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Practical, Evidence-Based Reviews in Pediatric Emergency Care

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Pediatric Stroke

Pediatric strokes, defined as strokes occurring in patients 28 days to 18 years of age, are classified as ischemic or hemorrhagic cerebral events, and are seen in two to three per 100,000 children per year. They have the same incidence as pediatric brain tumors.[1] *Early identification of stroke is paramount in patient outcomes, as therapy and subsequent recovery is often time dependent.*

Treatment is driven by the underlying etiology of the event and type of stroke sustained by the patient. The post-stroke course and rehabilitation has extensive implications beyond the patient's medical needs, with financial and societal impacts for the patient and his or her family.[2]

— Ann M. Dietrich, MD, FAAP, FACEP, Editor

Definition of the Problem

Stroke, a clinical syndrome of rapidly developing focal or global neurologic deficits lasting more than 24 hours, is diagnosed in more than 2000 children each year.[3,4] Pediatric strokes, defined as strokes that occur in individuals 28 days to 18 years, are classified as ischemic or hemorrhagic cerebral events. In 2013, the CDC's Web-based Injury Statistics Query and Reporting System (WISQARS™) reported that for children younger than 1 year and between 5-14 years of age, cerebrovascular accidents ranked in the 10 leading causes of death. Other adverse outcomes include persistent neurologic deficits, seizures, stroke recurrence, and psychosocial implications. Emergency physicians who care for pediatric patient populations should include stroke in the differential diagnosis when patients present with neurologic complaints, signs, or symptoms; however, a high index of suspicion is needed given the varied presentation depending on the location of the stroke and the patient's age. Due to the potentially devastating consequences of strokes and the time-sensitive nature of interventions, early identification of a stroke presenting in the emergency department (ED) is paramount for mobilizing resources and initiating treatment.[5]

Epidemiology

Pediatric strokes affect children of all ages, from birth through teenage years. While the incidence of childhood stroke is relatively rare, affecting two to three per 100,000 children per year, the diagnosis is still made as commonly as the diagnosis of childhood brain tumor.[1] The incidence of an arterial ischemic stroke (AIS) is 7.8 per 100,000 children and the incidence of hemorrhagic stroke is 2.9 per 100,000.[6] About one-third of all strokes are diagnosed in children younger than 1 year of age.[3] In the teenage population, subarachnoid hemorrhage (SAH) is the most common cause of stroke.[7]

Stroke mortality in children is high, and risk of death during the acute phase is predicted by level of consciousness on admission.[3] The total mortality of AIS, SAH, and intraparenchymal hemorrhage (IPH) is 5-7%.[4] The mortality of

EXECUTIVE SUMMARY

- The incidence of an arterial ischemic stroke is 7.8 per 100,000 children and the incidence of hemorrhagic stroke is 2.9 per 100,000. About one-third of all strokes are diagnosed in children younger than 1 year of age. In the teenage population, subarachnoid hemorrhage is the most common cause of stroke.
- Ischemic strokes commonly arise from medical conditions, whereas hemorrhagic strokes are more likely to occur from structural anomalies or severe head trauma.
- The typical presentation of arterial ischemic stroke in children is abrupt onset of hemiplegia or another focal neurologic deficit. For infants younger than 1 year of age, common presentations of stroke are seizure, encephalopathy, lethargy, or apnea, even without a focal neurologic deficit.
- The Pediatric National Institutes of Health Stroke Scale (PedNIHSS) has been validated for children ages 2-18 years and may be used in the ED to obtain a baseline for predicting stroke outcomes and to facilitate communication between emergency physicians and consulting neurologists. The scoring system and interpretation for PedNIHSS is the same as the adult version, although each section of the exam is modified for children.

cerebral sinovenous thrombosis (CSVT) is 3-12%.[8,9] This risk increases for children who suffer repeat strokes.[3] More than half of all survivors have persistent neurologic, cognitive, or psychiatric deficits. Nearly one-third of all survivors develop epilepsy. Universally, strokes are associated with significant financial costs to families and society.[2]

Etiology

Unlike adult strokes, pediatric strokes are associated with a myriad of conditions. One-half of children who experience a stroke have at least one premorbid illness.[10,11] Ischemic strokes commonly arise from medical conditions, whereas hemorrhagic strokes are more likely to occur from structural anomalies or severe head trauma.[5,12] The etiologies of AIS are shown in Table 1. The etiologies of hemorrhagic stroke are listed in Table 2.

In children with predisposing risk factors, a stroke may also be "triggered" by certain conditions. For example, head trauma may trigger arterial strokes, while dehydration may trigger venous strokes.[3] Common infectious processes such as otitis media, varicella, meningitis, and tonsillitis can also trigger strokes.[13] Notably, hypertension is not a common trigger for hemorrhagic stroke in children, although it is a common cause of IPH in adults.[3]

Arterial Ischemic Stroke

The most common cause of non-traumatic childhood stroke is a cardioembolic event from congenital or acquired heart disease.[14] In fact, cardiac disease is present in 27% of children in the United States who are hospitalized for a stroke.[15] Hematologic disorders are also important risk factors for both ischemic and hemorrhagic strokes. The link between sickle cell disease (SCD) and AIS is significant: SCD is the most common cause of stroke in African American children, and 11% of patients with SCD will experience a first stroke before the age of 20. If left untreated, two-thirds of these patients may experience recurrence.[15,16] Red blood cell transfusion for strokes in patients with SCD is the only well-studied recommended treatment for childhood stroke.[17] Anemia is present in more than 25% of children with AIS.[13] Prothrombotic disorders have also been associated with AIS.[18] Additionally, thrombocytopenia and coagulation disorders increase risk for IPH.[3]

Vascular disorders increase the risk for both ischemic and hemorrhagic strokes. Non-traumatic arterial dissection of the anterior circulation, which tends to occur intracranially in older children and adolescents (compared to extracranially in adults), accounts for 20% of AIS.[13,19] Though rare, children may also experience posterior circulation strokes, which are commonly due to vertebral artery dissections.[20] Children with autoimmune or inflammatory vasculopathies are at slightly increased risk of both ischemic and hemorrhagic stroke compared to the general population.[2]

Table 1. Etiologies of Arterial Ischemic Strokes in Children

- Sickle cell disease
- Cardioembolic
- Moyamoya syndromes
- Cervical arterial dissection
- Steno-occlusive cerebral arteriopathy
- Other determined etiology
- Multiple probable/possible etiologies*
- Undetermined etiology

* Cerebral angiitis, fibromuscular dysplasia, cerebral involvement in systemic vasculitis, bacterial meningitis, hypertension, prothrombotic disorders, clinical chicken pox, and hyperhomocysteinemia.

Adapted from: Gumer LB, Del Vecchio M, Aronoff S. Strokes in children: A systematic review. *Pediatr Emerg Care* 2014;30:660-664.

Table 2. Etiologies of Hemorrhagic Strokes in Children

- Vascular malformations
  - Arteriovenous malformation
  - Cavernous hemangioma
  - Aneurysm
  - Subarachnoid hemorrhage
  - Venous malformation
- Brain tumors
- Trauma/dissection
- Underlying medical disorder
- Undetermined

Adapted from: Gumer LB, Del Vecchio M, Aronoff S. Strokes in children: A systematic review. *Pediatr Emerg Care* 2014;30:660-664.

