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# PAEDIATRIC HYDROCEPHALUS


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## INTRODUCTION

Pediatric hydrocephalus is a major neurological and neurosurgical problem in childhood, with its heaviest burden falling on low- and middle-income countries (LMICs). A global systematic review and meta-analysis found that the incidence of pediatric hydrocephalus is higher in LMICs than in high-income countries, at 123 versus 79 cases per 100,000 births, and estimated that nearly 400,000 new pediatric cases occur worldwide each year.[1] More recent review literature likewise indicates that over 80% of new pediatric hydrocephalus cases arise in LMICs, underlining the marked global inequality in disease burden.[2,3]

The importance of this condition is particularly evident in sub-Saharan Africa, where higher birth rates, a larger child population, and a greater contribution from preventable or treatable acquired causes all intensify the burden of disease.[1,3] In KwaZulu-Natal, South Africa, Enicker and Aldous documented 3,325 children treated for hydrocephalus across four study periods, with infants comprising 60.2% of cases, 51.4% of patients coming from rural areas, and post-infectious hydrocephalus emerging as the commonest etiological category.[3] Their findings show that pediatric hydrocephalus is not only common, but also closely linked to referral patterns, infectious disease exposure, and access to neurosurgical care.[3] This makes it especially important in settings where children may present late, travel long distances for specialist treatment, or reach care only after complications have developed.

Why hydrocephalus matters so much in pediatrics is not simply its frequency, but the developmental stage at which it occurs. The condition commonly affects infants and young children during periods of rapid brain growth, so delays in diagnosis and treatment may worsen both immediate and long-term outcomes. Recent review evidence shows that the burden of pediatric hydrocephalus is shaped not only by etiology, but also by socioeconomic conditions, access to emergent care, access to specialized treatment teams, and the availability of coordinated longitudinal follow-up. In this sense, pediatric hydrocephalus in resource-limited settings is both a disease of the nervous system and a marker of broader health-system inequality.[2,3]

At a basic clinical level, hydrocephalus describes an excessive accumulation of cerebrospinal fluid (CSF) within the head, most often because CSF circulation is obstructed or its absorption is impaired.[5] However, the significance of this disorder extends beyond ventricular enlargement alone. Enicker and Aldous emphasize that hydrocephalus produces a dual insult to the brain: first from the underlying causative pathology, and then from the additional effects of raised intracranial pressure, especially when treatment is delayed.[3] Understanding this interaction between disturbed CSF dynamics, intracranial pressure, and injury to the developing brain provides the foundation for the sections that follow on ventricular anatomy, CSF physiology, and pathophysiology.

## 1. DEFINITION OF HYDROCEPHALUS

Hydrocephalus is most simply understood as a congenital or acquired disorder in which cerebrospinal fluid (CSF) accumulates excessively within the head, usually because CSF circulation is obstructed or its absorption is impaired.[4,5] A more precise modern formulation describes hydrocephalus as a disorder of CSF homeostasis that leads to active expansion or distension of the cerebral ventricular system rather than

merely passive enlargement of the ventricles.[2,6,7] This disturbance is described as an imbalance between the production and absorption of CSF, resulting in pathological accumulation of CSF, ventricular dilatation, and often raised intracranial pressure. Taken together, these sources show that hydrocephalus is not simply “too much fluid in the brain”, but a dynamic disorder of CSF circulation, absorption, and pressure regulation that may injure the developing brain if untreated.

Current literature also emphasizes that hydrocephalus should be distinguished from ventriculomegaly alone. Rekate argues that the term should be reserved for active ventricular distension resulting from inadequate passage of CSF from its site of production to its site of absorption, because this definition better reflects the underlying pathophysiology and avoids confusion with other causes of enlarged ventricles.[6] Similarly, recent reviews note that pediatric hydrocephalus reflects failed CSF homeostasis with active ventricular expansion, whereas other forms of ventricular enlargement may arise secondarily from cerebral atrophy or other structural abnormalities rather than true hydrocephalic disease.[2,7] This distinction is clinically important because not every enlarged ventricle represents active hydrocephalus requiring diversion of CSF.

Hydrocephalus may be described in several complementary ways:

- First, it may be congenital or acquired. Congenital hydrocephalus is present before or at birth and is associated with developmental abnormalities such as aqueductal stenosis, Dandy–Walker malformation, Chiari malformations, and some genetic disorders, whereas acquired hydrocephalus develops after birth, often following infection, hemorrhage, trauma, or tumor-related obstruction.[2,4,7]

- Second, it may be communicating or non-communicating, depending on whether CSF still communicates freely between the ventricles and subarachnoid spaces.[4,6]

- Third, its onset may be acute or chronic, reflecting the speed with which CSF disturbance develops and the degree to which compensatory mechanisms have had time to operate.[4]

Related descriptive terms also help avoid diagnostic confusion.

- Hydrocephalus ex vacuo refers to compensatory enlargement of the ventricles secondary to loss or atrophy of surrounding brain tissue rather than an active CSF circulation disorder.[4,6]

- External hydrocephalus, often termed benign external hydrocephalus in infancy, refers to enlargement of the subarachnoid spaces, usually with macrocephaly and normal or only mildly enlarged ventricles.[4,8]

- Arrested hydrocephalus describes a chronic state in which the balance between CSF production and absorption has stabilized, so that ventricular enlargement persists without clear evidence of progressive raised intracranial pressure.[9]

These distinctions are especially important in pediatrics, where the differential diagnosis of head enlargement and ventriculomegaly must be approached carefully.[4,6,8,9]

## 2. IMPORTANCE IN PAEDIATRICS

Pediatric hydrocephalus is important not only because it is common, but because it affects the brain during a period of rapid growth, structural maturation, and developmental vulnerability. Large reviews describe childhood hydrocephalus as a major disorder of CSF homeostasis with consequences that extend beyond ventricular enlargement to include risks to cognitive, motor, visual, and overall neurodevelopmental function if diagnosis or treatment is delayed.[1,3,10] In global burden terms, hydrocephalus is disproportionately concentrated in low- and middle-income countries (LMICs), where higher rates of infection, neural tube defects, delayed referral, and limited neurosurgical access increase both incidence and disease severity.[1,2,7]

### Infant predominance and early-life vulnerability

The importance of hydrocephalus in pediatrics is especially clear in infancy. In the KwaZulu-Natal study by Enicker and Aldous, 3,325 children were treated for hydrocephalus, and infants younger than 1 year constituted 60.2% of all cases, making infancy by far the dominant age group.[3] The same study also showed that 51.4% of patients came from rural areas and 47.9% were referred from regional hospitals, indicating that pediatric hydrocephalus is not only a clinical problem but also a referral-system and access-to-care problem.[3] Broader literature supports this infant predominance: infants with hydrocephalus commonly present with progressive head enlargement, whereas older children are more likely to present with symptoms of intracranial hypertension such as headache, vomiting, and visual disturbance.[2,7]

### Why children are uniquely vulnerable

Children are uniquely vulnerable because the disease occurs in the setting of an immature and developing nervous system. Rath and Dash emphasize that pediatric neurosurgical patients differ fundamentally from adults because of their developing neurological and physiological status, and in infants the open fontanelle and cranial sutures create a more compliant intracranial compartment.[10] This means that increasing intracranial pressure may initially be partly compensated for by expansion of the skull, masking the mass effect of hydrocephalus and allowing pathology to become relatively advanced before clear intracranial hypertension is recognised.[10] They also note that infants and children have age-specific cerebral hemodynamic and metabolic features, which contribute to their vulnerability during neurosurgical illness and treatment.[10]

A further reason hydrocephalus is so important in children is that the underlying causes differ across age groups and regions. In many LMIC settings, particularly across sub-Saharan Africa, post-infectious hydrocephalus remains a major contributor, whereas in higher-income settings congenital malformations, hemorrhage of prematurity, and tumor-related obstruction are relatively more prominent.[1-3,7] This geographic variation matters because it reflects preventable causes, unequal health-system capacity, and differing long-term outcomes.

### Clinical and health-system significance

From a pediatric perspective, hydrocephalus has implications that go beyond the initial diagnosis. Children with symptomatic hydrocephalus usually require definitive surgical treatment and long-term follow-up, including surveillance for shunt-related complications and developmental sequelae; repeated revision procedures are common in a substantial proportion of shunted patients. Enicker and Aldous reported that ventriculoperitoneal shunts were the mainstay treatment in KwaZulu-Natal, with a complication rate of 20.4% and mortality of 7.9%, highlighting the continuing burden of this disease on children, caregivers, and health services.[3] Recent reviews similarly stress that pediatric hydrocephalus is a chronic condition for many patients, with outcome influenced by etiology, timing of treatment, access to specialist care, and the quality of long-term follow-up.[11-15]

Key reasons are especially important in pediatrics:

- It commonly presents in infancy, when the brain is rapidly developing.
- Open sutures and fontanelles may delay obvious signs of intracranial hypertension, so disease may already be advanced at presentation.
- In LMICs, the burden is amplified by infection, referral delays, and limited neurosurgical access.[2,3,10]
- The condition often requires long-term follow-up, repeated procedures, and neurodevelopmental surveillance.

Overall, pediatric hydrocephalus is important because it combines biological vulnerability, developmental risk, and health-system inequality. Its prominence in infancy, together with the potential for delayed recognition due to skull compliance, make early diagnosis and timely management particularly critical in children.

