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INNOVATIVE APPROACHES AND TECHNOLOGICAL ADVANCES IN THE MANAGEMENT OF HYDROCEPHALUS

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ABSTRACT

Introduction and Purpose: Hydrocephalus is a neurological disorder caused by abnormal accumulation of cerebrospinal fluid (CSF) within the brain ventricles, leading to increased intracranial pressure and neurological impairment. Despite advances in neurosurgery and neuroimaging, optimal management remains a major challenge. This review aims to summarize current therapeutic strategies for hydrocephalus and highlight recent and emerging innovations to improve outcomes and patient quality of life.
Materials and Methods: A comprehensive literature search was conducted using PubMed, BioMed Central, Scopus, and Google Scholar. Keywords included: hydrocephalus, NPH, VP shunting, LP shunting, and endoscopic third ventriculostomy (ETV). Reference lists of selected papers were also reviewed.
Conclusion: Over the past century, hydrocephalus treatment has progressed from simple drainage to valve-regulated shunts and endoscopic procedures. However, no curative therapy exists, and CSF diversion remains the mainstay. Progress has been incremental due to heterogeneous etiologies, biological complexity, technical constraints, and lack of pharmacological options. Current approaches primarily include ventriculoperitoneal shunting and ETV, selected according to etiology and patient profile. Future directions involve hybrid surgical techniques (ETV with stenting), smart and biocompatible implants, molecular modulation of aquaporins and glial function, gene and stem-cell therapies, and prenatal neuroendoscopic repair demonstrated in experimental models. The next generation of therapy will likely unite mechanical precision with biological insight, shifting from simple drainage toward restoration of CSF physiology and brain health. Integrating surgical, molecular, and etiological perspectives is essential to move beyond symptomatic management.

KEYWORDS

Hydrocephalus, Cerebrospinal Fluid, Ventriculoperitoneal Shunt, Endoscopic Third Ventriculostomy, Neuroinnovation, Neurosurgery

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Introduction

Hydrocephalus is a pathological condition marked by an interruption in the normal circulation or absorption of cerebrospinal fluid (CSF), typically leading to elevated intracranial pressure and subsequent abnormal ventricular enlargement [1].

Hydrocephalus is regarded as one of the most prevalent neurological conditions. This condition can affect individuals of all age groups, including children, adults, and the elderly. It is estimated that the incidence of congenital and acquired infant hydrocephalus ranges from 80 to 125 cases (in some regions even more than 300) per 100 000 births, depending on the region of the world. Some models estimate that nearly 400 000 new cases of pediatric hydrocephalus occur each year. Infant hydrocephalus represents a significant public health concern, with its etiology being diverse and closely linked to socioeconomic conditions and access to medical care. Registry-based data suggest that in regions with more advanced perinatal and neurosurgical care, the number of infants requiring initial surgical treatment for hydrocephalus may show a decreasing trend, despite a stable or increasing number of diagnoses. In this age group, the most frequent causes include congenital malformations, hydrocephalus associated with spina bifida, as well as acquired forms stemming from intraventricular hemorrhage in premature infants and infections [2,3].

In the group of adults, on the other hand, hydrocephalus represents a growing global health concern, with an overall estimated prevalence of approximately 85 cases per 100 000 individuals. Although, the risk of developing hydrocephalus increases significantly with age. While the condition is relatively uncommon among individuals younger than 65 years, its prevalence rises markedly in older populations – reaching about 175 per 100 000 among those aged 65 to 80, and up to 400 per 100 000 in individuals over the age of 80. Unlike in children, where primary disturbances in cerebrospinal fluid circulation are most commonly observed, adult hydrocephalus is typically secondary and associated with other underlying conditions. The most frequent causes include cerebrovascular diseases (such as stroke) and tumors of the central nervous system. Other important etiologies involve post-traumatic hydrocephalus, normal pressure hydrocephalus (NPH), and pseudotumor cerebri [4–6].

Considering all of the above, this article aims to provide a comprehensive overview of the underlying pathophysiology of hydrocephalus, outline its common clinical presentations, and discuss the appropriate timing and methods for diagnostic evaluation. Furthermore, it explores current approaches to the management of adult patients with pediatric history of hydrocephalus, addressing those with an established history of hydrocephalus.

Types and causes of hydrocephalus

Hydrocephalus can be classified etiologically into two types: primary and secondary.

