Cerebral venous overdrainage: an under-recognized complication of cerebrospinal fluid diversion

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Understanding the altered physiology following cerebrospinal fluid (CSF) diversion in the setting of adult hydrocephalus is important for optimizing patient care and avoiding complications. There is mounting evidence that the cerebral venous system plays a major role in intracranial pressure (ICP) dynamics especially when one takes into account the effects of postural changes, atmospheric pressure, and gravity on the craniospinal axis as a whole. An evolved mechanism acting at the cortical bridging veins, known as the “Starling resistor,” prevents overdrainage of cranial venous blood with upright positioning. This protective mechanism can become nonfunctional after CSF diversion, which can result in posture-related cerebral venous overdrainage through the cranial venous outflow tracts, leading to pathological states. This review article summarizes the relevant anatomical and physiological bases of the relationship between the craniospinal venous and CSF compartments and surveys complications that may be explained by the cerebral venous overdrainage phenomenon. It is hoped that this article adds a new dimension to our therapeutic methods, stimulates further research into this field, and ultimately improves our care of these patients.

http://thejns.org/doi/abs/10.3171/2016.6.FOCUS16172

KEY WORDS cerebrospinal fluid diversion; hydrocephalus; posture; shunt; Starling resistor; cerebral venous overdrainage
changes in ICP and cerebral venous pressure with alterations in body posture. In healthy subjects, with upright posture there is a drop in ICP ranging between -5 and 5 cm H$_2$O with reference to the foramen magnum. The drop in ICP is related to the CSF loss from the cranial to the spinal compartment, usually about 3 ml, which is due to the differential in compliance of the 2 compartments. In contrast, in patients harboring shunts without antisiphon devices, pressures range from -15 to -35 cm H$_2$O when they are upright. One method that allows us to quantify the cerebral venous pressure component of ICP during CSF diversion is Davson's steady-state formula, 

\[ ICP = Pcv + (I_f \times R_o) \]

where $Pcv$ is cerebral venous pressure, $I_f$ is CSF formation rate (usually 0.3 ml/min), and $R_o$ is CSF outflow resistance. One can see that if CSF is withdrawn at the same rate as its production and because $R_o$ approaches 0, ICP will reflect cerebral venous pressure.

**Pathophysiology of Cerebral Venous Overdrainage**

The anatomy of the cranial venous outflow tracts is crucial in the pathophysiology of cerebral venous overdrainage. In the supine position the internal jugular veins are the main venous outflow pathways. During verticalization, because of atmospheric pressure, the internal jugular veins collapse and venous blood is rerouted to nonjugular pathways, specifically to the noncollapsible vertebral venous plexus. In a systematic ultrasonography and MRI analysis of the types and prevalence of human cerebral venous outflow, it was shown that there is predominantly jugular drainage in 72% of healthy volunteers. In 22% the jugular drainage equals the nonjugular drainage, and in 6% the drainage pattern is nonjugular. These findings suggest that in the general population there are variations in the anatomy of cranial venous outflow.

Human cerebral venous outflow depends on central venous pressure and posture. A marked increase in central venous pressure, such as any Valsalva maneuver, completely reopens the internal jugular veins. In an ultrasonography study of the postural dependency of venous outflow, it was shown that blood flow in the internal jugular veins decreases from 700 ml/min in the supine position to 70 ml/min at 90° elevation while flow in the vertebral veins is increased from 40 to 210 ml/min. Total venous outflow declines from 740 to 280 ml/min from 0° to 90°, with the largest decrease at 15°. One important finding is that flow in the vertebral veins exceeds internal jugular vein flow at 45° of head elevation. The rigid spine seems to prevent epidural vein collapse, and negative epidural pressure may promote venous flow from the brain to the vertebral column.

There is significant anatomical and functional conti-
nuity between venous structures of the brain and spine. Analogous to the CSF craniospinal axis, the venous outflow network of the cranium and spine has been referred to as the “cerebrospinal venous system,” which is further enhanced by the lack of venous valves. During postural changes this anastomosis serves an important function in pressure homeostasis of the cerebral venous system. The suboccipital cavernous sinus, situated in the deep muscular layers of the neck, serves as an important “relay station” anastamosing with the 2 main divisions of the craniospinal venous system (cranial and spinal) in the suboccipital region. It is connected to the jugular bulb and internal jugular veins via the condylar veins and continues inferiorly into the deep cervical veins. The craniospinal venous system is essentially a large-capacity, valvless venous network in which flow is bidirectional and freely communicates with the sacral and pelvic veins. It has been shown that during Valsalva maneuvers, blood is squeezed out of the intraabdominal veins into the vertebral system and thus increasing intracranial pressure.