## 3. BASIC VENTRICULAR ANATOMY AND NORMAL CSF PHYSIOLOGY

### Basic ventricular anatomy

The ventricular system is a continuous series of interconnected, fluid-filled spaces within the brain and brainstem. In simple terms, it consists of the two lateral ventricles, the third ventricle, the cerebral aqueduct (aqueduct of Sylvius), and the fourth ventricle.[16] The lateral ventricles are the largest cavities and lie within the cerebral hemispheres. They communicate with the midline third ventricle through the interventricular foramina of Monro.[16]

The third ventricle then continues caudally as the cerebral aqueduct, which traverses the midbrain and opens into the fourth ventricle, situated dorsal to the pons and medulla.[16] From the fourth ventricle, CSF leaves the ventricular system through the median aperture of Magendie and the paired lateral apertures of Luschka to enter the basal cisterns and the cranial and spinal subarachnoid spaces.[4,17-19]

The anatomy is particularly important in pediatric hydrocephalus because obstruction at different points produces different ventricular enlargement patterns. For example, aqueductal obstruction classically enlarges the lateral and third ventricles while the

fourth ventricle remains relatively normal in size, a point highlighted in the Mshayisa teaching document and consistent with standard hydrocephalus anatomy.[19]

### Key anatomical landmarks

- *Lateral ventricles:* largest CSF spaces, one in each cerebral hemisphere.[19]
- *Foramina of Monro:* narrow channels linking each lateral ventricle to the third ventricle.[18-19]
- *Third ventricle:* midline cavity between the thalami.[18-19]
- *Cerebral aqueduct:* the narrowest major ventricular channel and a common site of obstruction in hydrocephalus.[1,2,19]
- *Fourth ventricle:* communicates with the subarachnoid space through Magendie and Luschka.[1,18-19]

### Normal CSF physiology

Cerebrospinal fluid is more than a passive cushioning liquid. It contributes to homeostasis of the brain extracellular environment, helps maintain normal neuronal function, provides buoyancy, transports micronutrients, and assists in the clearance of metabolic waste products.[4,17-18] Krovvidi et al. emphasize both its biochemical and physical functions, including its role as a mediator of intracranial compliance because CSF moves freely between the cranial and spinal compartments.[4]

### CSF production

Most CSF is produced by the choroid plexus, with a smaller contribution from brain interstitial fluid and periventricular tissues.[4,17-18] Krovvidi et al. state that most CSF is produced in the choroid plexus and that a small proportion is formed around cerebral vessels from brain interstitial fluid.[4] Contemporary physiology reviews agree that choroid plexus secretion is dominant, while also recognizing additional extra-choroidal contributions.[18-19]

In adults, total CSF volume is approximately 150 mL, with roughly 25 mL within the ventricles and the remainder in cranial and spinal subarachnoid spaces.[4,18-19] Chakravarthi highlights the pediatric scale difference by listing an approximate newborn CSF volume of 5 mL and a formation rate of about 25 mL/day, whereas adult production is given as about 0.3–0.35 mL/min or 500–600 mL/day.[20] This developmental contrast is important in pediatrics because even modest disturbances in production, flow, or absorption can have major effects in a small intracranial volume.[2,20]

### CSF circulation and pulsatile flow

Under normal conditions, CSF does not move as a constant stream. Krovvidi et al. note that CSF flow throughout the neuraxis, including across the foramen magnum, is pulsatile and linked mainly to the cardiac cycle.[4] Recent pediatric and physiology reviews similarly describe CSF circulation as being influenced by ongoing secretion, arterial pulsations, respiratory forces, and pressure gradients.[17-19] In the developing

brain, Kahle et al. add that CSF dynamics are only partially mature at term gestation, that the choroid plexus is functional by about 22–23 weeks of gestation, and that ependymal maturation continues after birth.[2] This developmental immaturity helps explain why CSF disorders in infancy can have distinctive clinical and structural consequences.[17,20]

**CSF** absorption

The classical major route of CSF absorption is through arachnoid villi and arachnoid granulations into the dural venous sinuses. Krovvidi et al. also identify a secondary outflow route alongside cranial and spinal nerve roots, particularly along the olfactory nerves through the cribriform plate.[4] Recent reviews support the view that CSF drainage is not limited to arachnoid granulations alone and includes substantial lymphatic and nerve sheath pathways.[2,17-18]

**Table 2. Selected normal CSF facts relevant to pediatrics**

| Parameter | Pediatric/developmental note | Adult reference point |
|---|---|---|
| Main production site | Predominantly choroid plexus; the neonatal system is still developing | Predominantly choroid plexus |
| Total CSF volume | Newborn approximately 5 mL in Mshayisa teaching notes | Approximately 150 mL |
| Production rate | Newborn approximately 25 mL/day in Mshayisa notes | About 0.3–0.35 mL/min or 500–600 mL/day |
| Flow pattern | Pulsatile; influenced by cardiac cycle and respiration | Pulsatile; not a steady stream |
| Main absorption route | Arachnoid villi/granulations, with secondary lymphatic/perineural routes | Same general pattern |

Source: 4,17-18

## 4. EPIDEMIOLOGY

### Global burden

The epidemiology of pediatric hydrocephalus differs substantially by geography and national income level. In the most widely cited global meta-analysis, Dewan et al. estimated that the incidence of congenital hydrocephalus was higher in low- and middle-income countries (LMICs) than in high-income countries, at 123 versus 79 cases per 100,000 births, and that nearly 400,000 new pediatric cases occur worldwide each year.[1] The same analysis found that the highest congenital incidence rates were reported in Africa and Latin America, whereas the lowest rates were seen in North America.[1] Krovvidi et al. similarly cite a prevalence estimate of 0.5–0.8 per 1,000 live births for congenital and pediatric hydrocephalus in the USA and Europe, while noting that rates are likely to be higher in resource-poor settings with reduced access to care.[4]

## Burden in Africa and other LMIC settings

The African burden is particularly important because pediatric hydrocephalus is shaped not only by birth prevalence but also by acquired causes, especially infection. The study by Kahle et al. states that more than 80% of new pediatric hydrocephalus cases occur in LMICs and emphasizes that the epidemiology of the condition varies by geography, season for post-infectious disease, and income level of the country.[2]

In a systematic review and meta-analysis of African studies, Aukrust et al. included 38 studies from 18 countries and found that post-infectious hydrocephalus was the single most common cause across the continent, with a pooled proportion of 28%, compared with 21% for non-post-infectious hydrocephalus and 16% for hydrocephalus associated with spinal dysraphism.[21]

A more recent African meta-analysis by Darko et al., covering 12,355 patients, also showed that post-infectious hydrocephalus featured prominently and that the mean age of affected children was 12.3 months, reinforcing the strong infant and early-childhood burden seen across the region.[22]

## KwaZulu-Natal pattern

Within South Africa, Enicker and Aldous provide the strongest regional epidemiological data from your source set.[3] In their KwaZulu-Natal study, 3,325 children were treated for hydrocephalus, with infants constituting 60.2% of the cohort and a median age of 7 months.[3] Slightly more than half of the children came from rural areas (51.4%), and almost half were referred from regional hospitals (47.9%), showing that the burden is concentrated not only in early childhood but also among patients dependent on regional referral pathways.[3] Post-infectious hydrocephalus was the predominant etiological category (32.7%), and among infectious causes, tuberculous meningitis accounted for 54.1%, illustrating how local infectious disease patterns shape the epidemiology of pediatric hydrocephalus in KwaZulu-Natal.[3]

Key epidemiological points:

- Pediatric hydrocephalus is disproportionately concentrated in LMICs, where case volume is increased by higher birth rates, neural tube defects, infection-related disease, and reduced access to specialist care.[1-2,21]

- In Africa, post-infectious hydrocephalus remains a major driver of disease burden, unlike many high-income settings where congenital malformations, hemorrhage of prematurity, and tumor-related obstruction are relatively more prominent.[2,21-22]

- In KwaZulu-Natal, the epidemiological profile is characterized by infant predominance, rural origin, regional referrals, and a strong infectious component, particularly tuberculous meningitis.[3]

Overall, the epidemiology of pediatric hydrocephalus is not uniform. It changes according to geography, infectious burden, health-system capacity, and referral pathways, which is why local data such as those from KwaZulu-Natal are essential for planning prevention, referral, diagnostic, and treatment services.

## 5. PATHOPHYSIOLOGY

### Basic mechanism

Paediatric hydrocephalus develops when cerebrospinal fluid (CSF) homeostasis fails and the ventricular system expands actively rather than remaining in physiological balance. In practical terms, this usually reflects one of three mechanisms: obstruction to CSF flow, impaired CSF absorption, or, much less commonly, CSF overproduction.

