

Chapter 15

Hydrocephalus: New Theories and New Shunts?

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Introduction

The optimal and ideal management for any given clinical disorder should be fundamentally aimed at reversing or preventing the pathobiological mechanism underlying the disorder. For hydrocephalus, our current incomplete understanding of the pathophysiology is, in part, responsible for significant inadequacies of the current mainstay of treatment: the cerebrospinal fluid (CSF) shunt. The management of shunt-related problems and disorders has become a de facto subspecialty within neurosurgery. Although it is clear that the treatment of hydrocephalus was vastly improved with the introduction of the differential pressure valve by Nulsen and Spitz (25) half a century ago, it can be argued that little further improvement has occurred in the interim. A randomized, multicenter trial failed to show a benefit from “technologically advanced” valve designs compared with a standard differential pressure valve (7) similar to the one designed by John Holter.

Probably one of the most refractory problems of CSF shunt diversion has been that of over-drainage. In children, excessive CSF drainage by the shunt is an important cause of shunt failure caused by ventricular catheter obstruction resulting from ventricular collapse. Over time, shunted hydrocephalic children can develop the slit-ventricle syndrome—one of the most challenging conditions to treat in neurosurgery. In older adults, shunt over-drainage can result in the devastating complication of subdural hematoma. A similar degree of ventricular reduction occurs despite the implementation of valve designs touted to prevent it, including flow-limiting valves and antisiphon devices (31).

Why have efforts failed to prevent excessive CSF drainage by shunts? Although the answer is likely multifactorial, we argue that the fundamental problem lies in our incomplete and overly simplistic understanding of the hydrodynamics of hydrocephalus and in vivo shunt physiology. The next generation of CSF diversion shunts or other treatments for hydrocephalus, will arise from a better understanding of the complex interrelation between pulsatile cerebral perfusion and CSF movement dynamics. Biomedical engineers and neurosurgeons must transition beyond the overly simplistic, first-order CSF bulk circulation model that has dominated for the past 50 years.

The classic (first-order) model of CSF physiology is based on the assumptions that 1) CSF is produced primarily by the choroid plexus at a constant rate, 2) CSF circulates by bulk flow through the ventricles and around the convexities towards the vertex, where 3) CSF is absorbed by the arachnoid granulations (plus, possibly, lymphatic drainage [33]). By this classic theory, ventriculomegaly is caused by a backup of CSF flow, caused by an obstruction either within the ventricles (obstructive hydrocephalus) or at the arachnoid granulations (communicating hydrocephalus).

There is no doubt that bulk CSF flow occurs within and exiting the ventricular system. The fate and purpose of CSF, however, is still an open question. Greitz (18) and Greitz et al. (19) have demonstrated by high-resolution cine magnetic resonance imaging (MRI) that there is no net bulk flow of CSF over the cerebral convexities. Instead, it has been suggested that CSF water freely crosses all capillaries and pre-venules (5, 28). Therefore, the concept of CSF absorption should possibly be constrained to the uptake of CSF solutes rather than CSF water. Furthermore, it has been suggested that the prima facie evidence of classical CSF bulk circulation, the radionuclide cisternogram, is,

instead, a misinterpretation of the radioisotope movement. Rather than depicting the movement of CSF, the concentration of isotope at the midline convexity (and terminal sacral subarachnoid space) reflects a maximal dilution effect occurring at the foramen magnum (19).

If communicating hydrocephalus were truly caused by an increase in the CSF outflow resistance at the arachnoid granulations, then one would expect an increase in the pressure gradient between the pressure in the subarachnoid space and the sagittal sinus venous pressure. An increase in this pressure gradient has yet to be convincingly demonstrated (10, 29). Even the etiology of obstructive hydrocephalus, in classic first-order thinking, must be questioned. Tisell et al. (30) demonstrated that, in adult patients with documented aqueductal stenosis (complete obstruction), there was no difference in pressure and CSF outflow resistance between the ventricular system and the subarachnoid space.

The main problem with the first-order CSF circulation model is that it is overly simplistic, and, therefore, potentially misleading. In this chapter, a second-order CSF physiology approach is proposed that superimposes an important hydrodynamic component on the first-order model. Rather than viewing the CSF physiology as a simple slow-flow circulation, it is instead considered a highly dynamic and complex system in which the movement of CSF is intimately related to cerebral blood flow (CBF) hemodynamics. The second-order model also requires an alternative view of intracranial pressure (ICP) physiology. In the classic model of hydrocephalus, ventriculomegaly is considered to act as a space-occupying lesion, akin to neoplasms and hematomas, that increases ICP because of the added volume in the intracranial cavity. In the second-order model, the physical origin of ICP is considered the vascular system, and, therefore, it is the understanding of cerebral hemodynamics that is the most crucial element.