The site where the craniospinal axis is atmospheric, referred to as the “zero point,” is generally at the level of the foramen magnum. Magnaes has shown that in patients with shunts, during upright positioning the zero point is shifted caudally to the upper thoracic level; thus intracranial pressure can drop significantly relative to sagittal sinus pressures.

In hydraulic terms, if the ambient CSF pressure in the subarachnoid space surrounding the bridging veins is low enough, it cannot constrict venous drainage into the sagittal sinus. One can see that when CSF diversion is accompanied by upright positioning, CSF pressure can drop below sagittal sinus pressure. It follows that low CSF pressure can render the Starling resistor nonfunctional and thus allowing overdrainage of cerebral venous blood through nonjugal pathways. This is consistent with the mathematical model proposed by Piechnik et al. in which CSF and sagittal sinus pressures become dissociated. This could lead to exacerbation of intracranial hypotension and, depending on varying circumstances (such as head elevation, position, individual anatomy, and compensatory mechanisms), congestion in veins such as the suboccipital cavernous sinus that connects the jugular and nonjugal pathways and accommodates the diverted venous volume. A horizontal position would increase ICP with respect to the dural sinus pressure, in other words, reestablishing the normal hierarchical relationships in the pressures and thus terminating the cascade of intracranial hypotension.

The phenomenon of venous overdrainage from the cranium was first described in 2000 by Barami et al. They measured ICP and compliance in patients with chronic shunts that had been externalized. The data allowed for calculating whether displacements in CSF volume accounted for posture-related changes in ICP. A key finding was that in these patients with chronic shunts, the almost instantaneous drop in ICP in the upright position occurred even when the shunt was externalized and not allowed to drain. The data showed that for the pressure volume indices measured, the amount of fluid shift to explain pressure changes exceeded the amount of intracranial CSF. Therefore, it was hypothesized that the other liquid component of the cranial cavity, that is, venous blood, was responsible for the drop in ICP referred to as “venous overdrainage.”

Disease Entities Implicating Cerebral Venous Overdrainage

A survey of the relevant literature revealed several categories of complications that might be explained by cerebral venous overdrainage: 1) venous congestion or engorgement in the spine causing mass effect, 2) suboccipital venous congestion causing remote site intracranial hemorrhage, 3) low pressure hydrocephalus, and 4) ventriculomegaly associated with craniectomy (Fig. 2).

Venous Congestion–Associated Cervical Myelopathy After CSF Diversion: Miyazaki Syndrome

Cervical myelopathy associated with intracranial hypotension after ventriculoperitoneal shunting was first described by Miyazaki in 1998. Subsequently in 2002 Matsumoto presented a case of cervical radiculomyelopathy in a 69-year-old man with a ventriculoperitoneal shunt that had been placed 2 years earlier and in whom imaging showed an enhanced “extramedullary mass” causing cord compression. The patient’s symptoms resolved after removal of the shunt. Imaging findings and symptoms were thought to represent engorged epidural veins caused by intermittent intracranial hypotension due to postural changes of CSF pressure. In 2009 Martínez-Lage et al. presented the case of a 20-year-old woman with a ventriculoperitoneal shunt that had been placed 14 years earlier and who had an incidental finding of an engorged cervical epidural venous plexus. In a review of the literature on similar cases spanning patient ages from the late teens to the geriatric years and all harboring shunts, these authors documented an interval from 22 months to 27 years between CSF shunt placement and the appearance of symptoms. Ulrich et al. have performed a more current literature review of chronic overshunting-associated venous congestion causing myelopathy and also described a patient with congenital cervical canal stenosis who had presented with cervical myelopathy and dilated venous plexus at the cranio cervical junction. Symptoms and imaging findings in that case resolved after shunt revision and the addition of an antisiphon device leading to a gradual increase in ICP. The preferential shunting of blood through the vertebral plexus has been angiographically demonstrated in another case described by Wolfe et al., who found the collapse of both internal jugular veins and engorged muscular and pericondylar veins shunting blood into the epidural venous plexus after ventriculoperitoneal shunting.