Krovvidi et al. describe hydrocephalus as excess CSF accumulation caused mainly by obstructed circulation or impaired absorption,[4] Rekate explain the process as an active distension of the ventricular system caused by inadequate passage of cerebrospinal fluid from its site of production to its site of absorption, resulting in ventricular enlargement.[6] A recent pediatric review by Kahle et al. uses similar language, defining hydrocephalus as a failure of CSF homeostasis that results in active expansion of the cerebral ventricles.[2]

Obstruction is the commonest pathophysiological pathway. When a blockage occurs anywhere along the ventricular system, CSF accumulates proximal to the obstruction and the upstream ventricles enlarge.[23] Impaired absorption produces a similar end result, but the defect lies mainly at the level of CSF resorption, classically the arachnoid villi and associated venous pathways.[24] Overproduction is uncommon and is most typically associated with choroid plexus papilloma. Krovvidi et al. likewise note that communicating hydrocephalus is usually linked to impaired reabsorption, whereas non-communicating hydrocephalus arises when CSF flow is blocked within the ventricular system.[4]

A useful principle for understanding ventricular enlargement is the Law of Laplace, highlighted by Krovvidi et al.[4] According to this concept, the pressure required to maintain an already enlarged ventricle is lower than the pressure needed to initiate its expansion.[4] This helps explain why markedly enlarged ventricles may persist even when the measured CSF pressure is no longer dramatically elevated. In other words, high pressure may be required early to enlarge the ventricles, but once the cerebral mantle has been stretched and thinned, large ventricles can remain under relatively lower pressure. This is one reason why ventricular size and intracranial pressure do not always correlate perfectly, especially in chronic hydrocephalus.[4]

Another important point is that hydrocephalus is not merely a “plumbing problem”. Contemporary reviews emphasize that it can also reflect abnormal neurodevelopment, ependymal dysfunction, ciliary dysfunction, disturbed brain–CSF biomechanics, inflammation, and altered clearance pathways. Kahle et al. note that congenital hydrocephalus may arise from gene mutations that disrupt brain morphogenesis and alter the biomechanics of the CSF–brain interface, whereas acquired hydrocephalus is often linked to infection or hemorrhage with inflammation-dependent dysregulation of CSF secretion and clearance.[2] A molecular review likewise argues that hydrocephalus is more complex than a simple circulatory disorder and involves ependymal-cell dysfunction, inflammation, apoptosis, blood-flow abnormalities, metabolic changes, and, in some forms, glymphatic dysfunction.[25]

**Core mechanisms at a glance**

- Obstruction to CSF flow → ventricular enlargement proximal to the block.
- Impaired CSF absorption → progressive CSF accumulation, often in communicating hydrocephalus.
- Rare overproduction → usually related to choroid plexus tumours.
- Raised intracranial pressure and ventricular expansion → compression of surrounding brain tissue.
- Chronic enlargement may persist at lower pressure because of ventricular wall mechanics and the Law of Laplace.

**Table 1. Main pathophysiological mechanisms in hydrocephalus**

| Mechanism | What happens | Typical result |
|---|---|---|
| Obstruction | CSF cannot pass normally through the ventricular or subarachnoid pathways | Upstream ventricular dilatation |
| Impaired absorption | CSF resorption through arachnoid/venous pathways is reduced | Progressive CSF accumulation, often communicating hydrocephalus |
| Overproduction | Excess CSF formation, usually tumor-related | Enlargement of the whole ventricular system |
| Altered ventricular mechanics | Enlarged ventricles require less pressure to remain enlarged | Chronic ventriculomegaly despite less marked pressure elevation |

*Source: 2, 4, 25*

**Brain effects**

The neurological damage caused by hydrocephalus is multifactorial. Del Bigio explains that hydrocephalus-related brain injury results from a combination of mechanical distortion, ischemic compromise, and metabolic-toxic disturbance, with the severity depending on the rate and magnitude of ventricular dilatation and the developmental stage at which it occurs.[26] These changes include compression of the cortical mantle, stretching of periventricular white matter, cerebral ischemia, periventricular oedema, and barrier-related injury.[26] This framework is also supported by an empirical study on congenital and neonatal hydrocephalus.[27]

One of the earliest and most characteristic tissue responses is periventricular white matter injury. As the ventricles enlarge, the surrounding white matter is compressed and stretched, disrupting axonal organization and reducing perfusion in vulnerable periventricular regions.[28] Trans ependymal passage of CSF can contribute to periventricular oedema, especially in acute hydrocephalus. Older neuropathological

work showed that periventricular CSF oedema in acute hydrocephalus often reflects movement of fluid through the ependyma into adjacent white matter,[29] while more recent experimental studies have linked ventriculomegaly with oligodendrocyte loss, cell death, and white-matter inflammation.[30,31] This is clinically important because white matter supports motor, cognitive, and inter-regional brain connectivity, all of which may be affected in children with prolonged hydrocephalus.[27]

Secondary injury develops as hydrocephalus persists. The Mshayisa document lists neuronal cell loss, gliosis, demyelination, altered metabolism, impaired connectivity, and choroid plexus structural change. These points are consistent with the wider literature. McAllister notes major roles for gliosis and neuroinflammation in congenital and neonatal hydrocephalus,[27] while Del Bigio highlights impaired developmental processes, including myelin production.[26] A 2022 experimental study demonstrated that acquired hydrocephalus was associated with neuroinflammation, increased astrocytes and microglia, reduced oligodendrocytes, and reduced proliferative activity in the subventricular zone.[30] Together, these findings suggest that hydrocephalus injures not only mature tissue but also the developing cellular systems responsible for myelination, repair, and normal brain growth.

There is also growing evidence that ependymal and blood–brain barrier-related abnormalities contribute to pathophysiology. McAllister describes ependymal denudation and altered development of neural progenitors exposed to CSF,[27] while Guerra et al. discuss blood–brain barrier abnormalities in fetal-onset hydrocephalus and argue that shunting may limit further damage without correcting the underlying brain maldevelopment.[32] Kahle et al. further notes that ependymal cells mature through late gestation and infancy, and that developmental disturbance of the CSF–brain interface may be central in congenital hydrocephalus.[2] This is especially relevant in pediatrics because the immature brain is not only structurally smaller but biologically still under construction.

**Main tissue effects of hydrocephalus**

- Compression and stretch of the cortical mantle and periventricular white matter
- Reduced cerebral blood flow and microvascular injury with ischemic contribution.
- Periventricular oedema from trans ependymal CSF movement, particularly in acute hydrocephalus.
- Gliosis and neuroinflammation involving astrocytes and microglia.
- Demyelination and axonal injury, especially in periventricular white matter.
- Altered metabolism and impaired connectivity, contributing to long-term neurodevelopmental deficits.

**Table 2. From ventricular enlargement to brain injury**

| Stage | Pathophysiological event | Possible consequence |
|---|---|---|

| | | |
|---|---|---|
| Early | CSF accumulates because of obstruction, poor absorption, or overproduction | Ventriculomegaly and raised intracranial pressure |
| Mechanical phase | Ventricles expand and stretch surrounding tissue | Cortical thinning and periventricular white-matter distortion |
| Vascular phase | Reduced perfusion and microvascular compromise | Ischemic injury |
| Fluid-shift phase | Trans ependymal CSF movement into white matter | Periventricular oedema |
| Cellular phase | Gliosis, inflammation, oligodendrocyte loss, neuronal injury | Demyelination, impaired connectivity, developmental delay |

*Source: 2, 26, 27, 30*

## 6. CLASSIFICATION

Hydrocephalus can be classified in several complementary ways, but the most clinically useful starting point is the distinction between communicating and non-communicating hydrocephalus.

Krovvidi et al. note that hydrocephalus may be categorised into different types, with the communicating/non-communicating distinction remaining especially important because it reflects the site of CSF flow disturbance and has practical implications for diagnosis and drainage strategy.[4]

Rekate also points out that although many classification systems exist, a workable classification remains essential for clear communication in both clinical practice and research.[6]

### Communicating versus non-communicating hydrocephalus

In communicating hydrocephalus, CSF pressure waves and fluid can still pass from the ventricular system into the basal cisterns and spinal CSF spaces, but absorption is impaired, most commonly at the level of the arachnoid granulations or related venous outflow pathways. Krovvidi et al. define it as a state in which there is free flow and transmission of CSF pressure waves from the ventricles into the basal cisterns and spinal CSF channels, with impaired reabsorption being the usual problem.[4]. Agarwal et al. similarly state that the blockage in communicating hydrocephalus is effectively at the level of the arachnoid granulations, such as after intraventricular or subarachnoid hemorrhage, meningitis, or rarely with CSF overproduction from a choroid plexus papilloma.[33]

By contrast, non-communicating hydrocephalus, also called obstructive hydrocephalus, occurs when CSF flow is blocked within the ventricular system itself.[4] Krovvidi et al. state that this form develops when CSF flow is obstructed at any point within the ventricular pathway, for example by aqueductal stenosis, tumour, haemorrhage, or focal mass lesions.[4] A 2020 study gives the same principle, describing non-communicating hydrocephalus as a condition in which the ventricular system no longer communicates effectively with the site of CSF absorption because of obstruction somewhere along the normal CSF pathways.[34]

### Congenital versus acquired hydrocephalus

A second broad classification is congenital versus acquired hydrocephalus.

- Congenital hydrocephalus is defined as hydrocephalus caused by processes present before birth, including aqueductal stenosis, Dandy–Walker malformation, X-linked hydrocephalus, Chiari malformations, myelomeningocele, encephalocele, and prenatal infections such as CMV.[7,35]

- Acquired hydrocephalus, in contrast, develops after birth and includes hydrocephalus secondary to tumors, infections, intraventricular hemorrhage, subarachnoid hemorrhage, and head injury.[36]

Kahle et al. support this broad division, noting that congenital hydrocephalus is typically present at or near birth and may be linked to malformation, genetics, or in utero insult, whereas acquired hydrocephalus develops after birth and is often caused by CNS infection, hemorrhage, or tumour.[2] Rath and Dash also use this congenital/acquired distinction in their pediatric overview.[10]

### Acute versus chronic hydrocephalus

Hydrocephalus may also be described according to time course. Krovvidi et al. explain that acute-onset hydrocephalus often presents with symptoms of raised intracranial pressure and may require urgent intervention, whereas chronic forms usually involve more established ventricular enlargement and a slower evolution of symptoms.[4] Orešković et al. likewise state that hydrocephalus may be acute or chronic, depending on how rapidly the CSF imbalance develops and whether pressure has remained persistently elevated or later stabilised.[37]

### Related distinctions

Two related terms should be interpreted carefully.

- External hydrocephalus refers to abnormal CSF accumulation in the subarachnoid spaces over the cerebral hemispheres, often with normal or only mildly enlarged ventricles.
- Hydrocephalus ex vacuo refers to compensatory ventricular enlargement caused by loss or atrophy of surrounding brain tissue rather than by an active CSF circulation disorder.

