Theories of cerebrospinal fluid dynamics and hydrocephalus: historical trend

A review

NIGEL PETER SYMSS, M.B.B.S., D.N.B., AND SHIZUO OI, M.D., PH.D.

Division of Pediatric Neurosurgery, Jikei Women’s and Children’s Medical Centre, Jikei University School of Medicine, Tokyo, Japan

According to the CSF bulk flow theory, hydrocephalus is caused by an imbalance between CSF formation and absorption, or a block at various locations in the major CSF pathway. New theories, however, have been proposed in which minor CSF pathways may play a significant role in the development of congenital hydrocephalus. The authors review major contributions to the literature and analyze the evolution of theories of CSF dynamics in relation to hydrocephalus, dividing their development into 4 stages on the basis of historical trends.

In Stage I (prior to 1950), 2 systems of classifying hydrocephalus were proposed, namely Dandy’s classifications of communicating and noncommunicating hydrocephalus and Russell’s nonobstructive and obstructive hydrocephalus. In Stage II (1950–1974), based on these theories of major CSF pathway dynamics, treatment focused on ventriculostomy as an alternative to reduction of CSF production by choroid plexus coagulation. In Stage III (1975–1999), some of the specific forms of hydrocephalus, especially in premature infants, were found to be unsuitable for ventriculostomy. In Stage IV (2000–2008), selection of treatment modalities evolved further, with a focus on analysis of the chronological changes in CSF dynamics and the differences in absorption pathways in the developing and mature brains. The authors focus on “minor pathway hydrocephalus” in the immature brain, differentiating it from the conventional classification of obstructive and nonobstructive “major pathway hydrocephalus.”

Key Words • cerebrospinal fluid dynamics • major pathway • bulk flow theory • minor pathway • evolution theory • hydrocephalus

Historically, the study of CSF dynamics almost always takes us back to the Monro-Kellie doctrine, which states that the total volume of the 4 main intracranial components—that is, the brain, the arterial and venous blood, and the CSF (although in the era of Kellie the volume of CSF was not considered)—must be constant due to the fixed volume of the skull and spinal canal. Also, any volume increase in one component causes an equivalent decrease in the other components, a consequence of fluids being incompressible. The 3 components influencing CSF dynamics are the production, circulation, and drainage of CSF.

Research on hydrocephalus began in 1914 with Dandy and Blackfan, who produced hydrocephalus in dogs by plugging the cerebral aqueduct. The bulk flow theory and the concept of CSF malabsorption as the cause of hydrocephalus are based on their experiment. Dandy then classified hydrocephalus into communicating and noncommunicating types (Fig. 1). In 1925, Cushing’s important work on “the third circulation” was published, and then in 1949 Russell classified hydrocephalus as nonobstructive or obstructive (Fig. 2). Communicating hydrocephalus, however, remained an enigma. O’Connell and Bering suggested that increased pulse pressure within the ventricles is the cause for communicating hydrocephalus. The more recent theory proposed by Oi and Di Rocco focuses on the minor CSF pathway through the brain parenchyma, choroid plexus, perineural lymphatic channels, and deep venous structures as the dominant CSF absorption site in the embryo, fetus, and infants, differentiating it from the major CSF pathways (Fig. 3). The aim of the present study is to analyze the concepts of CSF dynamics, which have continually evolved, with initial concepts having been based on experimental studies conducted in animals and more recent concepts being based on state-of-the-art MRI techniques.

Stages in the Historical Evolution of CSF Dynamics Research

Based on a review of original publications in the field of CSF dynamics research, we have divided the evolution

Abbreviations used in this paper: HCA = hydrocephalus chronology in adults; ICP = intracranial pressure; NPH = normal pressure hydrocephalus.
of the field into 4 stages: the first stage being until 1950, the second stage being from 1950 to 1974, the third from 1975 to 1999, and the final stage from 2000 to 2008. A summary of the historical trend of proposed theories of CSF dynamics can be found in Table 1.

