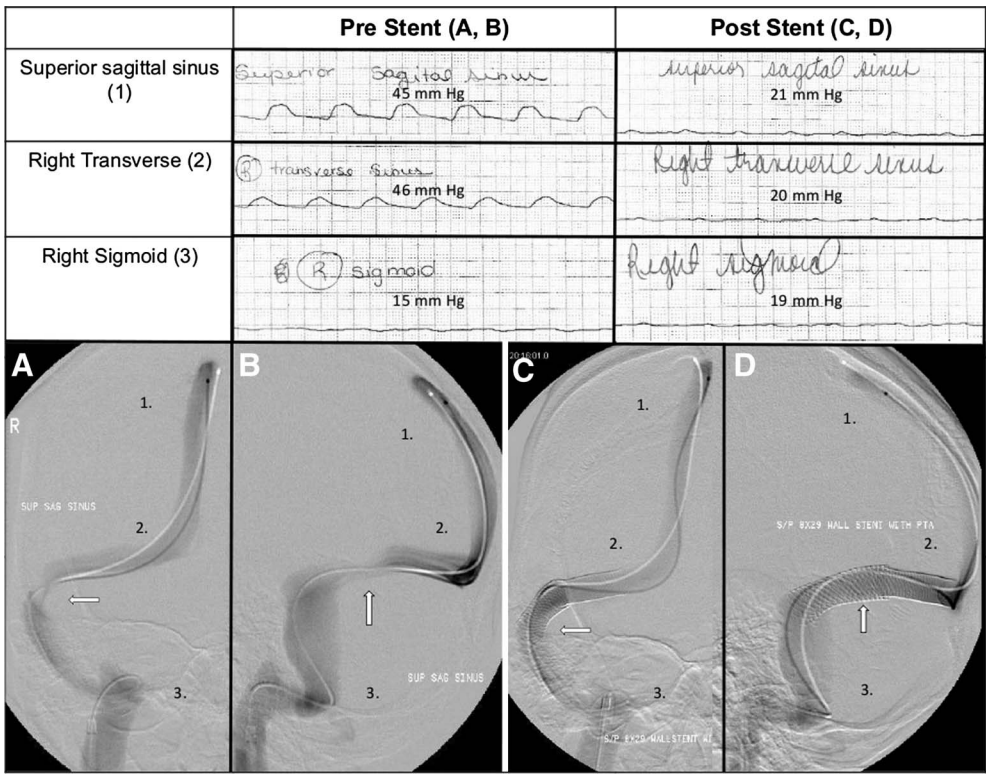

FIGURE 4. *Manometry tracings and reference venogram from case 3. Manometry tracings obtained at the superior sagittal sinus (1), right transverse sinus (2), and right mid-sigmoid sinus (3) demonstrate pulsatility proximal to the narrowing (prestent column), which resolved following stent implantation of the right transverse venous sinus narrowing (poststent column). Venography images show the focal narrowing (arrow) in frontal projection before (**A**) and after stent placement (**C**) and in lateral projection before (**B**) and after stent placement (**D**). Numbers are provided for reference of manometry locations.*

TABLE. Radiographic Lesion Characteristics, Manometry Values, and Follow-up[a]

	Case 1	Case 2	Case 3
Sinus narrowing	>80%	>75%	>70%
Target sinus	Left transverse	Left transverse	Right transverse
Prestent mean pressure proximal to narrowing in direction of flow, mm Hg	39	50	45
Prestent mean pressure distal to narrowing in direction of flow, mm Hg	9	10	15
Prestent mean pressure gradient, mm Hg	30	36	30
Poststent mean pressure gradient, mm Hg	5	5	1
Stent	Express	Wallstent	Wallstent (2)
Follow-up imaging, time interval	MRV, 18 mo	MRV, 12 mo	DSV, 1 mo
Stent lumen	Patent	Patent	Patent
Manometry	Not available	Not available	Normal pressures with no gradient
Longest available follow-up (all patients had papilledema remission)	21 months	12 months	1 month