Krovvidi et al.[4] make this distinction clearly, and Rekate[6] specifically argues that ex vacuo ventriculomegaly should be excluded from the core definition of hydrocephalus because it does not represent active ventricular distension from impaired CSF passage.

**Table 3. Practical classification of hydrocephalus**

| Classification axis | Category | Main idea |
|---|---|---|
| By CSF pathway | Communicating | CSF still communicates with subarachnoid spaces, but absorption is impaired |

| | | |
|---|---|---|
| By CSF pathway | Non-communicating | CSF flow is blocked within the ventricular system |
| By timing of origin | Congenital | Present before or at birth; often malformative, genetic, or prenatal |
| By timing of origin | Acquired | Develops after birth; often infection, hemorrhage, trauma, or tumor related |
| By time course | Acute | Rapid onset, often with raised ICP and urgent presentation |
| By time course | Chronic | Slower evolution, often with longstanding ventriculomegaly |

**Key points**

- Communicating hydrocephalus usually reflects impaired CSF absorption.
- Non-communicating hydrocephalus reflects obstruction within the ventricular system.
- Congenital and acquired are broad clinical categories based on whether the underlying cause existed before birth or developed later.
- Acute and chronic describe time course rather than cause.
- External hydrocephalus and hydrocephalus ex vacuo are useful related distinctions, but ex vacuo ventriculomegaly is not the same as active hydrocephalus.

## 7. CAUSES OF PAEDIATRIC HYDROCEPHALUS

The causes of pediatric hydrocephalus are best understood within the broad categories of congenital and acquired disease, although these groups can overlap in practice. Contemporary reviews describe the main causes as congenital malformations, neural tube defects, central nervous system haemorrhage, infection, and tumours.[2,7,10]

Overall, cause distribution varies markedly by geography:

- Congenital malformations and prematurity-related hemorrhage are prominent in high-income settings, whereas
- Post-infectious hydrocephalus remains a major driver in many African and other low- and middle-income settings.

### CONGENITAL CAUSES

Congenital hydrocephalus is typically present before or at birth and is associated with brain malformation, genetic disease, or an in utero insult such as infection or haemorrhage.[2,7] In practical pediatric neurosurgery, the most important congenital causes include aqueductal stenosis, myelomeningocele with Chiari II malformation, Dandy–Walker malformation, X-linked hydrocephalus, encephalocele, arachnoid cysts, and vascular malformations such as vein of Galen malformation.[2,7,10]

- **Aqueductal stenosis**

Aqueductal stenosis is one of the most important congenital causes of obstructive hydrocephalus in childhood. It results from narrowing or occlusion of the aqueduct of Sylvius, which prevents normal passage of CSF from the third to the fourth ventricle and causes enlargement of the lateral and third ventricle.[2,10] Cinalli et al.[38] and Giuseppe and D'armiento[39] describe aqueductal stenosis as the most common cause of hydrocephalus in children and note that it may be congenital or acquired, with congenital forms sometimes associated with Chiari malformation, vein of Galen malformation, or Dandy–Walker malformation. Modern reviews likewise identify aqueductal stenosis as the commonest congenital form of hydrocephalus and link it to abnormal brain growth, development, and, in some cases, known genetic mechanisms.[2,7]

- **Myelomeningocele and Chiari II malformation**

Hydrocephalus is strongly associated with myelomeningocele, usually in the context of Chiari II malformation. Myelomeningocele is a neural tube defect caused by failure of neural tube closure, and Chiari II malformation is present in almost all infants with myelomeningocele, although not all develop active hydrocephalus.[40] Rath and Dash also identify aqueductal stenosis, Arnold–Chiari malformation, and Dandy–Walker syndrome among the classic congenital causes of hydrocephalus in pediatric practice.[10] Mechanistically, hydrocephalus in myelomeningocele is thought to arise from crowding of the posterior fossa, fourth ventricular outlet obstruction, aqueductal abnormalities, venous hypertension, and impaired CSF flow at the craniocervical junction.[10]

- **Dandy–Walker malformation**

Dandy–Walker malformation is a posterior fossa developmental anomaly characterized by cystic dilatation of the fourth ventricle, vermian hypoplasia or dysgenesis, and enlargement of the posterior fossa.[41] In the Mshayisa document, hydrocephalus in Dandy–Walker malformation is described as multifactorial, related to aqueductal stenosis, abnormal subarachnoid space development, basal arachnoiditis, and venous hypertension from the posterior fossa cyst.[41] Recent literature likewise recognizes posterior fossa malformations as major congenital causes of pediatric hydrocephalus.[2,7]

- **X-linked hydrocephalus**

X-linked hydrocephalus is a rare inherited form of congenital hydrocephalus, classically associated with pathogenic variants in L1CAM and often linked to aqueductal stenosis.[41] The Mshayisa notes refer to Bickers–Adams syndrome as a recessively transmitted disorder carried by females and inherited by males.[41] More recent genetic reviews support L1CAM-related disease as the most common inherited genetic cause of congenital hydrocephalus and describe it as a syndromic form often associated with aqueductal stenosis and additional developmental abnormalities.[2]

- **Encephalocele, arachnoid cyst, and vein of Galen malformation**

Other congenital structural lesions can also lead to hydrocephalus. Encephalocele may distort normal CSF pathways or include herniated ventricular structures within the sac.[10,41] Arachnoid cysts can produce hydrocephalus when their expansion obstructs

CSF circulation, especially when they are located near key ventricular outflow pathways41. Vein of Galen malformation can cause hydrocephalus by direct aqueductal compression or, importantly, by venous hypertension that secondarily impairs CSF absorption.[41,42] Paramasivam et al. emphasize that hydrocephalus in vein of Galen malformation is often a hydrodynamic disorder related to high-flow vascular shunting and poor venous drainage reserve in early life.[42]

**Key congenital causes at a glance:**

- Aqueductal stenosis — classic obstructive cause.
- Myelomeningocele with Chiari II malformation — neural tube defect-associated hydrocephalus.
- Dandy–Walker malformation — posterior fossa developmental anomaly.
- X-linked hydrocephalus — inherited, often L1CAM-related.
- Encephalocele, arachnoid cyst, vein of Galen malformation — less common but important structural causes.

## ACQUIRED CAUSES

Acquired hydrocephalus develops after birth and is mainly caused by infection, hemorrhage, tumors, or trauma.[2,7,10] The Mshayisa document provides a detailed practical list of these causes, while modern reviews emphasize that acquired hydrocephalus is particularly shaped by local neonatal care, infection burden, and access to early diagnosis.[2,21,41]

- **Infections**

Infections are among the most important causes of acquired hydrocephalus in children. The Mshayisa notes state that bacterial, fungal, viral, and parasitic CNS infections can all lead to hydrocephalus by causing ventriculitis, blockage of CSF pathways, or impaired absorption within the subarachnoid spaces.[41] Tuberculous meningitis is particularly important because it can produce hydrocephalus either by basal cisternal inflammation causing communicating hydrocephalus or by tuberculoma-related mass effect.[41] Contemporary African evidence supports this emphasis: a large systematic review and meta-analysis found that post-infectious hydrocephalus was the single most common cause of pediatric hydrocephalus across Africa.[21] The study by Kahle et al. also notes that acquired hydrocephalus in children is frequently associated with infection and inflammation-dependent dysregulation of CSF secretion and clearance.[2]

- **Intraventricular hemorrhage of prematurity**

Post-hemorrhagic hydrocephalus of prematurity is a major acquired cause, especially in preterm and very-low-birthweight infants. The Mshayisa document describes germinal matrix hemorrhage as the commonest cause of hydrocephalus in the premature infant and notes that the risk of post-hemorrhagic hydrocephalus rises with the severity of the haemorrhage.[41] Rath and Dash likewise list intraventricular hemorrhage of prematurity among the major acquired causes of pediatric hydrocephalus.[10] Recent reviews explain that post-hemorrhagic hydrocephalus of

prematurity usually follows severe germinal matrix/intraventricular hemorrhage, with blood products, inflammation, ependymal injury, and impaired CSF circulation or absorption all contributing to progressive ventriculomegaly.[2,43]

- **CNS tumors**

CNS tumors are another important acquired cause, usually through obstruction of the ventricular system. The Mshayisa notes explain that hydrocephalus may be present at diagnosis of the tumor, may occur after treatment, or may develop later with tumor recurrence.[41] Posterior fossa tumors are especially important because of their proximity to the fourth ventricle and aqueduct, while suprasellar, pineal, intraventricular, and brainstem tumors can also interfere with CSF pathways.[41] Rath and Dash note that posterior fossa space-occupying lesions are classic acquired causes of hydrocephalus in children.[10] Rarely, tumors such as choroid plexus papilloma or carcinoma may cause hydrocephalus through CSF overproduction rather than obstruction alone.[7,41]

- **Head trauma**

Head injury can also result in hydrocephalus, particularly when it is complicated by intraventricular or subarachnoid hemorrhage. The Mshayisa document explains that blood breakdown products may render the arachnoid villi unable to reabsorb CSF adequately, while debris and clot may also obstruct normal CSF pathways.[41] Krovvidi et al. make a similar point in their classification discussion, noting that blood shed into CSF after trauma or hemorrhage can block arachnoid granulations and impair CSF reabsorption.[4] Although traumatic hydrocephalus is less dominant than infection or prematurity-related hemorrhage in most pediatric series, it remains an important acquired cause to recognise.[4,41]

