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EXPERT-CONSENSUS REPORT

Pediatric Intracerebral Hemorrhage Management—Consensus Statement of the International Pediatric Stroke Organization—Part 1: Acute Phase and Workup

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ABSTRACT: Pediatric intracerebral hemorrhage (pICH) is a rare but serious neurologic emergency associated with significant morbidity. Although pICH accounts for nearly half of all pediatric strokes, it remains understudied, and dedicated evidence-based management guidelines are lacking. To address this gap, the International Pediatric Stroke Organization convened a multidisciplinary international working group in 2020 to develop a comprehensive, consensus-based framework for the acute evaluation and management of pICH in children aged 28 days to 18 years. The working group included child neurologists, neurointensivists, neurosurgeons, neuroradiologists, and neurointerventionalists. Subgroups conducted systematic literature reviews and formulated key clinical questions. A modified Delphi process was used to derive consensus statements across 6 domains: prehospital and emergency care, diagnostic imaging and workup, neurocritical care and medical management, neurosurgical and neurointerventional approaches, and identification of knowledge gaps. Through rounds of structured review and voting, 21 consensus statements were developed and approved. The process was endorsed by multiple professional societies. This represents the first international, multidisciplinary, multisociety consensus statement focused on the acute management of pICH in children. It provides structured, expert-driven guidance to inform clinical decision-making, reduce practice variability, and highlight areas for future research. These consensus statements aim to support clinicians worldwide in improving outcomes for children with pICH.

Key Words: nontraumatic cerebral hemorrhage ■ pediatric hemorrhagic stroke ■ pediatric intracerebral hemorrhage

See Editorial by Jordan and Kirton.

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CLINICAL PERSPECTIVE

What Is New?

- This consensus statement from the International Pediatric Stroke Organization provides the first comprehensive, multidisciplinary recommendations for the acute management of pediatric intracerebral hemorrhage, emphasizing standardized diagnostic and therapeutic pathways.

What Are the Clinical Implications?

- Early recognition of pediatric intracerebral hemorrhage symptoms, prompt imaging, and multidisciplinary acute management is critical for improving outcomes. This statement highlights key interventions including neuroprotective strategies, targeted neuroimaging, and tailored neurosurgical and neurocritical care approaches.
- Further multicenter studies are needed to validate these recommendations, refine imaging and treatment protocols, and identify optimal strategies for long-term recovery and prevention of recurrent hemorrhage in children.

Nonstandard Abbreviations and Acronyms

COL4A	collagen type IV α
CTA	computed tomography angiography
DSA	digital subtraction angiography
ICH	intracerebral hemorrhage
ICP	intracranial pressure
IPSO	International Pediatric Stroke Organization
pICH	pediatric ICH

Hemorrhagic stroke is a rare but serious emergency in children.[1,2] The present consensus statements focuses on pediatric intracerebral hemorrhage (pICH), the most common form of hemorrhagic stroke in infants and children, defined as nontraumatic hemorrhage within the brain tissue or ventricles in children aged 28 days to 18 years.

Although pICH accounts for nearly half of all pediatric strokes, it remains an understudied condition with limited evidence to guide management. The incidence of pICH is estimated to range from 10 to 20 per million children per year,[3] with underlying causes differing significantly from those in adults.[4,5] Unlike adult intracerebral hemorrhage (ICH), which is often associated with hypertension and cerebral amyloid angiopathy microangiopathies, pICH is primarily caused by vascular malformations, coagulopathies, brain tumors, and systemic diseases. These differences impact diagnostic workup, acute management, and long-term care strategies. Additionally, pICH carries a high risk of morbidity, with many survivors experiencing lasting neurological, cognitive, and functional impairments that affect independence and quality of life.[6]

Despite advances in neuroimaging and critical care, clinical decision-making in pICH remains challenging due to a lack of standardized guidelines and consensus-based recommendations. Care pathways vary depending on clinical severity, underlying risk factors, available resources, and societal factors. Recognizing this gap, the International Pediatric Stroke Organization (IPSO) was founded in 2019 to foster international and multidisciplinary collaborations aimed at improving the understanding, care, and outcomes of childhood cerebrovascular disease. Through an IPSO-led international effort, this document provides consensus-based guidance for the diagnosis and acute management of pICH, addressing key knowledge gaps and standardizing best practices for clinicians worldwide.

This consensus statement covers key areas essential to pICH management:

1. Prehospital and emergency evaluation: recognition of pICH symptoms, stroke protocols, and initial neuroimaging recommendations.
2. Initial workup: optimal neuroimaging, vascular studies, coagulopathy assessment, and genetic considerations.
3. Neurocritical care and medical management: neuroprotective strategies, seizure monitoring and prophylaxis, and intracranial pressure (ICP) management.
4. Neurosurgical interventions: indications for external ventricular drainage, hematoma evacuation, and surgical management of underlying vascular lesions.
5. Neurointerventional approaches: role of diagnostic and therapeutic angiography in pICH management.

By addressing these critical aspects, this document aims to provide a structured, evidence-informed framework for improving pICH care globally.

CONSENSUS METHODOLOGY

A dedicated working group was launched within IPSO in 2020 with the goal to develop a comprehensive set of recommendations for the management of children with pICH.

pICH Definition

pICH was defined as nontraumatic hemorrhage within the brain tissue or ventricles in children aged from 28 days to 18 years. This definition encompasses intraparenchymal hemorrhage, with or without accompanying intraventricular hemorrhage, and isolated intraventricular hemorrhage. pICH may also be associated with subarachnoid hemorrhage, but pure subarachnoid hemorrhage without ICH is not addressed in this article because the pathophysiology and natural history differ from those of ICH. ICH in neonates has separate causes and management than in older children. This work does not address neonatal ICH except when specified.

Methodology

IPSO convened the Hemorrhagic Stroke Working Group to identify knowledge gaps and present a practical framework for health care professionals. The initial working group was divided into 6 subgroups based on participants' field of expertise. Each subgroup identified a set of relevant practical questions for each topic, with a maximum of 10 questions. The groups then performed a systematic literature search for articles pertaining to the management of pICH in children, using the following search equation in PubMed. ("pediatrics"[MeSH Terms] OR "pediatrics"[All Fields] OR "pediatric"[All Fields]) AND ("intracerebral hemorrhage"[All Fields] OR "cerebral hemorrhage"[MeSH Terms] OR ("cerebral"[All Fields] AND "hemorrhage"[All Fields]) OR "cerebral hemorrhage"[All Fields] OR ("intracerebral"[All Fields] AND "hemorrhage"[All Fields] OR "intracerebral hemorrhage"[All Fields]), restricting the search to articles published after 1990. The last literature search was performed in March 2022. Each team then summarized the literature and drafted recommendations to each question based on the best evidence and shared expertise.

An interventional neuroradiologist (G.B.) and a child neurologist (L.L.L.) reviewed all sections for harmonization and derived a set of 21 statements. A modified Delphi approach was then implemented to reach consensus for each statement.[7] All contributors were asked to vote (agree/disagree) on each statement and to provide feedback and comments for improvement in cases of disagreement. Statements that reached >80% agreement were approved and included in the present document. Conversely, statements that did not reach an 80% agreement were revised by G.B., L.L.L., and a second child neurologist (L.A.B.) following feedback provided by the study group and underwent a second round of consensus voting. The coordinating team drafted and circulated the final version of the article. Participants were asked to provide comments, disclosures, and other relevant information. All coauthors approved the submitted article on February 29, 2024.

Table Clinical Presentation of Pediatric Intracerebral Hemorrhage

Clinical presentation	Frequency
Headache	46%–80%
Nausea/vomiting	60%
Seizures	20%–40%
Focal neurological deficits	13%–50%
Altered level of consciousness	>50%

PREHOSPITAL PATHWAY AND PEDIATRIC STROKE PROTOCOL

In Children, What Symptoms Should Trigger a Code Stroke?

Typical symptoms that trigger a code stroke include most notably the occurrence of a focal neurological deficit (acute hemiparesis, aphasia); however, pICH can present with less specific symptoms such as vomiting, headache, or altered level of consciousness[8–10] (see the Table). Younger children are more likely to experience seizures and nonspecific symptoms.[1,11] Infants may present with lethargy and poor feeding. Vomiting and drowsiness in an infant can be mistaken for sepsis. Depending on the pICH cause, the onset can vary from rapidly progressing severe headache and neurological deficits to insidious symptoms or progression over days, which can cause a diagnostic delay.[9]

The most common causes of pICH are vascular malformations including arteriovenous malformations (AVMs), arteriovenous fistulas, cavernous malformations, and aneurysms. Patients with known vascular malformations warrant special attention when exhibiting the above-mentioned symptoms. Other conditions associated with pICH include arteriopathies (eg, moyamoya), disorders of coagulation (thrombocytopenia, congenital or acquired coagulopathy) including receiving anticoagulation and hemoglobinopathies (eg, sickle cell disease), and brain tumors.[12–14]

Statement 1: Specific symptoms that should trigger a code stroke include sudden onset focal neurologic deficits including motor (gait disturbances, hemiparesis, or facial weakness) or nonmotor (visual impairment or sensory symptoms), new onset seizure followed by or preceded by persistent focal neurologic symptoms, or unexplained alteration of mental status in the presence of stroke risk factors.

What Are the Optimal Prehospital Pathway and Destination for a Child With Suspected Stroke?

