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SECOND EDITION

CLINICAL PROTOCOLS IN PEDIATRICS

JAYDEEP CHOUDHURY
JAYANTA BANDYOPADHYAY

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Resuscitation

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2.1 CARDIOPULMONARY RESUSCITATION

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A sudden cardiac arrest is uncommon in children. Cardiac arrest is usually the terminal event of progressive respiratory failure or shock. Any emergency set up should be well equipped with procedures for cardiopulmonary resuscitation (CPR).

Airway

1. Mild neck extension is preferable (Child's head and occiput are proportionately large causing neck flexion). One can use a folded towel placed under the neck and shoulder.
2. Open the airway by head tilt—chin lift method. If cervical injury is suspected, open the airway using a jaw thrust without head tilt.
3. Clear airway from secretions, vomits and remove foreign bodies. In neonates and infants this can be effectively done with bulb mucus sucker (Fig. 2.1.1).
4. Oropharyngeal and nasopharyngeal airways for maintaining an open airway.
    i. Oropharyngeal airway in unconscious patient, i.e. with no gag reflex. The size is determined by the distance from the central incisors to the angle of the mandible.
    ii. Nasopharyngeal airway is better tolerated than oral airway by patients who are not deeply unconscious. The size is determined by the distance from the tip of the nose to the tragus of the ear.

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Figure 2.1.1 Bulb mucus sucker

Breathing

1. Use 100 percent oxygen during resuscitation.
2. Bag-mask ventilation can be as effective as endotracheal intubation:
   i. Use a self-inflating bag with a volume of 450 to 500 mL (Fig. 2.1.2)
   ii. Maintain oxygen flow of 15 L/min into a reservoir attached to a bag
   iii. The mask should fit over the mouth and nose to provide a tight seal and avoid any air leakage.
3. Ventilation through an endotracheal tube (ETT) (Fig. 2.1.3).
   Size for children 1 to 10 years of age is determined as ETT internal diameter (mm) = (age in years/4) + 4.
4. Laryngeal Mask Airway (LMA): When endotracheal intubation is not possible LMA is an acceptable adjunct for experienced providers.

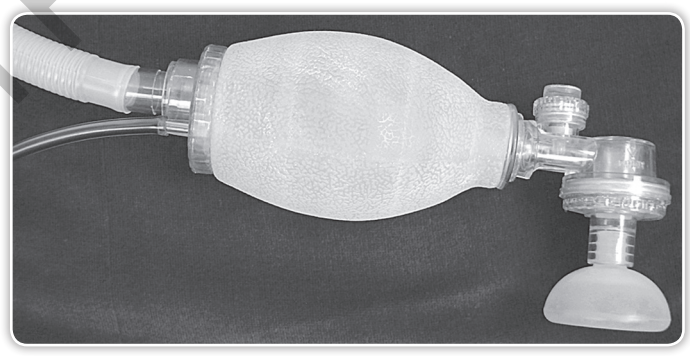

Figure 2.1.2 Bag and mask ventilation

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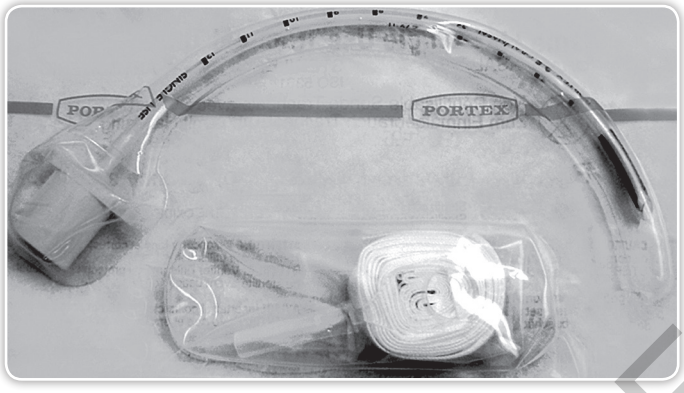

Figure 2.1.3 Endotracheal tube

To Minimize Gastric Inflation

i. Avoid excessive peak inspiratory pressures (e.g. ventilate slowly and watch chest rise, deliver only the volume needed to produce visible chest rise)
ii. Apply cricoid pressure to obstruct the esophagus
iii. Pass NG tube after intubation because a gastric tube interferes with the gastroesophageal sphincter, allowing possible regurgitation.