**Stage I: Prior to 1950**

In 1854 Faivre suggested that CSF was formed in the ventricles by the choroid plexuses. Cushing observed weeping of fluid from the surface of the choroid plexus during surgery, and this supported Faivre’s view. However, proof only came in 1919, following Dandy’s publication of his experimental work on hydrocephalus, in which he showed that dilation of a lateral ventricle that normally follows occlusion of a foramen of Monro did not occur when the choroid plexus was excised from that ventricle. Putnam subsequently confirmed these findings by producing amelioration or relief of hydrocephalus in infants through electrical coagulation of the choroid plexus. Regarding CSF absorption, in most anatomy and physiology textbooks it is stated that CSF passes from the subarachnoid space into the great venous sinuses through the pacchionian granulations. This view was based on the results of injections of Prussian blue suspensions into the spinal canal of cadavers by Key and Retzius in 1870 (published in 1875–1876). It was supported by Weed’s work on experimental animals (published in 1914), in which he found that Prussian blue granules in many intracranial sites, yet focused on their presence in the arachnoid villi. It has been repeatedly demonstrated, however, that under normal conditions granules of Prussian blue never pass through the pacchionian bodies. Also, the hypothesis that CSF is absorbed by the pacchionian granulations is discredited by the fact that these structures are acquired with age and do not exist in infants and young children.

In 1929, Dandy stated in his article “Where is CSF absorbed?” that the fluid passes into the capillaries that abound in all the radicles of the subarachnoid space. This was based on the fact that when dyes were injected into the spinal canal they could be detected in the bloodstream in less than 2 minutes. In 1925, Harvey Cushing in his Cameron lecture on “The Third Circulation and its Channels” described CSF flow as a circulation somewhat like blood flow and somewhat like lymphatic flow. He also supported Weed’s idea that CSF absorption takes place via the arachnoid villi. Sepp, in his 1928 book, regarded vascular contractions as the major factor responsible for the circulation of the fluid. According to Weed, in his 1935 article, CSF absorption into the venous sinuses depends upon 2 factors: 1) the small hydrostatic pressure gradient favoring the passage of CSF through the villi into the veins and 2) the osmotic pressure of the proteins in the blood. In 1943, O’Connell summarized the modern view of CSF dynamics: First, the normal positive ICP is due to the balance existing between the production and absorption of CSF. Second, during a given period of observation, the CSF pressure is relatively constant and subject to minimal variations with the cardiac and respiratory activity. Third, the circulation of CSF is a movement from its point of production toward the arachnoid villi to replace fluid that has been absorbed. O’Connell also added that a vascular factor is important in maintaining the cerebrospinal circulation. A new understanding of
this dynamic was proposed in the work of Russell in her 1949 monograph, “Observations on the Pathology of Hydrocephalus,” in which she stated that any obstruction to the flow of CSF, within or outside the ventricular system, causes obstructive and nonobstructive hydrocephalus, respectively (Fig. 2). In her work, Russell provided an encyclopedic collection of hydrocephalic specimens. However, the theory that nonobstructive hydrocephalus is a result of malabsorption of CSF at the arachnoid villi is not substantiated by experimental evidence or physical reasoning.

In summary, in Stage I, the classification of hydrocephalus was based on the major CSF pathway.

Stage II: 1950–1974

In 1958, Dott and Gillingham supported the view of Sepp on the basis of their observation on the formation of pouches along the major cerebral arteries in cases of localized obstructions of the subarachnoid space. In 1961, Bering demonstrated his experimental work on circulation of the CSF in the dog model. He found that symmetrical hydrocephalus did not occur in dogs when hydrocephalus was induced after the choroid plexus was removed from one lateral ventricle with the foramen of Monro left open. The ventricle without the choroid plexus remained small or did not enlarge. Following unilateral choroid plexectomy in dogs with established hydrocephalus, that ventricle collapsed, while the contralateral ventricle continued to enlarge. He concluded that the back pressure generated by obstructing the flow of CSF was not the force producing hydrocephalus, but rather was the result of a local force produced by the choroid plexuses. His experiments showed that the normal pressure of CSF in dogs was 99 ± 2.4 mm H2O, and after bilateral choroid plexectomy it fell to 72 ± 4.2 mm H2O, which was close to the sagittal sinus pressure, thereby demonstrating that CSF is circulated by the forces generated by the choroid plexuses as they fill with blood with each arterial pulse. Later, in 1963, Bering and Sato studied the formation, flow, and absorption of CSF in dogs that had one normal ventricle and one that was gradually enlarging (progressive unilateral hydrocephalus) by means of steady-state studies during ventricular perfusion. They found the formation of CSF to be constant, independent of hydrostatic pressure, and unaffected by the development of hydrocephalus. They also found the absorption of CSF to occur within the ventricles.