Second-Order CSF Physiology Hydrodynamics

The hemodynamic theory of ICP physiology (1) proposes a theoretical model of the intracranial compartment that establishes a relationship between the cerebrovascular system and ICP, and places greater emphasis on the impedance characteristics of the cerebrovascular system. There are three premises, which together establish the foundation by which the model is built:

1. Cerebrovascular impedance, and not exclusively resistance, determines CBF.
2. Dynamic CSF compliance, modulated primarily by CSF movement in ventricles and basal cisterns, is the primary determinant of cerebrovascular impedance.
3. Alteration in cerebrovascular impedance, either regionally or globally, may result in disorders related to ICP.

Cerebrovascular Impedance

Cerebrovascular impedance is the algebraic sum of cerebrovascular resistance plus cerebrovascular reactance. The concept of cerebrovascular resistance is easier to understand because it is analogous to the property of friction. Pouisseeille's Law relates vascular resistance as being linearly proportional to the length of the vessel and inversely proportional to the fourth power of the radius. Pure resistance, however, occurs only in the realm of nonpulsatile flow. When an acceleration (and/or deceleration) of fluid exists, then reactive forces are introduced.

One type of reactance results from inertia. It is easier to throw a beach ball than an equivalent size bowling ball because the bowling ball weighs more—not because there is more air resistance. The inertial reactance is much higher for accelerating the bowling ball, whereas the resistance would be roughly equivalent for each ball. Importantly, if surface friction is equivalent for the two balls, it takes an equivalent amount of energy to keep the balls rolling at a constant velocity. Inertial reactance “impedes” rapid changes in blood flow.

Capacitive reactance, like its inertial counterpart, is frequency dependent. The mechanical analog to capacitance is compliance—a term that means different things to different people. For the intracranial compartment, the static compliance curve, traditionally defined as the change in pressure that occurs if a certain volume is introduced, is pertinent for only very low-frequency inputs (experimentally, the addition is very slow compared with the rapidity of blood flow volume changes occurring during one heartbeat). The compliance characteristics of blood vessel walls also potentially confuse the terminology. At low pressures, venous compliance is 10 to 20 times greater than arterial compliance. However, changes in venous volume occur slowly over time. Regarding the second-order intracranial physiology concepts addressed here, the dynamic compliance is the most important consideration. The ability and degree of dynamic arterial expansion in response to the bolus of systolic blood flow will influence the frequency characteristics of blood flow and pressure downstream.

Is blood flow impedance important? Under normal circumstances, blood flow inertial reactance is probably minimal. This occurs as a result of a highly efficient vascular compliance system that converts the purely pulsatile blood flow emanating from the left ventricle of the heart to a nearly steady flow in the systemic venous system. The redistribution of systolic to diastolic flow occurs in the arterial system because of the Windkessel phenomenon (15). Expansion of the arteries during systole temporarily stores some blood volume (compliance), which is then propelled anterogradely during diastole when the arterial walls reflexively constrict. The elegance in the cardiovascular system lies in the fact that blood volume is unequally distributed within the systemic vasculature: approximately 76% of the total blood pool resides within the venous portion (20, 21). In other words, the large inertial component of the blood volume (the “bowling ball”) moves along with minimal acceleration and deceleration forces required.

To gain an appreciation of the theoretical importance of the Windkessel system, imagine a hypothetical situation in which the entire vasculature tree was completely noncompliant. The left ventricle of the heart would have to accelerate the entire 5-L blood volume from a standstill during a period of approximately 300 ms with every heartbeat—a likely impossible feat. From a hemodynamic viewpoint, the vascular impedance (caused by reactance, not resistance) would be prohibitively high—resulting in an inadequate systemic blood flow.

In the peripheral circulation, the Windkessel effect occurs because of the pliable nature of soft tissues and the skin. For example, during systole, the arm is able to circumferentially expand, thereby accommodating the expanding arteries. Because of the presence of the rigid cranial enclosure, however, the brain cannot expand outwardly in a similar fashion. It is argued here that the CSF dynamic translocation system serves as the critical mechanism that allows the Windkessel effect to take place.