In children with suspected pICH, the initial care pathway, prehospital management, and triage before hospital arrival is critically important to minimize delays

to treatment and optimize therapeutic opportunities. Recognizing and managing neurosurgical and neurological emergencies can be challenging in children. Institutional or regional code stroke pathways are designed to overcome this challenge. Locally and regionally tailored care protocols and specific care pathways have the potential to facilitate timely diagnosis and decrease time to hyperacute therapies including endovascular revascularization treatment in children with ischemic stroke.[15]

Timely assessment of any child with suspected stroke is best facilitated at centers with pediatric stroke care pathways that streamline assessment, determine appropriate neuroimaging, including potential need for anesthesia, and guided management by an interdisciplinary team including pediatric neurology, neurosurgery, and interventional neuroradiology,[16,17] as well as access to pediatric neuro-intensive care. Prehospital providers should try to notify the destination emergency, neurology, and neuroimaging teams of the arrival of a child with a suspected stroke.

Statement 2: Pediatric stroke protocols should be in place to optimize prehospital and early in-hospital care. They should be adapted to geographic specificities and available resources, and maximize opportunities for acute multidisciplinary decision-making in specialized environments.

Statement 3: Emergency medical services transporting a child with suspected stroke should specify a destination hospital and should contact the hospital before arrival, when possible, similar to trauma transports.

INITIAL WORKUP

In Children With Suspected Stroke, What Is the Optimal Imaging Modality for Initial Diagnosis?

Immediate brain imaging is essential to confirm or exclude hemorrhagic and ischemic stroke diagnoses, assist in consideration of reperfusion therapies in children with arterial ischemic stroke, and facilitate timely implementation of emergent treatments tailored to the cause of symptoms.[18,19] When feasible, without delaying care and in the absence of contraindications, magnetic resonance imaging (MRI) presents advantages over noncontrast computed tomography in suspected stroke. MRI is more sensitive to early ischemia[20,21] and is better at differentiating stroke from stroke mimics.[22] MRI is highly sensitive to the diagnosis of ICH.[23] Furthermore, MRI has a high yield in detection of underlying causes of hemorrhages, including vascular malformations, vasculitis, and cerebral venous thrombosis.[24,25] MRI is the most sensitive and specific imaging modality for detecting cerebral cavernous malformations, a frequent cause of ICH in children.[10,14,26] MRI has high sensitivity for identifying arteriovenous malformations, the most frequent underlying cause of pICH.[25] MRI sequences for a suspected stroke in a child should include, at minimum, diffusion-weighted imaging, T2* gradient recalled echo or its 3-dimensional derivatives,[27] T2/fluid attenuated inversion recovery, and time of flight magnetic resonance angiography (MRA).[28] MRI protocols should be optimized to be fast and with a diagnostic-level signal-to-noise ratio.

Noncontrast computed tomography (CT) and computed tomography angiography (CTA) have advantages in the emergency setting for pediatric patients with acute stroke symptoms including their wide availability, swiftness of imaging acquisition limiting the need for sedation, and high sensitivity for detecting pICH.[4] Drawbacks are ionizing radiation and lower sensitivity for non-pICHs or mimics. Both noncontrast CT and MRI are able to quantify hematoma volume and determine signs that can lead to early neurological deterioration such as intraventricular extension of hemorrhage, hydrocephalus, and/or brain herniation.[29,30]

Statement 4: In children with acute stroke symptoms, brain imaging should be performed as quickly as possible upon hospital arrival after patient stabilization, with MRI/MRA or CT/CTA based on hospital resources and patient factors. Vascular imaging should be included in imaging protocols.

Statement 5: In children with suspected acute ICH, urgent imaging with CT or MRI should be performed to rule out hemorrhage. This can be performed with CT/CTA or MRI/MRA, depending on the circumstances/clinical scenario and hospital resources.

After Imaging Diagnosis of an Acute Intracranial Hemorrhage in a Child, Is Immediate Vascular Imaging Needed?

After diagnostic confirmation of a pICH, vascular imaging should be performed as soon as possible, ideally within the same imaging session that identified the hemorrhage, to assess for a readily identifiable structural vascular cause, the most frequent cause.[14,31] Early identification of the underlying cause is crucial for guiding management and preventing recurrent hemorrhage. Bleeding patterns can inform further investigations, and input from an experienced pediatric neuroradiologist, neurologist, neurosurgeon, and interventional neuroradiologist is essential. This is usually best achieved at a referral center.

CTA performed <96 hours from symptom onset has a high accuracy for detecting underlying vascular anomalies, with sensitivities ≥95% and specificities approaching 100%.[4] Positive and negative predictive

values are reported to be >97% in younger adults.[32] CT venogram is similarly accurate to detect cerebral venous thrombosis.

Due to the high prevalence of AVMs, advanced MRI techniques, such as dynamic contrast MRA and arterial spin labeling, have shown promise in the initial causative workup of pICH.[25] The sequences require validation in larger multicenter studies.

Statement 6: At the time of ICH diagnosis, urgent vascular imaging should be performed using CTA or MRA to assess for an underlying structural vascular cause.

Role of Digital Subtraction Angiography in the Causative Workup

In the context of pICH, MRA/magnetic resonance venogram or CTA/CT venogram are reliable tools for initial diagnosis of vascular malformations associated with pICH. Digital subtraction angiography (DSA) is nonetheless commonly required for the precise characterization of vascular anomalies seen on noninvasive imaging and/or the identification of occult lesions such as small arteriovenous shunts.[16]

Subarachnoid hemorrhage, atypical hematoma configuration, and edema out of proportion seen on CT at admission or the presence of abnormal vascular structures in the brain are all distinctive radiological features of identifiable secondary causes of pICH. In these cases, DSA is necessary to identify the underlying cause, including AVMs and less commonly other types of vascular malformations or arteriopathies.

DSA is also indicated in patients with no obvious cause of bleeding despite adequate noninvasive workup. The DSA yield is significantly higher in young adult patients without preexisting hypertension[33] and is justifiable in children with no other identified cause of pICH, because AVM is the most common cause of pICH in children aged to 2 to 18 years.[14] Timing of DSA should take into consideration the clinical state of the patient, the neurosurgeon's judgment on the urgency of surgery, and the multidisciplinary assessment of the risk of early rebleeding based on noninvasive imaging.[4] In patients with elevated ICP due to an acute pICH, supine positioning for DSA can exacerbate intracranial hypertension. A lie flat trial (head of the bed at 0° for up to 30 minutes) before transporting the patient to the angiography suite can help determine whether the child will tolerate the procedure. When safe from an ICP standpoint, diagnostic investigations with DSA in the first 1 to 2 days in younger patients with no known preexisting risk factor should be considered due to the high prevalence of readily identifiable structural vascular lesions as the cause.[14,34] Delayed DSA can also show unexpected underlying structural lesions in patients without radiological suspicion of secondary causes of ICH, and therefore, a follow-up DSA should be considered for all patients when their first DSA does not identify the cause of their pICH and there is not another specific cause found for their ICH (Figure 1).[35,36]

Statement 7: Cerebral DSA should be performed on all children with no obvious cause on initial vascular workup or to better characterize vascular lesions identified on MRA or CTA. DSA should be performed, when available, by an experienced clinician.

Is There a Role for Systematic Imaging Follow-Up at the Acute Phase?

In children with ICH, the development of new neurologic symptoms should prompt immediate follow-up imaging by either MRI or CT. The decision to perform routine follow-up imaging in patients with pICH without a declining neurologic examination is not straightforward. The risk of rehemorrhage in the acute period is reported to be as high as 32% and depends on the underlying cause.[37,38] Additionally, hematoma expansion from continued bleeding increases the risk of a poor clinical outcome in pICH. Hematoma expansion occurs in approximately one-third of patients, as assessed between 48 and 72 hours following baseline imaging.[37,38] The risk of hematoma expansion is increased in children with platelet or coagulation abnormalities. This should be weighed with the concerns of radiation exposure and frequent need for sedation. Protocols for follow-up imaging of children at risk for clinical deterioration secondary to hematoma expansion do not currently exist.[39] Timing of repeat imaging should be individualized and depends on the type of hemorrhage, need to identify the cause, anticipated sequelae, and the patient's clinical and neurological status.[40,41] Follow-up imaging with a low-dose CT protocol could help identify children with postadmission hemorrhage expansion. For patients with associated intraventricular hemorrhage, the role of follow-up imaging is also focused on assessing hydrocephalus, which may require cerebrospinal fluid diversion.

Statement 8: Children at high risk for hemorrhage expansion, for example, with platelet or coagulation abnormalities, should be considered for repeat brain imaging with CT or MRI within 48 hours, even if there are no new symptoms.

Statement 9: Children with stable neurological examinations with normal mental status who are not at high risk of hemorrhage expansion do not systematically require repeat brain imaging with MRI or CT at prespecified early time points.

Is Genetic Testing Recommended in Children With pICH?