These cases demonstrate that in patients with CSF diversion, there could be increased venous pooling in the spinal compartment whereby a subset of the patients become symptomatic. The dominant hypothesis among the cases reported so far has been that, according to the Monro-Kellie doctrine, the loss of CSF there should be an increase in blood flow as a compensatory mechanism where selective uptake of contrast material during MRI occurs in the pachymeninges, as compared with that in the leptomeninges, due to the lack of a blood-brain barrier. Biopsy of the meninges in patients with intracra-
nial hypotension suggests that a thin subdural zone of fibroblasts, fibrocollagenous proliferation, and small, thinwalled dilated blood vessels form the anatomical basis of pachymeningeal enhancement suggestive of a reactive secondary phenomenon. Interestingly, Caruso et al. have also described the same engagement of cervical epidural veins causing symptoms after craniectomy that resolved after cranioplasty. Their case report highlights the fact that perhaps it is the increased compliance and loss of CSF outflow resistance associated with craniectomy that leads to the loss of the Starling resistor effect and thus the cerebral venous overdrainage via the vertebral venous plexus causing cervical epidural vein engorgement. Cranioplasty, in turn, restores compliance and CSF outflow resistance and resolves symptoms.

Several items argue against the explanation that there is venous hyperemia to compensate for CSF loss according to the Monro-Kellie doctrine. First, there is angiographic evidence during intracranial hypotension of a collapse of internal jugular veins and engorgement of venous structures such as muscular branches and pericondylar veins. This suggests selective shunting of venous blood into the vertebral venous plexus. Second, there is evidence that after CSF diversion from lumbar puncture, flow in the venous sinuses actually drops and is not increased. Third, in other situations in which there is significant loss of CSF or another component of the cranial cavity, such as brain tissue loss after hemispherectomy, there has been no demonstrated compensatory increase in cerebral venous volume. Fourth and last, as stated earlier, the same pattern of spinal epidural engorgement occurs in cases without CSF loss but in which brain compliance increases and CSF outflow resistance decreases, conditions that would promote cerebral venous overdrainage.

The phenomenon of cervical venous engorgement after CSF diversion seems to be more congruent with cerebral venous overdrainage when it is due to CSF loss, the Starling resistor at the bridging veins becomes nonfunctional, and there is cerebral venous overdrainage with preferential flow and engorgement in the vertebral venous plexus due to postural changes.

Remote Site Intracranial Hemorrhage

Remote site intracranial hemorrhage is a rare complication following intracranial surgery and spinal procedures with a reported overall incidence of 0.08% to 0.6% after supratentorial craniotomies. Its etiology remains controversial; therefore recommendations on avoiding it are difficult to outline. Reports of infratentorial bleeds following supratentorial surgery and vice versa have been recently reviewed by Garg et al. The most common hemorrhage pattern is cerebellar hemorrhage following supratentorial surgery. Specifically blood is seen in the sulci of one or both cerebellar hemispheres and vermis facing the tentorium in an alternating pattern of hyperdense (blood) and hypodense (cerebellum) stripes referred to as the “zebra sign.” This complication was first reported by Yaşargil and Yonekawa. Remote site intracranial bleeds can also occur less frequently after endonasal surgery, but hole evacuation of subdural hematomas, and spinal surgery at all levels that are associated with CSF loss.

With regard to adult hydrocephalus, remote intracranial bleeds (most commonly cerebellar hemorrhages) have also been seen following lumbarperitoneal shunting, simple lumbar puncture, reduction in the valve pressure of a ventriculoperitoneal shunt, multiple manual pumping of a ventriculoperitoneal shunt valve, and placement of an external ventricular drain. All seem to have CSF loss as a common denominator. Although the etiology remains controversial, several aspects of this complication seem to be agreed on: 1) involvement of CSF drainage, 2) hemorrhage of venous origin rather than arterial, 3) location of a bleed typically subarachnoid with a variable intraparenchymal element, and 4) predilection for superior vermian vein territory, that is, superior cerebellar surface and vermis. In general the incidence of severe neurological deficits is 9.7% and associated morbidity is 14.5%. Moreover 54% of the patients develop symptoms 10 hours postoperatively, and the most common presenting symptom is a decreased level of consciousness. Borkar et al. have recently performed a literature review on this complication. One interesting finding is that supratentorial bleeds after infratentorial surgeries are uncommon as compared with the converse, and nearly all reported cases of the former had been performed in the sitting position, suggesting that postural factors seem to be involved.