The functioning of an intracranial Windkessel effect mandates that the cerebral arteries be allowed to expand within the intracranial compartment. According to the Monro-Kellie doctrine, the cranium is regarded as a rigid container filled with three components: cellular matter (brain and supporting cells), extracellular water (which includes CSF),

and intravascular blood. The pressure–volume characteristics of this container are defined by assumptions regarding two fundamental physical properties of this system. First, the intracranial volume is a constant (in an adult, rigid cranium); and second, the components of this container are incompressible. At constant pressure, any volume added to this container must be accompanied by the displacement of an equal volume out of the container. The classic intracranial pressure–volume curves, initially described by Langfitt et al. (22, 23), are often misinterpreted as implying that volume can be added to the intracranial compartment. Instead, the Monro-Kellie constraints dictate that intracranial volume must be constant. The Langfitt curve better depicts the force required to displace either CSF or blood out of the intracranial compartment because the net flux of fluid movement in and out of the container must be zero.

From a physiological standpoint, the cerebral circulation constitutes the greatest challenge to the intracranial cavity in coping with attempted rapid addition of blood volume. In the average adult, each cardiac cycle is accompanied by the addition of approximately 14 ml of arterial blood into the intracranial compartment. Blood flow entering the intracranial cavity must be accompanied by an equal volume of fluid (blood and CSF) exiting at all times. As demonstrated in Figure 19.1, a phase shift occurs between the arterial and venous blood flow measured at the cranial base. It is clear that two intracranial phases exist during the cardiac cycle. During systole, arterial CBF exceeds venous CBF—resulting in a transient increase in cerebral blood volume. This relationship reverses during diastole, when venous CBF exceeds arterial CBF.

The net change in CBV during the cardiac cycle is accommodated by the intracranial cavity by the dynamic translocation of CSF (18). During systole, a volume of CSF equivalent to the differential blood volume exits the cranial vault via the foramen magnum. During diastole, CSF returns from the spinal subarachnoid space to the intracranial space. The venting of CSF during the cardiac cycle has been elegantly documented using MRI by Greitz (18) and others (3, 4, 11–13, 24, 26, 27).

The translocation of CSF out of the cranium is accommodated by the more compliant spinal subarachnoid space. The dura in the spinal compartment is distensible because of the presence of a collapsible epidural venous plexus (14, 32). These spinal epidural veins are valveless, therefore allowing rapid refilling and preventing permanent collapse of the epidural space. Moreover, the elastic nature of the spinal dura, with accompanying epidural veins, may act as a balloon mechanism—effectively recoiling during diastole to push CSF in a spinal–cranial direction.

Features of the dynamic CSF translocation mechanism may increase the efficiency of the intracranial Windkessel effect. The anatomic relationship of the cerebral arterial vasculature is well suited to maximize this effect. Unlike many other organs of the body, the majority of the large- and medium-sized arterial vasculature is situated outside the brain parenchyma. Specifically, these vessels run in the CSF-filled subarachnoid sulci and cisterns. As blood enters the cranium, arterial expansion occurs within these CSF spaces, not within the brain parenchyma. Therefore, the brain itself does not normally undergo significant changes in volume (19). Instead, the subarachnoid spaces expand, pushing the brain parenchyma inward. As the cerebral cortices are being pushed inward, CSF is translocated across the tentorium in both the basal cisterns and through the cerebral aqueduct.

Is the Windkessel phenomenon occurring within the intracranial compartment important? If CSF did not exist, the intracranial compartment would be noncompliant and, therefore, the internal carotid artery and superior sagittal sinus

blood flow waveform patterns would have to be nearly identical. A large pulsatility of venous blood, given its relatively larger intracranial blood volume proportion, would increase the inertial cerebrovascular reactance.

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INTRACRANIAL RESONANCE

Nonpulsatile venous blood flow in the intracranial compartment probably only occurs in nonphysiological conditions, such as during the months after a large decompressive craniectomy procedure (6). With respect to optimizing intracranial blood flow impedance, nonpulsatile venous flow may not be necessary. Egnor et al. (8–10) have elegantly suggested that the function of CSF system is to tune the primary harmonic resonance of the intracranial compartment to optimize the intracranial Windkessel phenomenon. Theoretically, a tuned oscillator could reduce the impedance mismatch by shifting the fundamental harmonic resonance frequency of an object while dampening high-impedance resonant frequencies.