Knowledge of genetic causes may aid in post-acute targeted treatment strategies and inform family planning decisions.[42–44] Cerebral vascular malformations

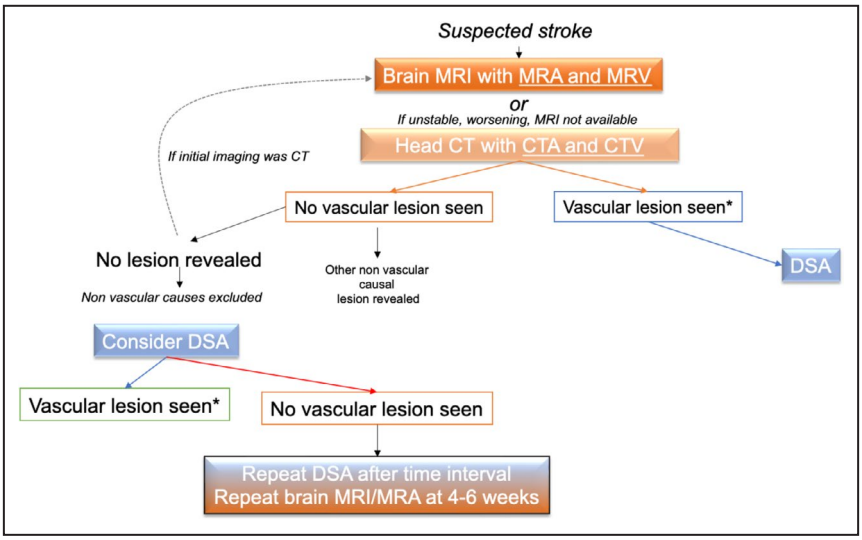

Figure 1. Proposed imaging protocols for children with suspected ICH.
Except if the vascular lesion is a cerebral cavernous malformation, then a DSA is not needed*. CT indicates computed tomography; CTA, computed tomography angiography; CTV, computed tomography venogram; DSA, digital subtraction angiography; ICH, intracerebral hemorrhage; MRA, magnetic resonance angiogram; MRI, magnetic resonance imaging; and MRV, magnetic resonance venogram.

are the most frequent causes for pICH, accounting for approximately half of the reported cases.[14,45] Genetic conditions predisposing individuals to vascular malformations include familial cerebral cavernous malformations and hereditary hemorrhagic telangiectasia.[46,47] A history of epistaxis or gastrointestinal bleeding in family members or a child with an AVM suggests the possibility of hereditary hemorrhagic telangiectasia.[48–51] When hereditary hemorrhagic telangiectasia is suspected, physical examination includes a detailed mucocutaneous assessment for vascular malformations and telangiectasias. Other clinical clues that may suggest a Mendelian, monogenic cause include skin capillary malformations and or limb overgrowth in capillary malformation-AVM syndrome,[52] neurocutaneous findings suggestive of neurofibromatosis type 1, skin rashes such as livedo reticularis (suggestive of deficiency of adenosine deaminase 2), as well as evidence of connective tissue abnormalities.

AVMs, cerebral cavernous malformations, and intracranial aneurysms are among the most frequent causes for pICH. Genetic mutations associated with AVM include *RASA-1*[52] and *EPHB4*[52,53], *ENG*, *ACVRL1* (ALK-1[54]), *SMAD4*, and presumably *GDF2* (BMPR9).[55] Familial cerebral cavernous malformations are caused by *KRIT1* (CCM1), *CCM2*, and *PDCD10* (CCM3) gene mutations.[56]

As the majority of sporadic brain AVMs harbor activating *KRAS* mutations in nidal endothelial cells,[57] novel mechanism-targeted medical treatments mainly related to mitogen-activated protein kinase (MEK) inhibition can potentially halt vessel remodeling and prevent growth of AVMs. However, data on efficacy or safety in intracranial AVMs are limited.[58] Identification of somatic mutations within brain AVMs can be considered using various techniques, including endovascular biopsy of the endothelial wall of intracranial AVMs before a planned endovascular treatment session. This can be carefully considered in selected cases when clinical and radiologic features are not characteristic or when the AVM is not amenable for surgical or endovascular treatment.[59] Because obtaining tissue from fast-flow vascular malformation is not without risk, cell-free DNA for genetic diagnostic testing is increasingly being used, but the yield in intracranial AVMs is currently largely unknown.

When pICH from a ruptured intracranial aneurysm is confirmed on neuroimaging studies, a genetic underpinning could be further investigated. Monogenic entities, such as autosomal dominant polycystic kidney disease, Loeys-Dietz syndrome, Marfan syndrome, vascular Ehlers-Danlos syndrome type IV, and rarely Klippel-Trenaunay-Weber syndrome can manifest as single or multiple intracranial aneurysms. Each of these entities has unique genetic foundations, and genetic investigation should be performed according to the specific clinical stigmata.[60,61]

Multiple studies have revealed *COL4A1* (type IV collagen α1)[62] and *COL4A2* (type IV collagen α2)[63] mutations to be associated with pICH, which can occur during the neonatal or fetal period and occasionally later.[64] Both genes code for collagen proteins that ensure the integrity of the cerebral vasculature. Small

vessels are often abnormal due to the mutated protein and present as deep parenchymal ICH.[65] Large vessel aneurysmal disease was also reported in a subset of patients with *COL4A1*-related disorder, but this rarely causes hemorrhagic stroke, particularly in children.[66] Systemic manifestations suggestive of *COL4A* (collagen type IV α)-related disorders include ocular (cataracts, microcornea, Axenfeld-Rieger anomalies)[67] or renal involvement. For children without identified vascular abnormalities, coagulation pathway genes could be explored[68,69] following a demonstration of a primary coagulopathy by clinical and laboratory evaluation. Disorders like hemophilia, factor X or XIII deficiency, or Von Willebrand disease should be ruled out and may be associated with severe pICH.[70]

Statement 10: Personal and family history for children with ICH should be documented to identify children who should undergo specific genetic testing (eg, for hereditary hemorrhagic telangiectasia, familial cavernous malformation syndromes).

Statement 11: For children whose vascular malformations are resected and in whom a germline mutation is not suspected, somatic testing of the lesion should be considered.

Is Workup for Coagulopathy Needed for Children With ICH? Who Should Get It, Which Laboratories to Send?

For children without an identified cause of hemorrhage after a thorough workup aimed at identifying a structural causal lesion, an inherited or acquired hematologic cause of pICH should be excluded. The identification of directly correctable risk factors, such as thrombocytopenia or coagulopathy, will facilitate rapid treatment, thereby limiting the risk for hematoma expansion and secondary brain injury. Hemophilia A (factor VIII deficiency) or B (factor IX deficiency) and von Willebrand disease are the most common inherited causes of hematologic causes of pICH, accounting for nearly 90% of hematologic disorders causing ICH in children.[71] Rarer forms (3%–5%) include factor VII deficiency, factor II, factor XIII deficiency, and vitamin K-dependent clotting factor deficiency.[71] Among acquired hematologic disease, idiopathic thrombocytopenic purpura and anticoagulant therapy-associated hemorrhage are the most frequent causes of pICH.[72,73] Anticoagulant therapy is particularly common in children with cardiac diseases, especially if requiring mechanical circulatory support including ventricular assist device and extracorporeal membrane oxygenation.[72] If a bleeding disorder is suspected, a complete blood count with platelets, coagulation studies, prothrombin time, international normalized ratio, and activated partial thromboplastin time, as well as higher-level studies including factor VIII, IX, and XIII levels and von Willebrand disease studies, should be performed. When a bleeding disorder is already known, a complete blood count with specific coagulation studies can be performed to help rapid correction.

NEUROCRITICAL CARE AND MEDICAL MANAGEMENT

The neurocritical care management of acute ICH requires a balance between ensuring adequate cerebral perfusion in the setting of increased ICP and mitigating the risk of rebleeding, all of which may be complicated by impaired autoregulation, disruptions in the blood–brain barrier, and systemic complications.[74]

General Principles of Acute Management

Early and consistent involvement of an interdisciplinary team can facilitate best practices for patient care. Establishing a cause of pICH is critical to prevent bleeding recurrence, improve outcome, optimize management, and develop follow-up strategy.

What Measures Should Be Used for Neuroprotection?

Pediatric studies in neuroprotection are lacking. Current practices are either extrapolated from adult guidelines or based on small, mainly retrospective, pediatric studies and expert opinions.[5,20,75] Neuroprotective measures should begin once a neurological/neurosurgical emergency is suspected and follow the principles of optimizing cerebral perfusion and decreasing oxygen consumption (eg, by managing pain and seizures) while obtaining a diagnosis and monitoring for specific complications.

The neuroprotective management of pICH follows similar principles as other types of brain injuries and focuses first on stabilizing the patient, assessing for the presence of elevated ICP, optimizing cerebral perfusion pressure, and identifying immediately correctable risk factors such as thrombocytopenia or coagulopathy (Figure 2).[4,37,38] An interdisciplinary approach is important for optimal acute management.

ICP Management

Monitoring for signs of increased ICP is imperative. ICP may become elevated in pICH due to the mass effect from the hemorrhage or from obstructive or communicating hydrocephalus from intraventricular hemorrhage.[76,77] In the unconscious, sedated, or intubated and ventilated children, signs of raised ICP are not always reliable; therefore, consideration of invasive ICP monitoring should be strongly considered and discussed with neurosurgery.[78] To avoid deterioration and secondary brain injury, the head

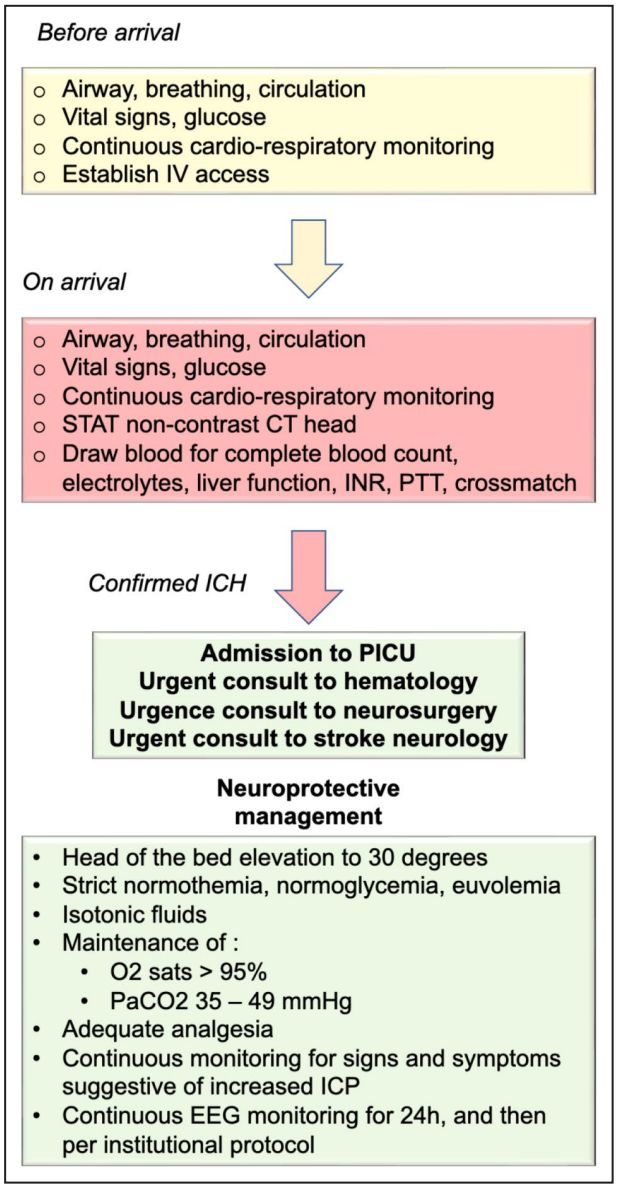

Figure 2. Neuroprotective measures in the context of suspected and confirmed pICH.
CT indicates computed tomography; EEG indicates electroencephalogram; ICP, intracranial pressure; INR, international normalized ratio; pICH, pediatric intracerebral hemorrhage; PICU, pediatric intensive care unit; and PTT, partial thromboplastin time.