The most common pathophysiological explanation for
this complication has been cerebellar “sag” due to CSF hypovolemia causing transient occlusion of posterior fossa bridging veins and secondary venous infarction. With regard to the cerebellar sag hypothesis, other authors have discussed the possibility of caudal migration of the cerebellum, which in turn causes stretching, kinking, or shearing of the superior vermian veins. Another mechanism proposed by Seoane and Rhoton is excessive rotation and extension of the head during perionial craniotomies, causing internal jugular venous compression by the transverse process of the atlas. This, in turn, occludes venous outflow with a resultant increase in intraluminal pressure in the veins, yet it seems an unlikely mechanism in cases of spinal procedures in which generally the head is kept neutral.

The above complications could be explained from the vantage point of cerebral venous overdrainage. The Starling resistor becomes nonfunctional during significant CSF loss. Should the loss of CSF occur in the supine position, cerebral venous pressures are low and there is slow flow in the outflow veins, which has been demonstrated by Canhão et al., as mentioned previously. On the other hand, if the loss of CSF is accompanied by head elevation without compensatory mechanisms to increase central venous pressure, there will be ensuing cerebral venous overdrainage from the noncollapsible major sinuses into the outflow veins with preferential congestion in the suboccipital cavernous sinus, the “relay station” between the cranial and spinal components of the craniospinal venous system as the venous outflow is redirected from the internal jugular veins to the vertebral venous plexus.

In summary, the cerebral venous overdrainage hypothesis may explain the phenomenon of remote cerebellar bleeds after rapid and/or excessive CSF loss associated with CSF diversion. During the loss of CSF, there is dissociation of CSF and cerebral venous pressures and the Starling resistor becomes nonfunctional. Compensatory mechanisms may be lacking, and depending on the degree of head elevation, individual variations in anatomy, including cranial venous outflow, there could be venous pooling in the posterior fossa veins causing hemorrhages. This may also explain why remote site hemorrhages are rare in the pediatric age group since their drops in CSF pressures are not as significant as those in adults during CSF loss given their smaller stature.

**Low Pressure Hydrocephalus**

Low pressure hydrocephalus is loosely defined as a paradoxical increase in the size of ventricles despite low to normal CSF pressures in patients with CSF diversion and is associated with neurological deterioration. Symptoms include postural headaches, lethargy, obtundation, and cranial neuropathies. Most cases involve patients with shunted hydrocephalus, whereas others involve patients with external ventricular drains as well as those with CSF rhinorrhea. The first case series on low pressure hydrocephalus was performed by Pang and Altschuler in 1994. They reported on 12 patients who had previously tolerated shunts but later presented with ventriculomegaly and low CSF pressures. All patients except one were treated with external ventricular drainage at subatmospheric pressures, which were slowly increased, and new shunts were finally reinserted. No correlation was found between ICP and ventricular size, and improvement in symptoms correlated with a decrease in the size of ventricles. The average calculated pressure volume index in 3 patients was 43.9 ± 4.6 (190% of predicted normal value). The authors proposed that the development of low pressure hydrocephalus is related to changes in the viscoelastic modulus of the brain due to the expulsion of extracellular water from the brain and due to structural changes caused by chronic overstretching. They asserted that susceptible patients are those who, due to bioatrophic changes, have innate low brain elasticity and that the goal of treatment is to allow the entry of water into the brain parenchyma to restore brain viscoelasticity. Subsequently other authors have argued against the viscoelastic hypothesis and the subatmospheric method of treatment. Vassilyadi et al. described 2 cases of low pressure hydrocephalus that occurred hours to days after spinal cysteopleural shunting in patients who had previously had shunts. These authors argued that the timing of the development of low pressure hydrocephalus did not correspond to that required for viscoelastic changes and that the transmantle pressure gradient causing excessively low convexity subarachnoid pressures also caused the ventriculomegaly. One patient improved with positive end-expiratory pressures and clamping of the cysteopleural shunt; the second patient was treated by changing to a flow-regulated valve. Clarke et al. described 2 patients with low pressure hydrocephalus who were treated with the subatmospheric method and whose symptoms improved during negative pressure drainage; however, despite the placement of ventriculoatrial shunts and the normalization of ventricular size, outcomes were poor. Another report on low pressure hydrocephalus in patients with shunts illustrates that subatmospheric drainage may be unnecessary and argues against any surgical intervention in the treatment of such patients. In this article, Dias et al. described 2 cases of low pressure hydrocephalus in patients with shunts who had presented with postural symptoms precipitated by lumbar puncture; interestingly both cases resolved, and ventricle size became normal with only enforced recumbency without the need for shunt revision. These authors advised against shunt revision in cases of low pressure hydrocephalus. It is possible that simple bed rest allows for the reestablishment of the normal CSF–venous system interaction.