The causes of primary hydrocephalus mainly include congenital malformations and developmental anomalies of the central nervous system (CNS) and neural tube, as well as genetic factors or syndromes such as Dandy-Walker syndrome and Arnold-Chiari malformation. Congenital hydrocephalus is also associated with primary hydrocephalus and, in addition to the aforementioned factors, may result from the presence of intracranial cysts or intrauterine infections during fetal development.

In contrast, secondary hydrocephalus develops as a consequence of acquired conditions. The causes of secondary hydrocephalus include intracranial hemorrhages, subarachnoid hemorrhages, traumatic brain injury (TBI), intracranial tumors, hematomas, cysts, vascular malformations, inflammatory conditions (e.g., meningitis), ischemic and hemorrhagic strokes, as well as neurosurgical procedures [7,8].

At present, four main types of hydrocephalus are recognized: communicating, non-communicating, normal pressure hydrocephalus (NPH), and hydrocephalus ex vacuo.

Communicating hydrocephalus

Communicating hydrocephalus is a relatively rare complication observed in patients with glioblastoma. Clinically and radiologically, it often manifests as a form of chronic hydrocephalus. While the exact pathophysiological mechanisms contributing to its development and its influence on overall prognosis remain insufficiently understood, emerging evidence suggests that cerebrospinal fluid diversion procedures, such as shunt placement, may lead to improvements in quality of life for affected individuals [9].

Non-communicating hydrocephalus

Non-communicating hydrocephalus arises due to intraventricular cerebrospinal fluid obstruction. The obstruction is commonly at the foramen of Monro, to the aqueduct of Sylvius, the fourth ventricle, or the foramen magnum. Non-communicating types can arise from hemorrhage, traumatic brain injury, brain tumor, cyst, or infection [10].

Normal pressure hydrocephalus

NPH is a form of communicating hydrocephalus that occurs with normal or only slightly elevated intracranial pressure. Primary NPH is believed to result from alterations in cerebrovascular flow dynamics, such as hypoperfusion, glymphatic system dysfunction, or microglial metabolic changes. In contrast, secondary NPH may arise due to non-communicating hydrocephalus or disturbances in cerebrospinal fluid circulation, secondary to conditions that impair cerebrospinal fluid absorption or drainage pathways, including meningitis, subarachnoid hemorrhage, or congenital malformations.

This type of hydrocephalus is clinically characterized by the Hakim triad of symptoms:

- Progressively worsening cognitive decline, typically mild and potentially reversible,
- Gait disturbances, such as ataxia or spastic paresis,
- Urinary incontinence.

It is important to note that not all components of the triad must be present to establish a diagnosis of NPH.

Radiological features of normal pressure hydrocephalus typically include disproportionately enlarged subarachnoid spaces, widening of the Sylvian fissures, narrowing of the high convexity subarachnoid spaces, and ventricular enlargement, often assessed using the Evans index [11,12]

Ex vacuo hydrocephalus

This type of hydrocephalus represents a degenerative reduction in brain parenchymal volume, accompanied by compensatory ventricular dilatation, typically seen in patients with neurodegenerative disorders [13]. Table 1. presents the classification of hydrocephalus.

Table 1. Classification of hydrocephalus.

Etiological classification	Anatomical/Pathophysiological Classification
Primary (idiopathic) hydrocephalus	Non-communicating (obstructive) hydrocephalus
Secondary (acquired) hydrocephalus	Communicating hydrocephalus

Diagnosis and differential diagnosis of hydrocephalus

Since hydrocephalus is a nonspecific symptom, it must be differentiated from a wide range of conditions. These include:

- Hematomas
- Age-related brain changes
- Frontal lobe epilepsy
- Frontotemporal dementia
- Frontotemporal lobe degeneration
- Alzheimer’s disease and other forms of dementia

- Glioblastoma
- Oligodendroglioma
- Brain abscesses
- Intracranial hemorrhage
- Bone marrow failure syndromes
- Meningiomas
- Migraine and its variants
- Craniopharyngioma
- Idiopathic intracranial hypertension
- Brain tumors
- Primary central nervous system lymphoma
- Disorders of the visual system [14].

The diagnostic process for hydrocephalus typically begins with a thorough neurological examination, tailored to the patient's age, clinical presentation, and any suspected anomalies within the central nervous system. This examination commonly includes assessment of muscle strength, reflexes, coordination, balance, cranial nerve function (including vision and hearing), as well as cognitive status and mood.

Neuroimaging plays a central role in confirming the diagnosis and excluding alternative pathologies. In neonates and infants, cranial ultrasound is often the initial modality of choice due to its safety, accessibility, and ability to visualize ventricular enlargement through the fontanelle. In prenatal care, ultrasound can also detect fetal hydrocephalus.