should be elevated to 30°[79] and maintained in a midline position to facilitate venous drainage, followed by strict control of arterial carbon dioxide to prevent excessive vasoconstriction/vasodilation.[80] Because the patient's head of bed is typically placed at 0° during DSA, a patient's current ICP control should be considered when deciding on the timing of DSA. As noted above, a lie flat trial in the intensive care unit can help determine whether a child will tolerate the supine position without elevated ICP. Neurosurgical interventions, including external ventricular drainage, hemorrhage evacuation, and craniectomy are discussed in the dedicated neurosurgical management section.

Nonsurgical interventions for increased ICP include administration of hyperosmolar therapy such as intravenous mannitol or hypertonic saline.[78] Plasma osmolality and electrolytes need to be closely monitored during hyperosmolar therapy to avoid hypotension and hypovolemia. To control ICP, pain should be prevented by adequate analgesia, and in intubated patients, shivering should be controlled with muscle relaxants. Hyperventilation can be used as a temporizing intervention but must be used with caution, because hyperventilation may cause worsening cerebral ischemia.[80]

Blood Pressure Management

Evidence is lacking about blood pressure management goals after pICH. The role of blood pressure reduction to limit hemorrhage expansion and avoid rebleeding in pICH is unknown. Excessive hypertension should be avoided, although exact blood pressure measurements associated with increased bleeding risk in children are unknown. Careful lowering of systolic blood pressure to a target of the 95th percentile for age, sex, height, and weight is reasonable while avoiding hypotension, which could inadvertently reduce cerebral perfusion leading to secondary brain injury.[20]

Other Neuroprotective Measures

Fever during the first 72 hours has been independently associated with poor outcomes in adult patients with ICH.[81] Therefore, temperatures >37°C should be aggressively treated with antipyretics and cooling blankets. Euvolemia and normoglycemia should be maintained and treatment with glucocorticoids avoided, because randomized controlled trials in adults with ICH showed no neuroprotective benefits[82,83] and a potential for subsequent hyperglycemia, which can worsen brain injury.[84,85]

Treating comorbidities such as coagulopathy, anemia, endocrine and electrolyte disturbances, as well as preventing in-hospital complications such as infections or deep vein thrombosis is important, because these contribute to poorer outcomes.[86] Because patients with pICH have high rates of invasive procedures and often require a prolonged pediatric intensive care unit admission,[87] treatment of other acute brain injury-related complications include pain management, mood disturbances, delirium, and sleep–wake cycle abnormalities.[88,89] Post-intensive care syndrome[90] can have a profound effect on a patient's health-related quality of life.[91]

Statement 12: An interdisciplinary approach is important for optimal acute management. The team should include, when possible, a pediatric intensivist,

neurosurgeon, pediatric neurologist preferentially a pediatric neurologist with stroke expertise, neurointerventionalist, neuroradiologist, and hematologist when no underlying vascular lesion is identified or when a platelet or coagulation disorder is present.

Statement 13: Signs of raised ICP are not reliable in unconscious, sedated, or intubated and ventilated children. Therefore, invasive ICP monitoring should be strongly considered and discussed with neurosurgery for children in whom increased ICP may occur but may not exhibit clear signs or symptoms.

What Is the Role of Electroencephalogram Monitoring in a Child With ICH? When? How Long?

Children with acute ICH are at risk of seizures,[92–94] which may, in turn, increase cerebral metabolic demand,[95] contribute to ongoing neuroinflammation,[96] and disrupt normal physiologic regulatory mechanisms, thereby causing secondary ischemia and exacerbating the underlying brain injury. In a mixed cohort of critically ill children, a maximum hourly seizure burden >12 minutes was associated with significant neurologic decline,[97] and evidence of seizure burden impacting outcome in adults with subarachnoid hemorrhage is accumulating.[98]

Although the impact of continuous electroencephalogram (EEG) monitoring after pICH on the detection and management of electrographic-only and subtle clinical seizures has not been well-studied, guidelines and consensus summary statements recommend that clinicians consider continuous EEG monitoring in children with acute supratentorial brain injury, including ICH, and altered mental status[99,100] as well as in those with abnormal movements and/or vital sign derangements concerning for seizure.[20,99,101,102] The ideal duration of monitoring for seizures in a critically ill child depends on the clinical scenario and EEG risk factors on the initial 1-hour recording. Of patients with nonconvulsive seizures, 80% to 95% can be identified within 24 to 48 hours,[92,103–106] and recording for at least 24 hours is recommended by the American Clinical Neurophysiology Society.[100] In patients who are younger, comatose, pharmacologically sedated, have periodic discharges, or an otherwise abnormal EEG background, nonconvulsive seizures may occur later, and monitoring for 48 hours or more may be necessary.[103,107,108]

In addition to detecting seizures, an EEG may identify new or ongoing cerebral ischemia, thereby facilitating neuroprotective strategies to optimize cerebral perfusion. This may be particularly relevant in the management of pICH when trying to balance cerebral perfusion and the risk of recurrent hemorrhage. The EEG background undergoes a predictable series of changes in the setting of decreasing blood flow,[109] and this precedes clinical symptoms indicative of the onset of delayed cerebral ischemia in adults with aneurysmal subarachnoid hemorrhage, thereby allowing for timely intervention.[110–112]

Statement 14: Continuous EEG monitoring should be considered for the first 24 to 48 hours after ICH in children who are not awake and alert or in whom mental status or neurological examination are fluctuating.

Statement 15: Clinicians should have a low threshold for continuous EEG monitoring, particularly in young infants with altered mental status, unexplained tachycardia, or unusual movements.

What Is the Role of Seizure Prophylaxis in a Child With ICH? For How Long?

The optimal duration of antiseizure treatment in pICH is unknown and depends on the clinical scenario, the electroencephalographic features, and the development of intracerebral posthemorrhagic complications. However, their use may be warranted if EEG monitoring is not readily available and the child does not have a reliable examination, because seizures are frequently reported in children with pICH.[113]

Studies that have examined prophylactic antiseizure medications (ASMs) in adults found that those treated had an increased risk of death and disability, hence prophylactic ASM is not recommended in this age group.[4,114–119] Furthermore, a small trial that randomized patients to 1 month of valproic acid did not demonstrate a reduction in seizures at 1 year.[120] A meta-analysis of 8 studies, which were mostly observational, found that prophylaxis did not prevent seizures <14 days from pICH or at longest follow-up.[121]

Few retrospective or prospective studies address epilepsy after pICH, and there are no clinical trials that examine the efficacy or safety of ASM prophylaxis. In a prospective cohort of 73 pediatric patients with spontaneous ICH, 19 of 53 children (36%) had seizures at presentation.[113] Of 7 children who had acute symptomatic seizures after presentation but within 7 days of hemorrhage, 4 had seizures despite treatment with an ASM. Four children did not have an acute symptomatic seizure yet were discharged on a prophylactic ASM, all of whom had vascular malformations. Nine of 67 survivors developed epilepsy (8 children and 1 neonate), with a 2-year epilepsy-free survival rate of 87%. The only risk factor for epilepsy was elevated ICP requiring surgical management. Of the 9 who developed epilepsy (8 children and 1 neonate), 5 had continued taking antiseizure medications after hospital discharge, a finding that indicated that continuation of ASMs does not prevent future seizures in all patients. Elevated ICP requiring intervention at the acute phase may increase the risk of developing remote symptomatic epilepsy.[113]

Out of the 53 children in the study, 27 were loaded with an ASM. In the 27 children loaded with a ASM, 5 children were loaded with phenobarbital (all aged <2 years), 11 children with phenytoin, and 11 children with levetiracetam. A majority of children who were started on a maintenance ASM in the hospital were started on levetiracetam (25 out of 30), and a majority of children who were discharged on an ASM were discharged on levetiracetam (21 out of 26).[113] This is not surprising given that levetiracetam has a better safety profile and similar efficacy in children presenting with benzodiazepine refractory status epilepticus compared with phenytoin.[122] Lacosamide is another ASM that has been used in critically ill children with minimal side effects.[123]

Statement 16: Although there are no data to support prophylaxis with ASMs for children with ICH without acute symptomatic seizures, it may be reasonable to use a short course of an ASM with a relatively benign side effect profile like levetiracetam for 1 to 2 weeks in children who are high risk for seizures or in whom a seizure might be particularly deleterious. Children at high risk for seizures may include those with altered mental status, those with elevated ICP, or those with large, untreated vascular malformations.

What Is the Role of Transcranial Doppler Ultrasound in the Management of an Acute pICH?