[a]MRV, magnetic resonance venography; DSV, digital subtraction venography.

generalized venous hypertension, and all causes of intradural sinus flow obstruction.[10] In patients with IIH and DSN, the role of DSN as either causative, contributory, sustaining, or merely a secondary phenomenon remains controversial. An early hypothesis in IIH pathophysiology attributes DSN to prolonged intracranial hypertension leading to weakening of the dural sinus and allowing collapse.[11] However, investigation of the hydrodynamics of DSN with focus on compliance has been limited.

Previous reports describe the use of transcranial sonography and MRV to evaluate venous sinus hemodynamics; however, pulsatility characteristics by direct manometry have been unexplored.[12-14] Sonographic investigations in normal subjects have shown that the resistance index of cerebral sinuses is higher than that of veins, likely because of a comparable increase in rigidity of the cerebral sinus walls; additionally, there is an increase in pulsatility with age.[12,14] Although data on flow have been reported, reference data for venous sinus pulsatility are not available. The cerebral venous sinuses are complex with considerable anatomic variability, which limits sonographic and MRV evaluation.[12]

In contrast, characterization of pulsatility on venogram may lend greater insight to the fundamental properties of dural sinus hydromechanics and the abnormalities that result from intracranial hypertension. Venous sinus waveform pulsatility may in part be a function of the sinus wall compliance. Two factors contribute to venous sinus compliance: distensibility of the surrounding dura and global intracranial compartment compliance governed by the Monro-Kellie doctrine.[15] Thin dura will result in net sinus compliance dominated by the global compartment compliance, whereas, in the presence of a thick and rigid dural sinus, compliance will predominantly be affected by dural distensibility.

The origin of the venous sinus pulse waveform within the enclosed intracranial space likely arises from direct transmission of the ICP waveform across the CSF-dural sinus interface. Theoretically, in the setting of a dura weakened by chronic ICP, thinning may allow for increased ICP waveform transmission. It is unlikely that the venous sinus waveform arises from the only other 2 possibilities, via arterial transmission through a capillary bed or retrograde venous transmission from the heart.

Following this understanding of venous sinus hydrodynamics, pulsatility would be increased in pathological states of weakening of the dural sinus, which allows for CSF pressure transmission across the dural interface. It has been proposed that the dural sinus in a healthy individual maintains a certain elasticity, which increases after prolonged increase in ICP, leading to a weakening of the wall that makes it susceptible to narrowing.[11] This could conceivably lead to a self-perpetuating cycle of the disease process in IIH. Additionally, a recent study suggested that the pulsatile ICP may be more relevant than the static ICP in the diagnosis of IIH.[16] Our findings are in accordance with these previous results. Furthermore, if the narrowing was simply due to a focal stenosis of an elastic venous system, then prestenotic dilatation would be expected. The absence of prestenosis dilatation likely represents presence of normal dural integrity that has been unaffected by increased ICP and yet maintains the normal rigidity threshold to prevent dilatation resulting from increased sinus pressure. Therefore, rather than a true stenosis, the focal narrowing of the dural sinus seen in IIH is due to collapse of the sinus from outward pressure imposed at a region predisposed to dural integrity weakening.

Pulsatility as a surrogate measure of dural weakening may aid in staging the disease process and selection of patients for treatment. Flow velocity and the pulsatility of the central retinal artery, for instance, have been shown to vary depending on IIH severity and papilledema.[17] Specifically, a biphasic distribution of central retinal artery pulsatility index was found. Although the authors hypothesized that this normalization was largely due to local

autoregulatory vascular changes, this may also represent progression of dural sinus weakening. Pulsatile audible tinnitus has also been proposed to reflect the transmission of systolic pulsations of the CSF under high pressure to the walls of the venous sinuses in IIH.[18] This may also be related to dural sinus compliance changes. Patients who do not demonstrate high pulsatility may represent those patients in an early stage of intracranial hypertension, those in whom dural sinus integrity is not yet threatened. However, increased pulsatility may reflect dural weakening in patients with significant compromise of dural sinus integrity, transmission of ICP, and therefore in need of structural support to restore venous outflow. Wide variation is present within the dural sinuses and apparent angiographic narrowing may be due to sinus trabeculation, whereas narrowing with high pulsatility in the presence of an appropriate clinical syndrome may improve selection of patients who would benefit from intervention.