Excessive Ventilation is Detrimental Because:

i. It impedes venous return and, therefore, decreases cardiac output, cerebral blood flow and coronary perfusion by increasing intrathoracic pressure
ii. Causes air trapping and barotraumas in patients with small airway obstruction
iii. Increases the risk of regurgitation and aspiration.

Circulation

1. Check pulse (brachial artery in infants—carotid or femoral artery in children)
2. Start cardiac compressions when heart rate <60 beats/min with signs of poor perfusion. Two thumb—encircling hands chest compression and bag and mask ventilation is shown in Figure 2.1.4.

Characteristics of Good Chest Compressions

1. "Push fast" at a rate of 100 compressions/min
2. "Push hard" to depress the chest 1/3 to 1/2 of the anterior-posterior diameter of the chest
3. Release completely to allow the chest to fully recoil after each compression
4. Minimize interruptions in compressions.

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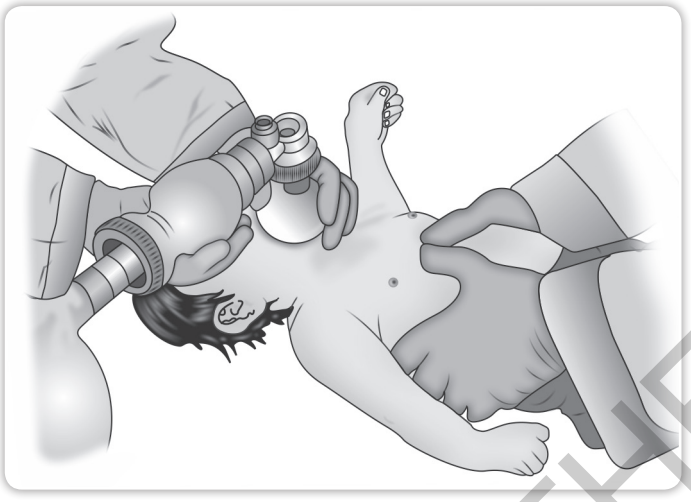

Figure 2.1.4 Two thumb—encircling hands chest compression and bag-mask ventilation

Recommended Chest Compression: Ventilation Ratio

1. One rescuer: Cycles of 30 compressions and two breaths
2. Two rescuers: Cycles of 15 compressions and two breaths
3. When an advanced airway is established (e.g. ETT or LMA):
   i. Give continuous chest compressions without pauses for breaths
   ii. Give 8 to 10 breaths/minute
   iii. Check rhythm every two minutes and change the compressor role to prevent fatigue and deterioration in quality and rate of chest compressions
   iv. The switch should be done in less than five seconds to minimize interruption in chest compression
   v. If the pulses are present but no breathing, give 12 to 20 breaths per minute (1 breath every 3 to 5 seconds).

   The method of chest compression is shown in Table 2.1.1.

Vascular Access

If one cannot achieve a reliable access quickly, an intraosseous (IO) access should be established.

Fluids

1. Isotonic crystalloid solution to treat shock (20 mL/kg of normal saline as quickly as possible). Repeated boluses may be necessary.

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TABLE 2.1.1
Method of chest compression in infants and children

Cardiac compressions	*Infants*	*Children*
Rate/min	100	100
Depth	1/3 of AP chest diameter	1/3 to 1/2 of AP chest diameter
Site	Lower half of the sternum not over the xiphoid (below intermammary line)	Lower half of the sternum not over the xiphoid
Technique	2 fingers technique. OR	Heel of one hand or two hands technique
	2 thumb—encircling hands technique (preferred)	

2. Hypotension—Systolic blood pressure less than 5th percentile of normal for age:
   i. <60 mm Hg in term neonates
   ii. <70 mm Hg in infants
   iii. <70 mm Hg + (2 × age in years) in children 1 to 10 years
   iv. <90 mm Hg in children >10 years of age.
3. Glucose containing fluids are not indicated during CPR unless hypoglycemia is present.

Drugs

1. Drugs are preferably administered through IV or IO route than by ETT.
2. If vascular access cannot be established, the following drugs can be given via ETT – "LEAN" – lidocaine, epinephrine, atropine and naloxone. Flush with a minimum of 5 mL normal saline followed by 5 assisted manual ventilations. If CPR in progress, stop chest compressions briefly during administration of medications.
3. Epinephrine dose in cardiac arrest = 0.01 mg/kg of 1:1000 epinephrine as the first and subsequent IV doses.