A mechanical equivalent of oscillating systems has been classically illustrated using a weighted block suspended from a spring. Stimulating the system at the fundamental harmonic frequency would result in wild, destructive oscillations of the block. With regard to blood flow physiology, the vasculature and its surrounding tissue is the “block” that is being stimulated by a pulsatile blood flow input. If this input were to occur at the natural harmonic frequency of the system, then the fragile capillary system might experience bounding blood pressure fluctuations.

One approach to minimize large capillary pulse pressures would be to place a damper on the system (analogous to a shock absorber used in automobiles). Dampers, however, absorb energy and, therefore, reduce the efficiency of energy transfer. In effect, vascular autoregulation is a feedback-controlled damping system that regulates the pressure drop over the arterial tree by either vasodilation or vasoconstriction of arterioles.

A different, more efficient, approach to counteract a potentially harmful stimulation at the fundamental frequency would be to add a tuned oscillator. When a properly chosen smaller weight and spring is attached to the original block suspended from a spring, the smaller block will vibrate in such a manner that the movement of the original larger block will be minimal. In the second-order hemodynamic model discussed here, the second block and spring is anatomically represented by the CSF system and the distensible spinal dura.

The CSF system is the most logical intracranial component comprising the (presumed) tuned oscillator. The complex, dynamic movement of CSF both within the cranium and to-and-from the spinal compartment could provide optimal impedance matching between the intracranial compartment and the cerebrovascular input. Similar to the added mass and spring, the CSF system has an inherent mass (because of its volume) and elastance (because of the recoil offered by the distensible spinal dural sac). From a teleological perspective, the CSF tuned oscillator is probably broadly tuned around the resting heart rate frequency. Deviations from the resting heart rate will result in incremental increases in the cerebrovascular reactance. Fine-tuning of the amount of CBF (cerebrovascular autoregulation), however, likely occurs at the arteriolar level by adjustments of cerebrovascular resistance. For example, an increase in cerebrovascular reactance could be compensated by a reduction in resistance, thereby maintaining a constant cerebrovascular impedance. As a result, cerebrovascular impedance can be maintained within a narrow range to maintain a constant CBF.

ICP PHYSIOLOGY RE-EVALUATED

Given that pressure is defined as force per unit area, ICP implies that a force is being exerted. In the case of the intracranial compartment, this force must originate from some source. Other than gravity, the only external force normally affecting the intracranial contents is derived from the cardiovascular system. To illustrate this principle, a simple mechanical model is useful for the basic understanding of how intravascular pressure is transmitted to ICP. Here, the cranium is modeled as a rigid steel sphere that has only one inlet and outlet (which correspond to the arterial and venous systems, respectively). Within the sphere container, the inlet and outlet are connected by a thin-walled balloon. The volume outside the balloon, but within the sphere, is completely filled with water. The “vascular” system (tubing plus balloon) is completely filled with water as well, and circulation is driven by a pump. Based on hydrostatic principles, the intrasphere pressure will nearly exactly equal the intraballoon pressure, assuming that the surface tension of the balloon wall is small. Increases in the inlet pressure will be associated with corresponding increases in the intrasphere pressure. It is important to note that this can occur without an associated measurable change in intraballoon volume. From a mechanical and conceptual standpoint, this balloon is analogous to the cerebral capillary network. Because capillaries are composed of only a single endothelial cell layer (with no fibrous, elastic, or muscular reinforcement), the transfer of hydrostatic capillary pressure to the interstitial fluid (and therefore brain parenchyma) should be maximal at this point. Although venous pressure transmission will also be efficient, the venous pressure is, by definition, lower than that of the capillaries (to maintain anterograde flow).

The second subcompartment exposed to capillary hydrostatic pressures is the intraventricular space. The choroidal plexus epithelial layers do not have an elastic or muscular layer, and, therefore, the choroidal capillary hydrostatic pressure will be linearly related to the intraventricular pressure (the difference being a consequence of the brain–CSF barrier–induced osmotic pressure).

A third intracranial subcompartment consists of the subarachnoid space. The subarachnoid space is also potentially subject to forces generated by the arteries and veins, which course within this subcompartment. The very nature of the Windkessel effect relies on an arterial transmural pressure gradient. In normal circumstances, it is proposed here that the arterial expansion is primarily limited by elements of the arterial tunica media. Nevertheless, the fact that the arterial volume transiently changes dictates a corresponding change in subarachnoid space pressure. Here, the pressure–volume relationship put forth by Langfitt is pertinent. The pressure generated by the Windkessel expansion is the driving force that pushes the brain parenchyma inward, therefore, secondarily impinging on the intraventricular subcompartment.