Other authors have supported the subatmospheric method for the treatment of low pressure hydrocephalus. Daniel et al. documented a case of low pressure hydrocephalus that developed after CSF rhinorrhea following hemispherectomy: the patient was successfully treated with subatmospheric drainage. Owler et al. demonstrated that 4 of their 5 low pressure hydrocephalus cases improved with subatmospheric drainage and conversion to low-pressure or valveless shunts, although they argued against Pang and Altschuler’s hypothesis on the loss of water from the parenchyma since ventriculomegaly during low pressure hydrocephalus was associated with con-
continued transependymal passage of CSF with an increase in brain extracellular fluid.

There is ongoing debate regarding the genesis of low pressure hydrocephalus. Akins et al. suggested, based on a poroelastic model, that the brain acts as a “sponge” and that due to increased brain permeability, extracellular fluid increases, leading to the flux of fluid in and out of the brain as well as an increased ventricular volume. Lastly, Lesniak et al. attempted to explain the paradoxical ventriculomegaly seen in patients with low pressure hydrocephalus by suggesting that in the acute phase of hydrocephalus, acute distension of the ventricular system increases compliance of the cranial vault, thus shifting the pressure-volume curve to the right.

The above reports show that there are clearly a group of patients with CSF diversion who develop low pressure hydrocephalus, respond well to CSF drainage at subatmospheric pressures, and are gradually transitioned to positive pressures. As regards the etiology and treatment of this condition, the cerebral venous overdrainage hypothesis offers several insights: CSF diversion causes CSF hypotension leading to the loss of the Starling effect on draining cortical veins. Interestingly in an MRI study of intracranial CSF volume after lumbar puncture, it was shown that most of the CSF loss occurred from the cortical sulci, suggesting a loss of pressure in the cortical subarachnoid space. Upright positioning (aggravated by the loss of compensatory mechanisms) leads to cerebral venous overdrainage and thus a parallel decrease in cerebral venous pressures. This derangement of the normal hierarchy of cerebral pressures initiates a vicious cycle of intracranial hypotension resolved by reestablishing the normal hierarchy of pressures. Ventriculomegaly could be explained by cerebral venous hypotension and low pressures in the superior sagittal sinus and similarly in the deep cerebral veins and periventricular veins (which are contiguous with the sagittal sinus and also lack Starling resistors), leading to an increased pressure gradient and CSF flow from ventricle to pachymena and ventriculomegaly. This also causes transependymal CSF flow leading to periventricular edema, as suggested by Portnoy et al. Bed rest (during external ventricular drainage) allows compensatory mechanisms to act (increase in central venous pressure), reducing this pressure gradient. Moreover, ventricular drainage contributes to normalization of the CSF to venous pressure gradient and reestablishment of the normal CSF—venous system relationship. The significance of posture to ventricular size was demonstrated by McCullough et al. In their report 5 patients with shunted hydrocephalus and harboring antisiphon devices developed postural symptoms and enlargement of the ventricles when they were upright; the ventriculomegaly and symptoms resolved after horizontal positioning. These authors also noted that all of the patients were relatively tall. The successful treatment of low pressure hydrocephalus cases with positive end-expiratory pressure and recumbency are also consistent with the fact that posture-related hydrostatic effects play a role in the pathophysiology of this disease entity.

More recently Hamilton and Price have reported their experience with inappropriately low pressure acute hydrocephalus and have provided insight into its treatment. In their series all patients had anatomical obstruction of CSF flow into the subarachnoid space. They concluded that, in addition to forced drainage of the ventricles, treatment should be directed at restoring communication between the cortical subarachnoid space and the ventricular system by endoscopic third ventriculostomy and by increasing subdural sinus pressures via a neck tourniquet and/or abdominal binder. From the vantage point of cerebral venous overdrainage, the application of a neck tourniquet and abdominal binder restricts cranial venous outflow and promotes blood flow into the vertebral venous system, respectively. Endoscopic third ventriculostomy diverts CSF out of the ventricles into the subarachnoid space, effectively reducing the ventricle to periventricular tissue gradient and increasing subarachnoid CSF pressures. Taken as a whole, these modalities restore the hierarchy of pressures as well as conditions for a functioning Starling resistor and thus reduce ventricular size and normalize intracranial pressure dynamics.