Medications for pediatric resuscitation and arrhythmias that should be available in pediatric emergency and medications to maintain cardiac output and for postresuscitation stabilization are shown in Tables 2.1.2 and 2.1.3, respectively.

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TABLE 2.1.2
Medications for pediatric resuscitation and arrhythmias

Medication	*Dose*	*Remarks*
Adenosine	0.1 mg/kg (max 6 mg)	Monitor ECG
	Repeat dose 0.2 mg/kg (max 12 mg)	Rapid IV/IO bolus
Amiodarone	In pulseless VT or VF—5 mg/kg IV/IO	Monitor ECG and BP
	Repeat up to 15 mg/kg, maximum 300 mμg.	Use with caution when administering with other drugs that prolong QT
	Infusion—25 μg/kg/min for 4 hours followed by 5–15 μg/kg/min continuous.	
Atropine	0.02 mg/kg IV/IO 0.03 mg/kg ET* repeat once if needed minimum dose 0.1 mg maximum single dose in child 0.5 mg, in adolescent 1 mg	Higher doses may be used with organophosphate poisoning
Calcium chloride (10%)	20 mg/kg IV/IO (0.2 mL/kg)	Slowly
Epinephrine	0.01 mg/kg (0.1 mL/kg 1:10 000) IV/IO 0.1 mg/kg	May repeat every 3–5 min
	(0.1 mL/kg 1:1000) ET* maximum dose 1 mg IV/IO; 10 mg ET*	
Glucose	0.5–1 g/kg IV/IO	10% dextrose diluted with 5–10 mL/kg 25% dextrose diluted with 2–4 mL/kg
		50% dextrose diluted with 1–2 mL/kg
Lidocaine	Bolus 1 mg/kg IV/IO	
	Maximum dose 100 mg	
	Infusion 20–50 μg/kg/min ET* 2–3 mg	
Magnesium sulfate	25–40 mg/kg IV/IO over 30 minutes, may be given faster in Torsades pointes	
	Maximum dose 2 g	
Naloxone	<5 years or ≤20 kg 0.1 mg/kg IV/IO/ET*	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1-15 μg/kg)
	≥5 years or >20 kg 2 mg IV/IO/ET*	
Procainamide	15 mg/kg IV/IO over 30–60 minutes adult dose 20 mg/min IV infusion up to total maximum dose 17 mg/kg	Monitor ECG and BP use caution when administering with other drugs that prolong QT
Sodium bicarbonate	1 mEq/kg/dose IV/IO slowly	After adequate ventilation
* flush with 5 mL of normal saline and follow-up with 5 ventilations		

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TABLE 2.1.3

Medications to maintain cardiac output and for postresuscitation stabilization

Medication	*Dose*	*Comment*
Dobutamine	2–20 µg/kg/min IV/IO‡	Inotrope, vasodilator
Dopamine	2–20 µg/kg/min IV/IO‡	Inotrope, chronotrope; renal and splanchnic vasodilator in low doses; pressor in high dose
Epinephrine	0.1–1 µg/kg/min IV/IO*	Inotrope, chronotrope, vasodilator in low doses; pressor in higher doses
Norepinephrine	0.1–2 µg/kg/min*	Inotrope, vasopressor
Sodium nitroprusside	1–8 µg/kg/min‡	Vasodilator; prepare only in D5W

‡6 × body weight (in kg) = mg of drug to add to 100 mL D5W then, an IV rate of 1 mL/h delivers 1 µg/kg/min of drug.
*0.6 × body weight (in kg) = mg of drug to add to 100 mL D5W, then, an IV rate of 1 mL/h delivers 0.1 µg/kg/min of drug.

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2.2 COMA

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Clinical status of a patient can be divided into four types—alert, lethargy, stuporous and coma. Coma is defined as the state of unarousable unresponsiveness.

Neurological Examination

Level of Consciousness

Modified Glasgow coma scale (GCS) as shown in Table 2.2.1 has been used in children and infants to assess the duration and depth of altered consciousness.