**Table 4. Major acquired causes of paediatric hydrocephalus**

| Cause | Main mechanism | Typical pediatric context |
|---|---|---|
| Infection | Ventriculitis, arachnoiditis, basal cistern scarring, impaired absorption | Bacterial meningitis, tuberculous meningitis, neonatal sepsis-related CNS infection |
| Intraventricular hemorrhage of prematurity | Blood products, inflammation, impaired CSF circulation/absorption | Preterm infants, especially severe germinal matrix hemorrhage |
| CNS tumors | Obstruction of aqueduct, fourth ventricle, foramina, or rarely CSF overproduction | Posterior fossa, pineal, suprasellar, intraventricular tumors |
| Head trauma | SAH/IVH, clot debris, arachnoid granulation dysfunction | Severe traumatic brain injury |

*Sources: 2, 4, 10, 43*

## 8. CLINICAL FEATURES

The clinical features of pediatric hydrocephalus vary with age, speed of onset, degree of ventricular enlargement, and the severity of intracranial pressure elevation.[4,10,41] Krovvidi et al. emphasize that acute-onset hydrocephalus usually presents with symptoms of raised intracranial pressure, whereas chronic hydrocephalus may evolve more gradually and, particularly in infants, may present with progressive head enlargement rather than dramatic acute neurological collapse.[4] Mshayisa similarly notes that signs and symptoms differ according to age, the degree of hydrocephalus at presentation, the underlying etiology, and the duration of the condition.[41]

### Clinical features in infants

In infants, the presenting picture is strongly influenced by the fact that the cranial sutures and fontanelles are still open. Rath and Dash explain that in infants, open fontanelles and sutures create a more compliant intracranial compartment, so the mass effect of intracranial pathology may initially be masked by skull expansion [2]. For this reason, infants often present with macrocephaly or rapidly increasing head circumference rather than with the full classical picture of acute intracranial hypertension seen in older children [1,2].

According to the Mshayisa teaching document, the common features in full-term infants include macrocephaly, rapid head growth, tense fontanelle, distended scalp veins, splayed cranial sutures, vomiting, poor feeding, increased drowsiness, poor head control, sun-setting eyes, and frontal bossing.[41] Krovvidi et al. add that in neonates the clinical signs include vomiting, irritability, drowsiness, downward gaze, bulging fontanelles, and increased head circumference, and that apnoeic episodes may be an important sign of raised intracranial pressure in this age group.[4] Rath and Dash likewise state that infants with intracranial hypertension may present with irritability, lethargy, decreased consciousness, failure to feed, bulging fontanelle, and cranial enlargement.[10]

Premature infants may present somewhat differently. Mshayisa lists apnoea, bradycardia, hypotonia, acidosis, seizures, rapid head growth, tense fontanelle, vomiting, and sun-setting eyes among the key signs in premature infants.[41] This reflects both the vulnerability of the preterm brain and the common association between prematurity, intraventricular hemorrhage, and post-hemorrhagic hydrocephalus.

### Common infant features

- Macrocephaly or rapidly increasing head circumference
- Bulging or tense anterior fontanelle
- Distended scalp veins and splayed sutures
- Poor feeding, vomiting, irritability, lethargy, or drowsiness
- Sun-setting eyes / downward gaze
- Apnoea as a concerning sign of raised intracranial pressure in neonates

A useful clinical point is that a normal head circumference at birth is approximately 33–36 cm in a full-term infant, and progressive deviation from the expected rate of

head growth should raise suspicion of hydrocephalus.[41] Mshayisa notes that head circumference normally increases by about 2 cm per month during the first 3 months, 1.5 cm per month in the fourth and fifth months, and approximately 0.5 cm per month from 6 to 12 months.[41] Thus, serial head circumference measurement is not merely descriptive but forms part of early clinical detection.

**Older children and signs of raised intracranial pressure**

In older children, the clinical picture shifts because the skull is no longer able to expand significantly. As a result, features of raised intracranial pressure (ICP) become more prominent. Rath and Dash state that children may present with early morning headache, vomiting without nausea, diplopia, and papilledema, and, in late stages, with Cushing's triad.[10] Krovvidi et al. likewise note that acute hydrocephalus usually presents with symptoms of raised ICP, and that clinical presentation depends on the speed of onset and age of the patient.[4]

The Mshayisa document expands this older-child profile to include headache, nausea, vomiting, irritability, lethargy, delayed development, decreased school performance, behavioral disturbance, papilledema, Parinaud's sign, sun-setting eyes, bradycardia, hypertension, and irregular breathing patterns.[41] These features reflect both the direct effects of raised pressure and the impact of long-standing ventricular enlargement on cognition, vision, and behavior. Parinaud's sign, or impaired upward gaze, may occur especially when the dorsal midbrain is affected, for example in obstructive hydrocephalus involving the aqueduct or posterior third ventricle.[4,41]

Morning headache and vomiting are particularly important red flags. They reflect the tendency for intracranial pressure to rise when the child is recumbent overnight and to manifest most clearly on waking.[10] Diplopia usually reflects sixth nerve palsy from raised ICP, while papilledema indicates sustained optic disc swelling from transmitted intracranial pressure.[10] Behavioral change, poor concentration, and declining school performance may signal more chronic hydrocephalus and should not be dismissed, especially when accompanied by headache or visual complaints.[41]

At the severe end of the spectrum, both Rath and Dash and Mshayisa identify Cushing's triad as a late and dangerous sign, consisting of bradycardia, systemic hypertension, and irregular breathing.[10,41] Its presence suggests markedly raised intracranial pressure and possible impending herniation, making it a neurosurgical emergency.[10,41]

**Table 5. Age-grouped clinical features of paediatric hydrocephalus**

| Feature group | Infants | Older children |
|---|---|---|
| Head size | Macrocephaly, rapid head growth | Usually absent because sutures are closed |
| Skull findings | Bulging fontanelle, splayed sutures, distended scalp veins | Not typical |
| Feeding / GI | Poor feeding, vomiting | Morning vomiting, often without nausea |
| Consciousness / behavior | Irritability, lethargy, drowsiness | Irritability, behavioral disturbance, poor school performance |

| | | |
|---|---|---|
| Eye signs | Sun-setting eyes, downward gaze | Diplopia, papilledema, Parinaud's sign |
| Severe ICP signs | Apnoea, decreased consciousness | Cushing's triad: bradycardia, hypertension, irregular breathing |

*Sources: 4,10,41*

### Key clinical take-home points

- In infants, the dominant clues are often head enlargement, fontanelle changes, and feeding or alertness disturbance rather than headache.
- In older children, raised ICP symptoms such as morning headache, vomiting, diplopia, and papilledema are more typical .
- Acute hydrocephalus tends to produce more obvious ICP symptoms, whereas chronic hydrocephalus may present more insidiously.
- Cushing's triad is a late emergency sign and suggests severe intracranial hypertension.

## 9.DIAGNOSIS

### Clinical and antenatal diagnosis

The diagnosis of pediatric hydrocephalus begins before birth in a proportion of cases and continues after delivery through a combination of clinical assessment and neuroimaging. Antenatally, ventriculomegaly is usually first detected on routine obstetric ultrasonography, and a distal lateral ventricular atrial width of 10 mm or more is generally used as the threshold for diagnosis. Mild ventriculomegaly is usually defined as 10–14.9 mm, while severe ventriculomegaly is 15 mm or more.[44-47] When ventriculomegaly is suspected, the next step is not simply to confirm enlarged ventricles, but to perform a detailed targeted neurosonography and a careful anatomic survey to look for associated central nervous system and extra-cranial abnormalities.[44-47]

Fetal MRI has an important complementary role in antenatal diagnosis. The ISUOG guideline states that fetal MRI is an adjunct to expert ultrasonography rather than a primary screening tool, and it is particularly useful when ultrasound is incomplete, technically limited, or suggests a brain abnormality that needs further characterization [2]. The MERIDIAN prospective cohort study showed that in utero MRI improved the diagnostic accuracy and confidence of prenatal assessment of fetal brain abnormalities compared with ultrasonography alone, and later analyses confirmed that MRI can provide additional clinically useful information in a substantial proportion of cases.[45-46] Therefore, in suspected fetal hydrocephalus or ventriculomegaly, ultrasound is the first-line test, but MRI is often the best second-line modality for defining the cause and identifying associated anomalies.[45-47]

After birth, diagnosis depends on the integration of history, physical examination, head growth, developmental status, and imaging. A key principle is that ventriculomegaly

alone is not enough; the diagnosis of hydrocephalus relies on radiographic findings interpreted together with clinical evidence suggesting raised intracranial pressure or disturbed neurological development.[48-49] In neonates and infants, important clinical clues include a rapidly enlarging head circumference, a full or tense anterior fontanelle, splaying of cranial sutures, upgazed palsy, lethargy, vomiting, irritability, and episodes of bradycardia.[49] Because hydrocephalus in infancy may present insidiously, serial occipitofrontal circumference (OFC) monitoring remains central to diagnosis. Recent pediatric review literature notes that screening in infancy relies heavily on repeated head circumference measurement, and developmental regression or delayed milestones should also prompt further assessment.[48,50]

Developmental assessment should not be treated as a separate issue from diagnosis. In infants, subtle problems with head control, alertness, feeding, motor organisation, and milestone progression may be the earliest signs that ventricular enlargement is already affecting brain function.[49-51] Gonț et al. note that structured neurological observation and general movement assessment can add useful information to imaging in newborns with hydrocephalus, especially when following progression over time.[51] Thus, postnatal diagnosis is strongest when the clinician combines physical signs, growth pattern, developmental observation, and timely imaging, rather than relying on any one element in isolation.[49-51]

**Practical diagnostic approach**

- Antenatal: routine ultrasound → targeted neuro sonography → fetal MRI when needed.
- Postnatal clinical assessment: history, fontanelle/suture examination, OFC plotting, neurological examination, developmental review.
- Confirmatory imaging: cranial ultrasound, CT in urgent settings, and MRI for fuller anatomical and etiological definition.