Figure 1a. Moyamoya Disease

Magnetic resonance angiography demonstrates multiple areas of stenosis in the internal carotid arteries, typical of moyamoya disease. This patient suffered multiple watershed region infarcts.

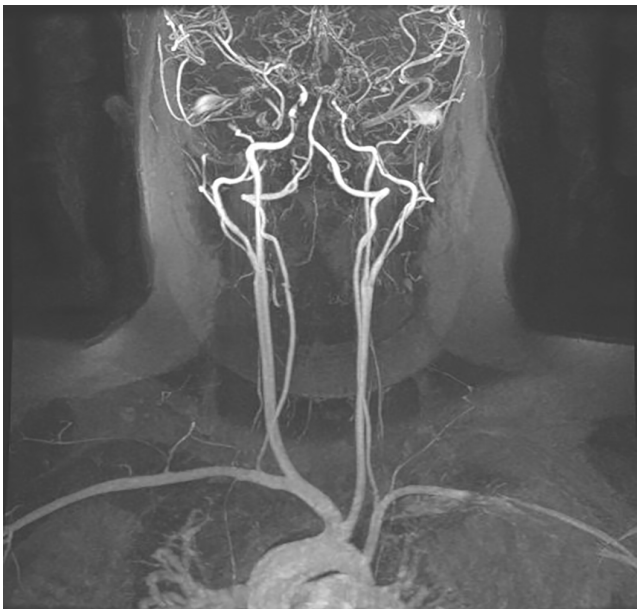
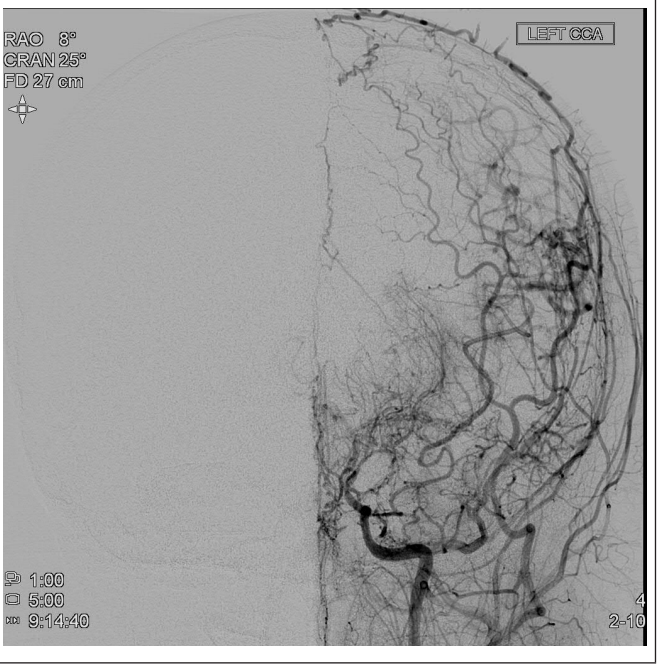


Figure 1b. Moyamoya Disease

Cerebral angiography of the same patient demonstrates 75% stenosis of the supraclinoid left internal carotid artery with moyamoya type collaterals, which result in a pathognomonic "puff of smoke" appearance.



Moyamoya vasculopathy, which is a radiologic term, is characterized by bilateral stenosis of the terminal internal carotid arteries (*see Figure 1a*) with resultant collateralization that gives a pathognomonic "moyamoya" (Japanese for "puff of smoke") appearance on angiography (*see Figure 1b*).[15] It is present in up to 20% of children with AIS. Although the radiologic finding was first described in Japan, the term "moyamoya" is often loosely used to describe any terminal internal carotid artery disease. Moyamoya disease is idiopathic; moyamoya syndrome describes a moyamoya appearance on angiography that is secondary to other conditions, such as Down syndrome, neurofibromatosis type 1, or head trauma.[19,21]

Hemorrhagic Stroke

Traumatic brain injury (TBI) is a preventable cause of hemorrhagic stroke. More than 40,000 children are treated in the ED for TBI each year, of which 2% are classified as moderate or severe.[22] Approximately one-half of all severe TBI patients require surgical hematoma evacuation.[23] Campaigns and provider-initiated instructions to promote the use of bicycle helmets and appropriate automobile restraints are worthwhile preventive tools to address this public health concern.

Children with vascular anomalies, including arteriovenous malformations, cavernous hemangiomas, and aneurysms, are at increased risk for hemorrhagic strokes. Any mass lesion, such as a brain tumor, also increases the risk for hemorrhagic stroke.[3]

Cerebral Sinovenous Thrombosis

CSVT can cause secondary ischemic or hemorrhagic events.[9] Risk factors for CSVT include chronic health disorders (50%), most notably congenital heart disease, inflammatory bowel disease, genetic disorders, hematologic disorders, prothrombotic states, and malignancies. They also occur in healthy children with acute systemic illness or head and neck infections (41%). Iron deficiency and leukemia were reported in 10% of cases. Sickle cell anemia is not a reported risk factor for CSVT.[24]

Pathophysiology

For the most part, the presumed mechanism of ischemic stroke is a cardioembolic event, especially if the patient has an underlying cardiac disease. However, because cardiac lesions can cause chronic hypoxia and polycythemia, thrombotic diseases may also factor into stroke mechanism. In patients with conditions that allow right-to-left shunting, such as a septal defect, patent foramen ovale, or single ventricle, venous blood may enter the systemic circulation and cause a paradoxical embolus. Stroke may also be a complication of cardiac catheterization or cardiac surgery.[25]

In traumatic strokes, the mechanism of stroke is from stretching or tearing of the vertebral or carotid arteries from extreme hyperextension, contralateral head rotation, or direct insult to the neck or orbit. These injuries cause dissection, arteriovenous fistula, or pseudoaneurysm formation, and ultimately disrupt blood flow directly or by causing artery-to-artery thromboembolisms.[26] Occlusion following trauma may also be related to preexisting risk factors, such as infection, that cause the cerebral anatomy to be vulnerable. When faced with even subtle shear or stretch, intimal lesions and thrombosis may result.[27] In children who experience AIS from vertebral artery dissection after trauma, the pathophysiology of the dissection may be related to anatomic variations caused by forceful accidents, such

Figure 2. Traumatic Vertebral Artery Dissection

Magnetic resonance angiography demonstrates narrowing of the right vertebral artery, consistent with dissection. This patient presented with complaints of dizziness and vomiting after slipping on ice and falling backward, hitting the head.