**TABLE 1: Summary of the historical trend of proposed theories of CSF dynamics**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Contribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faivre, 1854</td>
<td>suggested CSF was formed in the ventricles by the choroid plexuses</td>
</tr>
<tr>
<td>Key &amp; Retzius, 1875–1876</td>
<td>hypothesized that CSF is absorbed by the pacchionian granulations</td>
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<tr>
<td>Dandy &amp; Blackfan, 1914</td>
<td>proposed a classification for hydrocephalus in CSF dynamics: communicating vs noncommunicating</td>
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<tr>
<td>Cushing, 1925</td>
<td>proposed concept of the “Third Circulation”</td>
</tr>
<tr>
<td>O’Connell, 1943</td>
<td>suggested that increased pulse pressure in the ventricle causes communicating hydrocephalus</td>
</tr>
<tr>
<td>Russell, 1949</td>
<td>proposed classification of hydrocephalus as nonobstructive &amp; obstructive</td>
</tr>
<tr>
<td>Bering, 1962</td>
<td>demonstrated that the choroid plexus generated the force for flow of fluid for ventricular enlargement</td>
</tr>
<tr>
<td>Bering &amp; Sato, 1963</td>
<td>demonstrated changes in formation &amp; absorption of CSF within the cerebral ventricles</td>
</tr>
<tr>
<td>DiChiro, 1966</td>
<td>demonstrated cranially directed CSF circulation w/ radionuclide cisternography; also hypothesized 2-directional flow of CSF in spinal canal</td>
</tr>
<tr>
<td>Di Rocco et al., 1978</td>
<td>demonstrated that hyperdynamic ventricular pulsation was sufficient to cause ventricular dilation</td>
</tr>
<tr>
<td>Oi et al., 1991</td>
<td>proposed various forms of postshunting isolated compartments; proposed 5-compartment hydrocephalus</td>
</tr>
<tr>
<td>Greitz, 1991</td>
<td>emphasized importance of pistonlike action of brain for pulsatile flow &amp; mixing of CSF</td>
</tr>
<tr>
<td>Oi &amp; Di Rocco, 2006</td>
<td>proposed “evolution theory in CSF dynamics” &amp; minor pathway hydrocephalus in developing immature brain</td>
</tr>
<tr>
<td>Rekate et al., 2008</td>
<td>demonstrated importance of cortical subarachnoid space in understanding hydrocephalus</td>
</tr>
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tricles and in the subarachnoid spaces, and the resistance to absorption of CSF in the normal ventricle was much higher than in the enlarging (hydrocephalic) ventricle.

The introduction of radioactive tracers in the 1950s allowed for the detailed analysis of the circulatory dynamics of CSF. In 1962, Rieselbach et al.25 described the use of scintillation detection to study the subarachnoid distribution of 198Au following intrathecal administration, and showed that there is a consistent pattern to the flow of CSF, from the lumbar subarachnoid space cranially to the cisterna magna, and flowing through the basal cisterns, it becomes distributed approximately equally between the midline pathways of the callosal cistern and interhemispheric tissue and the lateral pathways of the sylvian cisterns and subarachnoid spaces over the convexities of the cerebral hemispheres.