**Imaging**

In infants with an open fontanelle, cranial ultrasound is the initial imaging modality of choice. It is portable, bedside, radiation-free, repeatable, and well suited to the neonatal intensive care setting.[49,51-52] It can demonstrate ventricular enlargement, asymmetry, intraventricular hemorrhage, post-hemorrhagic ventricular dilatation, and some associated structural abnormalities, while also allowing serial follow-up without the risks of transport or ionizing radiation.[49,51] Gonț et al. emphasize that cranial ultrasound remains one of the most accessible and useful tools for both diagnosis and monitoring of neonatal hydrocephalus, particularly where repeated assessment is needed.[51]

CT scanning is fast and widely available, and it is especially valuable in urgent or emergency situations when a rapid answer is needed, such as acute deterioration, suspected shunt malfunction, hemorrhage, or when MRI is not immediately available.[49,52] CT readily shows ventricular size and acute obstructive patterns, but its major limitation is exposure to ionizing radiation, which is particularly important in children who may need repeated imaging over time.[48,52] For this reason, CT is

generally not preferred for routine serial follow-up if ultrasound or MRI can provide the necessary information.[48-49,52]

MRI provides the most detailed anatomical assessment and is usually the best modality for identifying the cause of hydrocephalus and associated abnormalities. It is superior to CT for evaluating parenchymal injury, posterior fossa abnormalities, aqueductal stenosis, associated malformations, and the wider CSF pathway.[50,52] Krishnan et al. note that MRI helps define the nature of obstruction and provides functional and anatomical information that improves understanding of the underlying mechanism, while Raybaud describes MRI as a “road map” for analyzing pediatric hydrocephalus in relation to its pathophysiology.[50,52] MRI is therefore especially useful when the clinical question is not simply “Are the ventricles enlarged?” but rather “Why are they enlarged, what else is abnormal, and how should this guide management?”[50,52]

A central diagnostic principle is that imaging findings must always be interpreted in clinical context. Pindrik et al. stress that the diagnosis of neonatal hydrocephalus depends on both radiographic criteria and clinical findings suggesting raised intracranial pressure.[49].Wright et al. similarly recommend that hydrocephalus should initially be evaluated clinically and, when possible, with appropriate non-radiation imaging rather than by imaging alone.[48] In practice, this means that ventricular enlargement on ultrasound, CT, or MRI should be interpreted alongside the child’s OFC trend, neurological signs, symptoms, and developmental trajectory.[48-50]

**Key diagnostic points**

- Antenatal diagnosis usually starts with ultrasound, with fetal MRI as an adjunct when more detail is required.
- Postnatal diagnosis requires clinical assessment plus imaging, not imaging alone.
- OFC monitoring and developmental assessment are especially important in infancy.
- Cranial ultrasound is first-line in infants; CT is mainly for urgent situations; MRI best defines the cause and associated abnormalities.

## 9. MANAGEMENT

Management of pediatric hydrocephalus aims to relieve abnormal cerebrospinal fluid (CSF) accumulation, reduce intracranial pressure (ICP), preserve brain development, and minimize secondary neurological injury. Treatment is guided by symptoms, age, etiology, ventricular anatomy, disease progression, and available neurosurgical resources.2,4 In general, symptomatic hydrocephalus is primarily a surgical disease. Krovvidi et al. state that while some asymptomatic patients with chronic ventriculomegaly may not require intervention, symptomatic hydrocephalus usually requires surgery, and drug therapy alone is rarely sufficient.[4] Current reviews and guidelines support the same principle, framing management around temporary or definitive CSF diversion rather than prolonged medical treatment.[4,10,53]

The goals of management are to:

- restore safer CSF dynamics and lower ICP;
- protect the developing brain from further stretch and ischemic injury;
- treat the underlying cause where possible;
- provide durable CSF diversion with the fewest complications.

Broadly, treatment falls into three groups:

1. temporizing measures, when definitive surgery is unsafe or delayed;
2. permanent shunt diversion, most commonly the ventriculoperitoneal (VP) shunt;
3. internal diversion procedures, mainly endoscopic third ventriculostomy (ETV), with or without choroid plexus cauterization (CPC) in selected settings.

**Table 6. General management framework**

| Clinical situation | Main treatment principle |
|---|---|
| Asymptomatic ventriculomegaly without clear progression | Observation and reassessment |
| Symptomatic hydrocephalus | Surgical CSF diversion |
| Child not yet ready for permanent diversion | Temporizing CSF drainage |
| Clear obstructive anatomy | Consider ETV in selected cases |

A key practical point is that the aim of treatment is not simply to reduce ventricular size, but to improve CSF physiology and neurological well-being. Clinical status remains as important as radiological appearance.[4,10]

**Temporary medical and temporizing measures**

❖ **Medical therapy**

Medical treatment has historically included acetazolamide and furosemide, especially in posthemorrhagic ventricular dilatation (PHVD) of prematurity. However, higher-quality evidence does not support their routine use. Kennedy et al. found that acetazolamide plus furosemide did not reduce the need for shunt placement and was associated with increased neurological morbidity.[54] The Cochrane review reached the same conclusion, reporting that this combination was neither effective nor safe in posthemorrhagic ventricular dilatation.[55]

The evidence-based pediatric guideline therefore does not recommend these drugs as a method of reducing shunt dependence in premature infants with posthemorrhagic hydrocephalus (PHH).[53] They are best regarded as historical or occasional temporizing measures rather than standard treatment.[53-55]

### ❖ Lumbar puncture and lumbar drainage

Lumbar puncture may be used only in selected communicating hydrocephalus, particularly when the basal cisterns are patent and the hydrocephalus is not obstructive. Krovvidi et al. note that communicating hydrocephalus can usually be drained safely by the lumbar route, whereas high-pressure non-communicating hydrocephalus should be drained directly with an external ventricular drain (EVD).[4] In premature infants with PHH, lumbar puncture may be used during early temporization, but routine serial lumbar puncture is not recommended as a reliable strategy to prevent progression or avoid shunt surgery.[53,59]

### ❖ External ventricular drainage and other temporizing options

An EVD is an important temporary drainage method when urgent decompression is required or permanent shunting is unsafe. Common indications include acute new-onset hydrocephalus, infected shunt removal in a shunt-dependent child, and raised ICP with small or normal ventricles.[4] Krovvidi et al. report infection rates of 5–20% for EVDs,[4] and newer pediatric studies confirm that EVDs are useful but require strict infection-control protocols.[56]

Other accepted temporizing options in neonatal PHH include ventricular access devices (reservoirs) and ventriculosubgaleal shunts. Guidelines recognize VADs, EVDs, ventriculosubgaleal shunts, and lumbar punctures as reasonable options depending on the infant's condition and local expertise.[53] No single temporizing method has shown clear overall superiority.[53,57]

## Surgical management – ventriculoperitoneal shunt

The VP shunt remains the mainstay of treatment for pediatric hydrocephalus worldwide. It consists of:

- a proximal ventricular catheter placed in the lateral ventricle;
- a valve/reservoir assembly regulating flow;
- a distal catheter, usually placed in the peritoneal cavity.

Rath and Dash describe VP shunt surgery as the most commonly performed pediatric neurosurgical CSF-diversion procedure.[10] In KwaZulu-Natal, Enicker and Aldous found that 84.2% of children were managed with VP shunts, confirming their continued dominance in routine practice.[3]

### ❖ Valve types and shunt biomechanics

Shunt valves are generally classified as:

- fixed differential-pressure valves;
- programmable/adjustable valves;
- anti-siphon or siphon-resisting devices.

Fixed valves open at preset pressures, whereas programmable valves can be adjusted after surgery. Anti-siphon devices help reduce posture-related overdrainage, which may otherwise cause slit ventricle syndrome or subdural collections.[58] However, available evidence does not show consistent superiority of one valve type over another in paediatric outcomes.[58] Valve choice is therefore individualised according to age, anatomy, complication history, surgeon preference, and availability.[58]

- **Advantages and limitations of VP shunts**

VP shunts are widely used because they are familiar, rapidly effective, and suitable for many aetiologies. Their major disadvantages are lifelong shunt dependence and risks such as infection, obstruction, fracture, migration, over drainage, and repeated revision.[2-3,10] Enicker and Aldous reported an overall VP shunt complication rate of 20.4%, including 9.6% infections, in the KwaZulu-Natal cohort.[3]

**Surgical management – ETV and other options**

- **Endoscopic third ventriculostomy**

ETV is an internal CSF diversion procedure in which an opening is created in the floor of the third ventricle to bypass obstruction and re-establish CSF flow to the prepontine cisterns. Krovvidi et al. identify it as a useful alternative in non-communicating hydrocephalus, especially aqueductal stenosis and selected pineal-region lesions.[4] Prospective multicenter work from the Hydrocephalus Clinical Research Network confirms that ETV is a viable treatment for a subgroup of children.[59]

Its best indications are therefore mainly obstructive hydrocephalus. Outcomes depend on age, etiology, and prior shunt history, factors incorporated into the ETV Success Score (ETVSS). In the 2024 re-evaluation of the ETVSS, the 6-month success rate for first-time ETV was 76%.[60] Success is less predictable in very young infants and in postinfectious or posthemorrhagic hydrocephalus.[60-61]

**Contraindications, complications, and alternatives**

ETV is best suited to obstructive hydrocephalus and is less predictable in communicating hydrocephalus, severe prepontine scarring, very young age, and inflammatory or hemorrhagic disease.[4,61-60] Complications are uncommon but potentially serious. Bouras and Sgouros reported an overall complication rate of 8.5%, permanent morbidity of 2.4%, and mortality of 0.21%; serious events included intraoperative hemorrhage, vascular injury, neural injury, CSF leak, and infection.[63] Rath and Dash also note intraoperative bradycardia and arrhythmias during endoscopic manipulation.[10]

When the peritoneum cannot be used, alternative distal sites include the right atrium and pleural cavity.[4,10] Recent HCRN data showed broadly similar survival for ventriculoatrial and ventriculopleural shunts after adjustment, although pneumothorax was more frequent with ventriculopleural shunts.[64] These options remain important rescue procedures but are not first-line for most children.[64]

In summary, management of pediatric hydrocephalus is centered on timely CSF diversion, with VP shunts remaining the most widely used treatment and ETV offering

an important alternative in selected obstructive cases. Temporizing measures have a role when definitive surgery must be delayed, but prolonged medical therapy is not supported by current evidence.