In older children and adults, magnetic resonance imaging (MRI) is generally preferred due to its superior soft tissue resolution and ability to assess CSF dynamics. MRI enables detailed evaluation of ventricular enlargement, periventricular white matter changes, and potential underlying etiologies. In urgent cases presenting with sudden neurological decline, computed tomography (CT) is typically the first-line modality, as it allows for rapid assessment of ventricular size, potential obstructive lesions, and signs of intracranial hypertension.

A hallmark radiologic sign of hydrocephalus is ventricular dilation, with temporal horn enlargement considered an early and sensitive indicator. Widening of the temporal horns of the lateral ventricles >6 mm not accounted for by hippocampal atrophy. Additional features include ballooning or outward bowing of the third ventricle and rounding of the posterior horns. Notably, in hydrocephalus, external CSF spaces may appear disproportionately compressed relative to the dilated ventricles.

Evans index (Ei) serves as an indicator of ventricular size and has been suggested as a useful biomarker for diagnosing NPH. However, it is a relatively crude measure of ventriculomegaly and is highly dependent on the slice location and angle during imaging. Additionally, it lacks specificity for NPH and has been observed in other neurological disorders, including progressive supranuclear palsy and dementia with Lewy bodies. The Evans index is calculated as the ratio between the maximum width of the frontal horns of the lateral ventricles and the greatest internal diameter of the skull at the same axial level on CT or MRI scans. Interpretation guidelines for the Evans index are as follows:

- 0.20–0.25: normal
- 0.25–0.30: possible or early ventriculomegaly
- >0.30: ventriculomegaly

It is important to note that the Evans index may be elevated in some healthy elderly individuals and varies according to age and sex [15].

Transependymal CSF migration, resulting from elevated intraventricular pressure, can be visualized on CT as periventricular hypodensities and is more sensitively detected on T2-weighted and FLAIR MRI sequences. It is essential to distinguish these changes from age-related white matter lesions, which are generally smaller (less than 10 mm) and display a characteristic anteroposterior gradient in thickness.

When hydrocephalus is confirmed, further MRI sequences – particularly high-resolution sagittal T2-weighted images (such as CISS or SPACE sequences) – are recommended to assess anatomical structures including the corpus callosum, floor of the third ventricle, infundibulum, and cerebral aqueduct. Upward bowing and thinning of the corpus callosum, and downward bulging of the third ventricular floor, are indirect markers of chronic ventricular pressure. Evaluation of aqueductal patency is critical to identify potential obstructive mechanisms contributing to CSF accumulation.

In selected cases, additional tests such as lumbar puncture may be employed to measure CSF pressure and analyze its composition, particularly in suspected cases of NPH or postinfectious hydrocephalus. Intracranial pressure (ICP) monitoring, utilizing surgically placed sensors within the brain or ventricles, may be necessary in patients with suspected elevated intracranial pressure. Moreover, fundoscopic examination can help identify papilledema as an indirect indicator of increased intracranial pressure [16,17].

In the diagnosis of idiopathic normal pressure hydrocephalus (iNPH), increasing emphasis is placed on identifying patients who may benefit from neurosurgical treatment. iNPH should be considered in patients presenting with unexplained, symmetrical gait disturbances, frontal-subcortical type cognitive impairment, and urinary urgency incontinence – particularly when MRI imaging shows ventricular enlargement in the absence of other findings that could explain the symptoms. One of the most useful prognostic tools in assessing the potential effectiveness of shunt surgery is the so-called CSF tap test (Miller Fisher Test). This involves a single removal of a large volume of cerebrospinal fluid (typically 30–50 ml) via lumbar puncture, followed by evaluation of gait function (or other symptoms) before and after the procedure. An alternative with higher diagnostic sensitivity is the placement of a temporary lumbar catheter, allowing external CSF drainage over a period of several days, which provides a more reliable assessment of symptom reversibility. Another available method is the CSF infusion test, used to evaluate cerebrospinal fluid outflow resistance, although this technique is primarily employed in specialized centers [18]. Table 2. presents a summary of the diagnostic options for hydrocephalus.

Table 2. Diagnostic tools and imaging characteristics for hydrocephalus.