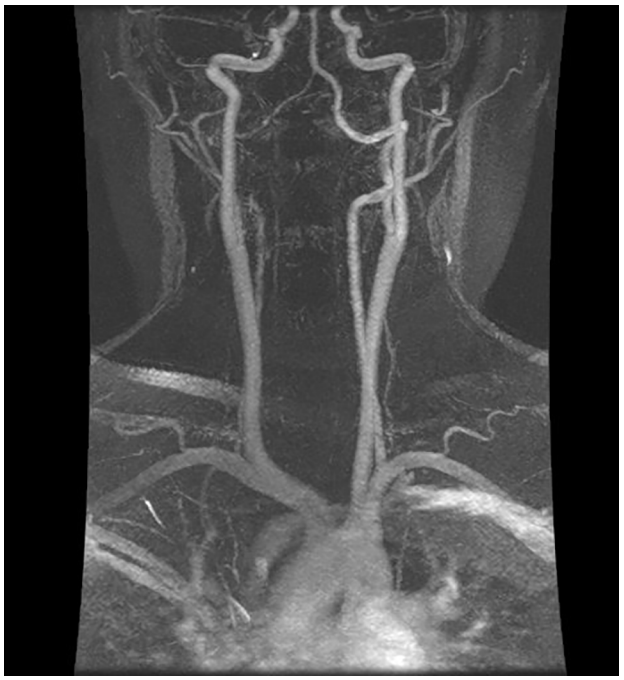
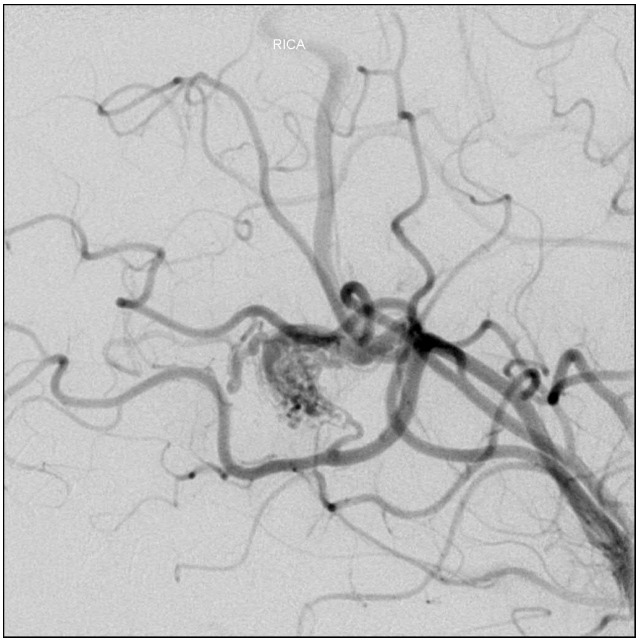


Figure 3. Cerebral Angiogram Demonstrating an AVM

Cerebral angiogram demonstrating an AVM fed primarily from the right middle cerebral artery



as atlanto-axial subluxation, bony lesions, or fibrous soft tissue disruption, which may compress the vertebral artery during head rotation.[2] Figure 2 illustrates findings typical of strokes related to traumatic vertebral artery dissection.

For children with sickle cell anemia, strokes result from development of cerebral arteriopathy. Large vessels, such as the distal internal carotid artery or the proximal segment of the middle or anterior cerebral arteries become stenotic, which then become a nidus for thrombus formation and artery-to-artery emboli. Additionally, these stenotic segments can cause decreased cerebral perfusion with resultant watershed ischemia.[2] Later in the course of sickle cell anemia as collateral blood supply forms (and moyamoya can be identified radiographically), the delicate, abnormal collateral vessels are at increased risk for hemorrhage.[28]

Though preceding infection was reported in 24% of AIS cases with highest prevalence in those younger than 5 years of age, the mechanism is not fully understood.[10] Two hypothesized mechanisms include involvement of an inflammatory response and direct vascular infection causing cerebral vascular injury and AIS.[2]

Vascular lesions, such as arteriovenous malformations (*see Figure 3*), cavernous malformations, and aneurysms, account for nearly one-half of all pediatric hemorrhagic strokes.[29] When vascular lesions are found after traumatic hemorrhagic strokes, the mechanism is likely explained by traumatic pseudoaneurysm formation rather than by the existence of underlying vascular malformations.[30]

Venous infarcts occur secondary to a low-flow state, including during systemic disease such as infection, dehydration, iron deficiency anemia, and prothrombotic states.[9,24,31] Retrograde venous pressure decreases cerebral blood flow, causing tissue damage.[9]

Clinical Features

While adult strokes tend to have classic presentations, such as focal motor or sensory deficits or altered mental status, strokes have varied presentations in children. Additionally, seemingly straightforward diagnoses such as headache, infection, or trauma do not preclude the presence of a stroke. A single-center study by Mackay et al found stroke was the fourth most common diagnosis in children without underlying epilepsy or trauma presenting with signs and symptoms of a "brain attack," such as seizures, headaches, altered consciousness, focal weakness, numbness, visual changes, speech changes, ataxia, or vomiting.[32] Even in children with an established seizure history, stroke must be suspected, especially if the characteristics of the seizure differ from prior seizures.[33]

Emergency physicians should recognize the signs of an acute stroke in children. The typical presentation of AIS in children is abrupt onset of hemiplegia or another focal neurologic deficit.[34] For infants younger than 1 year of age, common presentations of stroke are seizure, encephalopathy, lethargy, or apnea, even without a focal neurologic deficit.[35] The Pediatric National Institutes of Health Stroke Scale (PedNIHSS) has been validated for children ages 2-18 years and may be used in the ED to obtain a baseline for predicting stroke outcomes

Figure 4. Recurrent Ischemia from Moyamoya Disease

Magnetic resonance imaging with diffusion weighted imaging shows multiple areas of infarction in the right frontal and left parietal and occipital lobes in a patient with recurrent ischemia from moyamoya disease.

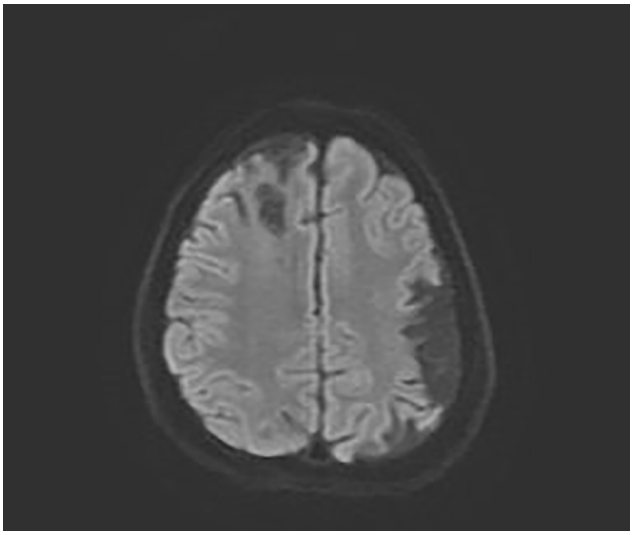
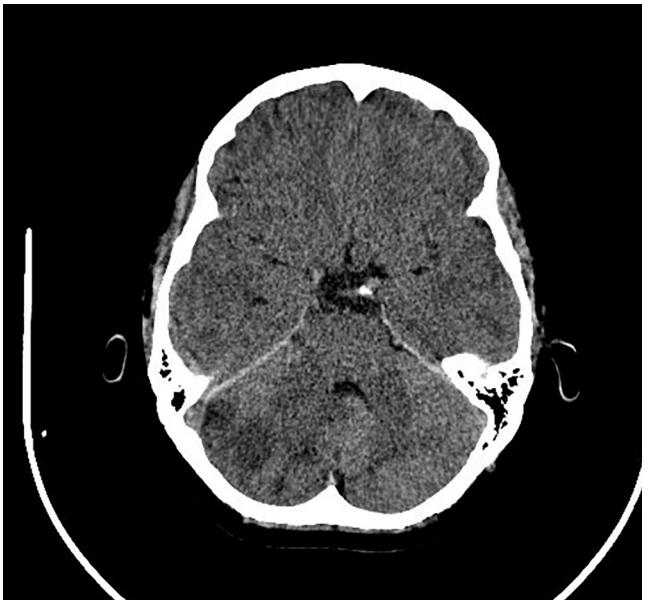