Given these considerations, the physiological definition of ICP must be precise. For the purposes of this second-order model, ICP will be equated to intraparenchymal pressure. Intraventricular and subarachnoid pressure will be considered separately. In practicality, pathological perturbations to the system generate brief net pressure gradients that likely dissipate once a new equilibrium state is reached. Therefore, the analysis of pressure gradients between subcompartments is important to the understanding of how the system changes. Once (if) equilibrium is reached, the net pressure gradients will again be zero. An additional consideration is that there is evidence that intraventricular CSF communicates with the parenchymal extracellular fluid (18, 28). Flow of CSF between these subcompartments likely occurs normally and may be enhanced in pathological situations.

As proposed, the mechanical containment of intraluminal cerebral capillary pressure is considered minimal. Interstitial pressures are, therefore, regulated by the Starling Equilibrium—a process tightly controlled by the blood-brain barrier (BBB). The difference between capillary and interstitial hydrostatic pressures (ICP) will, therefore, be predominantly related to the oncotic/osmotic pressure differential, which, in turn, will be a function of the BBB (within the parenchyma). Similarly, the intraventricular pressure will be primarily determined by the choroid capillary hydrostatic pressure and the state of the blood–choroid plexus barrier. A slight differential between the ventricular and brain pressures could explain a bulk flow of CSF out of the ventricles—a situation compatible with the first-order model of CSF physiology.

Given the above stipulations and definitions, ICP will mainly be a function of cerebral capillary pressure. It should be noted that this concept of ICP is significantly different from that expressed in most modern neurophysiological literature. The standard, accepted explanation of ICP, typically involves a scenario in which volume is added to the intracranial compartment. As stated previously, appreciable volume cannot be added to this compartment. Because the cranium is “leaky,” adding volume merely displaces fluid (either CSF or blood) out of the intracranial compartment. The error in reasoning occurs in the interpretation of the Langfitt pressure–volume curve. It is not the volume, per se, which causes the increase in ICP; instead, it is the force required to displace the equivalent volume from the intracranial compartment that determines the ICP. From a practical standpoint, the only such force is derived from the cardiovascular system (ignoring, for the moment, external forces, such as the impact during head injury). The only force an intracranial space-occupying lesion can exert is gravitational. We argue here that changes in ICP occur secondary to changes in capillary pressure or alterations of the BBB. Furthermore, it is proposed that intracranial space-occupying lesions interfere with cerebrovascular dynamics indirectly via obstruction of the CSF system (or alterations of the BBB).

THE PATHOPHYSIOLOGY OF HYDROCEPHALUS

What causes ventriculomegaly? For so-called obstructive hydrocephalus, the classic (bulk CSF flow) theory of hydrocephalus provides a simple, although possibly incomplete, explanation. Communicating hydrocephalus is poorly explained by the classic theory. If the interference with “CSF absorption” were indeed occurring at the arachnoid granulations, then expansion of all of the CSF spaces (ventricular and subarachnoid) would be expected. This, combined with the fact that there is no demonstrable increase in the subarachnoid–venous pressure gradient in communicating hydrocephalus, should lead one to critically question the validity of the classic hydrocephalus theory.

There is another plausible, but not intuitive, explanation for ventriculomegaly. Proposed by Egnor et al. (10), differential pressure pulsatility alone may result in compartmental volume changes. With respect to hydrocephalus, an increase in choroid plexus capillary hydrostatic pressure pulsatility, and not necessarily mean pressure, could be the driving force. The phenomenon can be demonstrated by placing a balloon within another balloon. Maintaining a constant pressure inlet pressure to the outer balloon, the diameter of the outer balloon can be changed by varying the degree of air pressure pulsatility within the inner balloon. The inner balloon does not add air volume to the outer balloon per se, and yet the outer balloon volume increases.

The second-order hemodynamic theory of hydrocephalus predicts that alterations in CSF dynamic movement may result in an increase in cerebrovascular reactance (primarily caused by venous inertial effects), which will be compensated by a reduction in arteriolar resistance (to maintain a constant cerebrovascular impedance). A decrease

in arterial resistance is synonymous with a reduction in the pressure drop occurring at the arteriolar level (caused by the increase in vessel diameter). As a result, capillary blood pressure pulsatility would be predicted to increase as a result of an obstruction of the free dynamic movement of CSF.