Diagnostic method	Utility	Key findings / Notes
Neurological examination	Baseline clinical assessment	Assesses motor function, reflexes, cranial nerves, cognition, balance, and mood
Cranial ultrasound	Initial imaging modality for young patients	Visualizes ventricular size via fontanelle; safe and accessible
MRI	Preferred modality for detailed evaluation	Evaluates ventricular size, CSF flow, periventricular changes, and underlying etiologies
CT scan	Fast and widely available	Detects ventricular dilation, mass effect, hemorrhage, and signs of elevated ICP
T2/FLAIR MRI sequences	Detect transependymal CSF flow	Shows periventricular hyperintensities; distinguishable from age-related white matter lesions
High-resolution T2 MRI (CISS/SPACE)	Detailed anatomical assessment	Thinning of corpus callosum, bowing of 3rd ventricle floor, aqueduct patency evaluation
Lumbar puncture/ CSF tap test (Fisher test)	Measures CSF pressure; sample for analysis; predicts shunt responsiveness	May aid diagnosis and therapeutic planning
Intracranial pressure monitoring	Direct pressure monitoring	Sensor-based assessment of ICP levels via surgical placement
Fundoscopic examination	Indirect assessment of ICP	Detects papilledema
Temporary lumbar drainage	High diagnostic sensitivity	Continuous external CSF drainage over several days to assess symptom reversibility
CSF infusion test	Measures CSF outflow resistance	Primarily used in research or tertiary centers

Complications

The extent of brain damage resulting from hydrocephalus can vary widely and is influenced by several factors. In newborns born with severe or advanced hydrocephalus, there is a high likelihood of neurological impairment and potential physical disabilities. Complications related to hydrocephalus can arise from the condition's progression itself, as well as from the medical management and surgical interventions used to treat it. Neurological manifestations may include cognitive dysfunction, visual changes, seizures, and gait disturbances, all of which can significantly impair a patient's quality of life. In cases of advanced or unmanaged hydrocephalus, serious consequences such as temporal lobe herniation may occur. Urinary incontinence is another common feature, particularly in patients with normal pressure hydrocephalus. From a surgical standpoint, complications related to ventriculoperitoneal shunting are varied and can include mechanical issues

such as shunt obstruction, disconnection, under-shunting, or over-shunting. Over-drainage may lead to the development of subdural hematomas or hygromas. Additionally, systemic complications such as electrolyte imbalances and metabolic acidosis may arise postoperatively or in the context of prolonged disease [14].

Long-term shunt use can lead to several complications. These include risks associated with the surgery itself, such as perioperative morbidity and mortality. Shunt infections – most commonly seen within the first three months post-operation – occur in roughly 5–9% of cases. Additionally, shunt malfunction is a significant concern, especially in pediatric patients, with around 40% experiencing issues within the first two years after placement [19].

Ventriculoperitoneal shunts can lead to several potential complications. One common issue is infection, often caused by skin flora such as Staphylococcus epidermidis entering the shunt system. Other complications include intracerebral or intraventricular hemorrhage, improper positioning of the shunt, and accidental perforation of abdominal structures during placement. Over time, the shunt may erode through the skin, exposing the system, and in some cases, over-drainage can occur, leading to slit ventricle syndrome [20,21].

In patients with idiopathic normal pressure hydrocephalus (iNPH), comorbid conditions like hypertension and type 2 diabetes mellitus are commonly observed. Type 2 diabetes, in particular, has been associated with increased mortality in this patient group [22]. Research from Finland by Vanhala et al. has shown that schizophrenia is approximately three times more prevalent in individuals with iNPH compared to the general elderly population [23].

Treatment

Treatment of hydrocephalus can be divided into two types: indirect and direct. Indirect treatment is aimed at addressing the underlying disease that causes disturbances in cerebrospinal fluid circulation, whereas direct treatment focuses on restoring proper CSF outflow or absorption, most often through neurosurgical techniques.

Depending on the etiology, indirect treatment may take various forms and primarily depends on the underlying condition and the secondary cause of hydrocephalus. For example, in hydrocephalus secondary to bacterial meningitis, intensive targeted antibiotic therapy is used with surgical EVD [24].

In cases involving tumors of the central nervous system, oncological treatment is implemented, including surgical resection, radiotherapy, or chemotherapy. In patients with post-traumatic or post-stroke hydrocephalus, causal treatment such as evacuation of intracranial hematoma or control of intracranial pressure may be appropriate. Indirect treatment is therefore an important component of the management of patients in whom the cause of hydrocephalus is potentially reversible and does not require the immediate use of neurosurgical techniques [25,26].