Attitude and Posturing

Resting position and spontaneous movements should be noted. Hemiparesis, focal motor seizure, or any asymmetrical finding indicates structural brain lesion, whereas symmetrical lesion is usually seen in metabolic coma. Decorticate rigidity indicates structural lesion of cerebral hemisphere or

TABLE 2.2.1
Modified GCS

Response	*Score*
Eye opening	
1. Spontaneous	4
2. To speech	3
3. To pain	2
4. None	1
Best verbal response	
1. Smiles, oriented to sound, follows object, interacts	5
2. Consolable cry	4
3. Inconsistently consolable cry	3
4. Inconsolable cry, restless, agitated, unaware of environment or parents	2
5. No response	1
Best motor response	
1. Spontaneous movement/obeys verbal command	6
2. Localizes to supraocular pain	5
3. Withdraws	4
4. Abnormal flexion to pain	3
5. Abnormal extension to pain	2
6. None	1
Grading—13–14 Mild, 9–12 Moderate, <8 severe	

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diencephalons whereas decerebrate rigidity indicates upper brainstem lesions. Medullary lesions cause flaccidity.

Respiration

Abnormal respiratory pattern in coma helps in localization:

i. Cheyne-Stokes respiration—Bilateral hemispheric or diencephalic lesions.
ii. Apneustic respiration—Upper pontine lesions.
iii. Cluster breathing—Lower pontine or upper medullary lesions.
iv. Ataxic respiration—Lower medullary lesions.
v. Central neurogenic hyperventilation—Midbrain or upper pontine lesion.
vi. Kussmaul breathing—Metabolic acidosis.

Brainstem Function

i. *Pupillary response:* Assuming the visual pathways to the lateral geniculate body are intact, assessment of the pupillary responses is important in localizing the site of coma and separating structural from toxic/metabolic causes, as pupillary responses in the latter are intact.
   a. Unilateral fixed dilated pupil—Uncal herniation or rupture of posterior communicating artery aneurysm
   b. Unilateral constriction—Horner's syndrome
   c. Bilateral pinpoint pupils—Pontine lesion or opiate poisoning
   d. Bilateral midposition—Nonreactive in midbrain lesion and Reactive in metabolic causes
   e. Bilateral dilated and fixed—Severe midbrain damage.
ii. *Ocular motility:* It is an important clinical indicator of the site of lesion in coma.

   If there is spontaneous eye movement, look for the following.
   a. Conjugate spontaneous roving movements indicate an intact brain stem
   b. Conjugate lateral deviation—Look towards the lesion indicates cerebral hemispheric lesion and away from the lesion is seen in brainstem damage
   c. Ocular bobbing indicates bilateral pontine or cerebellar lesion
   d. Skew deviation occurs with posterior fossa lesions
   e. Persistent adduction in paresis of VI cranial nerve
   f. Persistent abduction in paresis of III cranial nerve
   g. Dysconjugate gaze in horizontal plane indicates intoxication
   h. Downward deviation of the eyes indicates bilateral thalamic or subthalamic lesions and metabolic coma.

   If there is no spontaneous eye movement, then ocular motility can be tested by oculocephalic reflex (Doll's eye maneuvre) and oculovestibular reflex (Caloric testing). The methods of the tests are shown in Table 2.2.2.

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TABLE 2.2.2
Methods of testing oculocephalic and oculovestibular reflexes

Oculocephalic reflex	*Oculovestibular reflex*
Eyes move conjugately to the opposite direction to head movement: Normal	Nystagmous with fast component away from site of stimulation: Normal
Lateral gaze palsy: Brainstem lesion	No nystagmus or dysconjugate response:
No movement: Pontine lesion	Brainstem lesion
Full deviation towards the stimulated side: Cerebral hemispheric lesion with intact brainstem	

iii. *Fundoscopic examination:* Assess for papilledema and any hemorrhage.
iv. *Other cranial nerve reflexes:* Corneal reflexes (V and VII cranial nerves); gag and cough reflexes (IX and X cranial nerves) should also be assessed. Corneal reflex is retained until coma is very deep.

Signs of meningeal irritation: Presence of meningeal signs indicates meningitis or subarachnoid hemorrhage and parenchymal bleed with intraventricular extension.

Clinical differentiation of toxic, metabolic or infectious causes of coma from structural causes of coma like supratentorial and infratentorial coma are shown in Table 2.2.3.