In 1964 and 1966 publications, DiChiro reported on the use of another tracer, radio-iodinated serum albumin, in neurological patients injected intrathecally (isotope cisternography) and intraventricularly (isotope ventriculography). At 1 hour postinjection the basal cisterns were filled with activity, at 3 hours the activity had reached the sylvian fissures, at 12 hours it was over the convexity. He also found a fast migration of tracer from the lumbar region to the convexity, concluding there was an active cranially directed CSF circulation, at least from the foramen magnum and above. He considered the cranial transport to be a result of CSF bulk flow. In monkeys, DiChiro et al.15 found a rapid descent of the tracer into the spinal canal after it was injected into the lateral ventricles. This supported his hypothesis of a 2-directional flow of CSF in the spinal canal, directed downward in the posterior compartment and upward in the anterior compartment. DiChiro stated that CSF radionuclide scanning is the method of choice for studying the normal physiological pattern and altered CSF dynamics. Many papers have since been published about CSF radioisotope distribution after injection into the CSF pathways.30,34,54 Rubin et al. (1966), Cutler et al. (1968), and Lorenzo et al. (1970) were the first to use the method of ventriculovenricular or ventriculolumbar perfusion in human studies. By the addition of various tracer substances to the perfusion solution, CSF formation and elimination could be measured at different pressure levels. Du Boulay et al.19 using air encephalography, found pulsatile CSF movements in the third ventricle and basal cisterns and proposed that brain expansion was the cause of this pulsation.

In summary, in Stage II, the therapeutic modalities were discussed mainly on the basis of the concept of the major CSF pathway.

Stage III: 1975–1999

Ekstedt20 used the constant pressure method to study the hydrodynamics conductance of the CSF outflow pathways, infusing artificial CSF at constant pressures and recording the resultant flow. With the infusion pressure reduced below the pressure of the sagittal sinus, all the CSF produced could be collected, and the CSF formation rate could be was calculated. Ekstedt found a rectilinear relationship between CSF pressure and the flow necessary to maintain the pressure and concluded that the drainage of CSF was through open channels from the subarachnoid space into the dural sinuses and that colloid osmotic pressure had no influence on the flow. He also concluded that the channels of the arachnoid villi, once opened by pressure, are not further distended by increasing pressure. He found an upward bend in the pressure/flow curve, which was interpreted as being caused by elasticity of the arachnoid villi. Earlier, Portnoy and Croissant (1976)28 had confirmed a rectilinear relationship between pressure and flow in human experiments but with a slightly different technique.

In 1978, Ekstedt20 published his elaborate work on CSF dynamics in relation to CSF pressure and flow. The variables analyzed were the resting pressure, the conductance of the CSF outflow pathways, the CSF formation rate, the pressure difference across the CSF outflow pathways, and the sagittal sinus pressure. None of these variables showed any age dependence or sex difference. That same year, Marmarou et al.36 with the help of a mathematical model of the CSF system, explained how changes in volume (input) are related to changes in pressure (output). Their theory was applied to the CSF dynamics of the experimental animal, and they studied 4 parameters that influence ICP level and its rate of change: the intracranial compliance, dural sinus pressure, resistance to absorption, and CSF formation. Their results demonstrated a nonlinear behavior of the CSF system, which was attributed to the exponential curve relating pressure to volume (that is, the curve that pressure must follow when the normal volume equilibrium of the CSF compartment is disturbed). The compliance decreases as CSF volume is increased, and as a result, the hydrodynamic characteristics of the system are not uniform throughout the range of the pressure. They stated that, assuming the intracranial compliance to be constant, the structure of the nonlinear equation derived in their study mathematically reduces the linear approximation proposed by Guinane24 and Benabid et al.1 Based on the analysis of Marmarou et al.,36 dynamic tests were developed for rapid measurement of CSF formation, absorption, resistance, and the bulk intracranial compliance. In 1983, Cabanes et al.3 proposed a new model for the study of CSF kinetics—bicompartamental analysis of the CSF circulation. With this model, the study of each compartment separately and of the intercompartmental relationship was possible after intraventricular injection of a radiotracer. Radioisotope ventriculography was performed in 80 patients, divided into 4 groups: those with normal CSF circulation, those with hydrocephalus, those with infantile hydrocephalus, and those with functioning shunts. There was a significant difference between the normal and hydrocephalic patients and also between patients with NPH and cerebral atrophy with respect to the mean values for volume of compartment, flow out of the system, and intercompartmental flow. Communicating and obstructive hydrocephalus could also be differentiated by their method; however, no differences in mean values were found with respect to the etiology or clinical course of the hydrocephalus. Kosteljanetz25 studied CSF dynamics using quantitative techniques without the use of radioisotopes and focused on the interrelationship between resistance to outflow of CSF (Rout) and ICP in patients with communicating hydrocephalus. He concluded...
that the findings supported “the concept that abnormal CSF dynamics play an important role in communicating hydrocephalus, while the significance of abnormal pressure-volume relationship remains speculative.” The author further stated, “resistance to CSF outflow is a major determinant of the ICP, which … is not normal” and noted that one might question “whether the term ‘normal-pressure hydrocephalus’ is indeed appropriate.”