In obstructive hydrocephalus, where CSF flow is blocked, one common indirect treatment is to reduce CSF production. This is often done with medications like acetazolamide, which inhibits carbonic anhydrase, lowering CSF formation, and furosemide, a diuretic that blocks sodium-potassium-chloride transport in the kidneys, promoting fluid removal [27,28].

Before shunt surgery became standard in the 1950s and 60s, drug therapies aimed mainly to decrease CSF production or reduce brain swelling through diuresis.

Glycerol, an oral osmotic agent known since the early 1900s, was used to lower intracranial pressure by drawing fluid out of the brain and was suggested for hydrocephalus treatment [29].

Similarly, mannitol serves as an osmotic agent but is more commonly used today for short-term management of brain swelling rather than chronic hydrocephalus [30].

Glucocorticoids (steroids) have been used for many years to reduce inflammation and edema in neurological conditions with increased intracranial pressure. In hydrocephalus, they may help indirectly by reducing inflammation and fibrosis in the subarachnoid space, which can contribute to fluid buildup.

Finally, indirect treatment also includes addressing the root causes of hydrocephalus – such as infections, hemorrhages, or tumors-to prevent further CSF accumulation and progression of the condition [31].

VP Shunting

Ventriculoperitoneal (VP) shunts are instruments used by surgeon to shunt cerebrospinal fluid in the effective treatment of hydrocephalus. In a typical ventriculoperitoneal (VP) shunt procedure, a catheter is inserted with its proximal tip positioned within the cerebral ventricles to facilitate drainage of cerebrospinal fluid. This catheter is connected to a valve mechanism that regulates CSF flow based on intracranial pressure. Most modern valves are adjustable, allowing clinicians to fine-tune the pressure settings postoperatively to

better manage patient symptoms. Some systems also include a gravitational or anti-siphon component, which helps prevent over-drainage of CSF when the patient is in an upright position. The distal end of the catheter is then tunneled subcutaneously down to the peritoneal cavity, where the CSF is reabsorbed into the body [32].

Once a diagnosis of normal pressure hydrocephalus is confirmed, the primary treatment is cerebrospinal fluid diversion, most commonly through the placement of a ventriculoperitoneal (VP) shunt. However, the key clinical challenge lies in selecting patients who are most likely to benefit from this intervention. Several factors have been associated with more favorable outcomes following shunting. These include a relatively short duration of symptoms-typically less than six months-prior to surgery, as early intervention is often linked to better recovery. A clinical pattern where gait disturbance appears before cognitive decline also suggests a higher likelihood of improvement. Additionally, a positive response to a high-volume lumbar tap test, which temporarily relieves symptoms following the removal of about 40 mL of CSF, serves as a useful predictive tool. Neuroimaging findings such as the absence of significant cerebrovascular disease and the presence of an aqueductal flow void on T2-weighted MRI also support the potential for a good post-shunt response [33,34].

LP shunting

The lumboperitoneal (LP) shunt serves as an alternative method for diverting CSF, but its use has been relatively limited. Early reports highlighted a high rate of complications, primarily due to the absence of a reservoir or adjustable valve mechanism to fine-tune CSF flow. This lack of flow control often resulted in issues such as over- or under-drainage, contributing to its underutilization in clinical practice despite its utility in select cases like communicating hydrocephalus and idiopathic intracranial hypertension.

In lumboperitoneal shunting, a catheter is inserted with its proximal tip positioned in the intrathecal space of the lumbar spinal canal to divert CSF. The distal portion of the catheter is tunneled beneath the skin and directed into the peritoneal cavity, where the CSF is absorbed. These systems may include a pressure-regulated valve, although some configurations operate without one. LP shunts are primarily used in cases where there is no anatomical obstruction within the ventricular system-making them suitable for conditions such as communicating hydrocephalus, idiopathic intracranial hypertension, postoperative pseudomeningoceles, and cerebrospinal fluid leaks [35,36].

Endoscopic third ventriculostomy

Endoscopic third ventriculostomy (ETV) is a surgical intervention commonly used to treat obstructive hydrocephalus, particularly when the blockage occurs at the aqueduct of Sylvius, such as in cases of aqueductal stenosis. This procedure involves creating an opening in the floor of the third ventricle, allowing CSF to bypass the obstruction and flow directly into the subarachnoid space, thereby relieving intracranial pressure without the need for a shunt. Endoscopic third ventriculostomy has emerged as a preferred surgical option for treating obstructive hydrocephalus. The broader adoption of endoscopic procedures across neurosurgery has contributed to ETV's increasing popularity, particularly in cases where CSF flow is physically blocked. While ETV has shown encouraging outcomes across a range of causes of obstructive hydrocephalus, its success is notably reduced in patients with hydrocephalus resulting from hemorrhage or infection. Nevertheless, when patients are carefully selected and the procedure is supported by detailed imaging, experienced surgical techniques, and attentive post-operative management, ETV can be performed safely and effectively [37].