Figure 5. Right Cerebellar Infarct

Computed tomography imaging of a patient who presented with an unsteady gait demonstrates an acute right cerebellar infarct.



and to facilitate communication between emergency physicians and consulting neurologists. The scoring system and interpretation for PedNIHSS is the same as the adult version, although each section of the exam is modified for children.[36]

Children presenting with a hemorrhagic stroke may have sudden onset of severe headache, focal neurologic deficits, altered mental status, and seizures. Signs and symptoms of CSVT are nonspecific, but most commonly include headache, vomiting, depressed mentation, and seizures.[24,31]

Elevated body temperature can be seen in up to 25% of stroke patients and is associated with worse neurologic outcomes.[31]

Diagnostic Studies

Imaging Studies

Patients who present with signs and symptoms suggestive of a stroke should undergo immediate vascular imaging. In addition to mobilization of neurological and/or neurosurgical services, a radiologist should be immediately contacted, and resources should be mobilized for performing magnetic resonance imaging (MRI) if it is available (*see Figure 4*). The recommended initial imaging modality in the ED is computed tomography (CT) without contrast (*see Figure 5*). This is readily available in most EDs, and potentially bypasses the need for sedation due to its rapid nature. Diffusion weighted magnetic resonance angiography (MRA) and magnetic resonance venography (MRV) are highly sensitive and specific imaging modalities, although these techniques are more time-intensive and are often not available immediately. MRA has a 100% positive predictive value and an 88% negative predictive value in the assessment for arteriopathy.[3] MRV or CT venography should be obtained to look for CSVT, as 10% of hemorrhages are identified by this modality.[8]

Transcranial ultrasound with Doppler flow is useful for finding parenchymal hemorrhage and intraventricular hemorrhage in neonates and infants with open fontanelles, but its sensitivity for detecting pathology is user-dependent. The posterior fontanelle closes between 1-2 months of age and the anterior fontanelle closes between 9-18 months of age.[3]

Intracerebral hemorrhage carries the risk of hematoma expansion. Up to 32% of children with non-traumatic intracerebral hemorrhage had expansion of the hematoma, with 33% of those requiring emergent surgical evacuation. For institutions that receive transfers through the ED, a repeat CT scan should be considered in children with a known large hematoma or signs of increased intracranial pressure (*see Figure 6*).[37]

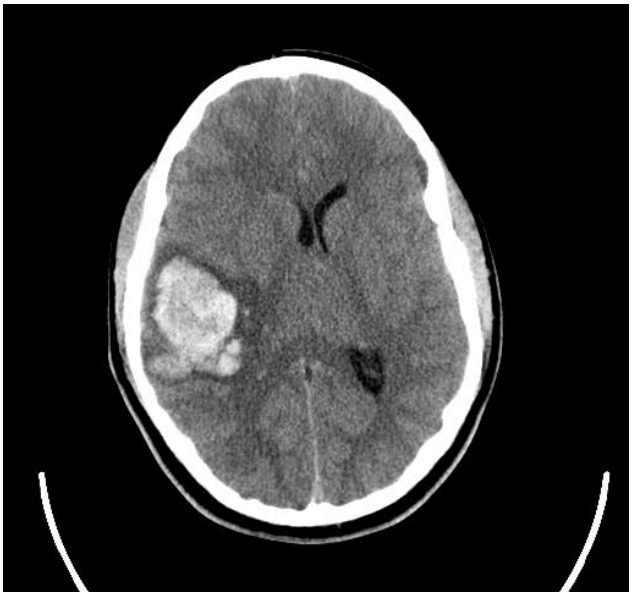
Finally, a chest X-ray is useful to determine if there is concomitant respiratory illness and to look for signs of aspiration. Cervical spine X-rays or CT may be considered when a posterior circulation stroke is present to assess for subluxation.[15]

Laboratory Analysis

The first and most essential laboratory value to obtain is a bedside glucose to rule out hypoglycemia as a quickly treatable cause of stroke-like symptoms in children. Serum glucose measurement can guide both diagnosis and therapy. Hypoglycemia is well-recognized as a non-stroke cause of focal neurologic deficits, and with correction of hypoglycemia, the neurologic exam may improve. However, hyperglycemia may also be useful in guiding the emergency physician. In one study, the combination of an abnormal neurologic exam with elevated serum glucose was highly sensitive and specific for intracranial hemorrhage in children younger than 3 years of age. For children who suffered non-accidental head trauma, the serum glucose was elevated to an even higher degree than in

Figure 6. Right Temporal Intraparenchymal Hemorrhage

CT of a patient who complained of acute onset of severe headache reveals right temporal intraparenchymal hemorrhage with midline shift. This patient required an emergent craniotomy and hematoma evacuation.



those who suffered accidental trauma.[38]

Additionally, a complete blood count (CBC) with differential should be obtained to assess for anemia, polycythemia, leukocytosis suggestive of infection, or malignancy. A comprehensive metabolic panel should be obtained to look for electrolyte disturbances (including sodium, potassium, calcium, magnesium, and phosphorous). An elevated BUN-to-creatinine ratio or hypernatremia may indicate dehydration. A coagulation panel will help assess for prothrombic state. When there is a history or suspicion of associated infection, blood cultures, urine culture, blood type and cross, and lactate levels also should be obtained. For suspected inflammation or vasculitis, erythrocyte sedimentation rate, C-reactive protein, and antinuclear antibody tests may be helpful.

For patients with risk factors, HIV and syphilis screening, plasma amino acids, urine amino acids, toxicology screen, liver function tests, and a fasting lipid panel should be considered.

Lumbar Puncture

For children in whom a stroke is attributed to meningitis, a lumbar puncture should be obtained after imaging has been done to rule out mass effect. However, if the patient's pretest probability that meningeal infection is high, empiric antibiotics should be started without confirmed diagnosis.

Lumbar puncture to identify xanthochromia in subarachnoid hemorrhage (SAH) has been studied in the pediatric and adult ED population. Therefore, the recommendation to perform a lumbar puncture to rule out SAH is the same in children as in adults. CT is 100% sensitive for the diagnosis of nontraumatic SAH when performed at or before 12 hours of symptom duration, but only 81% sensitive after 12 hours. In the pediatric population, symptom onset may be unknown; therefore, when subarachnoid hemorrhage is suspected, a lumbar puncture should be performed.[39,40]

Other Tests

An electrocardiogram (ECG) and transthoracic echocardiogram (TTE) should be obtained as soon as possible to evaluate for sources of cardioembolic emboli.[15] Diagnostic studies for the evaluation of pediatric stroke patients are listed in Table 3.

Differential Diagnosis

A significant problem with the diagnosis of acute stroke in children is the variability of signs and symptoms at presentation. Additionally, obtaining a good history of present illness in children who are unable to or too young to communicate is challenging. The differential diagnosis for neurologic symptoms and findings is broad. Twenty-one percent of children presenting with suspicion of stroke had a different disease, and 60% of children with neurologic deficits had another non-benign etiology.