Furthermore, understanding dural sinus anatomy and wall mechanics has implications for the appropriate selection of stent type for wall remodeling. This may prove important for treatment outcomes. Vessel compliance varies after implantation with different stents.[19] Therefore, stent selection seems to be important to achieve support that may allow for improved venous outflow and greater durability. Characterization of pulsatility before and after stent placement may be useful in stent planning, evaluating the immediate effect on wall strength, and surveillance.

The paucity of reports investigating the hydrodynamics of IIH in the setting of emerging potential endovascular therapies and increasing reports of stent implantation highlights the need for scientific validation. Manometry is a simple method of obtaining information that may prove to be an invaluable addition to criteria for the diagnosis and selection of patients with IIH who would benefit from endovascular therapy. Further study is needed to better characterize the association between venous sinus pulsatility and dural competence, the role of high pulsatility in the pathophysiology of IIH and treatment outcomes and to establish reference data for dural sinus pulsatility.

CONCLUSION

Dural sinus manometry showing a high-pressure gradient with a highly pulsatile waveform may represent weakening of the sinus wall leading to dural incompetence and ICP transmission. This finding may have an important role in understanding the pathophysiology of IIH and endovascular management. Further studies are needed to standardize manometric measurement thresholds and better define the role of venous sinus pulsatility as it relates to dural sinus pathology in IIH, disease severity, endovascular treatment planning, and outcomes.

Disclosures

Dr Zaidat serves as a consultant for Stryker Corporation. The other authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENT

In this article, the authors describe the pulse pressure changes that occur within the venous sinuses of the brain in patients with fulminant intracranial hypertension before and after sinus stenting. The findings of high pulse pressure before stent and reduced pulse pressure after stent have not previously been described. This adds further data toward the complex physiology of this disease process. One may argue about how important the pulsatile components of the disease are to the underlying physiology compared with the static venous pressure elevation; however, even taking

all of the pressure data together may not yield a complete picture of this process.

Careful review of the data presented shows that the average sigmoid sinus pressure before the intervention was 11 mm Hg, and this increased to 20 mm Hg after the stents (despite an overall drop in sagittal sinus pressure from the procedure). Given that the procedure did not affect the outflow resistance of the neck vessels or the right atrial pressure, the only other possibility is that the flow through the transverse sinus into the sigmoid sinuses increased by about 80%. This clearly vindicates the opinion of the authors that stent placement significantly improves the flow through the sinuses. Presumably, this increased flow comes about from the rerouting of collateral flow back to the main outflow channels.

However, even taking into account the stenoses and the collateral flow changes, we may not have a complete picture. I have no doubt that the procedure rendered these patients asymptomatic and preserved vision, but did it completely cure the disease? The average sagittal sinus pressure after the stent was 24 mm Hg which equates to 33 cm H_2O. Given that the CSF pressure cannot be less than the vein into which it drains, and that the cutoff for the diagnosis of IIH is 25 cm H_2O, some disease process clearly remains. Given that the average sigmoid sinus pressure was only 11 mm Hg before the procedure, and the right atrial pressure was presumably less than this for blood to flow toward the heart, we cannot blame right heart failure for all of the residual pressure elevation. I have described elevated total cerebral blood inflow in a percentage of these patients and believe this could be the missing component. I would encourage the authors to consider adding blood flow measurement to their MRI protocol in future cases.

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