In 1985, Oi and Matsumoto described the pathophysiology of nonneoplastic obstruction of the foramen of Monro causing unilateral hydrocephalus, and classified it into 4 categories: atresia of the foramen, morphological obstruction, functional obstruction, and patent foramen. They concluded that it is the change in the distribution of intracranial compliance caused by various lesions or therapeutic trials that plays a significant role in the pathophysiology of this condition. Oi and Matsumoto also proposed that the excess drainage of CSF via a ventricular shunt system causes morphological changes in the CSF pathways, possibly leading to the isolation of compartments. Overdrainage, which occurs most commonly in young infants, can lead to slit-ventricle syndrome. The presence of slit ventricles can, in turn, lead to the development of isolated ventricles, as in isolated unilateral hydrocephalus and isolated fourth ventricle. The pathogenesis for isolated unilateral hydrocephalus in such cases was described as a functional occlusion of the foramen of Monro, with shunting leading to a dynamic scenario in which overdrainage of the shunt-treated lateral ventricle causes a functional, rather than anatomical, obliteration of the foramen of Monro and subsequent enlargement of the contralateral lateral ventricle. For isolated fourth ventricle, Oi and Matsumoto proposed a “functional obstruction,” created by a pressure difference established between the supra- and infratentorial compartments. This arises when a shunt overdrains the supratentorial space, causing a low supratentorial intraventricular pressure, pulse pressure, and brain compliance. Occasionally, a trapped fourth ventricle can cause an associated hydromyelia. In 1991 Oi et al. reported a series of such cases and described 2 other conditions, isolated rhombencephalic ventricle and isolated central canal dilation. In isolated rhombencephalic ventricle, there is involvement of both the metencephalon and the myelencephalon; in isolated central canal dilation, only the central canal of the spinal cord is involved, with sparing of the fourth ventricle. They concluded that the pathophysiology of hydromyelia was closely related to hydrocephalus and proposed a new clinical category of hydrocephalus–hydromyelic hydrocephalus.

In 1985, the first study using gated MRI of the cerebral aqueduct was reported by Bergstrand et al., who described movements of pulsatile CSF through the aqueduct. Later, with the help of MR velocity imaging, Feinberg and Mark found systolic caudal movements of the diencephalon and brainstem, producing a wave of CSF down to the spinal canal, and systolic compression of the lateral ventricles from above, which they said moved CSF downward in systole. In their 1991 study of CSF circulation, Greitz et al. emphasized the importance of the pistonlike action of the brain for pulsatile flow and for the mixing of CSF within the cranium and in the spinal canal. This was followed by the 1992 article by Greitz et al., in which they reported on their study of brain tissue movement in the axial, sagittal, and coronal planes using a gated spin echo MRI sequence. The authors stated that arterial expansion is the driving force of pulsatile brain movements, remodeling the central and lower parts of the brain in a funnel-shaped manner required for the piston-like action. The tentorial hiatus and foramen magnum are important for the venting of brain and CSF during systole, with the return of CSF from the spinal canal occurring during diastole. Thus they concluded that the pulsatile brain movement was the result of the interaction of arterial expansion, brain expansion (by systolic capillary dilation), and volume changes in the veins and subarachnoid space, assuming the total volume of these components to be constant at every instant. In 1996, Greitz and Hannerz reported the findings of radionuclide cisternography (performed after injection of tracers into the spinal subarachnoid space) in a group of patients with venous vasculitis and higher than normal ICP. The flow phantom studies showed no tracer accumulation at an open outlet corresponding to the pacchionian granulations. The authors concluded that the normal radionuclide cisternography pattern “cannot be explained by a bulk flow transport of the tracer to an outlet at the pacchionian granulations but rather by a primary mixing caused by the pulsatile flow with a secondary dilution by newly formed CSF from the ventricular system.” They suggested “that the main absorption of CSF is through the central nervous system to the blood.”