Investigations

1. Blood sugar: In all patients presenting with coma or altered sensorium, blood sugar or blood glucose should be tested with Dextrostix. Treatment should not be delayed of symptomatic hypoglycemia awaiting this result
2. Full blood count, malarial parasite in peripheral smear
3. Blood culture
4. Consider coagulation screen before considering a lumbar puncture
5. If hypoglycemic: Measure blood ketones. Insulin, growth hormone and cortisol may be measured if possible.

TABLE 2.2.3
Differences between metabolic and structural coma

Toxic/metabolic/infectious	*Supratentorial lesions*	*Infratentorial lesions*
1. Confusion precedes motor signs	1. Initial focal signs	1. Preceding brainstem dysfunction
2. Pupillary reactions preserved	2. Rostral to caudal progression	2. Sudden onset
3. Symmetrical motor responses		3. Cranial nerve palsies
4. Asterixis, myoclonus		4. Early respiratory disturbances
5. Hyper/hypoventilation		

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6. Urea and electrolytes
7. Blood gas, including arterial lactate
8. Liver function tests: Consider serum ammonia in case of suspected hepatic coma, Reye's syndrome or urea cycle defect
9. Thyroid function tests
10. Urinalysis: Consider toxicology screen of urine and blood if indicated
11. CT scan or MRI brain: If trauma or clinical suspicion of child abuse, focal neurodeficit. Initial normal CT does not rule out an evolving lesion of an infection or metabolic disorder
12. CSF analysis: Lumbar puncture (LP) is contraindicated in presence of coma (GCS <9), raised intracranial pressure or unstable clinical state. If meningitis is suspected but LP is contraindicated, start antibiotics
13. Cervical spine imaging: Protect neck until injury has been excluded by standard criteria in cases of trauma or possible trauma. It is often not possible to exclude cervical spine injury in a comatose child. May need to CT scan of upper cervical spine in trauma
14. MRI provides better visualization of brainstem and cerebellar structures, venous sinuses, diffuse disorders, for example in inborn errors of metabolism or hypoxic encephalopathy
15. EEG: Serial EEG is more useful. Relatively specific in herpes simplex encephalitis. It helps in detection of nonconvulsive status epilepticus as a cause of coma.

Management

Early appropriate supportive care is essential to avoid preventable secondary insults and optimize the neurological outcome. Evaluation and stabilization of the patient's airway, breathing and circulation (ABCs) must proceed simultaneously with assessments of the depth of coma and the presence of raised intracranial pressure (ICP). Any rapidly correctable cause of coma must be immediately corrected.

Airway

Ensure adequate airway. Use airway adjunct oropharyngeal airway, nasopharyngeal airway, LMA or intubate if unable to maintain airway. If GCS <8 and clinical circumstances do not suggest improvement or imminent herniation, then intubation is indicated to secure airway. Even if spontaneously breathing with normal gas exchange, many comatose children will benefit from intubation, especially if they have intracranial hypertension.

Breathing

Give oxygen, monitor oxygen saturation, and assess rate and pattern of breathing. Supplemental oxygen is indicated for hypoxia, but should not be

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given routinely to comatose children with normal circulation and oxygen saturation.

Circulation

Assess for signs of shock and treat as indicated. The aim of fluid therapy in raised ICP is to maintain adequate cerebral perfusion pressure (CPP).

CPP = Mean arterial pressure – Intracranial pressure.

Give normal saline or Ringer's lactate to maintain BP and perfusion. What must be restricted are hypotonic fluids such as 1/5th normal saline in 5 percent dextrose (Isolyte P). The dextrose will be metabolized with a resultant hypotonic fluid that can exacerbate cerebral edema and ICP. Enteral feeds should be started at the earliest.

Hypoglycemia

Bolus of 10 percent dextrose 5 mL/kg IV, followed by a 10 percent dextrose infusion at 4 mL/kg/h (7 mg/kg/min) with close monitoring of blood glucose.

Hyperglycemia

Due to stress or diabetic ketoacidosis.

Drugs

If opioid toxicity is suspected—Naloxone 0.1–0.8 mg/kg IV (maximum dose 2 mg). Avoid flumazenil, which may induce convulsions in mixed overdoses, particularly if tricyclic antidepressants have been taken. Isolated benzodiazepine overdose does not cause significant respiratory depression and children are best managed with simple observation.

Specific Therapy

After stabilization, a rapid approach to diagnosis is imperative so that specific therapy can be given.