## 10. COMPLICATIONS

Complications remain a major part of the burden of pediatric hydrocephalus because treatment often creates long-term dependence on CSF diversion devices, repeated surveillance, and possible revision surgery. In the KwaZulu-Natal series, Enicker and Aldous reported an overall ventriculoperitoneal shunt (VPS) complication rate of 20.4%, including mechanical failure in 10.9% and infection in 9.6% of cases.[3] Other literature similarly shows that obstruction, infection, over-drainage, and distal catheter problems are the dominant shunt-related complications, while endoscopic third ventriculostomy (ETV) has a different but still important risk profile.[63-67]

### Shunt-related complications

***Infection*** is one of the most serious complications. In the KwaZulu-Natal cohort, VPS infection was independently associated with mortality, and the commonest organisms were *Staphylococcus aureus* and *Staphylococcus epidermidis*.[3] Mshayisa also identifies infection as a major complication of both newly inserted and established shunts.[41] Evidence-based guidelines emphasize that shunt infection is a major cause of shunt failure and often requires antibiotics together with partial or complete removal of hardware rather than antibiotics alone.[67]

***Obstruction and malfunction*** are also common. Enicker and Aldous found mechanical failure in 10.9% of shunted children.[3] Mshayisa lists obstruction, disconnection, breakage, malposition, distal catheter problems, and under-shunting among typical causes of malfunction.[41] Paff et al. describe obstruction, especially at the proximal catheter, as the commonest cause of VPS failure, while infection is usually second.[65] Riva-Cambrin et al. further showed that younger age is a significant risk factor for first shunt failure, which helps explain the high revision burden in infants and young children.[66]

***Over-drainage*** may occur when CSF is diverted too rapidly, especially because of posture-related siphoning. Mshayisa includes over shunting, slit ventricle syndrome, and subdural hematoma among recognized shunt complications.[41] Paff et al. similarly note that excessive drainage may cause ventricular collapse, headache, vomiting, and subdural collections or haematoma.[65]

***Distal catheter complications*** are another important group. Mshayisa lists peritonitis, hydrocele, inguinal hernia, CSF ascites, migration, volvulus, and intestinal strangulation.[41] Furtado et al. reported that common abdominal complications include abdominal pseudocyst, distal catheter migration, inguinal hernia, catheter disconnection, and intestinal obstruction.[68] These complications are particularly relevant in children because of growth, thin tissues, and abdominal vulnerability.

### Procedure and system complications

Procedure-related complications may occur during shunt insertion or endoscopic surgery. Mshayisa lists intraparenchymal or intraventricular hemorrhage, seizures, catheter malposition, and infection among shunt insertion complications.[41] Rath and Dash also warn that rapid intraoperative drainage of CSF should be avoided because it may provoke arrhythmia and hemodynamic instability.[10]

ETV avoids permanent shunt hardware but has its own risks. Mshayisa lists hypothalamic injury, pituitary stalk or gland injury, transient cranial nerve palsies, vascular injury, bleeding, cardiac arrest, and traumatic basilar aneurysm as possible complications.[41] Bouras and Sgouros reported an overall ETV complication rate of 8.5%, with intraoperative hemorrhage in 3.7%, permanent morbidity in 2.4%, and mortality in 0.21%.[68] Rath and Dash also note that bradycardia and arrhythmias may occur during irrigation or manipulation of the third ventricular floor.[10]

Because the peritoneum is the commonest distal site for shunting, abdominal complications deserve special attention. Enicker and Aldous reported intra-abdominal complications in 1.4% of cases, including acute abdomen/peritonitis in 0.8%, abdominal pseudocyst in 0.6%, and rectal protrusion in 0.3%.[3] Acute abdomen was associated with mortality.[3] Furtado et al. confirm that abdominal pseudocyst, migration, inguinal hernia, and bowel complications are important distal VPS problems.[68]

In summary, complications of pediatric hydrocephalus treatment are clinically significant because they increase morbidity, prolong hospital stay, and often lead to revision surgery or external drainage. The most important shunt-related problems are infection, obstruction, over-drainage, and distal catheter complications, while ETV carries risks of hemorrhage and neurovascular injury. Continuous follow-up is therefore essential in all children treated for hydrocephalus.

## 11. ANAESTHETIC OR PERIOPERATIVE CONSIDERATIONS

Pediatric hydrocephalus presents a distinct perioperative challenge because the anesthetist must manage raised intracranial pressure (ICP), vomiting, dehydration, aspiration risk, and, in some infants, a very large head that may complicate positioning and airway management.[4,10,69-70] The main goals are to preserve cerebral perfusion, avoid hypoxia and hypercapnia, facilitate safe CSF diversion, and allow early postoperative neurological assessment.

### Preoperative and induction concerns

Preoperative assessment should focus on signs of raised ICP and the physiological consequences of hydrocephalus. Rath and Dash note that infants may present with irritability, lethargy, poor feeding, bulging fontanelle, and cranial enlargement, whereas older children more often have morning headache, vomiting, diplopia, papilledema, and, in late stages, Cushing's triad.[10] Stendall et al. add that signs vary with age and papilledema may be absent despite raised ICP.[69]

Vomiting has major anesthetic implications because it may cause dehydration, electrolyte disturbance, and aspiration risk. Fluid balance and electrolytes should

therefore be assessed carefully before induction.[10,69-70] Airway assessment is also essential, since severe hydrocephalus may produce a large occiput and difficult positioning, while associated anomalies such as Chiari malformation may further complicate airway management.[70]

When ICP is raised, sedative and narcotic premedication should generally be avoided because respiratory depression and hypercapnia may worsen intracranial hypertension.[10] For induction, intravenous induction is preferred where possible because it allows smoother airway control and avoids struggling, although carefully controlled sevoflurane induction may be acceptable if IV access is difficult and agitation would be more harmful.[10,69] Ventilation should be controlled early, with emphasis on avoiding hypoxia and hypercapnia; brief mild hyperventilation should be reserved for acute ICP control rather than routine use.[10,69] If aspiration risk is significant, rapid-sequence induction is appropriate.[10,70]

### Intraoperative and postoperative care

If the child is dehydrated, rehydration should begin before induction and continue carefully intraoperatively.[10,70] During VP shunt surgery, the child is usually positioned supine with the head turned to one side, so the airway becomes less accessible after draping; the endotracheal tube and IV access must therefore be secured היט היט before incision.[10,69-70] Heat conservation is also important, especially in infants, and normothermia should be maintained with warm fluids and warming devices.[10,69-70]

Rapid drainage of CSF should be avoided because it may provoke haemodynamic instability, including bradycardia and arrhythmias, especially during ETV.[10,70] If an external ventricular drain is present, it should be managed as a closed sterile system because infection, dislocation, and dysfunction remain important risks.[71]

Postoperatively, the aim is rapid but safe awakening to permit neurological assessment.[10] Extubation is appropriate once airway reflexes and responsiveness are adequate, but some children require ICU-level observation, especially if there is poor mental status, brainstem dysfunction, blood loss, brain oedema, or postoperative bleeding.[10,70] Monitoring should focus on airway patency, aspiration risk, consciousness, pupils, vomiting, seizures, cardiorespiratory stability, and signs of persistent or recurrent raised ICP.[10,69-70]

## 12. PROGNOSIS, NEURODEVELOPMENT, AND LONG-TERM FOLLOW-UP

### Prognosis and outcome

The prognosis of pediatric hydrocephalus is variable and depends on far more than ventricular size alone. Outcome is influenced by the underlying cause, age at presentation, systemic illness, treatment-related complications, and the child's neurological condition before and after CSF diversion.[2-3,41,72] Recent reviews emphasize that long-term outcome is shaped by both intrinsic factors, such as developmental or genetic brain abnormalities, and extrinsic factors, such as infection burden, treatment delay, and access to specialist care.[2,72]

The strongest local data in your source set come from KwaZulu-Natal. Enicker and Aldous reported that 264 of 3,325 children (7.9%) died during the 20-year study period,

although mortality declined significantly over time.[3] Their multivariable analysis showed poorer survival with age ≥1 year, tertiary hospital referral, VP shunt infection, VP shunt mechanical failure, acute abdomen, and pneumonia.[3] Post-infectious hydrocephalus accounted for 47.3% of deaths, and tuberculous meningitis was the dominant contributor within that group.[3] These findings show that prognosis is influenced not only by hydrocephalus itself, but also by complications of treatment and the child's general medical condition.