Children with pICH are prone to increased ICP and its potential deleterious effects. Invasive ICP monitoring is the most common method for direct ICP assessment. Transcranial Doppler ultrasound is a noninvasive technique using acoustic windows of the skull to interrogate intracranial vessels and has been applied in attempts to noninvasively estimate ICP and cerebral perfusion pressure. In a survey of multiple pediatric intensive care units, the 2 most common (more than one-third of respondents) management changes after performing transcranial Doppler ultrasound were making decisions about performing additional neuroimaging and manipulation of cerebral perfusion pressure with fluids and vasopressors.[124]

The use of noninvasive detection methods, such as transcranial Doppler ultrasound as well as continuous EEG monitoring, may aid in distinguishing between compensated hydrocephalus with normal ICP and slowly progressive hydrocephalus with increased ICP[125] and may identify vasospasm if there is an associated subarachnoid hemorrhage.[126] Limitations in the routine use of transcranial Doppler ultrasound for vasospasm detection in children include lack of established criteria for vasospasm (age-dependent cerebral blood flow velocities, Lindegaard ratio), rarity of abundant subarachnoid hemorrhage outside the context of pure subarachnoid hemorrhage due to aneurysm rupture, poor characterization of the duration of risk for vasospasm,[124] and lack of interoperator reproducibility.

Statement 17: Transcranial Doppler ultrasound has not been studied adequately in intracerebral hemorrhage; therefore, there are insufficient data to provide firm recommendations for its use.

Timing and Principles of Anticoagulation Resumption After pICH

When to resume anticoagulation presumably depends on the cause of ICH, associated conditions (prosthetic cardiac valve), and remains controversial, even in the adult population. Careful assessment of the risk–benefit ratio is required in children on mechanical cardiac support, because clinicians may not feel it is possible to stop anticoagulation in these children. Intensivists might adjust left ventricular assist device parameters to ensure full ejection, with the goal of minimizing thrombus formation during reduced or no anticoagulation. Hematologic consultation is recommended to optimize and monitor coagulation parameters following identification of pICH, including monitoring daily lactate dehydrogenase and plasma hemoglobin levels to identify intrapump thrombus formation and hemolysis.

NEUROSURGICAL MANAGEMENT

In Children With Acute Intracerebral Hemorrhage, What Are the Clinical and Imaging Criteria for External Ventricular Drainage Placement?

Hydrocephalus is a strong determinant of poor outcome following pICH,[29,30,127] occurring in almost half of children during the initial hospitalization.[128,129] Acute hydrocephalus is a major driver of raised ICP and is a therapeutic target in the context of pICH. There is no high-level evidence for the selection of children with ICH for external ventricular drainage, but retrospective data indicate lower Glasgow Coma Scale score on admission (typically <8), visibility of temporal horns of ventricles, and higher modified Graeb scores (a semiquantitative scale for intraventricular hemorrhage volume measurement)[130] are strong predictors for the need of external ventricular drainage placement.

Given the risks of rapid deterioration due to increasing ICP in the context of hydrocephalus, external ventricular drainage placement should be considered before transfer whenever feasible and clinically indicated.

Statement 18: In children with acute ICH, the presence of altered consciousness, neurological

deterioration, hydrocephalus, or intraventricular blood on initial or subsequent neuroimaging should prompt consideration of external ventricular drainage.

In Children With Acute Intracerebral Hemorrhage, What Are the Clinical Imaging Criteria for Hemorrhage Evacuation/Hemicraniectomy?

Hemorrhage evacuation can play an important role in reducing ICP and secondary brain injury due to mass effect in patients with ICH in the posterior fossa and large or symptomatic subcortical lobar hemorrhages.[129,131] No high-level evidence exists in the pediatric population, but retrospective data have shown that despite more severe clinical presentations, children who were treated with hemorrhage evacuation and/or hemicraniectomy experienced similar long-term functional outcomes and mortality rates as those who did not receive these interventions.[129,132] In turn, it is important to consider hemorrhage evacuation in patients with significant midline shift, altered mental status, acutely worsening symptoms, or threatening cerebral herniation (temporal, tonsillar) due to mass effect. Hemicraniectomy may be considered in children with refractory increased ICP despite medical treatment optimization, hemorrhage evacuation, and adequate management of hydrocephalus.[129,132,133]

Statement 19: In children with acute ICH requiring decompression due to raised ICP and/or brain herniation, hemorrhage evacuation and hemicraniectomy should be considered.

Acute Surgical Management of the Underlying Cause?

In children with acutely ruptured AVMs, acute nidal resection is often not justified because of the low risk of early rebleeding proportional to the operative risks in an acutely hemorrhagic context.[129,134] Exceptions may include superficially located nidus, especially in low-grade lesions for which hemorrhage evacuation is performed[135] and when complete nidal resection is deemed feasible by an experienced team.[134] In patients with acutely hemorrhagic cavernomas identified on initial imaging, microsurgical excision is often performed within the first 2 weeks.[136]

Statement 20: In children with acute ICH caused by an AVM, acute surgical excision of the AVM is typically avoided unless there are high-risk features for early rerupture. When the causal lesion is a cavernous malformation, surgical excision is commonly performed in the first weeks following the hemorrhage, whenever a favorable risk–benefit ratio exists.

INTERVENTIONAL NEURORADIOLOGY

Considerations for Facilities Without Pediatric Neurointerventional Capability

Children who arrive at a facility without the capability for safe performance and/or interpretation of DSA should be transferred to a pediatric neurointerventioanl radiology (NIR) capable facility/center after stabilization within 24 hours.[16] Although it is well understood that dedicated pediatric NIR is rare, the capacity to treat children is important. If a facility is unable to provide the complete spectrum of cerebrovascular care to a child (eg, external ventricular drainage, DSA, embolization, clipping, AVM resection, vasospasm treatment, intensive care unit, and neurosurgical care), patients should be promptly transferred to a center that has these capabilities. For pediatric patients who are unable to undergo transfer due to ongoing instability and elevated ICP, it is advisable that repeat and/or advanced noninvasive imaging be performed and the patient transferred when stable.

General Principles for Therapeutic Angiography

Children who present with a pICH with a vascular cause and were diagnosed with noninvasive imaging should undergo DSA with a plan for intervention, unless the clinical condition requires urgent evacuation of the hematoma and decompressive craniectomy.[129] The proposed intervention depends upon the cause and the findings of the initial DSA runs.

For pICH secondary to high-flow vascular malformations such as AVM and arteriovenous fistulas, DSA is performed to define angioarchitecture of the vascular malformation for surgical planning but more importantly to identify and treat characteristics that suggest imminent rerupture/rebleed (eg, flow-related parent artery aneurysms and/or intranidal aneurysms).[16]

For pICH secondary to moyamoya, the goal of therapeutic DSA is to identify ruptured aneurysms in need of treatment, but it also can be used as a diagnostic tool to assess the severity of disease, external carotid artery collaterals, and related hemodynamics.[137]

In cases of pICH resulting from venous infarction due to cerebral venous thrombosis, and in the presence of clinical deterioration despite optimal medical therapy, such as worsening neurological status, refractory intracranial hypertension, or progressive infarction on imaging, one may contemplate the use of conventional DSA with potential interventions, including mechanical thrombectomy and/or thrombolysis that is in concordance with the American Heart Association's cerebral venous thrombosis consensus statement.[138]

We propose the standard of care for a readily identified vascular lesion amenable to endovascular therapy is management within 24 hours of diagnosis or following respiratory, hemodynamic, and ICP stabilization.

pICH With Concurrent Causes Where Urgent Conventional/Therapeutic Angiography Is Not Indicated

In the case of pICH secondary to cavernous malformation, brain tumor, or other readily identified nonmacrovascular cause, urgent DSA may not be necessary. Diagnostic and therapeutic cerebral angiography may be performed for preoperative evaluation and/or embolization of certain brain tumors (eg, hemangiopericytomas, intraventricular meningiomas), as per local expertise and neurosurgical anticipation of the perioperative bleeding risk. Last, DSA for diagnostic purposes only is not recommended for children aged <3 months. If DSA must be pursued in these patients, the purpose should be to perform an intervention.[16]

Timing of Angiography in Unstable Children or With Raised ICP

Priority should be given to respiratory and hemodynamic stabilization. Noninvasive imaging should be performed if it does not jeopardize stabilization efforts. If the patient has clinical and/or radiographic signs of increased ICP, strategies to reduce ICP should be attempted before DSA. DSA should not be performed in a hemodynamically unstable patient or with steadily increasing ICP, except in the rare case where it is required before surgical decompression.

Statement 21: Children with pICH who have a definite cerebral cavernous malformation identified as the cause on noninvasive imaging do not require a cerebral angiogram in the majority of cases.

Neurointerventional Access in Low-Resource Settings

Pediatric patients who present with ICH to hospitals without access to DSA should be transferred to capable centers, as mentioned previously, when stabilized. The increasing rise of centers performing thrombectomy for stroke management in low-income countries offers increasing opportunities for children with pICH to be transferred to centers capable of neurointervention. Establishing pathways and infrastructure in advance to help facilitate access to these resources is recommended. If transfer is not an option, clinicians should get in contact with an experienced team and consider repeat noninvasive imaging as often as needed to best determine the patient's medical care and avoid poor outcomes.

CONCLUSIONS

We have provided comprehensive updates on the acute diagnosis and management of pICH, with a specific focus on integrating current literature and expert consensus into clinical practice. Our aim is to equip health care professionals with the most current and relevant information, addressing existing controversies and identifying gaps in knowledge. This scientific statement is intended to serve as a resource for practitioners involved in the acute care of children presenting with ICH, ensuring informed and effective clinical decision-making. Further prospective, multicenter, international studies are needed to facilitate evidence-based practice.