The procedure is typically carried out with the patient lying on their back and the head flexed forward, which positions the burr hole at the highest point of the skull. This setup helps minimize the risk of CSF over-drainage and prevents air from entering the ventricular or subdural spaces – an important consideration in cases with significant ventricular enlargement. Excessive CSF loss during surgery is a known contributor to complications such as subdural hematomas. Some surgeons prefer the semi-sitting position to further reduce these risks.

For ETV to be feasible, the anatomical structures-specifically the lateral ventricles, foramen of Monro, and third ventricle-must be large enough to accommodate the endoscope. A minimum diameter of approximately 7 mm for both the third ventricle and foramen of Monro is generally considered necessary. In patients with slit-like ventricles, often due to chronic over-drainage from a shunt, externalizing the shunt temporarily may help restore ventricle size and facilitate endoscopic access. When needed, stereotactic navigation tools can assist in safely guiding the endoscope into the ventricular system [38].

Summary

Hydrocephalus is a complex condition caused by disturbances in the outflow, production, or absorption of cerebrospinal fluid (CSF). This condition can lead to deterioration of neurological function. It can occur at any age and may result from a variety of causes, including congenital malformations, infections, hemorrhages, tumors, trauma, as well as idiopathic mechanisms, as seen in idiopathic normal pressure hydrocephalus (iNPH).

Diagnosis relies on a combination of clinical features and neuroimaging findings, particularly ventricular enlargement disproportionate to cerebral atrophy. A key aspect of the diagnostic process is identifying the underlying cause of hydrocephalus and differentiating it from other neurodegenerative and structural brain diseases that may coexist with it. In some cases of hydrocephalus, the diagnostic workup should also be extended to include additional tests, such as the CSF tap test.

Management strategies can be broadly categorized into indirect treatments, which address the underlying etiology and direct interventions aimed at restoring CSF flow, most commonly through neurosurgical techniques like ventriculoperitoneal shunting or endoscopic third ventriculostomy (ETV).

Given the diversity of presentations and underlying causes, hydrocephalus remains a diagnostic and therapeutic challenge. A multidisciplinary approach is often necessary to ensure optimal patient care.

Discussion

Past and future

Hydrocephalus has been recognized since antiquity – Hippocrates and Galen described it as excess fluid in the skull, though its mechanisms were unknown. Early surgical attempts such as trephination or drainage were crude and usually fatal. A better understanding of CSF circulation in the 19th and early 20th centuries enabled more rational treatments. Walter Dandy and Kenneth Blackfan pioneered studies of the ventricular system, and in 1918 Dandy performed choroid plexus excision to reduce CSF production. Despite its theoretical basis, outcomes were poor and mortality high. By the mid-20th century, advances in anesthesia, asepsis, and materials made surgery safer, but treatments still focused on symptom relief rather than cure [39].

Shunting systems and Endoscopic Third Ventriculostomy

A major advance came in the 1950s with the introduction of silicone ventriculo-peritoneal (VP) and ventriculo-atrial (VA) shunts, which diverted CSF from the ventricles to absorptive cavities and greatly improved survival in children. The principle remains the same today: a catheter drains CSF while a valve regulates pressure. However, shunts often fail due to infection, blockage, or mechanical malfunction, leading to repeated revisions – sometimes dozens in childhood – and long-term failure rates over 50%. Though life-saving, shunts do not correct the underlying CSF imbalance [16].

Later, neuroendoscopy transformed management. **ETV**, which restores CSF flow through a small opening in the third ventricle floor, became the preferred option for obstructive hydrocephalus. Large studies show 70–90% long-term success, with around 80–85% of patients achieving shunt independence. Yet ETV is less effective in infants and post-infectious or post-hemorrhagic cases, where shunting is still required [16].

Despite improved safety and outcomes, no curative therapy has emerged since the 1950s, and progress has remained largely incremental.

Heterogeneous etiology and pathophysiology

As highlighted in the review *Hydrocephalus: Historical Analysis and Considerations for Treatment*, by Hochstetler et al., there have been only “incremental improvements in surgical treatments and little progress towards prevention or cure” over the past five decades [16].