The differential should include hypoglycemia, electrolyte disturbances (such as hyponatremia, hypo- or hypercalcemia, hypokalemia, or hypomagnesemia), infection (such as abscess, encephalitis, or meningitis), tumor, primary seizure disorder, autoimmune disorder, inborn errors of metabolism, and toxic exposure.[41]

Management

The time-sensitive goal in the management of any stroke is to rescue viable brain tissue, prevent complications, and minimize the risk of recurrent stroke by treating conditions that could cause recurrence.[42] There are comprehensive and specific multi-specialty guidelines for the management of ischemic and hemorrhagic strokes in adults. Unfortunately, the only proven strategy for acute management of childhood AIS is exchange transfusion for children with sickle cell anemia.[43] Much of the recommendation for managing strokes in children is extrapolated from adult studies.[36]

Immediate Management of All Strokes

In the emergency setting, the first goal in management is to ensure patient stability. This includes adhering to principles of emergency resuscitation. Protect the patient's airway from aspiration by elevating the head of the bed. Ensure that the patient is properly oxygenating and ventilating via pulse oximetry monitoring and direct observation of the patient's mental status and respiratory drive, and intervene if necessary. Confirm cardiovascular stability using electrocardiographic monitoring and blood pressure measurement. If the patient presents with stroke symptoms following a traumatic incident, immobilize the patient's neck using a cervical collar. Seizures should be medically managed if they are present.

The emergency physician's target should be homeostasis, providing supportive care to reduce metabolic demands

Table 3. Diagnostic Studies for Evaluation of Pediatric Stroke

Imaging options	• Computed tomography (CT) without contrast • Computer tomography angiography (CTA) • Computer tomography venography (CTV) • Magnetic resonance imaging (MRI) • Magnetic resonance angiography (MRA) • Magnetic resonance venography (MRV) • Transcranial ultrasound with Doppler flow in neonates and infants
Laboratory analysis (serum, urine)	• Bedside glucose • Complete blood count (CBC) with differential • Comprehensive metabolic panel • Coagulation panel (PT/INR, PTT) • Blood, urine cultures • Consider inflammatory markers (erythrocyte sedimentation rate, C-reactive protein), antinuclear antibody test, HIV, syphilis, plasma amino acids, urine amino acids, toxicology screen, fasting lipid panel
Laboratory analysis (cerebrospinal fluid)	• Cell count with differential • Protein • Glucose • Gram stain • Culture • Xanthochromia • Consider additional tests if infectious etiology is suspected
Additional testing	• Electrocardiogram (ECG) • Transthoracic echocardiogram (TTE)

in the setting of cerebral ischemia.[15] Effective therapies include maintaining normothermia, normal oxygenation, normotension, euvolemia, normoglycemia, and normal-range hemoglobin.[31]

A neurologist should be consulted immediately. If a hemorrhagic stroke is identified, a neurosurgical service should be consulted. Mobilize resources to obtain a MRI if available. If neurological or neurosurgical services are not available, plans to transfer the patient to a higher-level care center should be made immediately. Asses the patient's stability for transfer, with special consideration for protective intubation based on clinical evaluation and the severity and location of the stroke on imaging studies.

Management of Ischemic Stroke in Patients Without Sickle Cell Disease

High-quality clinical trials regarding drug treatment for AIS in children is limited. However, pediatric stroke guidelines regarding acute care and prophylactic management have been developed by three institutions, based on the available evidence and expert opinion: Royal College of Physicians (RCP; 2004), American College of Chest Physicians (ACCP; 2008), and American Heart Association (AHA; 2008).[3,44-46] Table 4 reviews antiplatelet and anticoagulation guidelines and recommendations. In most centers, patients who present with AIS without SCD are treated with aspirin.

Thrombolytics (Recombinant Tissue Plasminogen Activator or Alteplase)

All guidelines agree that there is no indication for thrombolysis in children outside of specific research protocols for clinical trials, although clinical practice in large centers may differ.[3,31,44,45] Mechanical thrombectomy trials using clot retrieval devices have also excluded patients younger than 18 years of age. However, institution-specific practices vary, and the immediate involvement of a neurologist is essential to determine appropriateness of therapy.[47]

Medical Management of Ischemic Stroke in Patients with Sickle Cell Disease

Chronic blood transfusion is effective for prevention of stroke in SCD. The landmark STOP trial showed that monthly transfusion therapy for children at high risk for stroke (MCA cerebral blood flow velocity greater than 200 m/s by transcranial Doppler) decreased the relative risk by 92% compared to standard care. In the emergency setting, these children should receive exchange transfusions in the setting of acute stroke.[15,43,48]

Management of Hemorrhagic Stroke

The diagnosis of a hemorrhagic stroke should prompt emergent consultation with a pediatric neurosurgeon. Management depends on the size and location of the hemorrhage. There are no controlled studies regarding osmotherapy (with mannitol or hypertonic saline) or decompressive craniectomy in children, but based on adult studies, these are reasonable interventions in life-threatening cases of increased intracranial pressure or signs of herniation. In prospective adult trials, the number needed to treat was 2 for one additional survivor with decompressive craniectomy. Surgeries performed before and after 24 hours showed similar benefits. However, there was no additional benefit seen if performed 48 hours after the insult. Decompressive craniectomy within 48 hours in children when there is extensive hematoma with middle cerebral artery distribution is recommended.[31]

If an arterovenous malformation is identified, early treatment with

Table 4. Antiplatelet and Anticoagulation Therapy[3,44-46]

	Antiplatelet Recommendations	Anticoagulation Recommendations
Royal College of Physicians	Aspirin 5 mg/kg/day in children with radiological evidence of stroke, excluding children with sickle cell disease or intracranial hemorrhage	Consider heparin in ischemic strokes when arterial dissection or CSVT is confirmed. Discuss heparin with pediatric cardiology and neurology before starting heparin in patients with ischemic strokes from a cardioembolic origin.
American Heart Association (AHA) Guidelines	Consider aspirin therapy in children with suspected cardioembolic cause of stroke unrelated to a patent foramen ovale.	Start heparin or low-molecular weight heparin as a bridge to warfarin in children with extracranial arterial dissection, cardioembolic stroke, and in neonates with severe thrombophilic disorders, multiple emboli, or propagation of a CSVT despite supportive care. Anticoagulation should not be started in children with intracranial dissection, subarachnoid hemorrhage from a dissection, or stroke due to native valve endocarditis.
American College of Chest Physician (ACCP) Guidelines	Empirically start initial treatment in children diagnosed with ischemic stroke not due to sickle cell disease with either aspirin (1-5 mg/kg/day) or anticoagulation (heparin or low-molecular weight heparin) until dissection and embolic causes of stroke are excluded. For neonates, treatment with aspirin or anticoagulation is not recommended except in cases of recurrent stroke	

embolization is recommended.[31] Cerebellar hemorrhages > 3 cm should be evacuated early; however, these recommendations are based on adult studies.[3,31]

Management of Cerebral Sinovenous Thrombosis

Initial antithrombotic therapy is recommended in all guidelines when CSVT without ICH is diagnosed.[9,31] The RCP does not specify what type of anticoagulation should be started, but recommends anticoagulation until recanalization or up to 6 months after the thrombosis. The ACCP and the AHA both recommend initial unfractionated heparin (UFH) or low molecular weight heparin (LMWH) until recanalization is seen, or until 3-6 months.[3,45] If no improvement is found, then thrombectomy, surgical decompression, or thrombolytic agent administration may be considered.[31]