ARTICLE INFORMATION

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EXPERT-CONSENSUS REPORT

Pediatric Intracerebral Hemorrhage Management—Consensus Statement of the International Pediatric Stroke Organization—Part 2: Outcomes, Rehabilitation, and Transition to Adulthood

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ABSTRACT: Pediatric hemorrhagic stroke can lead to significant neurologic, cognitive, and behavioral morbidities that often emerge over time and can impede long-term academic, vocational, and socioemotional function. While many of the existing data stem from studies in arterial ischemic stroke, functional outcomes in hemorrhagic stroke, and particularly pediatric intracerebral hemorrhage, remain largely understudied. Extrapolating findings from ischemic stroke can be challenging, as there are notable differences in care and potentially in outcomes for hemorrhagic stroke. The primary goal of this consensus statement by a multidisciplinary group of stroke experts is to provide a review of the current literature on neurologic, cognitive, behavioral, and socioemotional outcomes after hemorrhagic stroke. Neurologically, children with pediatric intracerebral hemorrhage often experience motor deficits, including hemiparesis and coordination issues, as well as cognitive impairments affecting attention, memory, and executive function. Behavioral and emotional problems, such as depression, and social difficulties can also occur. Data on academic attainment are also presented, along with considerations regarding long-term outcomes and the transition to adulthood. We further examine a variety of key determinants predicting outcomes, including medical, demographic, familial, and socioeconomic factors, as well as current research on rehabilitation, with an emphasis on gold-standard guidelines for clinical interventions. Given the complexity of outcome measurement in pediatric hemorrhagic stroke and the lack of uniform tools for assessing outcomes across diverse populations, we propose guiding principles for outcome measurement, along with examples of domain-specific tools. Finally, we discuss the limitations of the current literature and outline goals for future clinical practice and research.

Key Words: behavioral and socioemotional morbidities ■ neurologic and cognitive outcomes ■ outcome measurement ■ pediatric hemorrhagic stroke ■ rehabilitation

See Editorial by Jordan and Kirton.

Over the past two decades, our understanding of person-centered outcomes following arterial ischemic stroke (AIS) in children and newborns has dramatically improved. However, our knowledge of the neurologic, cognitive, behavioral, and functional outcomes following pediatric hemorrhagic stroke

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CLINICAL PERSPECTIVE

What Is New?

- Children with pediatric intracerebral hemorrhage often experience maladaptive neurologic, cognitive, behavioral, and socioemotional outcomes that can affect their learning and academic progress, social development, and independence in daily living.
- Key determinants for predicting outcomes in pediatric intracerebral hemorrhage include medical, demographic, familial, and socioeconomic factors.

What Are the Clinical Implications?

- Neurological examination and comprehensive neuropsychological assessment are critical first steps in planning interventions and should be a routine part of outcome evaluation to ensure timely intervention and consequently improved neurological function (eg, motor, visual), learning, academic achievement, emotional adjustment, and development of independence.

Nonstandard Abbreviations and Acronyms

ABI	acquired brain injury
AIS	arterial ischemic stroke
HS	hemorrhagic stroke
pICH	pediatric intracerebral hemorrhage

(HS) is much more limited even though it comprises >50% of childhood strokes[1] and ranks among the top 10 causes of pediatric deaths.[2] HS consists of intracerebral hemorrhage (ICH), defined as nontraumatic intraparenchymal hemorrhage with or without intraventricular bleed, and subarachnoid hemorrhage. In this consensus statement, we focus primarily on pediatric intracerebral hemorrhage (pICH) and state where the limited outcomes data also included other types of HS.

The field of pediatric HS has shifted its focus from survival to survivorship, including monitoring for challenges and focusing on appropriate supports, rather than just describing the neurological sequelae in more general terms, such as *good* or *poor*.[3,4] This shift has led to local, national, and international interdisciplinary initiatives to address knowledge gaps related to pICH outcomes in children.[1] Although death after pICH is lower than in adults,[5] it remains significant compared with childhood AIS or even compared with the burden of pediatric diseases overall.[2,3,6,7] There are important differences in the initial provision of care between hemorrhagic and ischemic strokes including the involvement of different interdisciplinary teams. Specifically, children with pICH frequently require neurosurgical or interventional neuroradiological management in addition to neurocritical care. Since pICH encompasses a heterogeneous group of conditions manifesting across a broad developmental range, from infancy to adolescence, studying outcomes remains complex. This challenge necessitates an adaptive approach, including long-term assessment in multiple domains with age-related adjustments. The chronic care continuum includes a wide range of interdisciplinary patient- and family-centered approaches such as rehabilitation therapies, neuropsychology, and behavioral therapies to support the optimization of outcome and development. This comprehensive focus on long-term outcomes highlights the need for further coordinated research and quality improvement in this field.

To date, HS-specific reports of long-term outcomes are scarce and even less studied in pICH alone. Studies are retrospective, cross-sectional, and based on small samples,[8–10] with data often derived from heterogeneous HS populations. Furthermore, they also frequently combine several conditions (eg, AIS, HS, or more broadly all types of acquired brain injury [ABI]), and often define the HS population with notable variability [eg, intracerebral hemorrhage, subdural and subarachnoid bleeds; or ruptured and unruptured arteriovenous malformation [AVM]). Thus, reliable evidence-based neuroprognostication remains challenging.

Here, members of the Hemorrhagic Stroke Working Group of the International Pediatric Stroke Organization present a summary of the limited available data on motor, cognitive, emotional, and adaptive outcomes following pICH. While all referenced studies were focused on pICH, due to the paucity of published data, some also included other types of hemorrhage (eg, subarachnoid). We propose general guidelines regarding interventions aimed at children and adolescents with ABI, including stroke, as these apply to HS/pICH as well. Finally, we provide guidance for measuring outcomes in both clinical practice and research settings.

NEUROLOGIC, MOTOR AND GLOBAL FUNCTIONAL OUTCOMES

Although improvement has been reported over the first year following pICH,[11] most children (≈75%) have persistent neurological deficits. One of the largest HS outcome studies (the majority with pICH) found that up to 48% of children with HS occurring between ages 1 and 20 years had no long-term motor impairment, but hemiparesis was the most frequent pattern of weakness in those with motor impairment (≈36%).[12] Another outcome

study including 106 children with ruptured AVM measured clinical outcomes using the King Outcome Scale for Childhood Head Injury; 76% had a good outcome, 13% had mild disability, 4% had a severe disability, 2% remained in a persistent vegetative state, and 5% did not survive.[13] Risk factors for unfavorable outcomes included volume of intracranial hemorrhage (≥30 cm^3) and development of hydrocephalus.[13] The significant uncertainties and potential for ongoing recovery are important to consider when planning care and discussing neuroprognostication with families.

A retrospective study from a pediatric rehabilitation center reported better motor and functional outcomes in children admitted for acute rehabilitation after HS (n=82) compared with those after AIS (n=46).[14] The HS patients in this cohort all had pICH (the large majority from ruptured AVM, 4 from ruptured cavernoma, 5 from ruptured aneurysm, and 5 with "unexplained" pICH) (author personal communication). Even though impaired adaptive skills are seen in about half of pediatric HS survivors, most children reach at least some level of functional independence in activities of daily living.[12,15,16]

COGNITIVE, BEHAVIORAL, AND SOCIOEMOTIONAL OUTCOMES

About half of children with HS experience cognitive and adaptive deficits,[12,15,16] impeding their academic and vocational outcomes. In a study of 34 children with spontaneous intracerebral hemorrhage, even those with seemingly "good recovery" on global outcome scales experienced cognitive and behavioral problems.[17] Memory and attention difficulties are common and reported in up to 45% of survivors of pediatric pICH.[17,18] Murphy et al found longitudinal improvement in some higher-order intellectual function and overall neurologic function in 7 children with pICH.[11] In contrast, cognitive proficiency (eg, processing speed, working memory) was impaired and showed a further decline over time, suggesting these children were not progressing in an age-expected manner in all aspects of executive functions.[11] Children with ruptured AVMs performed poorly on language-based measures of working memory, verbal fluency, and aphasia screening; their performance was worse than that seen in patients with severe traumatic brain injury,[19] implying more diffuse brain injury than initially apparent. A recent large rehabilitation center–based retrospective study including a mixed sample of 79 children with AIS and 105 with HS (the large majority with pICH due to AVM) found overall intellectual ability falling in the low average range.[20] Moreover, up to 53% of children exhibited impairments in formal language assessment of lexical and syntactic expression, as well as comprehension.[20] School function was strongly associated with performance on language and intellectual assessments. The authors further reported that after >3 years of median follow-up, only 27% of children did not require any educational support, while another 27% needed special education over time.[20] No significant differences in intellectual ability between AIS and HS were found in a subsample of 128 children from the same cohort, which included 82 children with HS (the large majority with pICH).[14]

Most HS survivors experience low self-esteem, and emotional and behavioral problems, even years after the injury.[12,21] In a case series of 5 children with ruptured AVMs, adaptive functions remained below age expectation, and parents frequently identified concerns in their child's social skills despite seemingly good initial adjustment.[22] In contrast, a longitudinal study of 29 children with ruptured and unruptured AVMs demonstrated "favorable" outcomes in social functioning, with most returning to school or work settings, despite persistent cognitive difficulties.[18]

ACADEMIC/RETURN TO SCHOOL

Impaired academic performance after childhood HS is commonly observed as a consequence of a combination of motor, cognitive, behavioral, and adaptive deficits. While most children return to school within a year, more than half require adaptations, additional support or special educational interventions in the long term.[14,23] In one study of patients with pICH, persistent motor deficits as measured by the Pediatric Stroke Outcome Measure at 3 and 12 months after stroke accurately predicted the need for educational support.[23] However, another study of mixed types of HS (mostly pICH) found that when outcomes were adjusted for general intellectual function, intellectual function remained the only robust significant predictor of the need for special education services in the long term.[14]