In summary, in Stage III, the CSF circulation other than the major CSF pathway was further investigated.


In their 2001 paper, Czosnyka et al. reported on the use of computerized CSF infusion tests to analyze whether CSF compensatory parameters are dependent on age in patients who have symptoms of hydrocephalus with apparently normal ICP. They found the mean ICPs to be independent of the age of the patient. Resistance to CSF outflow demonstrated a nonlinear increase with advancing age, with an associated decrease in the CSF production rate, as a result of increasing age. Both the pulse amplitude of the ICP waveform and the slope of the amplitude-ICP regression line increased significantly with advancing age. There was also a nonlinear increase in the elastance coefficient, indicating increasing brain stiffness, with older age groups. The authors concluded, “CSF circulation and pressure-volume compensation are age dependent in patients who present with hydrocephalus without intracranial hypertension.” Earlier, in 1978, Ekstedt published the findings of a study of CSF hydrodynamics in “normal” patients; although he noted that age-dependent changes in the connective tissue of the arachnoid villi would be expected to cause decreased conductance in older patients, the results failed to demonstrate such an effect. By draining CSF at a low pressure, the CSF production rate was determined, and the following variables were analyzed: resting pressure, conductance of CSF outflow pathways, CSF formation rate, pres-
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The pressure difference across CSF outflow pathways, and sagittal sinus pressure. He found that none of the above variables showed any age dependence, nor was there any difference based on patient sex.

Egnor et al. developed a model of intracranial pulsations based on the analogy between the pulsatile motion of electrons in an electrical circuit and the pulsatile motion of blood and CSF in the cranium. The authors noted that existing experimental evidence and physical reasoning did not substantiate the theory that communicating hydrocephalus is caused by CSF malabsorption at the arachnoid villi. They found that communicating hydrocephalus could be simulated, with considerable accuracy, as a redistribution of vascular and CSF pulsations. The clinical and experimental manifestations of communicating hydrocephalus, such as ventricular dilation, ICP waves, narrowing of the CSF-venous pressure gradient, diminished cerebral blood flow, an elevated resistive index, and malabsorption of CSF, emerge naturally from their model. They proposed an explanation for communicating hydrocephalus as a disorder of intracranial pulsations. Stephensen et al. examined whether a transmantle pressure gradient existed in adult patients with communicating and noncommunicating hydrocephalus. Microsensors were used to measure the ICP for 17–24 hours—during sleep and awake periods—in the right lateral ventricle and in the subarachnoid space over the right cerebral convexity simultaneously. The measured pressure difference between the 2 sensors was always within the limits of the maximum possible hydrostatic pressure difference, and it correlated well with the expected difference for the various body positions. The authors found that ICP waves caused by cardiac pulse, respiration, and B waves were identical in both spaces, and demonstrated no factual support for the existence of a transmantle pressure gradient in hydrocephalus. In 2004, Greitz proposed a new hydrodynamic concept, dividing hydrocephalus into 2 main groups, acute and chronic, with acute hydrocephalus being caused by intraventricular CSF obstruction. Chronic hydrocephalus was further subdivided into communicating hydrocephalus and chronic obstructive hydrocephalus, and malabsorption of CSF was not considered the causative factor. Instead Greitz suggested that the increased pulse pressure in the brain capillaries maintains the ventricular enlargement, and chronic hydrocephalus is the result of decreased intracranial compliance, causing restricted arterial pulsations and increased capillary pulsations. In a 2007 publication, Greitz reemphasized the hydrodynamic concept of hydrocephalus, which is based on Dandy’s observation of capillary absorption of the CSF. The proposed concept represented a paradigm shift in the view of hydrocephalus, which Greitz described as opening “a new avenue in that ETV [endoscopic third ventriculostomy] may be an effective treatment also in communicating hydrocephalus.”