For patients with CSVT with ICH, the recommendations differ between groups. The RCP does not make any recommendations. The ACCP recommends no initial anticoagulation, performing repeat imaging within 5-7 days, and initiation of anticoagulation if the thrombus has progressed. The AHA recommends initiating UFH or LMWH, bridging to warfarin, and continuing therapy for 3-6 months for CSVT with ICH.[3,31,45]

Ineffective Therapies

There are certain therapies that have not been shown to be effective in human studies. Oxygenation in the absence of hypoxemia, thrombocyte aggregation inhibitors such as GP-IIb/IIIa-receptor antagonists, antiepileptic drugs in the absence of clinical or electrographic seizures, high-dose insulin therapy, corticosteroids, and hypothermia are all considered ineffective therapies and are not recommended as general therapeutic measures in childhood stroke.[31]

Additional Aspects

Between 40-60% of children will have neurologic deficits following a stroke, the most common sequelae being hemiparesis.[3,34,41] Outcomes are most severe in AIS, and are somewhat better for patients who survive CSVT, hemorrhagic strokes, and posterior circulation strokes.[41]

In addition to the devastating effects childhood stroke has on patients, it causes an immense burden to families and society. Stroke patients tend to be hospitalized for a week or more. The average length of stay (LOS) for AIS is 7 days, while the average LOS for ICH is 11 days. The average LOS for SAH

Table 5. Pediatric Intracerebral Hemorrhage Score

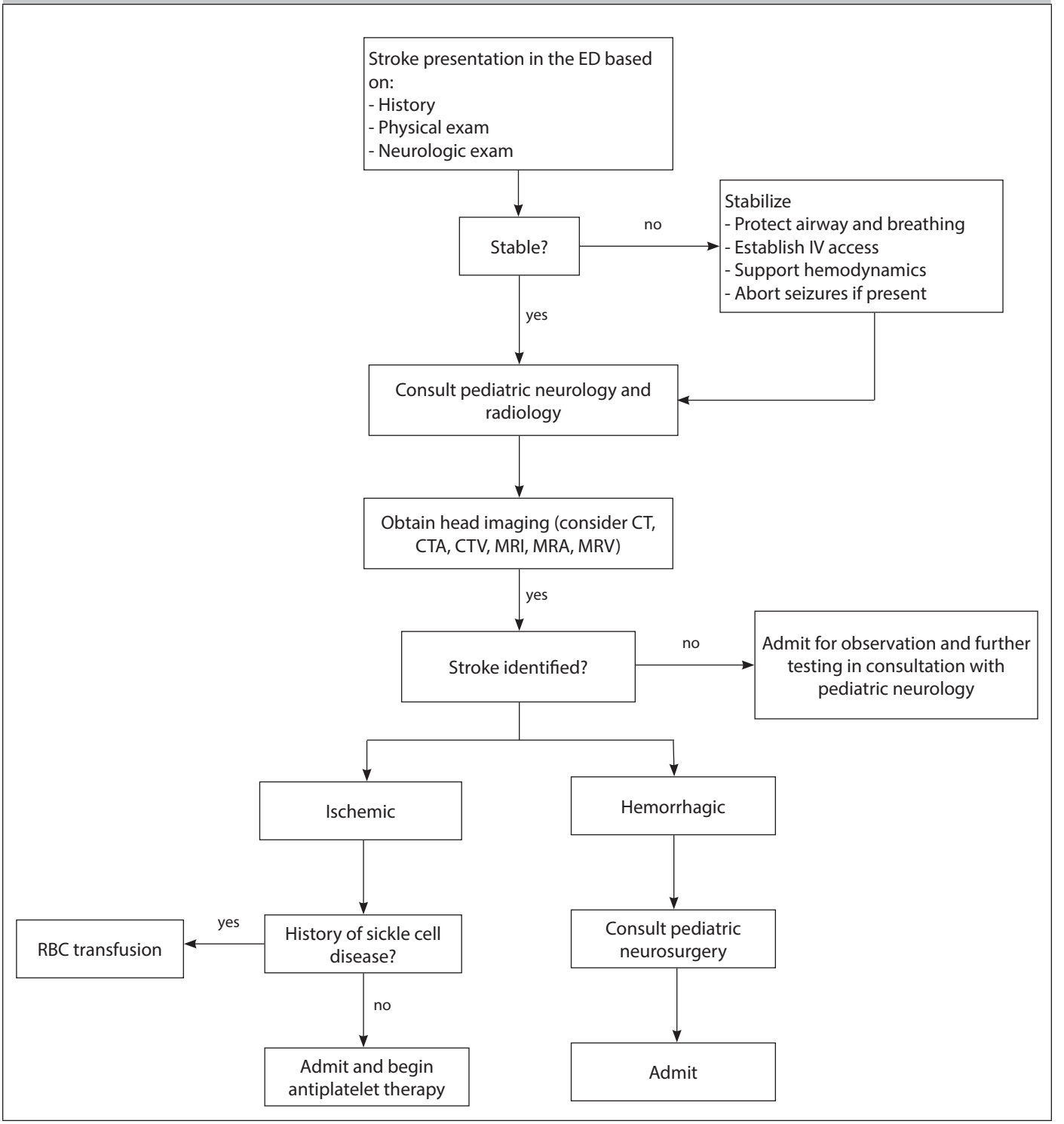
Component	ICH Scoring
IPH volume, % of TBV*	
≤ 2	0
> 2-3.99	1
≥ 4	2
Hydrocephalus	
No	0
Yes	1
Herniation	
No	0
Yes	1
Infratentorial	
No	0
Yes	1
Total ICH Score	0-5

* IPH = intraparenchymal hemorrhage; TBV = total brain volume.

A score of ≥ 2 is sensitive for predicting severe disability or death.

Adapted from: Beslow LA, Ichord RN, Gindville MC, et al. Pediatric intracerebral hemorrhage score: A simple grading scale for intracerebral hemorrhage in children. *Stroke* 2014;45:66-70.

Figure 7. Basic Approach to Pediatric Stroke Patients



is > 11 days, often in an ICU setting, as patients are monitored and treated for cerebral vasospasm.[4] The cost of a stroke for the year following an event ranges from $15,000 to $140,000, depending on the type of stroke.[49]

Expenses to families are significant. In one study, the total median out-of-pocket cost for the year after an AIS was $4354, while the median American household had a cash savings of $3650. This represents 6.8% of annual income for most families. Out-of-pocket costs are greatest in the first 3 months after the stroke, due to lost wages, transportation costs, and non-reimbursed health care. An additional, less tangible factor is the cost of decreased parental productivity while caring for a child following a stroke.[50]

Additionally, childhood stroke victims are at risk for poor social adjustment and having more social problems than their counterparts. Lesion volume is

not associated with social outcomes, but subcortical strokes are linked to reduced participation. It appears that age at onset of stroke is inversely related to degree of social interaction and level of self-esteem, with the worst prognosis for adolescents and teenagers. However, the sole predictor of social adjustment is family function, highlighting the importance of family support for stroke patients.[51]

Regarding long-term care, the AHA recommends the following:[3]

1. Age-appropriate rehabilitation and therapy programs are indicated for children after a stroke (Class I, Level of Evidence C).