Analysis of the above articles reveals several other interesting observations. Pressure volume index is a measure of neural axis compliance with a normal value of 25.9 ± 3.7 in adults and a range from 8.2 to 30.1 in the pediatric population. Increased brain compliance indices calculated by Pang and Altschuler are almost identical to those in the Barami et al. report (43.9 ± 4.6 vs 42.8 ± 4.5), yet ventricle sizes in the former study were mostly small and were large in the former study. This suggests that increased brain compliance can occur in patients with shunts without ventriculomegaly, and as suggested by Magnaes, it is the loss of CSF that causes an increase in compliance and not necessarily enlargement of the ventricles. The term “subatmospheric” may be a misnomer if one considers Magnaes’ findings in which in patients with shunts there was caudal displacement of the zero (atmospheric) point, thus the subatmospheric method of lowering the external ventricular drain below the external auditory meatus might have been a matching of the drip chamber to the lowered zero point. Interestingly several patients in previous series developed low pressure hydrocephalus hours or days after CSF diversion, which makes it unlikely that chronic inflammatory and atrophic changes (as suggested by Pang and Altschuler) have taken place. Lastly, some of the symptoms described in Pang and Altschuler’s series, specifically gait ataxia and hypertonicity, could be explained by cervical myelopathy associated with cerebral venous overdrainage (Miyazaki syndrome), as mentioned earlier.

Ventriculomegaly Associated With Cranietomy

Another condition in which the normal hierarchy of pressures is deranged follows the removal of a large portion of the skull known as “cranietomy,” which is performed as a life-saving maneuver during malignant intracranial hypertension. In a study in cats Shapiro et al. showed that when a portion of the skull is removed, the compliance (pressure volume index) of the brain increases and the CSF outflow resistance decreases; therefore, according to the Davson formula above, since CSF outflow resistance approaches 0, again ICP becomes dependent on cerebral venous pressure. Moreover, dural open-
Brain compliance and cerebrospinal fluid (CSF) outflow resistance are key factors in the development of chronic post-traumatic hydrocephalus after head injury. Increased brain compliance, as seen after craniectomy, may contribute to an imbalance in cerebral fluid dynamics leading to CSF overdrainage and subsequent hydrocephalus. This phenomenon, termed cerebral venous overdrainage, has been observed in patients after craniectomy, where the sagittal sinus pressure gradient is lost, leading to increased CSF outflow and hydrocephalus.

**The Role of Cerebral Venous Overdrainage**

Cerebral venous overdrainage is a phenomenon where the cerebral venous system is hyperfunctioning, leading to increased cerebral venous outflow and decreased CSF outflow resistance. This imbalance results in a net loss of CSF, causing hydrocephalus. The sagittal sinus, a major venous sinus, plays a crucial role in this process. When the sagittal sinus pressure gradient is lost after craniectomy, there is an increased risk of hydrocephalus due to the decreased resistance to CSF outflow.

**Factors Contributing to Cerebral Venous Overdrainage**

Several factors contribute to cerebral venous overdrainage, including cranial bone defects, increased intracranial volume, and changes in cerebral compliance. Patients with craniectomies too close to the midline are at higher risk due to the loss of CSF outflow through the sagittal sinus.

**Therapeutic Implications and Future Directions**

Therapeutic strategies to manage cerebral venous overdrainage include the use of antisiphon devices to prevent CSF loss and management of lower pressure hydrocephalus. Antisiphon devices may also help prevent cerebral venous overdrainage and may be useful in the ongoing management of hydrocephalus.

**Conclusion**

Cerebral venous overdrainage is a significant factor in the development of hydrocephalus after craniectomy. Understanding the mechanisms underlying this phenomenon is crucial for the development of effective therapeutic strategies. Further research is needed to identify modulating factors and to refine management protocols.
Acknowledgments

I thank Bahar Hanjani for the illustration in Fig. 2.

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**Disclosures**

The author reports no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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