The particular clinical disorder of CSF-related problems that occurs will depend on the differential response of choroid plexus versus brain parenchyma capillary hydrostatics. If the pulsatility in the choroidal capillaries predominates, then ventriculomegaly may occur. The degree of ventricular enlargement will depend on multiple factors, including the ventricular wall compliance. Moreover, it has been demonstrated in experimental hydrocephalus models that intracranial compliance decreases (improves) in conjunction with ventriculomegaly (16, 17). If intracranial compliance could be sufficiently lowered to re-stabilize the intracranial tuned oscillator system, then the forces driving the pathological hemodynamic changes could abate. Such a state would result in compensated hydrocephalus. Symptomatic hypertensive hydrocephalus would represent a continued hemodynamically pathological state in which autoregulatory arteriolar vasodilatation continues to generate high capillary hydrostatic pressures.

Other conditions, including pseudotumor cerebri, can be explained by this second-order hemodynamic theory. In selected cases, venous outflow obstruction would predictably result in an increase in capillary hydrostatic pressure, resulting in elevated ICP. Small ventricular size would occur if the process occurred primarily at the brain capillary level (more so than that at the choroid plexus). Primary alterations in BBB function (possibly associated with steroid use/withdrawal, hypervitaminosis states, etc.) could result in increases in brain tissue pressure resulting from a decreased pressure drop from the capillary hydrostatic pressure across the BBB.

CSF SHUNT PHYSIOLOGY

The classic concept of CSF physiology was presumably the basis behind the design of the currently used CSF shunt—where the differential pressure valve was designed to mimic the pressure-dependent drainage thought to occur at the arachnoid granulations. In very general terms, CSF was likened to a gently flowing river, and hydrocephalus was the lake that formed when a dam was introduced. A shunt was meant to provide an alternative drainage route—specifically at the identical rate of CSF formation. If this scenario indeed exists, why is over-drainage such a problem? The primary explanation likely lies in fact that CSF pressure is pulsatile—and therefore the concept of a gentle, smooth CSF circulation is incorrect. For historical reasons, all CSF shunt valves are required by the FDA to incorporate a unidirectional check-valve mechanism. The combination of a one-way valve mechanism and CSF pulsatility likely results in shunt over-drainage. Peak systolic CSF pulsations are vented unidirectionally by the shunt, resulting in a significant reduction in CSF pressure below that which would occur if CSF pressure were constant (2).

A second etiology of shunt over-drainage occurs because of gravity-dependent CSF flow. This phenomenon, misnamed “siphoning,” was recognized decades ago and spawned an industry aimed at counteracting its effect. The antisiphon device, originally touted as the panacea for over-drainage, has convincingly been shown not to prevent over-drainage complications. In normal pressure hydrocephalus, recent evidence suggests that exaggerated negative ICPs are unlikely to occur (2). Flow-limiting valve designs have fared marginally better than standard differential pressure valves, in that reduction in ventricle size occurs during a longer period of time, although, unfortunately, to a similar degree.

DEVELOPING THE NEXT GENERATION THERAPIES FOR HYDROCEPHALUS

Disturbances of the intracranial tuned oscillator, in effect, detuning it, will typically occur as a result of changes in intracranial compliance. In the second-order hemodynamic model, it is the interference with the free dynamic flow of CSF that is pathological. The difference between this and the classic CSF bulk flow theory lies in the fact that the two observers are looking at phenomenon at two very different time scales. In reality, the physiology and biomechanics at both time scales must be considered. However, restricting oneself to the myopic view of the classic model likely ignores the relatively more important dynamic forces that strongly influence intracranial CSF and blood flow dynamics.

As we gain a better understanding of the complex relationship between dynamic CSF movement and clinical disorders, including hydrocephalus, innovative treatments will arise. It is possible that future shunts may not need to shunt any significant amount of CSF at all—but instead may provide an effective alternative CSF translocation mechanism that re-tunes the intracranial compartment. This may be the primary mechanism by which ventriculocisternostomies (third ventriculostomies) function.

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Fig. 15.1 A, envelope tracings of blood flow velocity measurements obtained using Doppler ultrasonography of the internal carotid artery and internal jugular vein (immediately distal to the foramen lacerum). Here, the peak flow velocity is considered proportional to the CBF in these major vessels, and it is assumed that the CBF profiles obtained using the major vessels closely approximate the total CBF entering and exiting the intracranial space. B, overlapping the above tracings with equivalent areas under the curve for each per cycle (total volume in = total volume out), note that during systole, the incoming blood volume exceeds that exiting blood volume, whereas during diastole, the opposite occurs. This difference in volume is compensated by CSF movement out and in of the cranium, respectively.