1. Known poisoning—specific antidotes
2. Hypertensive encephalopathy—antihypertensives
3. Space occupying lesions—neurosurgery.

The stepwise approach to diagnosis and management of coma in children is shown in Flow chart 2.2.1.

Emergency Management of Raised Intracranial Pressure

GCS <9 and Cushing's triad of hypertension, bradycardia, abnormal respiration and/or fixed, dilated pupil(s).

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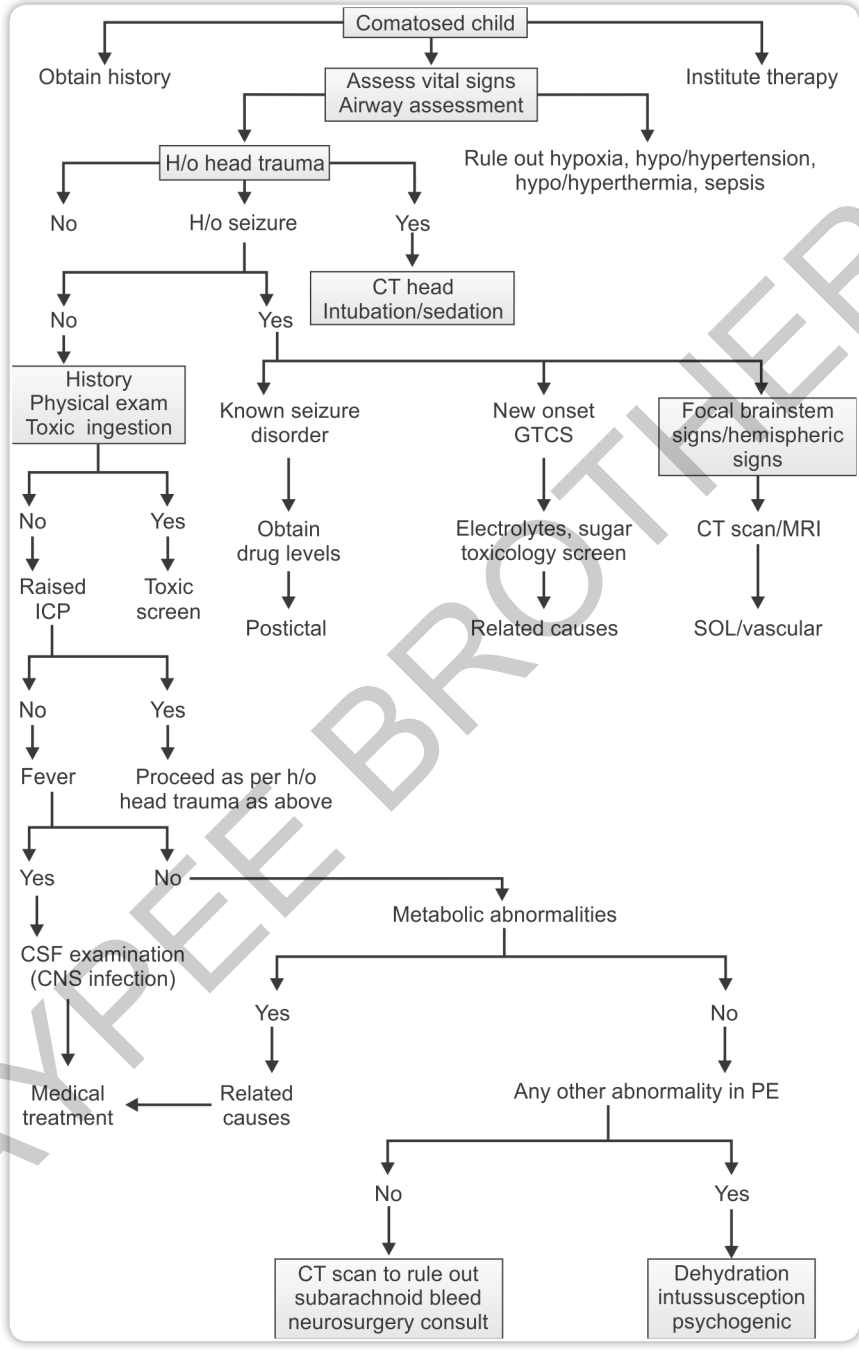
Flow chart 2.2.1 Algorithm for the diagnosis and management of coma in children



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