2. Psychological assessment to document cognitive and language deficits is useful for planning therapy and educational programs after a child's stroke (Class I, Level of Evidence C).

Disposition

All pediatric patients with suspected stroke require hospital admission for further evaluation and management. Transfer to tertiary care centers with access to pediatric neurologists and neurosurgeons is common in this setting.

Clinical grading scales may be useful for predicting outcomes, relaying information during transitions of care, re-evaluation, and clinical management. The PedNIHSS is a validated scoring system used to predict outcomes in AIS.[36]

The Pediatric Intracerebral Hemorrhage Score is a simple scoring system that has been proposed to predict disability or death in ICH.[52] A score of ≥ 2 is sensitive for predicting severe disability or death. Table 5 shows the Pediatric Intracerebral Hemorrhage Score.

Summary

Pediatric stroke presents a diagnostic challenge to the emergency physician given the varied presentation by the patient, dependent on the patient's age and the etiology of the stroke. A high index of suspicion is necessary when evaluating these patients. Initial treatment in the ED includes stabilization of the patient, followed by laboratory and imaging diagnostics to further guide treatment and disposition. Therapy is variable dependent on stroke etiology; however, all patients require admission for further evaluation, management, and risk stratification. Figure 7 reviews the basic approach to a pediatric stroke patient.

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CME Questions

1. Hemorrhagic strokes in children are frequently caused by:
   a. hypertension.
   b. severe head trauma.
   c. sickle cell disease.
   d. cardioembolic event from congenital or acquired heart disease.
2. Which of the following is not a risk factor for cerebral sinovenous thrombosis?
   a. Sickle cell disease
   b. Congenital heart disease
   c. Inflammatory bowel disease
   d. Hematologic disorders and malignancies
3. Which of the following is the most common etiology of ischemic strokes in children?
   a. Thrombotic disease
   b. Cardiac catheterization
   c. Congenital or acquired heart disease
   d. Sickle cell disease
4. What is the most common presenting symptom of stroke in the pediatric patient older than 1 year of age?
   a. Altered mental status
   b. Focal neurological deficit
   c. Lethargy
   d. Seizure
5. A 12-year-old girl presents complaining of a "thunderclap" headache that began 16 hours ago. It was worst at onset. She has no focal neurologic deficits on exam. A head CT without contrast is obtained. What is the next best step in management?
   a. Lumbar puncture
   b. MRV
   c. Ibuprofen and dismissal with one week neurology follow-up
   d. Antibiotic therapy
6. Which of the following can present with focal neurological deficits?
   a. Anemia
   b. Leukocytosis
   c. Hypokalemia
   d. Hypoglycemia
7. When should a physician consider starting anticoagulation in pediatric stroke patients?
   a. The etiology of the stroke is thought to be sickle cell disease.
   b. There is a confirmed cerebral sinovenous thrombosis.
   c. Intracranial dissections are identified.
   d. Endocarditis from a native valve is considered the etiology.
8. When should thrombolytics in childhood strokes be given?
   a. When a child presents with focal neurological deficits and no evidence of hemorrhage on imaging
   b. When a child is found minimally responsive with left sided hemiplegia
   c. To children with sickle cell disease and focal neurological complaints
   d. Never; there is no clear research showing benefit of recombinant tissue plasminogen activator or alteplase in the pediatric patient population

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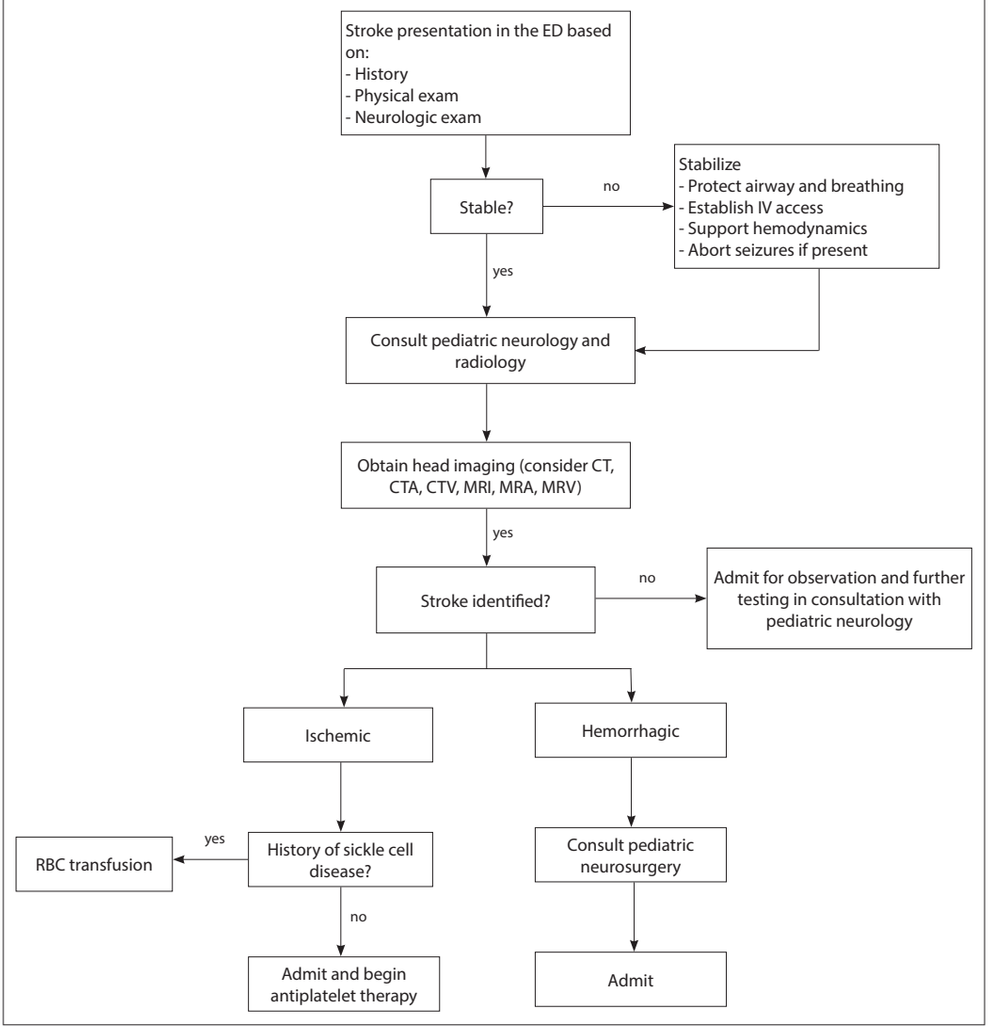
PEDIATRIC EMERGENCY MEDICINE **REPORTS**

Practical, Evidence-Based Reviews in Pediatric Emergency Care

Pediatric Stroke

Basic Approach to Pediatric Stroke Patients



Etiologies of Arterial Ischemic Strokes in Children

- Sickle cell disease
- Cardioembolic
- Moyamoya syndromes
- Cervical arterial dissection
- Steno-occlusive cerebral arteriopathy
- Other determined etiology
- Multiple probable/possible etiologies*
- Undetermined etiology

* Cerebral angiitis, fibromuscular dysplasia, cerebral involvement in systemic vasculitis, bacterial meningitis, hypertension, prothrombotic disorders, clinical chicken pox, and hyperhomocysteinemia.