TRANSITION TO ADULTHOOD

Very little research is available on how children with HS, or pICH specifically, function over time as they progress into adulthood. A long-term follow-up study of 29 young adults with a history of either childhood ischemic stroke (2/3) or HS (1/3) found that 88% of survivors of stroke graduated from high school, although 45% required special education, and 64% of those aged >18 years were attending college. Furthermore, 79% of those aged >16 years were driving, and 60% were employed. In contrast, only 28% of those aged 18 years were living independently, away from their parents, and only 2 young adults in the cohort were financially independent. Mobility outcomes were overall good, while functioning

fell in the low to moderate range for communication, activities of daily living, and socialization.[24] For comparison, a recent study reporting long-term outcomes for survivors of pediatric AIS reported impairments in executive functioning and work productivity, yet no differences in quality of life, depression, or fatigue compared with healthy controls.[25] However, some studies suggest that mood problems can persist into adulthood and impact long-term outcomes and independence. One report described mood problems, such as depression and anxiety requiring treatment in more than a quarter of young adult survivors of AIS, despite independence in driving, relationships, and employment for most patients.[26]

DETERMINANTS OF OUTCOME AND NEUROPROGNOSTICATION

Limited evidence suggests that predictors of adverse outcomes following pICH include the severity of presenting symptoms (including decreased Glasgow Coma Scale score on admission), length of coma, volume of hemorrhage, infratentorial location of hemorrhage, presence of an aneurysm, need for neurosurgical intervention, rebleeding, length of stay in the pediatric intensive care unit, neurosurgical complications leading to increased intracranial pressure and the development of acute hydrocephalus.[8–10,27–29] The presence of coagulopathy and underlying hematological disease has also been associated with worse outcomes.[17,30,31] In another study, the rate of recovery of sensorimotor function after the acute phase was the best predictor of favorable outcome.[9]

Younger age at the time of the brain injury is associated with worse outcomes in some[17,20] but not all studies.[30,31] This discrepancy could be a result of variations in sample size, type of outcomes measured, and timing of the follow-up assessment. Specifically, younger age at time of stroke has been associated with poorer cognitive outcomes, a finding well established in childhood AIS.[32] Complex cognitive and socio-emotional functions develop over time, and therefore deficits in these domains may not become apparent until children are expected to meet developmental milestones through the school and social settings.

Personal, family, and environmental factors, such as lower premorbid functioning, lower socioeconomic status, and poorer parental mental health and family functioning, have been consistently associated with worse outcomes in childhood ABIs,[33] and some studies have confirmed these findings for pediatric stroke as well,[34–36] although data specifically for pICH remain limited.[37]

Regarding poststroke factors affecting outcome, epilepsy is known to have an adverse impact on outcomes after brain injury. This was specifically shown to be true in a mixed cohort of children with pICH, AIS, and perinatal stroke studied for cognitive and academic outcomes, which can in turn significantly impact the long-term quality of life.[38] Another study including 53 pediatric survivors of intracerebral hemorrhage from 3 tertiary centers across the United States found that a single remote symptomatic seizure occurred in 23% of survivors of pICH, and epilepsy developed in 13%, all within 2 years after the pICH.[39] The risk of remote symptomatic epilepsy was higher in survivors requiring surgical intervention for elevated intracranial pressure.[39]

As in other types of brain injury, neuroprognostication should be multimodal, combining information collected from neuroimaging, electroencephalography, and comprehensive multidisciplinary clinical exam. Key factors to consider include premorbid state, age at injury, laterality of injury, recurrent bleeds, and presence of an additional global insult or secondary brain injury. Despite high variability in outcomes, serial assessments (radiologic, neurologic, neuropsychological), adequately timed and tailored to the patient's progress, can help to improve the accuracy of short- and long-term prognostication.

REHABILITATION

The evidence base supporting improved outcomes with rehabilitation for pediatric HS is limited. Most available data stem from studies in AIS for motor rehabilitation (eg, constraint-induced movement therapy, hand–arm intensive bimanual training), and from traumatic brain injury/ABI (including HS) for cognitive/behavioral interventions (eg, attention/working memory training, behavior modification, family-focused interventions).[40,41] For many of these interventions, however, the data only show small to moderate effects in the near term and limited long-term and generalizable effects. Therefore, several clinical consensus guidelines have been developed globally to apply more general, integrative approaches.[42–44]

Limited evidence suggests that effective rehabilitation extends beyond current evidence-based training of function and includes compensatory strategies, environmental adaptation, and education of families and schools regarding childhood stroke outcomes. Guidelines now recommend (1) to include caregivers/educators and allied health professionals in rehabilitation; (2) to repeat neuropsychological assessments to capture changing developmental needs; and (3) to individually tailor interventions that foster *everyday* function. In the acute phase, initial rehabilitation is often multidisciplinary and hospital based. Adequate discharge planning is crucial and should include key family members and identified professionals from health, education, and psychosocial care to support rehabilitation and reintegration into

the home and school life. Assessments and interventions should consider core domains of the *International Classification of Functioning, Disability and Health: Child and Youth Version of the World Health Organization* (*ICF-CY*, 2007) and take child and family priorities and preferences into account. Interventions should be goal-oriented and adapted to individual and environmental factors (eg, developmental abilities, social, family, and educational demands).[45]

Common interventions include medications for symptom management (eg, anticonvulsants, mood stabilizers, botulinum toxin, baclofen); motor therapy (eg, occupational and physical therapy, constraint-induced movement therapy, bimanual training, serial casting, bracing, mobility and strength training; speech/language therapy (motor–speech, aphasia); educational interventions for cognitive and academic weaknesses (eg, tutoring/special educational instruction, classroom and test accommodations such as extended time, modified materials and curriculum); and psychological interventions for behavioral or socioemotional problems (eg, behavior therapy, counseling). Although limited, available studies on motor interventions in the acute and subacute phases indicate improvement in functional recovery.[46]

A central focus in "rehabilitation" after childhood stroke is the child's reintegration into age-appropriate environments (academic, social, family), and foremost return to school. Many children (up to 50%) require adaptations or special educational services after HS, often long term.[23] Thus, serial neuropsychological and rehabilitative assessments are important to monitor the child's overall cognitive development, independence, as well as learning and social integration, and identify needs as they arise, especially at major transition points (eg, entering school, moving to secondary school, transition to the workforce). This should occur regularly until the transition to adult services.[44,47] These assessments allow accurate determination of the current functional status in the context of the environment and collaborative intervention planning (medical, therapeutic, educational) according to the patient's and family's goals.

MEASURING OUTCOME

Currently, no validated outcome instruments exist specifically for HS. Measures for global outcome have been validated only in AIS (eg, Pediatric Stroke Outcome Measure, Pediatric National Institutes of Health Stroke Scale). Assessment of more specific functional domains is highly dependent on several factors including the child's age, development, level of impairment, the aims of the assessment (eg, initial assessment to inform rehabilitation, subsequent comprehensive neuropsychological assessment to inform school reentry), the outcome measures available in a country or language, and the clinician's judgment and observation (eg, need for adapted assessment in case of motor, visual, and speech impairments). Within this context, most "toolkits" rely on measures that may have been previously used in studies of stroke, regardless of their reliability and validity in a specific population or context (eg, short versus long term, single time point versus repeated testing, specific function studied). Therefore, "prescribing" a battery of specific measures/tools remains difficult; the appropriate tools vary largely on the basis of the outcome studied, age/developmental stage, and country (eg, measures developed in US populations with US reference norms are not generalizable to other countries).

To assess long-term outcomes in both the research and clinical context where variability in development and outcomes makes a uniform approach challenging, the following principles are suggested:

1. Longitudinal, repeated neuropsychological assessments and neurological examination. Close follow-up is needed to respond to clinical concerns, monitor skill development over time, and individualize SMART (specific, measurable, achievable, realistic, time-bound) goal-oriented interventions and supports provided during "critical windows" of development, including times of transition (ie, start of kindergarten, middle school, high school, college).
2. Use of norm-referenced measures for specific domains of study.
3. Use of "hallmark" measures most commonly administered for each domain to allow cross-comparison (see Table [48–147] for specific examples by domain of outcome).
4. Prospectively ascertained outcomes are preferred over retrospective ones in the research setting.
5. Implementation of longitudinal (repeated) over cross-sectional (single time point) protocols, with relevant long term follow-up (ie, every 2 years and at transition points) including long-term monitoring following patients into adulthood.
6. Clear HS definition and prospective follow-up of all patients with HS to avoid bias introduced by clinically referred samples.

KNOWLEDGE GAPS, IMPORTANT NEEDS, AND FUTURE DIRECTIONS

Despite an increasing body of literature on neurologic, cognitive, behavioral, and adaptive outcomes after

Table Specific Examples of Measures by Domain of Outcome.