Hydrocephalus is not a single disease but a collection of conditions that share ventricular enlargement as a common endpoint. It can arise from congenital malformations (aqueductal stenosis, neural tube defects), intraventricular hemorrhage, infections, tumors, trauma, or vascular causes. The pathophysiology ranges from obstructed CSF flow to impaired absorption or even altered CSF production [4].

Because of this diversity, a single surgical technique rarely addresses the root cause. Procedures such as ETV are effective only for obstructive forms; when hydrocephalus results from subarachnoid scarring or impaired CSF absorption, a bypass does not correct the underlying dysfunction. Thus, current treatments remain mechanical solutions to a biological problem.

Technical limitations and complications

Even with modern materials, shunt systems remain failure-prone. Infection rates vary between 5–15%, and mechanical obstruction remains common, particularly in infants. Each shunt revision increases the risk of neurological injury and infection [4].

ETV, while conceptually elegant, is technically limited by patient anatomy and disease type. It may close spontaneously due to gliosis or membrane formation. Moreover, it cannot correct microstructural changes in brain tissue, such as interstitial edema, ependymal loss, or gliosis, which persist despite restored CSF flow [16].

Thus, the surgical armamentarium has plateaued: we can divert or re-route CSF, but we cannot yet repair the underlying physiological defect.

Pharmacological and molecular therapies

Over the past decades, numerous pharmacological attempts have been made to influence CSF dynamics – including acetazolamide, furosemide, isosorbide, and corticosteroids. None has proven sustainably effective.

The 2022 review underscores that "no medical therapy has been shown to reliably prevent or reverse hydrocephalus." The absence of viable drug targets reflects the limited understanding of the molecular and cellular mechanisms driving CSF accumulation and brain injury. Consequently, hydrocephalus remains almost entirely within the domain of surgery [4].

Research barriers and data limitations

Conducting large, randomized trials in hydrocephalus is inherently difficult:

- the disease is relatively rare,
- etiologies vary widely,
- most patients are infants or young children,
- and long-term follow-up is required.

This heterogeneity makes it hard to compare outcomes across studies or to develop unified guidelines. Moreover, the lack of reliable biomarkers or imaging metrics for early disease progression further hampers progress.

In summary, despite improved instrumentation, safer anesthesia, and more refined materials, the fundamental paradigm of CSF diversion has not changed since the 1950s. The persistence of complications, biological heterogeneity, and absence of causal therapies explain why no transformative progress has been achieved [4].

Future Directions in Hydrocephalus Treatment

Current research on hydrocephalus focuses on three main directions. The first involves improving mechanical and endoscopic devices, such as advanced stents, sensors, and new shunt systems designed to better regulate cerebrospinal fluid and reduce complications. The second explores molecular and cellular therapies targeting the underlying disease mechanisms, including modulation of aquaporins, inflammation control, and regenerative approaches using gene or stem-cell techniques. The third focuses on experimental and preclinical studies, especially animal models, used to test these innovations before clinical application. Together, these approaches aim to move treatment from simple fluid diversion toward more effective, biologically based solutions [4].

Advances in surgical and implant technologies

Stents and hybrid endoscopic techniques

Recently, *endoscopic aqueductoplasty and stenting* have been proposed as solutions for recurrent or complex hydrocephalus. In a pediatric series (n = 33) involving isolated fourth-ventricle hydrocephalus, long-term success reached 87% at 2 years and 73% at 4 years. A 2024 study evaluating stented ETV (sETV) in 67 patients achieved an 88% success rate. The rationale is straightforward: the stent maintains patency of the new CSF pathway, preventing closure due to gliosis. Such "hybrid" approaches may extend the effectiveness of ETV, especially in patients at high risk of stoma re-closure [4].

Improved shunt designs and smart monitoring

Modern shunts use programmable valves that allow non-invasive pressure adjustments. Research prototypes now integrate pressure and flow sensors capable of wireless telemetry, enabling real-time monitoring of shunt performance. These "smart shunts" could allow earlier detection of malfunction, potentially reducing morbidity from delayed diagnosis. In addition, new biocompatible materials with anti-biofilm coatings aim to lower infection rates. However, these innovations remain evolutionary rather than revolutionary – the underlying dependence on mechanical diversion persists [4].