A cautious conclusion is that prognosis in pediatric hydrocephalus is heterogeneous. Some children do well with timely and durable CSF diversion, whereas others experience neurological, cognitive, or functional sequelae related to the original brain injury and the later burden of complications.[2,72]

**Main factors associated with poorer outcome**

- delayed or severe presentation, especially infection-related disease
- shunt infection and mechanical failure
- major systemic complications such as acute abdomen and pneumonia
- associated brain disease or congenital malformation

**Neurodevelopment and follow-up**

Long-term outcome in paediatric hydrocephalus must be considered in functional, developmental, and educational terms, not only survival. Mshayisa notes that functional outcome is influenced by prematurity, CNS malformations, other congenital anomalies, seizures, and sensory or motor impairment.[41] This is supported by wider literature showing that neurodevelopment depends on underlying etiology, associated brain injury, treatment complications, and broader neurological context.[2,72]

Prematurity is especially important because post-hemorrhagic hydrocephalus often affects infants already vulnerable to white-matter injury. In a 2023 follow-up study, Wu et al. reported that among infants with neonatal post-hemorrhagic hydrocephalus, 21.4% died, 42.9% had moderate-to-severe dysfunction, and only 35.7% had good outcomes after a median follow-up of 3 years.[73] Associated anomalies also matter. Fletcher et al. showed that hydrocephalus remains relevant to later school-age neurodevelopmental outcomes in children with myelomeningocele.[14]

Repeated revisions also influence long-term outcome. Mshayisa notes that seizures are frequent in shunted children and vary with underlying aetiology.[41] Wendling-Keim et al. found that the number of revision procedures had a significant adverse effect on motor development, even though valve type did not.[13] This suggests that prognosis reflects both the original disease and the cumulative burden of repeated shunt failure and reoperation.

Children treated for hydrocephalus therefore require long-term follow-up after either shunt insertion or ETV. Follow-up should continue beyond the immediate postoperative period because both shunt and ETV failure may occur later in childhood.[72,74] It should also extend beyond neurosurgical review to include developmental, cognitive, behavioral, visual, motor, and educational monitoring.

Prakash et al. found that school problems, cognitive difficulties, and reduced quality of life may persist after VP shunt surgery, supporting multidisciplinary long-term care.[75]

**Key follow-up priorities**

- monitor for late shunt or ETV failure
- assess motor, cognitive, language, and behavioral development
- screen for visual, sensory, and seizure-related problems
- review school progress and support need

## 13. PREVENTION AND PUBLIC HEALTH

Prevention and public-health action in pediatric hydrocephalus must be framed realistically, because not all causes are preventable. However, the KwaZulu-Natal data show that there are important opportunities to reduce burden through infection prevention, earlier recognition, and better access to specialist services. In the KwaZulu-Natal series, post-infectious hydrocephalus was the predominant etiological category (32.7%), tuberculous meningitis accounted for 54.1% of infectious cases, 51.4% of children came from rural areas, and 47.9% were referred from regional hospitals.[3] These findings suggest that hydrocephalus in this setting is partly a neurosurgical problem and partly a health-system problem involving infection burden, referral pathways, and treatment delay.[3]

A first public-health priority is the prevention of central nervous system infections, especially meningitis. The WHO “Defeating Meningitis by 2030” roadmap sets explicit goals to eliminate bacterial meningitis epidemics, reduce cases of vaccine-preventable bacterial meningitis by 50%, reduce deaths by 70%, and improve disability outcomes, with prevention, diagnosis, surveillance, and access to care forming the core pillars of action.[76] In practical terms, this supports strong routine immunization coverage, prompt recognition of meningitis, and rapid treatment to reduce the risk of hydrocephalus as a neurological sequela.[76] In settings such as KwaZulu-Natal, where tuberculous meningitis is a major contributor, childhood tuberculosis control also matters. WHO guidance for children and adolescents emphasizes integrated, decentralized models of TB case detection, prevention, and treatment, including specific recommendations for TB meningitis management.[77]

A second priority is early diagnosis and referral. Recent review literature notes that over 80% of new pediatric hydrocephalus cases occur in low- and middle-income countries and that the epidemiology varies by geography, infection burden, and income level.[2] Across Africa, a systematic review and meta-analysis found that post-infectious hydrocephalus is the single most common cause of pediatric hydrocephalus and concluded that improved access to diagnostic services is needed for better targeted investment and care.[21] In public-health terms, this means children with progressive head enlargement, bulging fontanelle, persistent vomiting, altered consciousness, or suspected meningitis need earlier identification at primary and district level, followed by rapid referral to centers that can confirm diagnosis and provide CSF diversion when needed.[2,3,21]

A third priority is improving access to pediatric neurosurgical services and reducing delays in treatment. Enicker and Aldous explicitly note that hydrocephalus produces a dual insult on the brain: injury from the underlying causative pathology and further injury from raised intracranial pressure, especially when treatment is delayed.[3] This point is central in LMIC settings, where geography, transport, referral hierarchy, and limited specialist capacity can all prolong time to treatment.

Therefore, a modest but evidence-based public-health message is that reducing the burden of pediatric hydrocephalus requires action at multiple levels: preventing CNS infections where possible, strengthening early diagnosis and referral, and ensuring that children in rural and regional settings can access timely pediatric neurosurgical assessment and treatment.

**Key public-health priorities**

- strengthen prevention and early treatment of meningitis and childhood TB
- improve early recognition and referral of suspected hydrocephalus
- expand equitable access to pediatric neurosurgical services, especially for rural populations
- reduce treatment delay, because delayed diversion increases the risk of secondary brain injury

In conclusion, prevention and public-health action in pediatric hydrocephalus should focus on realistic and context-specific priorities rather than assuming that all cases can be prevented. In settings such as KwaZulu-Natal, where infection-related hydrocephalus, rural residence, and referral delays remain important, effective public-health response requires stronger infection control, earlier recognition of warning signs, faster referral pathways, and more equitable access to pediatric neurosurgical care. Taken together, these measures can help reduce avoidable brain injury, improve survival, and lessen the long-term developmental burden associated with delayed diagnosis and treatment.

## 14. CONCLUSION

Pediatric hydrocephalus is a serious disorder of cerebrospinal fluid (CSF) dynamics in which disturbed CSF production, circulation, or absorption leads to ventricular enlargement and, in many children, raised intracranial pressure.[3,4] It is an important pediatric neurological condition because it commonly presents in infancy, when the brain is still rapidly developing and therefore especially vulnerable to compression, stretch, ischemia, and secondary neurodevelopmental injury.[4,10,41] In addition, the burden of disease is particularly high in low- and middle-income settings, where delayed access to diagnosis and treatment may worsen outcome.[3]

As this booklet has shown, pediatric hydrocephalus arises from both congenital and acquired causes. Important congenital causes include aqueductal stenosis, myelomeningocele with Chiari II malformation, Dandy–Walker malformation, X-linked hydrocephalus, and other developmental abnormalities, while acquired causes include infection, intraventricular hemorrhage of prematurity, tumors, and head trauma.[10,41] The relative importance of these causes varies by setting. In KwaZulu-Natal, post-

infectious hydrocephalus was the predominant category, with tuberculous meningitis making a major contribution, illustrating how local infectious burden shapes the real-world pattern of disease.[3]

Diagnosis depends on the combination of clinical assessment and radiological evaluation. Antenatal ultrasound and fetal MRI can identify ventriculomegaly before birth, while postnatal diagnosis relies on head circumference monitoring, developmental assessment, neurological examination, and imaging with cranial ultrasound, CT, or MRI.[10,41] Imaging is essential, but it must always be interpreted in clinical context because ventricular enlargement alone does not fully define severity or treatment need.[4] Clinical presentation is strongly age-dependent: infants often show macrocephaly, rapid head growth, bulging fontanelle, poor feeding, lethargy, and sun-setting eyes, whereas older children more commonly present with headache, vomiting, diplopia, papilledema, and other signs of raised intracranial pressure.[10,41]

Management is centered mainly on surgical CSF diversion. Symptomatic hydrocephalus usually requires operative treatment, most commonly by ventriculoperitoneal shunt insertion, while endoscopic third ventriculostomy has an important role in selected cases of obstructive hydrocephalus.[4,10,41] Medical treatment has only a limited and mainly temporizing role. The overall aim of treatment is not merely to reduce ventricular size, but to restore safer CSF dynamics, lower intracranial pressure, and protect the developing brain from further damage.[4,10,41] In KwaZulu-Natal, VP shunts were the mainstay of treatment, confirming their continued central role in pediatric practice.[3]

Despite these advances, complications remain a major challenge. Shunt infection, obstruction, mechanical failure, over-drainage, slit ventricle syndrome, subdural hematoma, and distal catheter complications continue to contribute substantially to morbidity.[3,41] Procedure-related complications such as hemorrhage, seizures, ETV-related bleeding or neurovascular injury, and abdominal complications such as acute abdomen and peritonitis also remain important.[10,41] In the KwaZulu-Natal series, VP shunt infection, mechanical failure, acute abdomen, and pneumonia were all associated with poorer outcomes and mortality, showing that prognosis depends not only on the initial hydrocephalus but also on the complications of treatment and the child's overall clinical condition.[3]

Finally, pediatric hydrocephalus should be understood as a condition that requires long-term follow-up, not simply a one-time procedure. Functional outcome depends on factors such as prematurity, associated anomalies, seizures, sensory and motor impairment, and repeated shunt revisions.[41] Long-term care should therefore include not only neurosurgical review, but also developmental, neurological, visual, behavioral, and educational monitoring. In this sense, the management of pediatric hydrocephalus extends from early diagnosis and timely surgical treatment to ongoing multidisciplinary support aimed at preserving survival, function, and quality of life.[3,41]

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