Functional domain	Subdomain	Example measures*	Age range†	Method	Informant	Norms available
Global outcome	Overall level of disability severity Functional impairment	Functional Status Scale[48]	0–16	Rating scale–inpatient	Physician or trained health professional	No
		Hammersmith Infant Neurological Examination[49–53]	3–24 mo	Rating scale	Physician or trained health professional	No
		King's Outcome Scale for Childhood Head Injury[54,55]	2–16	Rating scale	Physician or trained health professional	No
		Modified Rankin Scale[56]	0–18	Rating scale	Physician or trained health professional	No
		Pediatric Cerebral Performance Category/ Pediatric Overall Performance Category[57–59]	0–18	Rating scale	Physician or trained health professional Medical records or caretaker's information can be used	No
		Glasgow Outcome Scale Pediatric Version (Adapted version of Glasgow Outcome Scale)[60,61]	0–18	Rating scale	Physician or trained health professional	No
		Pediatric Stroke Outcome Measure[62–66]	0–2, 2–18	Rating scale	Neurologist or trained health professional	Age norms
		Pediatric Stroke Recurrence and Recovery Questionnaire[67]	0–18	Rating scale	Parent, child	No
		Pediatric Functional Independence Measure	6 months – 7 years	Performance scale	Trained professional or parent or both	Age norms
Motor function	Gross motor	Action Research Arm Test[68]	13–18	Rating scale	Trained health professional	No
		Assisting Hand Assessment[69,70]/Hand Assessment for Infants/Mini-Assisting Hand Assessment[71]	18 mo–18 y/8–18 months (Mini- Assisting Hand Assessment)	Criterion reference test	Trained and certified health professional	Age norms
		Bruininks–Oseretsky Test of Motor Proficiency[72]	4–21	Performance measure, age-based standard scores	Trained health or research professional	Norm referenced
		Community Mobility and Balance Test[73–77]	13–adulthood	Rating scale	Trained health professionals	No
		Fugl–Meyer[78–82]	13–17	Scoring based on direct observation of performance	Trained physical therapist, occupational therapist or rehabilitation professional	No
		Gross Motor Functional Measure[83–85]	5 mo–16 y	Rating scale, criterion reference observational assessment	Trained pediatric therapists	No
		6-Minute Walk Test[86–88]	2–5 6–12 13–18	Rating scale	Trained health or research professionals	Age norms
	Oromotor	Dysphagia Disorder Survey[89–91]	18–24 mo 2–adulthood	Task analysis tool, rating scale	Caregiver, trained health professionals	Age norms
	Fine motor	Test of Arm Selective Control[92]	4–adolescence	Rating scale	Trained pediatric therapists	No
		Quality of Upper Extremity Skills Test[93–95]	18 mo–18 y	Performance measure, rating scale	Trained health professionals	No
		Mini-Manual Ability Classification System[96,97]	1–4 4–18	Rating scale	Trained health professionals	Age norms
		Melbourne Assessment of Upper Limb Function[98]	2.5–15	Rating scale	Trained health or research professionals	Age norms (from >4 y)
		Pediatric Arm Function Test[99]	2–6	Rating scale	Trained health or research professionals	No
		Pediatric Balance Scale (modified from the Berg Balance Scale)[77]	5–15	Rating scale	Trained health or research professionals	No
		Pediatric Neuromuscular Recovery Scale[100]	1–12	Rating scale	Trained pediatric health professionals	No
General cognitive	Cognitive, language, motor development	Bayley Scales of Infant and Toddler Development (Bayley-III)[101,102]	1–42 mo	Standardized test	Psychologist/Psychometrist	Age norms
	Intellectual ability	Wechsler Intelligence Scales (WPPSI-IV, WISC-V, WAIS-IV, WASI-II)[103–109]	3–7, 6–16;11, 16+	Standardized test	Psychologist/Psychometrist	Age norms

(*Continued*)

Table. (Continued)

Functional domain	Subdomain	Example measures*	Age range†	Method	Informant	Norms available
Executive/ Attention	Attention Processing speed Working memory Planning Organization Monitoring Behavior regulation	Developmental Neuropsychological Assessment (NEPSY-II)[110,111] Delis–Kaplan Executive Function System[112,113]	3–4, 5–16 8–99	Standardized test	Psychologist/Psychometrist	Age norms
		Conners' Continuous Performance Test-3,[114] Test of Everyday Attention[115]	5–15	Standardized test	Psychologist/Psychometrist	Age norms
		NIH Toolbox Cognition[116]	3–6, 7+	Standardized research tasks	Psychometrist/Trained research staff	Age, educational
		Behavior Rating Inventory of Executive Functions (BRIEF-2/Preschool/Adult)[117,118]	3–5, 5–18, 18+	Rating scale	Parent, child	Age norms
Memory	Verbal Visual Spatial	Children's Memory Scale[119]	5–16	Standardized test	Psychologist/Psychometrist	Age norms
		Wechsler Memory Scale (WMS-IV)[120]	16–90	Standardized test	Psychologist/Psychometrist	Age norms
		California Verbal Learning Test (CVLT-Children's Version/CVLT-3)[121,122]	5–16, 16+	Standardized test	Psychologist/Psychometrist	Age norms
		Rey-Osterrieth Complex Figure (ROCFT)[123,124]	5–14, 6–89	Standardized test	Psychologist/Psychometrist	Age norms
Visual–spatial	Visual-motor Visual planning Spatial orientation Perceptual organization	Beery-Buktenica Developmental Test of Visual-Motor Integration (VMI-6)[125]	2–99	Standardized test	Psychologist/Psychometrist	Age norms
		Judgment of Line Orientation[126]	7–74	Standardized test	Psychologist/Psychometrist	Age norms
		Rey-Osterrieth Complex Figure (ROCFT)[123,124]	5–14; 6–89	Standardized test	Psychologist/Psychometrist	Age norms
Language	Word retrieval Expressive language Receptive language Language formulation	Children's Acquired Aphasia Screening Test[127]	3–7	Standardized test	Psychologist/Psychometrist	Age norms
		Boston Naming Test[128]	6+	Standardized test	Psychologist/Psychometrist	Age norms
		Expressive One-Word Picture Vocabulary Test[129]	2–70+	Standardized test	Psychologist/Psychometrist	Age norms
		Receptive One-Word Picture Vocabulary Test[130]	2–70+	Standardized test	Psychologist/Psychometrist	Age norms
		Clinical Evaluation of Language Fundamentals (CELF-5)[131]	5–21	Standardized test	Psychologist/Psychometrist	Age norms
Behavior	Broadbend: Externalizing Internalizing Adaptive	Behavior Assessment System for Children (BASC-3)[132]	2–21 6–25	Rating scale	Parent, teacher child self-report	Age norms
		Achenbach Child Behavior Checklist/Youth Self-Report (CBCL/YSR)[133,134]	1.5–5, 6–18 11–18	Rating scale	Parent, teacher youth self-report	Age norms
	Attention Hyperactivity	Vanderbilt ADHD Diagnostic Rating Scale[135]	6–12	Rating scale	Parent, teacher	No
Specific Mood	Depression	Children's Depression Inventory (CDI-2)[136]/ Beck Depression Inventory (BDI-II)[137]	7–17 13–80	Rating scale	Child self-report, parent	Age norms
	Anxiety	Multidimensional Anxiety Scale for Children (MASC-2)[138,139]	8–19	Rating scale	Child self-report, parent	Age norms
		NIH PROMIS measures[140–142]	1–5, 5–17 8–17, 18+	Rating scale	Parent Child self-report	Age norms
Adaptive		Adaptive Behavior Assessment System (ABAS-3)[143] Vineland Adaptive Behavior Scales (Vineland-3)[144]	0–89; 0–5, 5–21 0–90, 3–18	Rating scale/interview	Parent, teacher	Age norms
		Ages and Stages Questionnaire (ASQ-3)[145]	0–6	Rating scale	Parent	Age norms
		Pediatric Quality of Life Inventory Generic Core Scales and Cerebral Palsy Module[146,147]	2–18	Rating scale	Parent/child	No

ADHD indicates attention deficit/hyperactivity disorder; NIH, National Institutes of Health; PROMIS, Patient-Reported Outcomes Measurement Information System; WAIS-IV, Wechsler Adult Intelligence Scale, Fourth Edition; WASI-II, Wechsler Abbreviated Scale of Intelligence, Second Edition; WISC-V, Wechsler Intelligence Scale for Children, Fifth Edition; and WPPSI-IV, Wechsler Preschool and Primary Scale of Intelligence, Fourth Edition.

*The listed instruments represent example measures commonly used in the United States and Canada yet are not meant as an exhaustive list of available measures in North America or globally. For specific batteries see the NIH Common Data Elements for Traumatic Brain Injury and Stroke.

†If not otherwise specified, age is listed in years.

pediatric HS, there remain important knowledge gaps and needs for future research and clinical care to overcome them. These include the following:

1. Inconsistencies in published data within HS and between HS and AIS highlight the need for larger and longitudinal studies with more

homogenous HS samples to understand the immediate functional outcomes in this population and their long-term neurodevelopmental needs.
2. Neurological examination and comprehensive neuropsychological assessment are critical first steps in planning interventions and should be a routine part of outcome evaluation to ensure timely intervention and consequently improved neurological function (eg, motor, visual), learning, academic achievement, emotional adjustment, and development of independence.[17]
3. Service models and research that address the transition to adulthood and advise on topics such as reproductive health (eg, contraception, pregnancy, labor) and potential restrictions (eg, sports, activities) are urgently needed to inform care. Outcome prediction should account for global/diffuse injury secondary to complications associated with pICH versus focal brain injuries related to pICH.[19]
4. Standardization of timing and duration of follow-up as well as a minimal data set with clearly defined outcomes is required to better understand rehabilitation and learning needs.
5. Research on prognostic factors needs to include assessment of social determinants of health, which has been shown to have similar if not larger predictive power for cognitive and behavioral outcomes.
6. This also should include preinjury functional (cognitive, behavioral, academic) status as a potential contributor for poststroke outcome.
7. Multicenter research is needed to provide larger samples and comparisons across samples and across HS versus AIS populations to determine potential commonalities or differences in functional outcome, immediately and long term.

In conclusion, given that pediatric HS is a rare condition, large studies on specific and general outcomes (including participation, quality of life, and specific interventions) are needed. Many lessons can be learned from current knowledge and practices in childhood ABI (including AIS), and some clear and practical recommendations are available for informing clinical practice. Neuroprotection measures in the acute phase can be planned based on already identified prognostic factors as well as general interventions for acute ABI. However, multicenter, collaborative research on HS outcomes would be beneficial, similar to those successfully developed and implemented for childhood traumatic brain injury/ABI or brain tumors. Multisite, ideally international, HS registries such as the ones developed by the International Pediatric Stroke Study, the research arm of the International Pediatric Stroke Organization, will provide especially useful vehicles to answer these important questions.

ARTICLE INFORMATION

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