Choosing the right treatment based on etiology

Current reviews emphasize the importance of etiology-based treatment selection. For example, endoscopic methods are most effective for purely obstructive hydrocephalus (aqueductal stenosis, posterior fossa tumors). In post-hemorrhagic or post-infectious cases, success is far lower. Correct patient selection, therefore, remains the key determinant of outcome, as stressed in the paper of Jensen et al [39].

Molecular and cellular therapeutic perspectives

Growing knowledge of CSF physiology and brain interstitial fluid transport reveals that hydrocephalus involves complex cellular responses – not just altered fluid flow. This insight opens several new research directions.

Aquaporins and the glymphatic system

Water channels, especially Aquaporin-4 (AQP4) in astrocytic end-feet, play a crucial role in brain water homeostasis and glymphatic clearance. Numerous studies have shown dysregulation of AQP4 expression in hydrocephalus models. Typically, AQP4 upregulation occurs in periventricular astrocytes, possibly as a compensatory mechanism for disturbed CSF–interstitial fluid exchange. Experimental data suggest that AQP4 deficiency exacerbates ventricular dilation, while its overexpression may help buffer extracellular fluid accumulation. Thus, modulation of AQP4 or related pathways could become a pharmacological target. However, no clinically applicable agents exist yet [4].

Genetic and developmental mechanisms

Recent genomic studies have identified several genes associated with congenital hydrocephalus, such as L1CAM, MPDZ, and ATP1A3, which influence ependymal development, cell adhesion, and ion transport. Mutations in these genes disrupt ventricular wall integrity and CSF flow regulation. Understanding these pathways could allow gene-targeted or prenatal interventions in the future [4].

Glial and ependymal responses

Hydrocephalus induces profound reactive astrogliosis, microglial activation, and loss of ependymal cilia – all of which impair CSF circulation and brain development. Research on astrocyte and ependymal cell biology shows that their dysfunction contributes both to CSF flow obstruction and to white-matter damage. Restoring ependymal integrity, possibly through stem-cell-based therapies, could mitigate cognitive and neurological sequelae.

In short, hydrocephalus involves not just CSF flow imbalance but also cellular miscommunication between glia, ependyma, and vascular elements. Future treatment may combine surgical restoration of CSF pathways with pharmacological or molecular interventions targeting aquaporins, inflammation, and neurodevelopmental signaling. Yet as of 2025, all such therapies remain experimental [4].

Experimental and animal models – including the ovine study

Animal models are crucial for preclinical evaluation of both mechanical devices and biological therapies. The ovine (sheep) brain is particularly valuable due to its size and gyrencephalic structure, more similar to humans than rodent models [40].

Kaolin-induced hydrocephalus in adult sheep

One of the well-established models involves injecting kaolin into the cisterna magna, producing obstructive hydrocephalus. In a study of 42 adult sheep, 20 underwent shunt implantation. Observations revealed typical complications such as tissue obstruction, fibrosis, and infection – mirroring human shunt failures. The study validated the ovine model as realistic for testing novel shunt materials, flow sensors, or stents before clinical use [40].

Fetal ETV feasibility in an ovine model

A particularly innovative study demonstrated that fetal neuroendoscopic intervention is possible. Researchers induced hydrocephalus in ovine fetuses by injecting a polymeric glue (BioGlue) into the aqueduct at embryonic day 85 and performed fetal ETV at day 105. The study achieved technical success in 32 out of 50 procedures, representing a 64% success rate. When deliberately obstructed cases were excluded, the success rate increased to 80%. Importantly, there were no significant maternal or fetal deaths associated with the procedure, indicating that the intervention was both feasible and safe in the experimental setting. These results are highly experimental but suggest the possibility of prenatal hydrocephalus repair, potentially preventing irreversible brain damage before birth [40].

Interstitial or intraparenchymal stent concepts

The notion of interstitial stenting – implanting micro-stents within brain tissue to facilitate CSF diffusion – has so far been explored only in animal prototypes. The ovine brain provides a realistic platform for such testing, but these devices have not yet reached human trials. The concept remains promising, especially as an adjunct to ETV or to prevent periventricular edema by promoting interstitial drainage [40].

Implications from animal research

The key lessons from ovine studies are:

- The model replicates human complications such as fibrosis and blockage, making it suitable for device testing.
- Even technically successful drainage (ETV or shunt) does not fully prevent tissue injury – highlighting the need for biological co-therapies.
- Prenatal interventions may one day prevent the neurodevelopmental consequences of congenital hydrocephalus [40].

Disclosure

Author's contribution:

All authors contributed to the article.

All authors have read and agreed with the published version of the manuscript.

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