Madsen et al. stated that CSF movements include both bulk flow (representing direct current components in an electrical circuit analogy) and rapid pulsations (representing the alternating current components) in the CSF and in the vascular system. He suggested that the passage of pulsations through the brain constitutes a kind of “fourth circulation,” an idea he developed from Cushing’s “third circulation.” Oi proposed a perspective concept of hydrocephalus as “hydrocephalus chronology in adults” (HCA), clarifying aspects of the confusion in the classification and terminology of hydrocephalus (Fig. 4). In this view, the hydrocephalic state is classified into HCA Stages I–V based on the symptomatological features in relation to the chronological changes in ICP dynamics. Because of a variety of ICP dynamics recognized in NPH patients, a new term, “hydrocephalic dementia,” was proposed, since reference to the misleading pathophysiological aspect may cause confusion. It was also emphasized that ICP dynamics are varied and change chronologically in this specific type of hydrocephalus. The symptoms of the definitive triad of dementia, gait disturbance, and urinary incontinence are not specific to a particular kind of ICP dynamics, but may also be seen in the period of high ICP (HCA Stage III). In 2006, Oi and Di Rocco proposed the “evolution theory in CSF dynamics” based on a significant role for the transependymal intraparenchymal CSF pathway (“the minor pathway”) in various degrees as an alternative CSF pathway (Fig. 3). They found a high incidence of “failure to arrest hydrocephalus” by neuroendoscopic ventriculostomy in fetal, neonatal, and infantile periods. This was considered to depend on the specific CSF dynamics, in which the major CSF pathway has not developed and the minor pathway has a significant role. They proposed a new aspect of classification for hydrocephalus with special reference to the CSF circulation in the minor CSF pathway—that is, “minor pathway hydrocephalus”—differentiating it from the conventional classification by Dandy (communicating and noncommunicating) or Russell (nonobstructive and obstructive), which they described as referring to “major pathway hydrocephalus.” They also went on to propose a hypothesis that the CSF dynamics develop from the immature brain, as in the animals with the minor CSF pathway predominance, toward the mature adult human brain together with completion of the major CSF pathway: the “evolution theory in CSF dynamics.”

More recently Rekate et al. have defined the importance of CSF dynamics in the cortical subarachnoid space in understanding the pathophysiology of various forms of hydrocephalus. They proposed ventricular enlargement at the time of shunt failure implies a failure of flow between the ventricles and CSAS (cortical subarachnoid space), implying that patients who show this phenomenon are potential candidates for endoscopic third ventriculostomy.

In summary, in Stage IV, a new aspect of classification for hydrocephalus was proposed, with special reference to the CSF circulation in the minor CSF pathway—that is, “minor pathway hydrocephalus”—differentiating it from the conventional classification, described as “major pathway hydrocephalus.”

Conclusions

We can conclude that CSF is absorbed by the capillaries, and not by the arachnoid villi in human fetuses and neonates as well as in many animal species. Acute hydrocephalus is caused by an intraventricular obstruction to CSF flow in adults, but chronic hydrocephalus is the result of malabsorption of CSF mainly in the minor CSF pathway in experimental animal hydrocephalus models and clinically in fetal/neonatal hydrocephalus in humans. Based on the evolution theory of CSF dynamics there is a “minor pathway hydrocephalus” in the developing immature brain, which is differentiated from the classical or conventional classification of communicating and non-communicating or “major pathway” hydrocephalus.

Disclosure

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

Author contributions to the study and manuscript preparation include the following. Conception and design: Oi. Acquisition of data: Symss. Analysis and interpretation of data: both authors. Drafting the article: Symss. Critically revising the article: Oi. Reviewed submitted version of manuscript: both authors. Approved the final version on behalf of both authors: Oi. Statistical analysis: Symss. Study supervision: Oi.

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