Adapted from: Gumer LB, Del Vecchio M, Aronoff S. Strokes in children: A systematic review. *Pediatr Emerg Care* 2014;30:660-664.

Etiologies of Hemorrhagic Strokes in Children

- Vascular malformations
  - Arteriovenous malformation
  - Cavernous hemangioma
  - Aneurysm
  - Subarachnoid hemorrhage
  - Venous malformation
- Brain tumors
- Trauma/dissection
- Underlying medical disorder
- Undetermined

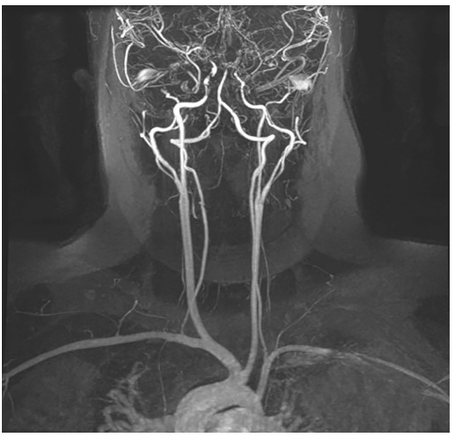
Adapted from: Gumer LB, Del Vecchio M, Aronoff S. Strokes in children: A systematic review. *Pediatr Emerg Care* 2014;30:660-664.

Antiplatelet and Anticoagulation Therapy[3,44-46]

	Antiplatelet Recommendations	Anticoagulation Recommendations
Royal College of Physicians	Aspirin 5 mg/kg/day in children with radiological evidence of stroke, excluding children with sickle cell disease or intracranial hemorrhage	Consider heparin in ischemic strokes when arterial dissection or CSVT is confirmed. Discuss heparin with pediatric cardiology and neurology before starting heparin in patients with ischemic strokes from a cardioembolic origin.
American Heart Association (AHA) Guidelines	Consider aspirin therapy in children with suspected cardioembolic cause of stroke unrelated to a patent foramen ovale.	Start heparin or low-molecular weight heparin as a bridge to warfarin in children with extracranial arterial dissection, cardioembolic stroke, and in neonates with severe thrombophilic disorders, multiple emboli, or propagation of a CSVT despite supportive care. Anticoagulation should not be started in children with intracranial dissection, subarachnoid hemorrhage from a dissection, or stroke due to native valve endocarditis.
American College of Chest Physician (ACCP) Guidelines	Empirically start initial treatment in children diagnosed with ischemic stroke not due to sickle cell disease with either aspirin (1-5 mg/kg/day) or anticoagulation (heparin or low-molecular weight heparin) until dissection and embolic causes of stroke are excluded. For neonates, treatment with aspirin or anticoagulation is not recommended except in cases of recurrent stroke	

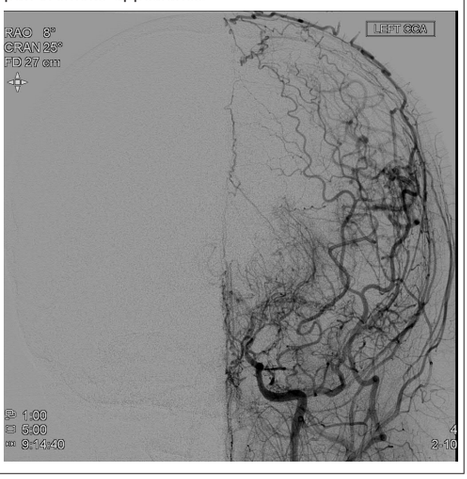
Moyamoya Disease

Magnetic resonance angiography demonstrates multiple areas of stenosis in the internal carotid arteries, typical of moyamoya disease. This patient suffered multiple watershed region infarcts.



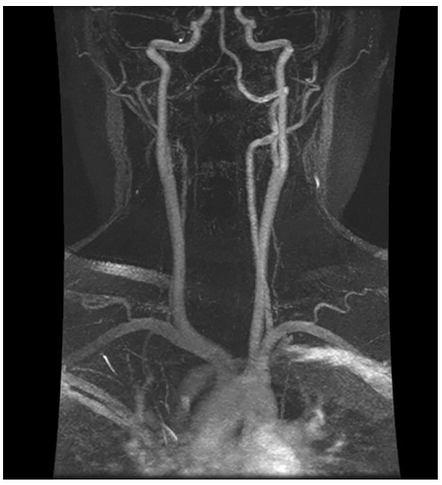
Moyamoya Disease

Cerebral angiography of the same patient demonstrates 75% stenosis of the supraclinoid left internal carotid artery with moyamoya type collaterals, which result in a pathognomonic "puff of smoke" appearance.



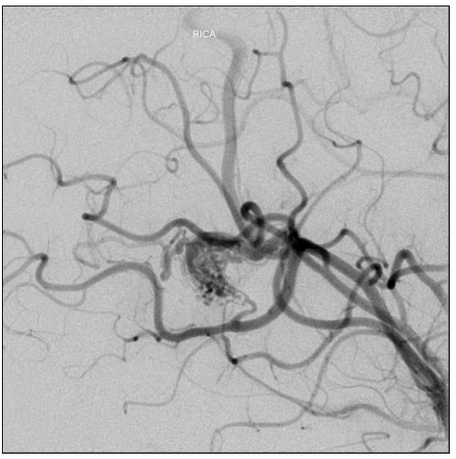
Traumatic Vertebral Artery Dissection

Magnetic resonance angiography demonstrates narrowing of the right vertebral artery, consistent with dissection. This patient presented with complaints of dizziness and vomiting after slipping on ice and falling backward, hitting the head.



Cerebral Angiogram Demonstrating an AVM

Cerebral angiogram demonstrating an AVM fed primarily from the right middle cerebral artery



Pediatric Intracerebral Hemorrhage Score

Component	ICH Scoring
IPH volume, % of TBV*	
≤ 2	0
> 2-3.99	1
≥ 4	2
Hydrocephalus	
No	0
Yes	1
Herniation	
No	0
Yes	1
Infratentorial	
No	0
Yes	1
Total ICH Score	0-5

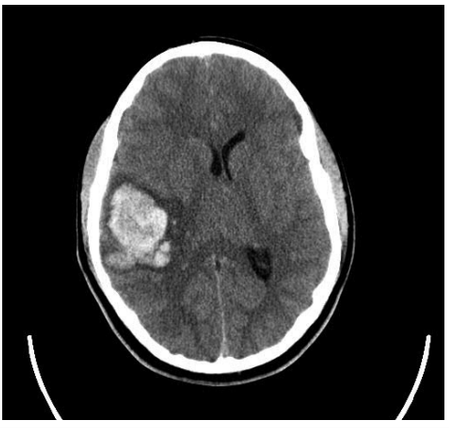
* IPH = intraparenchymal hemorrhage; TBV = total brain volume.

A score of ≥ 2 is sensitive for predicting severe disability or death.

Adapted from: Beslow LA, Ichord RN, Gindville MC, et al. Pediatric intracerebral hemorrhage score: A simple grading scale for intracerebral hemorrhage in children. *Stroke* 2014;45:66-70.

Right Temporal Intraparenchymal Hemorrhage

CT of a patient who complained of acute onset of severe headache reveals right temporal intraparenchymal hemorrhage with midline shift. This patient required an emergent craniotomy and hematoma evacuation.



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